

# Curricular Guide for Podiatric Medical Education

AACPM Council of Faculties 2014 Edition

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- Knowledge objectives developed by the American Society of Medical Pharmacology Chairs, with the permission of the president at that time, Bonnie Sloane.
- Booth SJ, Burges G, Justeman L, Knoop F. Design and Implementation of Core Knowledge Objectives for Medical Microbiology and Immunology. *J Int Assoc Med Sci Educ*. 2009;19:100-138.
- GRIPE, Group for Research in Pathology Education, with permission.
- McHanwell S, Davies DC, Morris J, et al. A core syllabus in anatomy for medical students adding common sense to need to know. *Eur J Anat.* 2007;11:3-18.
- Hyland, KM et al. Medical School Core Curriculum in Genetics 2013. Association of Professors of Human and Medical Genetics.

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## **Background**

The 2005 APMA House of Delegates adopted Resolution No. 2-05. This resolution charged APMA to do the following:

RESOLVED, That the APMA commit itself to achieving the goal by 2015 of podiatrist being defined as physicians who treat patients in the physician's specialty without restrictions; and RESOLVED, That the APMA create a master plan to accomplish this goal and report its progress to the House of Delegates starting in 2006 and in subsequent years.

A Plan to Obtain National Recognition of the Podiatric Physician Working Document reported in May 2009 the following:

#### **Overall Mission**

Podiatrists are universally accepted and recognized as physicians consistent with their education, training, and experience

#### Objectives

- 1. Evaluate and ensure that podiatric medical education is comparable to that of allopathic and osteopathic physicians.
- 2. Demonstrate to the entire health care community that the education, training, and experience of a podiatric physician are comparable to that of allopathic and osteopathic physicians.
- 3. Obtain state and federal government recognition that podiatrists are physicians.
- 4. Market and promote podiatrists as physicians.
- 5. Attract high quality applicants to colleges of podiatric medicine and thereby to the profession.

#### AACPM Council of Faculties Curriculum Review and Educational Objective Development Project

On March 4, 2009, the AACPM Council of Deans (COD) received the report of the March 2 meeting of the AACPM Council of Faculties (COF) which proposed a plan and timeline for completion of a comprehensive review of core competency criteria that might provide guidelines for the scope of concepts essential to present and future preparation of practitioners of Podiatric Medicine. The COF recommended that the currently examined areas of Part I and Part II of the National Board of Podiatric Medical Education serve as the organizing framework for the creation of a comprehensive set of educational objectives. The Deans approved the COF recommendations and the Board of Directors voted unanimously to fully fund the project.

Preclinical Science Areas	Clinical Areas
General Anatomy	General Medicine
Lower Extremity Anatomy	Radiology
Biochemistry	Orthopedics/Biomechanics/Sports Medicine
Physiology	Surgery/Anesthesia
Microbiology/Immunology	Community Health/Jurisprudence/Research
Pathology	Geriatrics**
Pharmacology	
Neuroanatomy**	
Embryology**	

#### Core Competency Review

Genetics**	
Histology**	

#### \*\*Section added in 2014.

This living document contains a comprehensive set of weighted learning objectives in each of the ontent areas above.

Even as version 1.0 of the Curriculum Guide was being finalized, content areas that were not part of the original list were identified, with plans for starting work on them for inclusion in version 2.0. These additional content areas include Neuroanatomy, Histology, Embryology, Genetics and Geriatrics. Consideration was also given to separating the individual components in the area of Community Health, Jurisprudence and Research when that area is updated.

The weighted ranking of the learning objectives was carried out by content area experts from each of the schools and colleges of podiatric medicine with the aim of identifying those objectives important for each graduating podiatric medical student to master prior to beginning residency training. The ranking scale ranged from 0-4, with 4 being most important.

These objectives were developed using Bloom's *Taxonomy of Objectives for the Cognitive Domain* (1956), to categorize cognitive tasks, usually in increasingly sophisticated order.

#### Bloom's Taxonomy

Bloom's Taxonomy breaks education into 6 different areas: Knowledge, Comprehension, Application, Analysis, Synthesis, and Evaluation. These levels are increasingly complex—that is, Knowledge is the most basic of areas and Evaluation is the most complex.

A comprehensive mix of learning objectives takes specificity and focus into account, as well as education areas and complexity. The mix also depends on the actual content; an introductory text will tend to be more heavily weighted on the Knowledge, Comprehension, and Application learning objectives, whereas a text on advanced thermodynamics will tend to be focused on Analysis, Synthesis, and Evaluation.

To provide more detail on Bloom's areas and the verbs often associated with each level, you can refer to the table in Appendix I.

## **GENERAL ANATOMY LEARNING OBJECTIVES**

Basic Anatomy of the Back Clinical Anatomy of the Back Basic Anatomy of the Upper Limb Clinical Anatomy of the Upper Limb Basic Anatomy of Pelvis and Perineum Clinical Anatomy of Pelvis and Perineum Basic Anatomy of Thorax Clinical Anatomy of Thorax Basic Anatomy of Abdomen Clinical Anatomy of Abdomen Basic Anatomy of Head and Neck Clinical Anatomy of Head and Neck

## I. Basic Anatomy of the Back

1.	List the functions of the vertebral column.	3.9
2.	Describe the osteological features of vertebrae.	3.9
3.	Describe the osteological features of the atlas, axis, sacrum, and coccyx.	3.9
4.	Describe the osteological features of the scapula.	3.6
5.	Differentiate between the primary and secondary curvatures of the spine.	3.3
6.	Describe the intervertebral joints.	3.7
7.	Describe the features of the vertebral column that control its mobility.	3.7
8.	Describe the cutaneous innervation of the back and posterior neck.	3.6
9.	Differentiate between the extrinsic and intrinsic back muscles.	3.6
10.	Describe the deep muscles of the back in terms of their origins, insertions, innervations,	and
	major actions.	3.4
11.	Describe the vascular supply and venous drainage of the back, vertebral column, and sp	inal
	cord.	3.3
12.	Contrast the movements found in the cervical, thoracic, and lumbar regions of the spine	2.
		3.3
13.	Describe the attachments locations of the ligaments of the vertebral column.	3.6
14.	Explain the structure and function of an intervertebral disc.	3.7
15.	Explain the structure and function of the zygopophysial (facet) joints and compare them	in the
	cervical, thoracic, and lumbar regions.	3.4
16.	Describe the atlanto-occipital and atlanto-axial joints with emphasis on their movement	s.
		3.3
17.	Describe the innovation of the muscles of the back.	3.6
18.	Identify the major surface features of the back.	3.9
19.	Identify the surface anatomy landmarks for locating the spinous processes of the C7, T3	, T7, L4,
	L5, and S2 vertebrae.	3.6
20.	Describe the osteological features the boundaries of the suboccipital triangle and its con	ntents.
		2.6
21.	Describe the boundaries of the intervertebral foramen and its contents.	4.0
22.	Describe the relationship between the vertebral levels and spinal cord levels in the adul	t and
	child.	3.7
23.	Identify the spinal nerves in relation to the adjacent vertebrae above and below.	3.6
24.	Describe the major features of the spinal cord and meninges.	3.9
25.	Describe the origin, course, and termination of the vertebral artery.	3.4
26.	Define pars interarticularis.	3.4
27.	Differentiate between the CNS and PNS.	3.7
28.	Define <i>dermatome</i> .	3.9
29.	Diagram a transverse section through the vertebral canal demonstrating the meninges a	and the
	meningeal spaces.	3.4
30.	Describe the fascia of the back, including the thoracolumbar fascia.	3.3
31.	Describe the lymphatic drainage of the back and vertebral column.	3.1
32.	Draw the structure of a typical spinal nerve.	3.9
33.	Describe the superficial muscles of the back in terms of their origins, insertions, innerva	tions,
	and major actions.	3.6

### II. <u>Clinical Anatomy of the Back</u>

1	Intograto bacic anatom	y with the following clinical correlat	00.
<b>1</b> .	IIILE EI ALE DASIC AHALUIT	v with the following tinntal toneiat	es.

<b>-</b> .			
	a.	low back pain	4.0
	b.	spina bifida	4.0
	с.	laminectomy	4.0
	d.	lumbar puncture	4.0
	e.	herniated nucleus pulposus	4.0
	f.	scoliosis	4.0
	g.	kyphosis	4.0
	h.	lordosis	4.0
	i.	spondylosis	4.0
	j.	spondylolisthesis	4.0
2.	Explain	lumbarization and sacralization.	3.6
3.	Identify	the osteological features of the back as demonstrated on diagnostic imaging.	3.7
4.	Identify	soft-tissue structures of the back on sagittal and transverse CTs and MRIs.	3.3
5.	Define	pars interarticularis and identify the features of the "Scotty dog" as seen on oblig	que
	radiogr	aphs of the lumbar spine.	3.4
6.	Rationa	alize the choice of sites for lumbar puncture and epidural anesthesia.	3.9
7.	List, in	order, the structures and spaces pierced in a lumbar puncture and epidural anes	thesia.
			3.9
0	Doccrit	the veneus anastemeses associated with the vertebral column and discuss the	ir clinic

Describe the venous anastomoses associated with the vertebral column and discuss their clinical significance.
 4.0

## III. Basic Anatomy of the Upper Limb

1.	Describe the osteological features of the scapula, humerus, and clavicle.	3.7
2.	Describe the structure associated with pectoral and scapular region.	3.3
3.	Describe the muscles of the shoulder complex in terms of origins, insertions, actions,	
	innervations, and blood supply.	3.6
4.	Describe the muscles of the anterior and posterior compartments of the arm in terms of	of origins,
	insertions, actions, innervations, and blood supply.	3.6
5.	Describe the axillary artery and its branches.	3.7
6.	Describe the brachial artery and its branches.	3.7
7.	Describe the brachial plexus, including roots, trunks, divisions, cords, and branches.	3.7
8.	Describe the osteological features of the ulna and radius.	3.7
9.	Describe the muscles of the anterior and posterior compartments of the forearm in ter	ms of
	origins, insertions, actions, innervations, and blood supply.	3.6
10.	Describe the structure of synovial tendon sheaths.	3.7
11.	Describe the radial artery and its branches.	3.7
12.	Describe the ulnar artery and its branches.	3.7
13.	Describe the osteological features of the carpel, metacarpals and felangial bones.	3.7
14.	Describe the intrinsic muscles of the hand in terms of origins, insertions, actions, inner	vations,
	and blood supply.	3.6
15.	Explain the structure and function of extensor expansion (aponeurosis).	3.7
16.	Describe the structure of the flexor retinaculum, as well as the carpal tunnel and its con	ntents.
		3.6
17.	Describe the branches of the radial and ulnar arteries at the wrist and in the hand.	3.4
18.	Describe the superficial and deep venous drainage of the upper extremity.	3.6

19	Describe the superficial and deep lymphatic drainage of the upper extremity.	3.0
20	Describe the avillary lymph nodes	37
20. 21	Evaluin the structure and function of the joints of the upper extremity	3.7 2.6
Z1.	explain the structure and function of the joints of the upper extremity.	5.0
22.	Describe the structure and function of the rotator cuff.	3.6
23.	Explain the structure and function of the interosseous membrane.	3.1
24.	Describe the boundaries and contents of the axilla.	3.4
25.	Describe the boundaries and contents of the quadrangular space, triangular space, and	
	triangular interval.	2.9
26.	Describe the boundaries and contents of the cubital fossa.	3.7
27.	Describe the boundaries and contents the anatomical snuffbox.	3.3
28.	Identify the surface anatomy and palpable bony landmarks of the upper extremity.	3.9
29.	Describe the superficial and deep fascia of the upper extremity in terms of myofascial	
	compartments and their contents.	3.3
30.	Describe the innervation of the upper extremity in terms of dermatomes and cutaneous	
	domains.	3.7

## IV. <u>Clinical Anatomy of the Upper Limb</u>

1.	Identify the osteological features of the upper extremity as demonstrated on diagnostic		
	imaging.	3.7	
2.	Explain winging of the scapula.	3.6	
3.	Identify sites where the pulse is taken in the upper extremity.	3.9	
4.	Describe the functional deficits resulting from the most common brachial plexus injuries	5.	
		3.9	
5.	Diagnose probable legion sites of the brachial plexus from motor and sensory deficits.	3.7	
6.	Define Colles' fractures.	3.1	
7.	Explain cubital tunnel syndrome and its clinical significance.	3.4	
8.	Explain carpel tunnel syndrome and its clinical significance.	3.6	
9.	Describe the anatomical basis for wrist drop.	3.3	
10.	Identify the sites at which pulses in the radial and ulnar arteries may be located.	4.0	
11.	Explain the clinical significance of scaphoid fractures, including radiographic diagnosis.	3.6	
12.	Explain the clinical significance of lunate dislocation.	3.1	
13.	Identify the structures found in a transverse cross-section of the carpal tunnel.	3.4	
14.	Explain Dupuytren's contracture and its clinical significance.	2.4	
15.	Describe the clinical importance of the arterial anastomoses of the shoulder, elbow, and	d hand.	
		2.9	
16.	Identify common sites used for venipuncture.	4.0	
17.	Describe the clinical significance of the lymphatic drainage of the breast and axilla in relation	ation to	
	the metastatic spread of breast cancer and melanoma.	3.7	
18.	Compare and contrast a separated shoulder and a dislocated shoulder.	3.3	
19.	Identify soft tissue structures of the shoulder, arm, elbow, forearm, wrist, and hand on (	CT and	
	MRI images.	3.4	
20.	Describe the clinical significance of rotator cuff injuries.	3.6	
21.	Describe common routes for the spread of infection from the hand to the forearm	3.3	
22.	Describe DeQuervain's tenosynovitis.	3.3	

## V. Basic Anatomy of Pelvis and Perineum

1.	Describe the skeletal and ligamentous components of the pelvis, pelvic inlet, and pelvic	outlet. 3.9
2.	Compare and contrast the male and the female pelvis.	3.3
3.	Explain the structure and function of the lumbosacral and sacroiliac joints and pubic syn	nphysis. <b>4.0</b>
4.	Describe the openings that permit passage of structures to and from the pelvis, perineu lower extremity and identify the structures that pass through them	m, and
5	Describe the anatomical walls and floor of the pelvic cavity	4.0 2.7
5. 6	Describe the polyic muscles in terms of their origins insertions actions innervations are	J./
0.	supply	3.7
7.	Describe the pelvic (fascial) ligaments and the structures that they support and transmit	.3.3
8.	Describe the inferior boundaries of the peritoneum and peritoneal cavity/pouches with male and in the female pelvis.	in the
		3.7
9.	Describe the organization and relationships of the pelvic viscera in sagittal, frontal, and	
	transverse sections of the male and the female pelvis.	3.6
10.	Relate internal pelvic viscera to its continuity into the perineum.	3.4
11.	Describe the internal iliac artery and its branches.	4.0
12.	Describe the venous drainage of the pelvis and perineum.	3.4
13.	Describe the lymphatic drainage from the pelvis and perineum.	3.4
14.	Describe the sacral plexus and its branches.	4.0
15.	Describe the autonomic innervations of the pelvis and perineum.	3.4
16.	Identify the boundaries of the perineum.	3.4
17.	Identify the boundaries and contents of the urogenital and anal triangles.	3.3
18.	Describe the pudendal nerve and its branches.	3.9
19.	Describe the internal pudendal artery and its branches.	<b>3.</b> b
20.	Describe the blood supply, lymphatic drainage, and innervation of the sigmoid colon an	
21	with respect to empryonic origin.	Z.I
21.	torms of arterial supply voncus drainage, and innonvation	2 A
22	Compare and contract the internal and external anal sphinsters in terms of location, str	<b>3.4</b>
22.	and innorvation	<b>2 1</b>
72	Describe the course of the unstars in the polyis	2.6
23. 24	Explain the structure and function of the urinary bladder	3.0
24.	Describe the anatomy of the urethra in male and in female	3.0
25.	Compare and contrast the external urethral sphincter in the male and female	3.4
20.	Describe the testicular arteries	3.6
27.	List in order the veins through which venous blood originating in the testes would be r	eturned
20.	to the inferior vena cava (IVC) on both right and left sides of the body	3.3
29.	Describe the path taken by spermatozoa from the testes to the penile urethra.	3.3
30.	Describe the anatomy of the scrotum, testes and epididymis including the arterial suppl	V,
	venous, and lymphatic drainage.	3.3
31.	Describe the course and contents of the spermatic cord.	3.6
32.	Explain the structure and function of the seminal vesicles.	3.4
33.	Explain the structure and function of the prostate gland.	3.4
34.	Describe the general anatomy of the penis including blood supply of the erectile tissues	. 3.4

35. Describe the ovarian arteries.	3.6
36. Describe the broad ligament.	3.6
37. Describe the anatomy of the ovary and associated ligaments.	3.3
38. Describe the uterine tubes.	3.3
39. Describe the uterus including the cervix.	3.4
40. Describe the uterine arteries, emphasizing their relationships to the transverse (ca	rdinal)
ligament and the ureters.	3.6
41. Describe the vagina and the fornices.	3.3
42. Describe the vulva.	3.1
43. Describe the structure of the clitoris and vestibular bulbs, including blood supply t	o the erectile
tissues.	3.3
44. Identify the homologous structures of the male and female reproductive systems.	2.7

### VI. Clinical Anatomy of Pelvis and Perineum

1.	Identify the osteological and soft tissue features of the pelvis and perineum in diagnostic	2
	imaging.	3.7
2.	Describe the palpable anatomical landmarks of the pelvis and perineum, and explain the	ir
	clinical significance.	3.9
3.	Explain the functional importance of the pelvic diaphragm in the male and female pelvis.	
		3.6
4.	Relate urinary stress incontinence or uterine prolapse to weakness of the pelvic diaphrage	gm.
		3.4
5.	Explain the clinical significance of an open female peritoneal cavity versus a closed male	
	peritoneal cavity.	3.1
6.	Explain the clinical significance of the vascular anatomoses between vessels in the pelvis	and
	perineum.	3.6
7.	Explain the functional and clinical significance of the perineal body.	3.0
8.	Describe the pudendal nerve in terms of clinically relevant sites for nerve block.	3.9
9.	Describe the clinical significance of the ischioanal fossae.	3.4
10.	.0. Compare and contrast internal hemorrhoids from external hemorrhoids in terms of location,	
	venous drainage, and possible causes.	3.1
11.	Compare and contrast the internal and external anal sphincters in terms of fecal contine	nce.
		3.0
12.	Explain the changes in position of the urinary bladder and its overlying peritoneum durin	ng
	pregnancy.	2.9
13.	.3. Describe the basic patterns of sympathetic and parasympathetic innervation in the urinary	
	bladder and internal urethral sphincter during bladder filling (urinary continence) and er	nptying
	(micturition).	2.6
14.	Describe the role of the external urethral sphincter in urinary continence in the male and	b
	female.	2.9
15.	Describe the common disorders of the scrotum, testes, and epididymis.	2.7
16.	Describe the roles of sympathetic and parasympathetic innervation during male and fem	nale
	sexual response.	2.0
17.	Explain the role of Pap smears in detecting and preventing cervical cancer.	3.0

## VII. Basic Anatomy of Thorax

1.	Describe the osteological features of the thoracic cage and identify those that are palpa	ble.
		3.7
2.	Demonstrate the anatomic landmarks of the thoracic vertebrae, sternum, ribs, and clave	icle.
		3.9
3.	Describe the sternal angle and its use as a reference point.	3.7
4.	List the vertebral levels of suprasternal notch, sternal angle, and xiphisternal joint.	3.6
5.	Describe the costovertebral, costotransverse, sternocostal, and sternoclavicular joints.	3.3
6.	Explain the movement of the thoracic cage during respiration.	3.6
7.	Describe the vertical reference lines for the follwoing thoracic walls:	
	a. midsternal	3.7
	b. parasternal	3.7
	c. midclavicular	3.7
	d. anterior axillary	3.7
	e. midaxillary	3.7
	f. posterior axillary	3.7
	g. scapular lines	3.7
8.	Identify the surface projections of the heart and great vessels, the margins of the pleura	i, and
	the lobes and fissures of the lungs.	4.0
9.	Describe the boundaries of the thoracic inlet and outlet, and identify the structures pass	sing
	through them.	3.7
10.	Describe the pectoral region and associated structures.	3.6
11.	Describe the female breast.	3.1
12.	Describe the layers of the thoracic wall from the superficial to the deep.	3.6
13.	Describe the segmental innervation (dermatomes) of the skin of the thoracic wall.	3.6
14.	Describe the origins, insertions, innervations, and actions of the muscles of the thoracic	wall.
		3.4
15.	Describe the intercostal nerves and vessels.	3.6
16.	Describe the lymphatic drainage of the thoracic wall, with emphasis on the axillary lymp	h nodes.
		3.4
17.	Explain the structure and function of the diaphragm.	3.7
18.	Describe the surface projection of the diaphragm.	3.1
19.	Describe the mechanisms by which the thoracic cavity diameters are altered during insp	iration
	and expiration.	3.1
20.	Describe the divisions of the thoracic cavity.	3.3
21.	Describe the location of the organs within the thoracic cavity and their relationship to o	ne
	another.	3.7
22.	Describe the pleural cavity.	3.7
23.	Describe the endothoracic fascia and suprapleural membrane.	2.7
24.	Describe the visceral pleurae parietal.	3.3
25.	Describe the costomediastinal and costodiaphragmatic recesses.	3.6
26.	Describe the surface projection of the pleural.	3.3
27.	Explain the structure and function of the lungs.	3.9
28.	Compare and contrast the right and left lung.	3.7
29.	Describe the surface projections of lungs and pleura to the thoracic wall.	
		3.6

30.	Describe the innervation of, and the blood flow to and from, the lungs.	
		3.7
31.	Describe the lymph drainage of the lungs, trachea, and primary bronchi.	3.0
32.	Describe the structures in the hilum and the mediastinum of each lung.	3.6
33.	Define cardiac notch and cardiac fossa.	3.3
34.	Describe the trachea and bronchi.	3.7
35.	Characterizethe primary bronchi.	3.4
36.	Describe a bronchopulmonary segment.	3.1
37.	Label structures on cross-sections through the mediastinum.	3.7
38.	Describe the superior mediastinum.	3.6
39.	Describe the anterior mediastinum.	3.1
40.	Describe the thymus.	3.3
41.	Describe the middle mediastinum.	3.6
42.	Describe the pericardium.	3.7
43.	Describe the phrenic nerves.	3.7
44.	Identify and describe the oblique and transverse pericardial sinuses.	2.9
45.	Describe the external and internal anatomy of the heart with emphasis on the chambe	rs and
	valves.	4.0
46.	Describe the surface projections of the heart.	3.7
47.	Describe the pathway of blood flow through the heart.	4.0
48.	Explain the structure and function of the cardiac valves.	4.0
49.	Describe the arterial and venous coronary circulation.	3.9
50.	Describe the conducting system of the heart.	3.9
51.	Describe the autonomic innervation of the heart.	3.4
52.	Describe the lymphatic drainage of the heart and epicardium.	3.0
53.	Describe the cardiac skeleton.	2.7
54.	Describe fetal circulation and the changes that occur at birth.	3.6
55.	Describe the posterior mediastinum.	3.4
56.	Describe the thoracic aorta.	3.7
57.	Describe the esophagus.	3.7
58.	Describe the esophageal plexus.	3.6
59.	Describe the azygos system of veins.	3.6
60.	Explain the lymphatic drainage of the thorax.	3.4
61.	Compare and contrast the right lymphatic duct and the thoracic duct.	3.6
62.	Describe the thoracic portion of the sympathetic chain.	3.6
63.	Describe the thoracic splanchnic nerves.	3.6
64.	Describe the vagus nerves in the thorax.	3.7
65.	Compare and contrast the left and right recurrent laryngeal nerves.	3.6
66.	Explain the distribution of autonomic fibers to the upper extremity.	1.4
67.	Identify the branches of the subclavian arteries that supply structures in the thorax.	3.4
68.	Describe cervical rib syndrome.	3.0

## VIII. <u>Clinical Anatomy of Thorax</u>

1.	Identify bony features and soft tissue structures of the thorax on radiographs, MRI, CT	, and
	angiograms.	3.9

2. Describe the lymphatic drainage of the breast in relation to the spread of breast cancer. **3.7** 

3.	Explain the functional significance of the bronchial tree and bronchopulmonary segments i	n
	relation to inhalation injury and surgical resection. 3.	1
4.	Describe the clinical significance of the differences in innervation of the parietal and viscer	al
	pleura. 3.	3
5.	Define <i>pneumothorax</i> and <i>pleurisly</i> . <b>3.</b>	6
6.	Describe the clinical significance of the costomediastinal and costodiaphragmatic recesses	in
	relation to thoracocentesis. 3.	6
7.	Describe the surface projection of the heart as related to sites of auscultation of the cardia	C
	valves and describe the placement of ECG electrodes. 4.	0
8.	Describe the functional consequences of coronary artery obstruction. 4.	0
9.	Describe the mechanism of referred pain as related to thoracic organs. <b>3.</b>	7
10.	Describe the lymphatic drainage of the heart and epicardium. <b>1.</b>	6
11.	Explain the cardiac tamponade and routes of pericardio centesis. <b>3.</b>	3
12.	Identify the function, communications, and clinical significance of the azygos venous system	m.
	3.	6
13.	Describe the anomalies of the heart and great vessels. 2.	9
14.	Explain the movements that the thorax makes during ventilation and describe the motor a	nd
	sensory nerve supply, as well as pleural and peritoneal coverings. <b>1.</b>	9

### IX. Basic Anatomy of Abdomen

1.	Describe structure and function of the abdominal wall.	3.7
2.	Define aponeurosis.	3.4
3.	Relate surface landmarks of the abdominal wall to underlying structures and organs.	3.9
4.	Describe the regional and quandrant reference systems of the abdomen and identify the	eir
	contents.	3.7
5.	Describe the muscles of the abdominal wall in terms of origins, insertions, actions, inner	vations,
	and blood supply.	3.6
6.	Explain the structure and function of the rectus sheath.	3.3
7.	Describe the blood supply of the abdominal wall.	3.6
8.	Describe the dermatomes of abdominal wall.	3.7
9.	Describe the inguinal canal, including contents in both males and females.	3.7
10.	Describe the descent of the gonads.	2.9
11.	List the components of the spermatic cord.	3.6
12.	Identify the arterial and venous structures of the anterior abdominal wall.	3.6
13.	Describe the boundaries of the abdominal and peritoneal cavities.	3.6
14.	Compare and contrast the visceral and parietal peritoneum.	3.7
15.	Describe the lesser and greater peritoneal sacs and their relationships to the epiploic for	ramen.
		3.6
16.	Describe the paracolic (lumbar) gutters.	2.7
17.	Explain the structure and function of the gastrointestinal abdominal viscera and spleen.	3.7
18.	Explain the structure and function of the portal-caval system, including significant anaster	omoses.
		3.7
19.	Describe the abdominal mesenteries and relationship to the abdominal viscera; contrast	t intra-
	versus retroperitineal structures.	3.6
20.	Describe the blood supply, lymphatic drainage, and innervations of the abdominal visce	ra with
	reference to the divisions of the embryonic gut.	2.7

21. Describe the collateral circulation of the abdominal organs.	3.0
22. Relate the portal vein, common bile duct, and proper hepatic artery within the hepato	oduodenal
ligament.	3.6
23. Explain the structure and function of the kidneys and ureters.	3.9
24. Explain the structure and function of the suprarenal glands.	3.6
25. Describe the abdominal aorta and its branches.	3.9
26. Describe the inferior vena cava and its tributaries.	3.9
27. Describe the lymphatic drainage of the abdominal viscera and wall to cisterna chili.	3.3
28. Describe the muscles of the posterior abdominal wall in terms of origin, insertion, act	ion,
innervations, and blood supply.	3.6
29. Describe the autonomic innervations of the abdomen.	3.1
30. Describe the lumbar plexus and its branches.	4.0
31. Explain the structure and function of the thoracic diaphragm.	3.7

#### X. <u>Clinical Anatomy of Abdomen</u>

1.	Describ	be the palpable anatomical landmarks of the abdomen and explain their clir	nical
	signific	ance.	3.7
2.	Identifi	y the osteological features of the abdomen with diagnostic imaging.	3.7
3.	Identify	y the abdominal viscera in CTs and MRIs.	3.7
4.	Define	hydrocele and hematocele.	3.1
5.	Describ	be pain referral patterns of the abdominal viscera and the diaphragm.	3.4
6.	Describ	be the clinical significance of diaphragmatic herniation.	3.3
7.	Compa	re and contrastthe inguinal (Hesselbach's) triangle in relation to the diagno	sis of indirect
	versus	direct inguinal hernias.	3.9
8.	Compa	re and contrast inguinal and femoral hernias.	3.4
9.	Integra	te basic anatomy with the following clinical correlates:	
	a.	appendicitis	3.7
	b.	billary inflammation and stones ulcers	3.7
	с.	pancreatitis	3.7
	d.	pyelonephritis	3.7
	e.	portal hypertension	3.7
	f.	renal calculi	3.7

## XI. Basic Anatomy of Head & Neck

### A. Head

1.	Identify the position, palpable and imaging landmarks of the bones of the skull and ca	lvaria.
		3.7
2.	Describe the boundaries, walls, floors, and contents of the cranial fossae.	3.6
3.	Describe the external and internal features of the cranial foramina and list the structu	ires that
	each transmits.	3.6
4.	Describe the relationships of three meningeal coverings of the brain.	3.7
5.	Describe the arrangement of the dura mater, and its main reflections within the crani	al cavity,
	as well as therelationship to the major venous sinuses and the brain itself.	3.6
-		

6. List bones that develop from endochondral and intramembranous ossification. **1.3** 

7.	Identify the sutures of the skull.	3.6		
8.	Define fontanel. Locate and give the times of closure of the anterior and posterior fonta	inels.		
		2.6		
9.	Identify the major grooves for the intracranial venous sinuses.	3.3		
10.	Describe the origins, and summarize the courses and major branches of the facial and m	naxillary		
	arteries, including the course and intracranial relations of the middle meningeal artery a	and its		
	significance in extradural hemorrhage.	3.6		
11.	Describe the cutaneous innervation of the face.	3.7		
12.	Discuss the muscles of facial expression.	3.6		
13.	Describe the parasympathetic innervation of the parotid gland.	2.9		
14.	Describe the sympathetic innervations of the face.	3.0		
15.	Describe the course and branches of the internal carotid artery. Describe the tributaries	of the		
	internal jugular system of veins to the face.	3.6		
16.	Describe the parotid gland and its relationship to the facial nerve.	3.6		
17.	Describe the lymphatic drainage from the face and scalp.	2.9		
18.	Describe the layers of the scalp and the anatomical basis of scalping injuries.	3.0		
19.	Describe the extraocular muscles, in terms of their attachments, innervations, and actio	ns.		
		3.6		
20.	Describe the parasympathetic and sympathetic innervation of the orbit and its contents			
		3.0		
21.	Describe the arterial supply and venous drainage of the orbit.	3.6		
22.	Describe the muscles responsible for opening and closing the palpebral fissure.			
		3.3		
23.	Name the extraocular muscles, their actions, and their innervation.	3.6		
24.	Describe the intrinsic muscles of the eye, as well as their actions and innervation.	3.6		
25.	Describe the superior and inferior ophthalmic veins, and identify possible anastomotic			
	connections with the cavernous sinus, pterygoid plexus, facial veins and veins of the sca	lp.		
		2.7		
26.	List the name and Roman numeral for each cranial nerve.	3.7		
27.	Describe where each cranial nerve emerges from the brain.	3.7		
28.	Identify the foramen through which each cranial nerve passes to or from in the cranial of	avity.		
		3.7		
29.	List each cranial nerves functional component(s) and associated ganglia.	3.7		
30.	Describe the structure(s) (motor and/or sensory distribution) that (are) present in each	cranial		
	nerve.	3.7		
31.	Describe the boundaries of the infratemporal fossa.	3.0		
32.	Describe the temporomandibular joint (TMJ) and explain how the mandibular condyles	and the		
	articular disc work together in jaw opening and closing.	3.0		
33.	Describe the maxillary artery and its branches in the infratemporal fossa.	3.3		
34.	Describe the contents of the infratemporal fossa.	3.3		
35.	Describe the muscles of mastication, including attachments, innervations, and actions.	3.4		
Nack				
ive		• •		
1.	Describe the boundaries of the anterior and posterior triangles of the neck.	3.6		
2.	Describe the location and anatomic relations of the thyroid and parathyroid glands.	3.7		
3.	Describe the courses of the accessory, vagus, and phrenic nerves in the neck.	3.7		
4.	Describe the major structures passing between the neck and the thorax.	3.4		

5. Describe the courses and important relationships of the subclavian arteries and veins. **3.4** 

В.

	6. Describe the cervical plexus and its distribution.	3.0
	7. Explain the arrangement of the lymphatic drainage of the head and neck, and identify the	he major
	groups of lymph nodes and potential routes for the spread of infection and malignant d	isease.
		3.4
	8. Describe the borders of the anterior triangle of the neck and its contents.	3.6
	<ol> <li>Describe the carotid sheath and its contents.</li> <li>Describe the muscles in the anterior pack including their attachments, actions, and</li> </ol>	3./
	innervations	3 1
	11. Describe the dermatomes and the cutaneous innervation of the neck.	3.3
	12. Describe the carotid artery and its branches.	3.3
	13. Describe the carotid sinus and carotid body.	3.7
	14. Describe the brachiocephalic, external jugular and internal jugular veins.	3.7
	15. Describe the fascias of neck.	2.7
	16. Describe the boundaries of the posterior triangles of the neck and its contents.	3.1
	17. Diagram the origin and distribution of the cutaneous and muscular branches of the cerv	/ical
	plexus.	2.7
	18. Describe the autonomic nervous system in the neck.	3.0
	19. Describe the relationship between the trachea and the esophagus.	3./
С.	Pharynx and Palate	
	1. Explain the structure and function of the pharynx.	3.7
	2. Describe the roles the soft palate, its musculature, and the tongue in swallowing.	3.3
	3. Describe the blood supply and venous drainage of the pharynx.	2.9
	4. List the components and functions of the pharyngeal plexus.	3.0
	5. Give the nerve and blood supply to the palatine tonsil.	2.6
	6. Describe the anatomic arrangement and functional significance of the lymphoid tissue i	n the
	tonsils, pharyngeal, and posterior nasal walls.	1.7
D.	Oral Cavity	
	1. Describe the oral cavity.	3.6
	2. Describe the functional anatomy of the tongue, including its motor and sensory innerva	tions,
	and the role of the extrinsic and intrinsic muscles.	3.4
	3. Describe the function of the submandibular ganglion.	3.1
	4. Describe the salivary glands.	3.4
Ε.	Nasal Cavity, Paranasal Sinuses & Pterygopalatine Fossa	
	1. Describe the paranasal sinuses and their innervations.	3.3
	2. Describe the nasal cavity.	3.3
	3. Describe the pterygopalatine fossa and its contents.	3.1
F.	Larynx	
	1. Explain the structure and function of the hyoid bone and larynx.	3.7
	<ol> <li>Describe the muscles of the larynx, in terms of attachments, actions, and innervations.</li> </ol>	3.4
	3. Describe the course of the right and left recurrent laryngeal nerves.	3.7
G.	Ear	
	1. Explain the structure and function of the ear.	3.0
		-

## XII. Clinical Anatomy of Head and Neck

1. 2	Describe the clinical significance of skull fractures.	3.7 2 7	
2. २	In the posterior triangle demonstrate the position of the spinal accessory perve, the ro	J.7	
5.	trunks of the brachial plexus, the external jugular vein and subclavian vessels in relation to		
	penetrating neck trauma.	3.6	
4.	Locate the carotid pulse.	3.9	
5.	Describe the "danger area" of the face and explain how infections in this area may lead	to	
	cavernous sinus thrombosis.	3.6	
6.	Demonstrate the test for an injury of the facial nerve and describe the manifestations of	such an	
	injury.	3.7	
7.	Explain dislocation of the temporomandibular joint.	3.1	
8.	Explain the spread of infections from the oral cavity into the neck.	3.1	
9.	Describe the deviation of the tongue after hypoglossal nerve injuries.	3.6	
10.	Explain resulting affects of nerve injuries of the larynx.	3.3	
11.	Describe how fractures of the cribriform plate can result in meningitis.	2.9	
12.	Differentiate the appearance of extradural and subdural hematomas on transverse CT s	cans.	
		3.0	
13.	Define <i>Bell's palsy</i> .	3.7	
14.	Explain the clinical significance of emissary veins.	3.6	
15.	Describe inferior alveolar nerve block procedure.	2.7	
16.	Describe mandibular deviation after mandibular nerve injury.	3.4	
17.	Describe venus and lymphatic spread of infection.	2.6	
18.	Describe the clinical significance of the fascias of the neck.	2.9	
19.	Identify all bony features of the skull as indicated on radiographs (X-rays).	3.7	
20.	Describe the appearance of the carotid arteries on radiographs and magnetic resonance	2	
	angiographs.	3.1	
21.	Describe the entrance of cerebral veins into the superior sagittal sinus in subdural hemo	orrhage,	
	and explain how connections between dural venous sinuses and extracranial veins may	permit	
~~	intracranial infection.	3.7	
22.	Describe the standard anatomical and radiographic views of the skull.	3.4	
23.	Describe the blood supply of the scalp and its significance in laceration injuries.	2.0	
24	Differentiate between the correct reflex, the numillary light reflex, and the corrected	3.U	
24.	Differentiate between the corneal reflex, the pupiliary light reflex, and the accommodat		
<b>Э</b> Е	Describe the clinical testing of the ovtraccular muscles	5.4 2 7	
25.	Describe the child testing of the extraocular muscles.	5./ 2 /	
20.	Describe how the extraocular muscles can be clinically tested	3. <del>4</del> 1 0	
27.	Discuss possible observed manifestations of a lesioned nerve, and dentify whether the	1.0	
20.	manifestations would be insilateral contralateral or bilateral	34	
29	Identify the positions of the external and internal jugular veins and the surface landmar	s that	
25.	are used when inserting a central venous line	3.6	
30	Describe the venous spread of infections and the way in which they can become system	ic 2.9	
31	Relate the topographic anatomy of the neck to nalpable and radiographically visible ske	letal	
51.	structures.	3.4	
32.	Describe the anterior scalene syndrome (Scalenus Anticus Syndrome).	2.9	

33.	Describe the clinical importance of the cervical pleura in relation to trauma at the base of	
	neck.	3.0
34.	Explain the gag reflex.	3.6
35.	Identify common sites that foreign bodies can become lodged.	3.6
36.	Describe the retropharyngeal space and its clinical significance.	3.7
37.	Describe the clinical anatomy of procedures that open the airway.	3.9
38.	Explain the cough reflex.	3.6
39.	Describe the major arteries that supply the lateral wall and nasal septum in relation to	
	nosebleeds.	3.6
40.	Describe the palpable surface features of the larynx.	4.0

## **NEUROANATOMY LEARNING OBJECTIVES**

Gross Structure of the Brain Gross Structure of the Spinal Cord and Peripheral Nerves Clinical Correlations of Neuroanatomy

#### I. Gross Structure of the Brain

Describe the central axis of the portions of the brain and the anatomical direction	s for each
portion.	3.0
Explain the divisions of the CNS.	4.0
Describe the external (topographical) anatomy of the lobes of the cerebrum.	3.0
Explain the general function of the lobes of the cerebrum.	4.0
Describe the distribution of the gray and white matter of the cerebrum.	4.0
Describe the external (topographical) anatomy of the cerebellum.	4.0
Describe the distribution of the gray and white matter of the cerebellum.	3.0
Identify and describe the structure and functions of the diencephalon.	3.0
Describe the external anatomy of each region of the brainstem.	4.0
Describe the nuclei and tracts of each region of the brainstem.	4.0
Describe the sensory components of the cranial nerves.	4.0
Describe the motor components of the cranial nerves.	4.0
	Describe the central axis of the portions of the brain and the anatomical directions portion. Explain the divisions of the CNS. Describe the external (topographical) anatomy of the lobes of the cerebrum. Explain the general function of the lobes of the cerebrum. Describe the distribution of the gray and white matter of the cerebrum. Describe the external (topographical) anatomy of the cerebellum. Describe the distribution of the gray and white matter of the cerebellum. Describe the distribution of the gray and white matter of the cerebellum. Identify and describe the structure and functions of the diencephalon. Describe the external anatomy of each region of the brainstem. Describe the nuclei and tracts of each region of the brainstem. Describe the sensory components of the cranial nerves.

### II. Gross Structure of the Spinal Cord and Peripheral Nerves

1.	Describe the external (topographical) anatomy of the spinal cord.	4.0
2.	Discuss the relationship of spinal nerves to roots and rami upon entrance and exit of the cord.	spinal <b>4.0</b>
3.	Compare and contrast the effects of lesions to a dorsal root, ventral root, and spinal ner	ve.
		4.0
4.	Identify and describe the divisions (Rexed laminae) in the gray matter regions of the spin	al cord
		3.0
5.	Identify and describe the funiculi (dorsal, lateral, and anterior) in the white matter of the cord.	e spinal <b>3.0</b>
Me	eninges and Ventricles	
1.	Describe the dura mater, its reflections, and the formation of venous sinuses.	4.0
2.	Describe the relationship of epidural and subdural hematomas to the layers of the menin	nges.
		4.0
3.	Describe the arachnoid mater and the formation of the subarachnoid space.	4.0
4.	Explain the differences between the cranial and spinal meningeal layers.	3.0
5.	Describe the ventricular system of the brain and the production and flow of cerebrospin	al fluid.
		4.0
6.	Discuss the structural and functional basis of the blood brain barrier.	4.0
Vas	sculature of the Central Nervous System	
1.	Discuss the vascular supply to the brain.	4.0
2.	Explain the venous drainage of the brain.	4.0
3.	Discuss the vascular supply to the spinal cord.	4.0
4.	Explain the venous drainage of the spinal cord.	4.0
Sor	natosensory Systems of the Body	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li><i>Mee</i></li> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li><i>Vas</i></li> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li><i>Sor</i></li> </ol>	<ol> <li>Describe the external (topographical) anatomy of the spinal cord.</li> <li>Discuss the relationship of spinal nerves to roots and rami upon entrance and exit of the cord.</li> <li>Compare and contrast the effects of lesions to a dorsal root, ventral root, and spinal nervel.</li> <li>Identify and describe the divisions (Rexed laminae) in the gray matter regions of the spin describe the funiculi (dorsal, lateral, and anterior) in the white matter of the cord.</li> <li>Meninges and Ventricles</li> <li>Describe the dura mater, its reflections, and the formation of venous sinuses.</li> <li>Describe the arachnoid mater and the formation of the subarachnoid space.</li> <li>Explain the differences between the cranial and spinal meningeal layers.</li> <li>Describe the ventricular system of the brain and the production and flow of cerebrospin</li> <li>Discuss the structural and functional basis of the blood brain barrier.</li> <li>Vasculature of the Central Nervous System</li> <li>Discuss the vascular supply to the brain.</li> <li>Explain the venous drainage of the brain.</li> <li>Discuss the vascular supply to the spinal cord.</li> <li>Explain the venous drainage of the spinal cord.</li> <li>Explain the venous drainage of the spinal cord.</li> </ol>

Describe the peripheral receptors and sensory modalities of the somatosensory systems of the body.
 4.0

	2.	Describe the sensory neurons and the nerve fibers of the somatosensory systems of the	body.		
	3. 4. 5.	Discuss the dorsal column-medial lemniscus pathway. Describe the tracts of the anterolateral system. Describe the spinocerebellar tracts.	4.0 4.0 4.0 3.0		
D.	Sor	matosensory of the Head			
	1. 2. 3. 4.	Describe the peripheral receptors and sensory modalities of the trigeminal system. Describe the mesencephalic, principal (main, chief) sensory, and spinal trigeminal nuclei. Describe the central pathways of the trigeminal system. Identify and describe trigeminal reflexes.	4.0 .3.0 3.0 3.0		
Ε.	Мс	otor Systems – Somatic Motor System, Cerebellum, and Basal Ganglia			
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> </ol>	Describe the components of the somatic motor system. Describe the cortical descending pathways (corticospinal and corticonuclear tracts). Identify the components of the basal nuclei. Describe the connections of the components of the basal nuclei and their function. Recognize the neuroanatomical and functional relationships of major brainstem descend pathways. Identify the three histological layers of the cerebellar cortex. Describe the divisions of the cerebellum, their functions, and the symptoms of their dysfunctions. Describe the connections of the cerebellum.	4.0 4.0 3.0 ding 4.0 3.0 4.0 4.0		
F.	Vis	ual System			
	1. 2. 3. 4.	Describe the functional anatomy of the eye, the retina, and photoreceptors. Discuss the conversion of visual images to neural impulses. Describe the visual pathways and the visual cortex. Identify and describe pupillary light and accommodation reflexes.	4.0 4.0 4.0 4.0		
G.	Vestibular System and Medial Longitudinal Fasciculus				
	1. 2. 3. 4.	Describe the functional anatomy of the vestibular apparatus. Discuss vestibular pathways and the associated nuclei. Describe the neuroanatomical basis of the vestibuloocular reflex. Describe the anatomy and function of the ascending and descending portions of the medolongitudinal fasciculus. Describe neuroanatomical basis of nystagmus.	4.0 4.0 4.0 dial 3.0 4.0		
Н.	Auditory System				
	1. 2. 3. 4. 5.	Describe the functional anatomy of the ear (outer, middle, inner). Describe the conversion of sound waves to neural impulses and their tonotopic relations Describe the neuroanatomical basis for localization and recognition of sound. Describe the auditory pathways and auditory cortex. Identify and describe the auditory reflexes.	4.0 ships. 4.0 3.0 3.0 3.0		
Ι.	Hypothalamus				
	1.	Describe the functional anatomy of the hypothalamus.	3.0		

	<ol> <li>Describe the afferent and efferent pathways of the hypothalamus.</li> <li>Describe the supraopticohypophyseal tract and the tuberoinfundibular tract.</li> <li>Identify and describe the hypothalamic reflexes.</li> <li>Describe the role of the hypothalamus in the control of ANS function.</li> </ol>	3.0 3.0 3.0 3.0
J.	Autonomic Nervous System	
	<ol> <li>Describe the functional anatomy of the central nervous system and peripheral nervous sportions of the autonomic nervous system.</li> <li>Describe the functional anatomy of the enteric nervous system.</li> <li>Describe the central regulation of the autonomic nervous system.</li> <li>Identify and describe the autonomic reflexes.</li> </ol>	ystem 4.0 3.0 3.0 4.0
К.	Limbic System	
	<ol> <li>Describe the functional anatomy of the limbic lobe and the limbic system.</li> <li>Describe the functional anatomy of the hippocampal formation.</li> <li>Describe the functional anatomy of the amygdaloid nuclear complex.</li> <li>Describe the functional anatomy of the septal nuclei.</li> <li>Describe the functional anatomy of the nucleus accumbens.</li> </ol>	4.0 3.0 3.0 3.0 4.0
L.	Reticular Formation	
	<ol> <li>Identify the location of the reticular formation.</li> <li>Describe the reticular formation's contribution to: modulation of pain transmission, con movement, autonomic reflexes, and the ascending reticular activating system (ARAS).</li> </ol>	<b>3.0</b> trol of <b>4.0</b>
М.	Cerebral Cortex	
	<ol> <li>Describe the six histological layers of the neocortex.</li> <li>Compare and contrast archi-, paleo-, and neo-cortices.</li> </ol>	4.0 3.0
III.	Clinical Correlations of Neuroanatomy	
А.	Gross Structure of the Spinal Cord and Peripheral Nerves	
	<ol> <li>Describe peripheral nerve neuropathies.</li> <li>Explain spinal shock.</li> </ol>	4.0 3.0
В.	Meninges and Ventricles	
	<ol> <li>Discuss the disorders associated with formation, circulation, and reabsorption of cerebrilluid.</li> <li>Describe the neuroanatomical basis of meningitis.</li> <li>Describe subarachnoid hemorrhage.</li> </ol>	ospinal 4.0 4.0 4.0
С.	Vasculature of the Central Nervous System	
	1. List the criteria used for localizing brainstem lesions due to hemorrhage and vascular oc	clusion.
	<ol> <li>Describe Weber syndrome.</li> <li>Describe Parinaud syndrome.</li> </ol>	4.0 4.0 3.0

D. Somatosensory Systems of the Body

	1.	List and describe the neurological deficits seen with unilateral lesions at different points dorsal column-medial lemniscus pathway.	in the <b>4.0</b>
	2.	Describe tabes dorsalis.	3.0
	3.	Describe sensory ataxia.	4.0
	4.	List and describe the neurological deficits related to occlusion of the posterior spinal arts	ery.
			3.0
	5.	List and describe the neurological deficits related to unilateral lesions at different points	in the
		anterolateral system.	4.0
	6.	Describe referred pain.	4.0
	7.	Explain the neuroanatomical basis of phantom limb pain.	4.0
	8.	Describe syringomyelia and identify the neurologic defects characteristic of this disorder	
			4.0
	9.	Discuss central or thalamic pain.	3.0
	10.	Compare and contrast lesions in the primary and association somatosensory cortices.	4.0
F.	Sor	matosensory of the Head	
<b>L</b> .	50,		
	1.	Identify and describe the neurological deficits associated with lesions to the different cra	anial
	•	nerves and their nuclei and pathways.	4.0
	2.	Describe trochlear nerve palsy.	3.0
	3.	Discuss the underlying cause of lateral gaze paralysis.	3.0
	4.	Discuss the underlying cause of one-and-a-hair syndrome.	3.0
	5.	Discuss the underlying cause of internuclear ophthalmoplegia.	3.0
	ь. ¬	Compare and contrast oculomotor nerve paisy and Horner's syndrome.	4.0
	/. 0	Describe Bell's palsy.	3.0
	٥.	Describe trigeminal neuralgia.	4.0
F.	Мс	otor Systems – Somatic Motor System, Cerebellum, and Basal Ganglia	
	1.	Discuss the effects of a unilateral lesion in the primary motor cortex.	4.0
	2.	Discuss the effects of a unilateral lesion in the supplementary motor cortex.	4.0
	3.	Discuss the effects of a unilateral lesion in the premotor cortex.	4.0
	4.	Discuss the effects of lesions in the corticonuclear tract as it descends through the genu	of the
		internal capsule and basis pontis to terminate in brainstem nuclei.	4.0
	5.	Compare and contrast central facial (central VII) palsy with Bell's palsy.	4.0
	6.	Discuss the effects of lesions in the hypoglossal nucleus, hypoglossal nerve, nucleus amb	oiguus,
	_	and pharyngeal plexus.	4.0
	7.	Describe the effects of lesions at different points in the corticospinal tract.	4.0
	8.	Describe alternating hemiplegia.	4.0
	9.	Compare and contrast the effects of lesions in the anterior and lateral corticospinal tract	ts.
	10	Company and contract upper motor normal lower motor normal losion sizes	3.0
	10.	Compare and contrast upper motor neuron and lower motor neuron lesion signs.	4.0
	11.	Discuss clinical signs accoriated with a homicastion of the spinal cord (og. Brown Seguar	4.0 d
	12.	viscuss cinical signs associated with a nemisection of the spinal cord (eg, Brown-Sequar	u 10
	10	Synurome). Describe the neuroanatomical basis of amyotrophic lateral sclerosis	4.U 1 0
	13. 11	Describe the neuroanatomical basis of poliomyolitic	4.0
	⊥4. 1⊑	Identify the neuroanatomical nathways affected in subacute combined degeneration	4.U 2 0
	15. 16.	Describe the neurological deficits seen with occlusion of the anterior spinal artery.	5.0 4.0

	17. Discuss the symptoms and the mechanisms underlying Parkinson's disease, Huntingt	on chorea,
	Sydenham chorea and hemiballism.	4.0
	10. Describe the cause and mannestions of tardive dyskinesia.	4.0
	20. Describe Friedrich's ataxia.	4.0
G.	Visual System	
	<ol> <li>Describe the results of lesions at all points along the visual pathways.</li> <li>Compare and contrast central and peripheral lesions of the auditory pathways.</li> </ol>	3.0 3.0
Н.	Vestibular System and Medial Longitudinal Fasciculus	
	<ol> <li>Compare and contrast the mechanisms, symptoms, and tests for conduction deafness deafness.</li> </ol>	s and nerve <b>3.0</b>
Ι.	Hypothalamus	
	<ol> <li>Identify and describe the signs of damage to the major hypothalamic nuclei.</li> <li>Describe the neuroanatomical basis of autonomic dysreflexia.</li> </ol>	3.0 4.0
J.	Autonomic Nervous System	
	1. Identify and describe the hippocampal memory disorders.	4.0
	2. Describe the neuroanatomical basis of Alzheimer's disease.	3.0
	3. Describe the neuroanatomical basis of Korsakoff syndrome.	3.0
К.	Limbic System	
	1. Describe the neuroanatomical basis of Klüver-Bucy syndrome.	3.0
L.	Cerebral Cortex	
	1. Define <i>apraxia</i> .	4.0
	2. Discuss Broca's aphasia.	4.0
	3. Discuss Wernicke's aphasia.	4.0
	<ol> <li>Identity and describe the different types of agnosias.</li> <li>Describe the neuroparatemical basis of a controlatoral particulatoral and describe the neuroparatemical basis of a controlatoral particulatoral partis particulatoral particulatoral particulatoral particulatoral</li></ol>	4.0
	5. Describe the neuroanatomical basis of a contralateral neglect syndrome.	4.0

## LOWER EXTREMITY ANATOMY LEARNING

## **OBJECTIVES**

Anatomical Terminology and Gait Cycle Osteology of the Thigh and Gluteal Region Joints of the Thigh and Gluteal Region Muscles of the Thigh and Gluteal Region Vascularization of the Thigh and Gluteal Region Lymphatics of the Thigh and Gluteal Region Innervation of the Thigh and Gluteal Region Osteology of the Leg Joints of the Leg Muscles of the Leg Vascularization of the Leg Lymphatics of the Leg Innervation of the Leg Osteology of the Foot Joints of the Foot Muscles of the Foot Vascularization of the Foot Lymphatics of the Foot Innervation of the Foot Cross Sections of the Lower Extremity Lumbosacral Plexus *Surface Anatomy of the Lower Extremity* Prenatal Development of the Lower Extremity

#### I. Anatomical Terminology and Gait Cycle

1.	Describe the anatomical position of the lower extremity.	3.4
2.	Describe the major regions of the lower extremity and the skeletal structure of each re	gion.
		3.3
3.	Identify the longitudinal axis of the thigh, leg and foot regions.	2.6
4. 5.	Apply anatomical terms to their related anatomical positions of the lower extremity. Apply anatomical terms to their related movements of the lower extremity.	3.8
		3.8
6.	Describe axes and planes of motion for functional joints.	2.8
7.	Describe the stance and swing phases of the gait cycle.	2.3

#### II. Osteology of the Thigh and Gluteal Region

1.	Describe the osseous structure of the sacrum.	3.5
2.	Describe the osseous structure of the body and ala of the ilium.	3.3
2.	Describe the osseous structure of the body and ramus of the ischium.	3.1
3.	Describe the osseous structure of the body, superior ramus, and inferior ramus of the p	oubis.
		3.1
4.	Describe the osseous structure of the proximal extremity, shaft, and distal extremity of	the
	femur.	3.6
5.	Describe the angle of inclination and the angle of declination (femoral torsion).	
		3.5
6.	Identify the osteology of the thigh and gluteal region on radiographs.	3.5
7.	Describe the ossification of the femur and os coxae.	3.4

## III. Joints of the Thigh and Gluteal Region

1.	Describe the formation and ligamentous structure of the sacroiliac joint.	3.3
2.	Describe the formation, axes of motion, and ligamentous structure of the hip joint.	3.9
3.	Identify the components of the hip joint on radiographs.	3.5

#### IV. Muscles of the Thigh and Gluteal Region

1.	Describe the superficial fascia of the thigh and gluteal regions.	2.8
2.	Describe the deep fascia of the thigh and gluteal regions.	3.6
3.	Describe how the deep fascia forms the anterior, medial and posterior compartments of	of the
	thigh.	3.8
4.	Describe the formation and contents of the muscular and vascular compartments bene	ath the
	inguinal ligament (subinguinal space).	3.8
5.	Describe the formation and list the contents of the femoral sheath.	3.5
6.	Describe the origin, course, insertion, and action of the iliacus and psoas major muscles	5.
		3.8
7.	List the muscles in the gluteal region and describe the origin, course, insertion, and acti	on for
	each muscle.	3.8

8. List the muscles in the anterior compartment of the thigh and describe the origin, course,

iı	nsertion, and action for each muscle.	3.9
9. L	ist the muscles in the medial compartment of the thigh and describe the origin, course,	
ir	nsertion, and action for each muscle.	3.6
10. L	ist the muscles in the posterior compartment of the thigh and describe the origin, cours	se,
ir	nsertion, and action for each muscle.	3.9
11. C	Describe the femoral triangle and its contents.	3.8
12. C	Describe the adductor canal and its contents.	3.8
13. C	Describe the anatomical basis of a Trendelenberg Gait.	3.5

### V. <u>Vascularization of the Thigh and Gluteal Region</u>

1.	Describe the superficial veins of the thigh.	3.4
2.	Describe the superior gluteal artery and its branches in the gluteal region.	3.4
3.	Describe the inferior gluteal artery and its branches in the gluteal region.	3.5
4.	Describe the obturator artery and its branches in the thigh region.	3.4
5.	Describe the femoral (superficial femoral) artery and its branches in the thigh region.	3.8
6.	Describe the profunda femoral artery and its branches in the thigh region.	3.8
7.	Describe the collateral circulation of the hip joint.	3.6
8.	Describe avascular necrosis of the head and neck of the femur and their anatomical ba	sis.
		3.5

## VI. Lymphatics of the Thigh and Gluteal Region

1.	Describe the superficial lymphatic drainage of the thigh and gluteal region.	3.3
2.	Describe the deep lymphatic drainage of the thigh and gluteal region.	3.3
3.	Describe the inguinal lymph nodes.	3.5
4.	Describe the lymphatic flow from the inguinal lymph nodes to the cisterna chyli.	2.9

## VII. Innervation of the Thigh and Gluteal Region

1.	Describe the superior gluteal nerve and its branches in the gluteal region.	3.6
2.	Describe the inferior gluteal nerve and its branches in the gluteal region.	3.6
3.	Describe the femoral nerve and its branches in the thigh region.	3.8
4.	Describe the obturator nerve and its branches and variations (accessory obturator) in t	he thigh
	region.	3.6
5.	Describe the lateral femoral cutaneous nerve and its branches in the thigh region.	3.1
6.	Describe the posterior femoral cutaneous nerve and its branches in the thigh region.	3.3
7.	Describe the sciatic nerve and its branches in the thigh region.	4.0

## VIII. Osteology of the Leg

1.	Describe the osseous structure	of the proximal	extremity, shaft,	and distal extremity	/ of the tibia.
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		3.9
2.	Describe tibial torsion and its relationship to malleolar torsion.	3.5
3.	Describe the osseous structure of the proximal extremity, shaft, and distal extremity of	the
	fibula.	3.9

4.	Describe the osseous structure of the patella.	3.6
5.	Identify the osteology of the leg on radiographs.	3.6
6.	Describe the ossification of the tibia, fibula, and patella.	3.8

#### IX. Joints of the Leg

1.	Describe the formation and	l ligamentous structure	of the tibiofibular	joint (superior	tibiofibular).
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- 3.8
   Describe the interosseous membrane.
   3.8
   Describe the formation and ligamentous structure of the tibiofibular syndesmosis (inferior tibiofibular).
   4.0
   Describe the formation, axes of motion, ligamentous structure (extracapsular, capsular, and intracapsular), and bursa of the knee joint.
   3.8
   Identify the components of the tibiofibular joint, tibiofibular syndesmosis, and knee joint on radiographs.
   6. Explain common ligamentous, meniscal, and articular damage to the knee joint.

#### X. <u>Muscles of the Leg</u>

Describe the superficial fascia of the leg region.	3.4
Describe the deep fascia of the leg region.	3.9
Describe the formation of compartments in deep fascia of the leg.	3.9
List and describe the origin, course, insertion, and action of the muscles in the anterior	
compartment of the leg.	4.0
List and describe the origin, course, insertion, and action of the muscles in the lateral	
compartment of the leg.	4.0
List and describe the origin, course, insertion, and action of the muscles in the superficia	al and
deep posterior compartments of the leg.	4.0
Describe the relationship of the retrocalcaneal (deep) and superficial bursa to the tendo	)
calcaneus.	3.9
Describe the boundaries and contents of the popliteal fossa.	3.5
Describe anterior, deep posterior, and lateral compartment syndromes.	3.5
	<ul> <li>Describe the superficial fascia of the leg region.</li> <li>Describe the deep fascia of the leg region.</li> <li>Describe the formation of compartments in deep fascia of the leg.</li> <li>List and describe the origin, course, insertion, and action of the muscles in the anterior compartment of the leg.</li> <li>List and describe the origin, course, insertion, and action of the muscles in the lateral compartment of the leg.</li> <li>List and describe the origin, course, insertion, and action of the muscles in the lateral compartment of the leg.</li> <li>List and describe the origin, course, insertion, and action of the muscles in the superficient deep posterior compartments of the leg.</li> <li>Describe the relationship of the retrocalcaneal (deep) and superficial bursa to the tendor calcaneus.</li> <li>Describe the boundaries and contents of the popliteal fossa.</li> <li>Describe anterior, deep posterior, and lateral compartment syndromes.</li> </ul>

#### XI. Vascularization of the Leg

1.	Describe the superficial venous return of the leg and the function of the calf pump.	3.9
2.	Describe the popliteal artery and its branches.	3.8
3.	Describe the collateral circulation (genicular anastomosis) of the knee joint.	3.5
4.	Describe the anterior tibial artery and its branches.	3.9
5.	Describe the posterior tibial artery and its branches.	3.9
6.	Describe the fibular (peroneal) artery and its branches.	3.9
7.	Describe the collateral circulation (medial and lateral malleolar anastomoses) of the ankl	e joint.
		3.9
8.	Describe the anatomical basis for the formation of varicosities and thromboses.	3.5

#### XII. Lymphatics of the Leg

1.	Describe the superficial lymphatic drainage of the leg.	3.5
2.	Describe the deep lymphatic drainage of the leg.	3.8
3.	Describe the popliteal lymph nodes.	3.5

#### XIII. Innervation of the Leg

1.	Describe the common fibular (peroneal) nerve and its branches in the leg region.	4.0
2.	Describe the deep fibular (peroneal) nerve and its branches in the leg region.	3.9
3.	Describe the superficial fibular (peroneal) nerve and its branches in the leg region.	3.9
4.	Describe the tibial nerve and its branches in the leg region.	4.0
5.	Describe the formation of the sural nerve and its branches in the leg region.	4.0
6.	Describe the saphenous nerve and its branches in the leg region.	4.0
7.	Explain the anatomical basis for foot drop.	3.5

#### XIV. Osteology of the Foot

1.	Describe the anatomical, biomechanical (medial and lateral column), and surgical (forefoot	- -)
	midfoot, and rearfoot) divisions of the osteology of the foot.	3.5
2.	Describe the osseous structure of the head, neck, and body of the talus.	4.0
3.	Describe the osseous structure of the calcaneus.	4.0
4.	Describe the osseous structure of the cuboid.	4.0
5.	Describe the osseous structure of the navicular.	4.0
6.	Describe the osseous structure of the medial, intermediate, and lateral cuneiforms.	4.0
7.	Describe the osseous structure of the first, second, third, fourth, and fifth metatarsals.	4.0
8.	Describe the osseous structure of the proximal, middle, and distal phalanges.	4.0
9.	Describe the osseous structure of the constant sesamoids of the foot.	4.0
10.	Describe the ossification of the bones of the foot.	4.0
11.	List and indicate the location of the accessory ossicles and sesamoids of the foot.	4.0
12.	Identify the normal osteology, ossification, and major accessory bones of the foot on radio	graphs.
		3.6
13.	Describe the following clinical aspects of the osteology of the foot: heel spur, neutral triang	gle of the
	calcaneus, talar torsion, talar vascularization, Steida's process, metatarsal stress fractures,	fusion of
	the middle, and distal phalanges of the fifth toe.	3.5

#### XV. Joints of the Foot

1.	Describe the formation and ligamentous structure of the ankle joint.	4.0
2.	Describe the formation, axis, and motion of the functional subtalar joint.	3.5

3. Describe the formation, axes, and motion of the functional midtarsal joint. **3.5** 

4. Describe the formation and ligamentous structure of the posterior subtalar articulation. **3.9** 

5. Describe the formation and ligamentous structure of the talocalcaneonavicular articulation.

4.0

- 6. Describe the formation and ligamentous structure of the calcaneocuboid articulation. **4.0**
- 7. Describe the formation and ligamentous structure of the cuboideonavicular, cuneonavicular,

	intercuneiform, and cuneocuboid articulations.	3.8
8.	Describe the formation and ligamentous structure of the tarsometatarsal articulations.	4.0
9.	Describe the formation and ligamentous structure of the intermetatarsal articulations.	3.8
10.	Describe the formation and ligamentous structure of the lesser metatarsophalangeal articu	lations.
		4.0
11.	Describe the formation and ligamentous structure of the metatarsophalangeal articulation	of the
	hallux.	4.0
12.	Describe the formation and ligamentous structure of the interphalangeal articulations.	4.0
13.	Identify the components of the joints of the foot on radiographs.	3.8
14.	List the synovial cavities of the foot and indicate the articulations found within each synovial	al cavity.
		3.9
15.	Describe the formation, ligamentous support, and muscular support of the longitudinal and	t
	transverse arches of the foot.	3.8
16.	Describe the anatomical basis of lateral ankle sprains.	3.5

## XVI. <u>Muscles of the Foot</u>

1.	Describe the histological structure of the integument on the dorsum of the foot.	2.8
2.	Describe the structure of the hair follicles, sebaceous glands, sweat glands, and nails on the	dorsum
	of the foot.	2.8
3.	Describe the superficial fascia on the dorsum of the foot.	3.5
4.	Describe the deep fascia on the dorsum of the foot, the dorsal subfascial space, and the co	ntents of
	the dorsal subfascial space.	4.0
5.	Describe how the deep fascia forms the superior and inferior extensor retinacula.	4.0
6.	Describe the origin, course, insertion, and action of the extensor hallucis brevis and extensor	or
	digitorum brevis muscles.	4.0
7.	Describe the formation, relationship of the tendons of intrinsic muscles, and the functio	n of the
	extensor hood (expansion) of the hallux and lesser digits.	4.0
8.	Describe the histological structure of the integument on the plantar surface of the foot.	3.8
9.	Describe the structure of the sweat glands on the plantar surface of the foot.	2.8
10.	Describe the superficial fascia on the plantar surface of the foot.	3.5
11.	Describe the deep fascia (plantar aponeurosis) on the plantar surface of the foot and the fo	rmation
	of the flexor sheaths.	4.0
12.	Describe the formation of the flexor and fibular (peroneal retinacula and the contents of the	е
	tunnels formed beneath them.	3.9
13.	Describe how the deep fascia forms compartments on the plantar surface of the foot and l	st the
	contents of each compartment.	3.9
14.	List the muscles in the first layer of plantar muscles and describe the origin, course, insertic	on, and
	action for each muscle.	4.0
15.	List the muscles in the second layer of plantar muscles and describe the origin, course, inse	rtion,
	and action for each muscle.	4.0
16.	List the muscles in the third layer of plantar muscles and describe the origin, course, inserti	on, and
	action for each muscle.	3.9
17.	List the muscles in the fourth layer of plantar muscles and describe the origin, course, inser	tion, and
	action for each muscle.	3.9
18.	Describe the relationship between the tendons of the extrinsic muscles and the intrinsic m	uscles on
	the dorsal and plantar surfaces of the foot.	3.9

19.	Describe the synovial sheaths of the extrinsic muscles found on the dorsal, medial, lateral,	
	posterior, and plantar surfaces of the foot.	3.9
20.	List the common muscular variations found in the foot.	3.5
21.	Explain the spread of infections within and between compartments of the foot and leg.	4.0

#### XVII. Vascularization of the Foot

1.	Describe the superficial venous return on the dorsum of the foot.	4.0
2.	Describe the superficial venous return on the plantar surface of the foot.	3.9
3.	Describe the dorsalis pedis artery and its branches on the dorsum of the foot.	4.0
4.	Describe the medial plantar artery and its branches on the plantar surface of the foot.	4.0
5.	Describe the lateral plantar artery and its branches on the plantar surface of the foot.	4.0
6.	Describe the dorsal proper digital and plantar proper digital arteries of each digit.	4.0
7.	Describe the major anastomoses in the rearfoot and the forefoot.	4.0

8. Identify common variations in the vascular supply to the dorsum of the foot. **3.5** 

#### XVIII. Lymphatics of the Foot

1.	Explain the superficial lymphatic drainage of the foot.	3.5
2.	Explain the deep lymphatic drainage of the foot.	3.6

#### XIX. Innervation of the Foot

1.	Describe the deep fibular (peroneal) nerve and its branches on the dorsal surface of the foot.
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4.02. Describe the superficial fibular (peroneal) nerve and its branches on the dorsal surface of the foot.

- 3. Describe the lateral dorsal cutaneous nerve and its branches on the dorsal surface of the foot.
- 4.4.04.Describe the saphenous nerve and its branches on the dorsal surface of the foot.4.0
- 5. Describe the medial plantar nerve and its branches on the plantar surface of the foot. **4.0**
- 6. Describe the lateral plantar nerve and its branches on the plantar surface of the foot. **4.0**
- 7. Describe the dorsal proper digital and plantar proper digital nerves of each digit. **4.0**
- 8. Describe the medial and lateral calcaneal nerves and their branches on the foot. **4.0**
- Describe the anatomical basis for tarsal tunnel syndrome, medial calcaneal nerve entrapment, Morton's neuroma, and digital blocks.
   3.5

#### XX. Cross Sections of the Lower Extremity

- 1. Label the osteology, integument, superficial fascia, deep fascia, compartments,<br/>muscles/tendons, vessels, and nerves on a cross section through the mid-thigh.3.5
- Label the osteology, integument, superficial fascia, deep fascia, compartments, muscles/tendons, vessels, and nerves on a cross section through the tibial tuberosity of the right and left leg.
   3.6
- 3. Label the osteology, integument, superficial fascia, deep fascia, interosseous membrane,

4.0
compartment muscles/tendons, vessels, and nerves on a cross section through the middle one third of the right and left leg. **3.6** 

- 4. Label the osteology, integument, superficial fascia, deep fascia, muscles/tendons, vessels, and nerves on a cross section through the malleoli of the right and left leg. **3.6**
- Label the osteology, integument, superficial fascia, deep fascia, ligaments, muscles/tendons, vessels, and nerves on a cross section trhough each of the metatarsophanlangeal joints of the right and left foot.
   3.6
- 6. Label the osteology, integument, superficial fascia, deep fascia, ligaments, muscles/tendons, vessels, and nerves on a cross section through the proximal and distal interphalangeal joints of the right and left foot.
   3.6
- Label the osteology, integument, superficial fascia, deep fascia, ligaments, muscles/tendons, vessels, and nerves on a cross section through the mid metatarsal shaft regions of the right and leg foot.
   3.6
- Label the osteology, integument, superficial fascia, deep fascia, ligaments, muscles/tendons, vessels, and nerves on sagittal and frontal section of the foot.
   3.6

### XXI. Lumbosacral Plexus

1.	Describe the lumbar portion of the lumbosacral plexus and its branches.	3.5
2.	Describe the sacral portion of the lumbosacral plexus and its branches.	3.5
3.	Describe the dermatomes of the entire lower extremity.	3.8
4.	Describe the peripheral nerve innervation of the skin of the entire lower extremity.	4.0
5.	Describe the muscular innervation of the entire lower extremity.	4.0
6.	Describe the deep tendon reflexes of the lower extremity.	3.8
7.	Describe the superficial reflexes of the lower extremity.	3.3
8.	Describe the anatomical basis of radiculopathies and peripheral neuropathies of the lower	
	extremity.	3.5

### XXII. Surface Anatomy of the Lower Extremity

1.	Describe the surface anatomy of the thigh region.	2.9
2.	Describe the surface anatomy of the gluteal region.	2.8
3.	Describe the surface anatomy of the popliteal fossa and knee region.	3.4
4.	Describe the surface anatomy of the leg region.	3.6
5.	Describe the surface anatomy of the dorsal surface of the foot and ankle.	3.9
6.	Describe the surface anatomy of the medial surface of the foot and ankle.	3.9
7.	Describe the surface anatomy of the posterior surface of the foot and ankle.	3.9
8.	Describe the surface anatomy of the lateral surface of the foot and ankle.	3.9
9.	Describe the surface anatomy of the plantar surface of the foot.	3.9
10.	Describe Langer's lines (relaxed skin tension lines) and Shenton's lines (lines of maximum	
	extensibility).	3.5

### XXIII. <u>Prenatal Development of the Lower Extremity</u>

- 1. Explain the embryonic and fetal portions of prenatal development. **3.1**
- 2. Describe the early development of a limb bud and its differentiation into a foot, leg, and thigh

	region.	3.3
3.	Describe the development of the vascular system of the lower extremity.	3.0
4.	Describe the development of the innervation of the lower extremity.	3.1
5.	Describe the chondrification and ossification of the skeleton of the lower extremity.	3.5
6.	Describe the development of the muscles of the lower extremity.	3.4
7.	Describe the development of the joints of the lower extremity.	3.4
8.	Describe the rotation of the lower extremity.	3.6

# **BIOCHEMISTRY LEARNING OBJECTIVES**

Biological Acids, Bases and Buffers Amino Acids and Protein Structure Enzymes Molecular Biology Lipids and Biological Membranes Hormones, Second messengers, Signal Transduction Introduction of Metabolism **Bioenergetics and Energy Metabolism** Carbohydrate Metabolism Lipid Metabolism Protein and Amino Acid Metabolism Nucleotide Metabolism Heme Metabolism Hemostasis and Blood Coagulation Diabetes Free Radicals and Antioxidants Nutrition Integration of Metabolism

# I. Biological Acids, Bases and Buffers

1.	Define <i>pH</i> .	4.0
2.	Differentiate between strong acid, weak acid, strong base, weak base, and buffer.	4.0
3.	Explain the Henderson-Hasselbach equation and its applications.	4.0
4.	List the buffer systems that predominate in blood and in the interior of cells.	3.0
5.	Define acidosis and alkalosis.	4.0
6.	Explain the physiological significance of carbonic anhydrase.	1.0
7.	Explain the classification of the bicarbonate buffer as an open system.	1.0
8.	Relate plasma $CO_2$ concentration and pH.	1.0
9.	Explain the effects of hyperventilation and hypoventilation on blood pH.	1.0
10.	. Identify common disorders that lead to an acid-base imbalance.	1.0
11.	. Explain the role of the kidney in maintaining acid-base balance.	1.0

# II. <u>Amino Acids and Protein Structure</u>

## A. Amino Acids and General Concepts of Protein Structure

<ol> <li>Identify the basic structure of alpha amino acids.</li> <li>Describe the stereochemistry of amino acids.</li> </ol>	4.0 3.0
<ol> <li>List examples of neutral, polar, acidic, basic, hydrophobic, aromatic, and sulfur-cor acids.</li> </ol>	ntaining amino <b>4.0</b>
4. Describe acid-base properties of amino acids in terms of pk <sub>a</sub> , isoelectric point capacity.	and buffering <b>3.0</b>
5. Describe the properties of the peptide bond.	4.0
6. Define primary, secondary, tertiary and quaternary structures of protein.	3.0
7. Explain protein domains.	4.0
8. Describe stabilizing factors of protein structures.	4.0
9. Describe protein denaturation and conditions that can contribute to this process.	4.0
10. Explain the role of chaperones in the protein folding process.	1.0
11. Explain protein folding diseases.	1.0

# B. Relationship of Protein Structure and Function

1.	Describe structural and functional differences between hemoglobin and myoglobin.	4.0
2.	Explain the role of heme in both hemoglobin and myoglobin.	4.0
3.	Explain the oxygen dissociation curve of hemoglobin and myoglobin.	4.0
4.	Summarize the effects of $H^+$ , CO, CO <sub>2</sub> and 2,3-BPG (2,3-bisphosphoglycerate) on the a	affinity of
	hemoglobin for oxygen.	4.0
5.	Relate the unique amino acid composition of collagen to its molecular structure and	function.
		4.0
6.	Explain the role of ascorbic acid and copper in collagen synthesis.	4.0
7.	Correlate altered protein structures to sickle cell anemia, thalassemias, osteogenesis im	perfecta,
	Ehlers Danlos syndrome, and Scurvy.	3.0
8.	Distinguish between the oxygen binding capacities of HbA and HbF.	1.0

# III. <u>Enzymes</u>

1.	Explain	oxidoreductases, transferases, hydrolases, lyases, isomerases, and ligases.	4.0		
2.	Define	co-factor, coenzyme, prosthetic group, holoenzyme, and apoenzyme.	4.0		
3.	Explain	the active site and its significance to enzymatic function.			
			4.0		
4.	Describ	e the effect of enzymes on the energy of activation for the forward and reverse	e reaction,		
	and the	e equilibrium constant of a reaction.	4.0		
5.	Explain	how temperature alters enzyme-catalyzed reactions.	3.0		
6.	Relate	the importance of pH to enzyme function.	3.0		
7.	Define	V <sub>max</sub> and K <sub>m.</sub>	4.0		
8.	Describ	e Michaelis-Menten enzyme kinetics in terms of $V_{max}$ and $K_{m.}$	4.0		
9.	Explain	the Lineweaver-Burk (double-reciprocal) plot.	4.0		
10.	Graph	the effects of competitive inhibition and noncompetitive inhibition using	Michaelis-		
	Menter	n and Lineweaver-Burk double-reciprocal plots.	4.0		
11.	Explain	irreversible inhibition.	3.0		
12.	Explain	allosteric enzymes.	3.0		
13.	Contra	st allosteric kinetics and Michaelis-Menten kinetics.	4.0		
14.	Define	isoenzyme.	4.0		
15.	Define	zymogen.	4.0		
16.	Describ	e and provide specific examples for mechanisms of enzyme regulation, includin	g:		
	a.	feedback (or product) inhibition and forward activation;	4.0		
	b.	phosphorylation/dephosphorylation;	4.0		
	с.	calcium-binding proteins;	4.0		
	d.	proteolytic activation/deactivation;	4.0		
	e.	allosteric regulation;	4.0		
	f.	induction/repression;	4.0		
	g.	substrate availability; and	4.0		
	h.	compartmentalization.	4.0		
17.	Define the turn-over number and catalytic efficiency of enzymes. <b>3.0</b>				

# IV. Molecular Biology

Α.	Structure	and	Organization	of	Nucleic Acids
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1.	Describe the basic structural unit of DNA and RNA molecules.	4.0
2.	Distinguish between the primary and secondary structure of DNA and RNA.	4.0
3.	Differentiate between euchromatin and heterochromatin.	4.0
4.	Explain base pair complementarity.	4.0
5.	Explain the denaturation and renaturation of the DNA molecule.	4.0
6.	Explain nucleosome complex.	4.0
7.	Define gene and pseudogene.	3.0
8.	Contrast the organization of genes in prokaryotes and eukaryotes.	4.0
9.	Define introns and exons.	4.0
10.	Describe the structure and function of each type of RNA (mRNA, rRNA, tRNA, and Micro	RNA).
		4.0
11.	Explain ways in which various RNAs are modified.	3.0

	12. 13.	Compare mitochondrial and bacterial DNA. Explain the significance of repetitive DNA sequences.	1.0 1.0	
В.	DN	IA Replication		
	1. 2. 3. 4. 5.	Describe semi-conservative DNA replication. Define <i>origin of replication, replication fork, primer,</i> and <i>template</i> . Outline the major functions and properties of bacterial DNA polymerases I and mammalian DNA polymerases alpha, beta, and gamma. Discuss the functions of helicase and topoisomerases I and II in DNA replication. Describe the role of single-strand DNA-binding proteins in DNA replication.	3.0 4.0 I III ai 3.0 4.0	nd
	6. 7. 8. 9.	Distinguish between the leading and lagging strands of DNA. Describe Okazaki fragments. Explain telomeres in relationship to DNA replication. Rationalize DNA replication as a point of attack in chemotherapy.	3.0 4.0 4.0 4.0 3.0	
С.	Μι	utations		
	1. 2. 3. 4.	<ul> <li>Define the following types of mutations: <ul> <li>a. silent mutation</li> <li>b. nonsense mutation</li> <li>c. missense mutation</li> <li>d. read-through mutation</li> <li>e. insertion and deletion</li> <li>f. frame-shift mutation</li> </ul> </li> <li>Give examples of mutations caused by physical agents, such as UV light and X-rays.</li> <li>Define <i>mutagen</i>.</li> <li>Describe the following DNA damage repair: <ul> <li>a. base excision repair</li> <li>b. mismatch repair</li> <li>c. repair of DSBs (double stranded breaks)</li> </ul> </li> </ul>	4.0 4.0 4.0 4.0 4.0 3.0 4.0 4.0 4.0 4.0	
D.	Tro	anscription and RNA processing		
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> </ol>	<ul> <li>Define transcription.</li> <li>Differentiate between coding and non-coding (template) strand of a gene.</li> <li>Describe post-transcriptional processing and modifications of mRNA, rRNA, and eukaryotes.</li> <li>Compare and contrast the regulation of transcription in eukaryotes and prokaryotes.</li> <li>Define basal transcription factors.</li> <li>Describe the relationship between mRNA and coding strand of DNA.</li> <li>Explain how errors in RNA modifications can lead to β-thalassemia and phenylketonuria Identify the target of alpha-amanitin.</li> </ul>	4.0 4.0 tRNA 4.0 4.0 1.0 1.0 1.0	in
Ε.	Tro	anslation and Protein Processing		
	1. 2. 3.	Explain the translation process (initiation, elongation, and termination). Identify properties of genetic code, codons and anticodons. Explain the "Wobble Hypothesis."	4.0 4.0 4.0	

	4.	Outline the basic components required for protein synthesis and outline their roles.	4.0					
	5.	Explain the role of tRNA in translation.	4.0					
	6.	Explain the "proofreading" function of amino acyl-tRNA synthetases.	3.0					
	7.	List post-translational modifications of proteins.	4.0					
	8.	Describe the role of the signal peptide in protein translocation and secretion.	3.0					
	9.	Discuss protein turnover with reference to the role of ubiquitin and the proteasome.	4.0					
	10.	Describe the effects of antibiotics on translation in prokaryotes.	3.0					
F.	Reg	gulation of Gene Expression						
	1.	Define:						
		a. chromatin remodeling	4.0					
		b. acetylation/deacetylation of histone	4.0					
		c. methylation/demethylation of DNA	3.5					
		d. epigenetics	3.5					
		e. gene rearrangement	1.5					
		f. gene amplification	1.5					
		g. gene expression	4.0					
		h. operon	3.5					
		i. promoter	3.5					
		j. operator	3.5					
		k. inducers	3.5					
		I. responsive elements	3.5					
	2.	Differentiate between the following terms repressors and co-repressors; and transcript	tion					
		activators and co-activators.	3.5					
	3.	Describe the regulation of <i>lac</i> operon.	3.5					
	4.	Explain the regulation of eukaryotic gene expression at multiple levels.	4.0					
	5.	Describe the gene regulatory functions of the steroid/thyroid hormone receptor super	family.					
			3.5					
	6.	Describe the basic functional motifs/domains of DNA-binding proteins.	1.0					
	7.	7. Characterize mRNA transport and stability as important to the regulation of gene expression.						
	0. Characterize the initiation of the relation of the second structure to the theory of the second structure of							
	8. Characterize the initiation of translation as important to the regulation of gene expr							
	_	eukaryotes.	3.0					
	9.	Describe the regulation of <i>Trp</i> operon.	1.0					
	10.	Explain stringent response in bacteria.	1.0					
	11. Explain the regulation of gene expression by extracellular factors. <b>1.0</b>							
	12.	Describe RNA editing using the expression of ApoB-48 as an example.	1.0					
	13.	Define <i>microRNA</i> (miRNA).	1.0					
	14.	Explain small interference RNA (siRNA).	1.0					
	15.	Explain KINA INTERTERENCE (KINAI).	1.0					
	16.	Describe the effect of mikina and sikina on gene expression.	1.0					
G.	Bio	technology						
	1.	Explain gel electrophoresis.	4.0					
	2.	Explain Sanger's dideoxynucleotide DNA Sequencing method.	4.0					
	3.	Explain the significance of using dideoxynucleotides in DNA sequencing technique.	4.0					
	4.	Explain how restriction enzymes work.	4.0					

5. Describe how restriction enzyme digests of a given DNA sequence are used in re	combinant DNA
molecule generation.	4.0
6. Explain the use of plasmids as cloning vectors.	4.0
7. Describe how to produce a genomic library.	4.0
8. Describe how to produce a cDNA library.	4.0
9. Explain the production of recombinant proteins.	4.0
10. Describe the following techniques:	
a. Southern blotting analysis	4.0
b. Northern blotting analysis	4.0
c. Western blotting analysis	4.0
d. ELISA	4.0
e. Immunohistochemistry	4.0
11. Explain PCR.	4.0
12. Explain RT-PCR.	4.0
13. Explain RFLP analysis.	4.0
14. Explain the usefulness of allele-specific oligonucleotide (ASO) probes.	4.0
15. Explain GeneArrays (or Microarrays).	4.0
16. Explain how microarrays are used to determine the differing patterns of gene explanation of gene expla	pression in two
different types of cell.	4.0
17. Explain gene targeting and transgenic animals.	1.0

# H. Cancer

1.	Define proto-oncogenes and oncogenes.	4.0
2.	List classes of proteins coded for by proto-oncogenes.	3.0
3.	Summarize the mechanisms through which proto-oncogenes become oncogenes.	3.0
4.	Identify a tumor suppressor gene.	4.0
5.	Outline the process of carcinogenesis (using colorectal cancer as an example).	3.0
6.	Describe the role of telomerase in cancer etiology.	3.0

# V. Lipids and Biological Membranes

1.	Define:	
	a. amphipathic	4.0
	b. emulsification	4.0
	c. liposome	4.0
	d. <i>micelle</i>	4.0
2.	Describe the structural features of fatty acids, phospholipids, triglycerides, and ch	olesterol.
		4.0
3.	Describe the functions of biological membrane.	4.0
4.	Evaluate the main functions of biological membranes.	4.0
5.	Analyze the role of membrane proteins.	4.0
6.	Distinguish between intrinsic and extrinsic membrane proteins and describe the	structural
	properties of each.	4.0
7.	Evaluate the role of cholesterol in biological membranes.	4.0
8.	Compare active transport, secondary active transport, symport, and antiport.	3.0
9.	Distinguish between facilitated diffusion and simple passive diffusion.	4.0
10.	Identify the defective ion channels in cystic fibrosis.	1.0

### VI. Hormones, Second Messengers, Signal Transduction

1.	Define hormone and distinguish between	endocrine, paracrine,	and autocrine signaling. 4.0
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2.	Differentiate between the properties and mode of action of the hydrophilic and h	ydrophobic
	hormones.	4.0

- Define second messenger.
   Describe the structure and function of monomeric and trimeric G-proteins.
   Describe how cAMP mediates signal transduction between the plasma membrane and the cytosol.
   4.0
- 6. Describe the IP<sub>3</sub>/DAG signal transduction system.
- Outline the mode of action of growth factors and the role of receptors with endogenous enzyme activity.
   4.0
- 8. Distinguish between the modes of action of insulin and glucagon. **4.0**
- 9. Explain the mode of action of cholera and pertussis toxin. **3.0**

### VII. Introduction of Metabolism

- Contrast the roles of anabolic and catabolic pathways.
   Explain the functions of NAD<sup>+</sup>, NADP<sup>+</sup>, FAD, and FMN in enzymatic reactions.
   Explain the central roles of glucose 6-phosphate, acetyl-CoA, and pyruvate in the integration of metabolic pathways.
   4.0
- 4. Differentiate between substrate-level phosphorylation and oxidative phosphorylation. **4.0**
- 5. Describe the regulation of anabolic and catabolic pathways by insulin and glucagon. **4.0**

### VIII. Bioenergetics and Energy Metabolism

1.	Describe the concept of free energy change of the reaction.	4.0
2.	Explain the relationship between the free energy change ( $\Delta G$ ) of the reaction and stand	dard free
	energy change ( $\Delta G^0$ ) of the reaction.	4.0
3.	Explain reaction coupling.	4.0
4.	Describe high-energy bonds.	4.0
5.	Differentiate exergonic and endergonic reactions.	4.0
6.	Explain oxidation and reduction.	4.0
7.	Describe the structure and function of mitochondrion and its various compartments.	4.0
8.	Determine the localization and function of the components of the mitochondrial	electron
	transport chain (ETC).	4.0
9.	Identify common inhibitors of ETC.	4.0
10.	Explain the concept of transporting reducing equivalents across mitochondrial men	nbranes.
		4.0
11.	Explain chemiosmotic potential (or proton motive force) and its relation to mitochone	drial ATP
	production.	4.0
12.	Describe mitochondrial ATP synthase.	4.0
13.	Explain oxidative phosphorylation.	4.0
14.	Explain uncoupling proteins and other uncoupling agents.	3.0
15.	Explain P/O ratio.	3.0

4.0

16.	Explain OXPHOS diseases.	1.0
17.	Explain standard oxidation reduction potential ( $E_0$ ).	1.0
18.	Explain how oligomycin inhibits ATP synthase and ultimately inhibits the activity of ETC.	1.0

# IX. Carbohydrate Metabolism

1.	Differentiate between mono-, di-, oligo-, and poly-saccharides.	4.0
2.	Define <i>aldose</i> and <i>ketose</i> .	4.0
3.	Explain the breakdown of carbohydrates in the digestive system.	4.0
4.	Describe carbohydrate uptake by the cells of the intestinal epithelium.	4.0
5.	Identify disorders related to carbohydrate absorption.	3.0
6.	Describe phosphorylation-coupled trapping of carbohydrates.	4.0
7.	Describe the glycolytic degradation of glucose, galactose, and fructose.	4.0
8.	Outline regulated steps in glycolysis and identify the regulatory factors. 4.0	
9.	Identify the glycolytic reactions that consume or generate ATP.	4.0
10.	Explain the significance of oxidation of NADH in anaerobic glycolysis.	4.0
11.	Describe the Cori cycle.	4.0
12.	Explain the consequences of the following	
	a. Pyruvate kinase deficiency	3.0
	b. Fructose intolerance	3.0
	c. Classic galactosemia	3.0
	d. Arsenic poisoning	3.0
13.	Describe gluconeogenesis.	4.0
14.	Explain the regulation of gluconeogenesis.	4.0
15.	Explain how impaired gluconeogenesis causes lactic acidosis and fasting hypoglycemia.	4.0
16.	Characterize the importance of insulin- and glucagon-dependent regulation of glyco	lysis and
	gluconeogenesis.	4.0
17.	Describe the pentose phosphate pathway (HMP).	4.0
18.	Describe the consequences of glucose-6-phosphate dehydrogenase deficiency.	4.0
19.	Explain how insulin, glucagon, and epinephrine influence carbohydrate metabolism to	maintain
	blood glucose level.	4.0
20.	Compare the physiological functions of liver and muscle glycogen stores.	4.0
21.	Differentiate between glycogenesis and glycogenolysis.	4.0
22.	Identify and describe glycogen storage diseases (von Gierke and McArdle diseases).	3.0
23.	Describe the influence of alcohol on carbohydrate metabolism.	3.0
24.	Describe the structures and functions of GAGs and proteoglycans.	4.0
25.	Define mucopolysaccharidoses.	1.0
26.	Describe the synthesis of lactose.	1.0

# X. Lipid Metabolism

# A. Fatty Acid Oxidation (Beta-oxidation) and Ketogenesis

1.	Identify when and where lipolysis, fatty acid oxidation, and ketogenesis occur.	4.0
2.	Describe the function and regulation of hormone-sensitive lipase in lipolysis.	4.0
3.	Explain fatty acid activation.	4.0
4.	Explain the function and regulation of carnitine shuttle.	4.0

	<ol> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> <li>11.</li> <li>12.</li> </ol>	Describe β-oxidation of various types of fatty acids (saturated fatty acids, unsaturated facids, and branched-chain fatty acids). Describe the metabolic fate of the products of fatty acid oxidation. Compare and contrast the fatty acid oxidation that occurs in the mitochondria versus in peroxisomes. Identify the ketone bodies produced in the liver and explain their metabolic fates. Explain the decreased rate of gluconeogeneis from ketone body oxidation. Characterize fatty acids as unusable precursors for the net synthesis of glucose. Explain why limited food intake can trigger disease conditions in individuals with the metabolic fates deficiency. Characterize dietary intake of medium-chain and short-chain fatty acids as beneficial to individuals with carnitine deficiency.	atty 3.0 4.0 the 3.0 3.0 4.0 4.0 edium- 3.0 2.0
В.	Fat	tty Acid Biosynthesis	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> </ol>	Identify when and where fatty acid synthesis occurs. List enzymes involved in the pathway from citrate to fatty acyl-CoA and identify the first committed step. Explain the significance of NADPH as substrate and palmate, and CO <sub>2</sub> as products in this pathway. Describe the reactions catalyzed by ATP-citrate lyase and acetyl CoA carboxylase and the regulations. Describe the reaction carried out by FA synthase and explain the structural properties of enzyme. Explain how fatty acids are elongated and desaturated. Explain why essential fatty acids are required in the human diet.	4.0 t- 4.0 eir 4.0 of this 3.0 4.0 4.0
С.	ΤA	G and Membrane Lipid Biosynthesis	
	1. 2. 3. 4. 5. 6. 7.	Describe TAG synthesis. Describe the biosynthesis of eicosanoids. Describe the principal regulatory enzymes, such as phospholipase A <sub>2</sub> and the cyclooxyge (COX-1 and COX-2). Describe the mechanism of action of anti-inflammatory steroids and NSAIDs (non-stero inflammatory drugs) in modulating the biosynthesis of the eicosanoids. Describe the functions of leukotrienes, prostaglandins, and thromboxanes. Compare the biological potency of the prostaglandins and thromboxanes made from or and omega-3 fatty acids. Explain biochemical defects associated with sphingolipidoses, such as Tay-Sachs, Gauch Niemann-Pick diseases.	4.0 4.0 enases 4.0 idal anti- 4.0 1.0 nega-6 1.0 er, and 1.0
D.	Cho	olesterol Metabolism	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> </ol>	Describe the structure of cholesterol. Compare and contrast cholesterol and cholesterol ester in terms of chemical characteric cellular significance. Identify when and where cholesterol synthesis occurs. Describe the pathway of cholesterol synthesis in three phases: synthesis of HMG-CoA, so of mevalonic acid, and synthesis of cholesterol. Explain the regulation of the cytosolic HMG-CoA reductase. Explain the biochemical basis of how the statin drugs lower serum cholesterol.	4.0 stics and 4.0 4.0 synthesis 4.0 4.0 4.0

7. Explain the occurrence of rhabdomyolysis in some patients on statin drugs.

### E. Cholesterol Derivatives

1. Describe the function and physiological significance of bile acid/bile salt synthesis and excretion.

		4.0
2.	Identify and describe the two-phase reactions that convert cholesterol into bile acids a	nd bile
	salts.	4.0

- Explain how bile salt is recycled.
   Explain the regulation of bile acid synthesis via cholesterol 7-α-hydroxylase.
   Describe the mechanisms and cellular locations of the synthesis of cholecalciferol, 25-
- hydroxycholecalciferol, and 1,25-dihydroxycholecalciferol.
  6. Describe the mechanism of action of cholestyramine, HMG-CoA reductase inhibitors (satins), niacin, and weight loss in managing hypercholesterolemia.
  1.0
- 7. Explain the etiology of cholelithiasis.

### F. Plasma Lipoproteins and Lipid Transport

- Compare and contrast chylomicron (CM), chylomicron remnant, VLDL, IDL, LDL, and HDL in terms of composition, function, location of synthesis, and delivery of lipid contents.
   4.0
- Describe the reactions catalyzed by the following enzymes: lipoprotein lipase (LPL); phosphatidylcholine: cholesterol acyltransferase (PCAT, also known as LCAT, in which "L" stands for lecithin); acyl-CoA: cholesterol acyltransferase (ACAT); and hepatic lipase.
   3.0
- Describe the role of cholesterol ester transfer protein (CETP) and PCAT in reverse transport of cholesterol by HDL.
   Explain the etiology of familial hypercholesterolemia.
   3.0
- 5. Describe the process of atherosclerosis and the roles played by LDL and HDL. **3.0**
- 6. Relate apoE to Alzheimer's disease.

### XI. Protein and Amino Acid Metabolism

### A. Protein Digestion

1.	Describe the process of dietary protein digestion.									4.0			
2.	Describe	the	transport	systems	involved	in	the	uptake	of	amino	acids,	dipeptides,	and
	tripeptides.								1.0				

3. Explain the disorders of amino acid absorption/reabsorption (hartnup, cysteinuria). **1.0** 

### B. Transamination and the Urea Cycle

	1.	Describe the basic function of transaminases and the role of pyridoxal	phosphate	in					
		transamination reactions.	4.0						
	2.	Describe the metabolic processes that produce ammonia.	4.0						
	3.	Explain the role of the urea cycle in ammonia detoxification.	4.0						
	4.	Identify the enzymes and their respective locations of the urea cycle.	4.0						
	5. List the sources of nitrogen incorporated into urea.								
	6. Describe the regulation of the urea cycle.								
	7.	Describe the disorders of the urea cycle (OTC deficiency, Arginase deficiency).	2.0						
С.	Me	etabolism of Individual Amino Acids							

1.0

1.0

1.0

	1.	Outline the steps in the metabolism of branched-chain amino acids.	4.0
	2.	Describe the common biochemical defect involved in Maple Syrup Urine disease.	4.0
	3.	Describe the pathways of creatine synthesis, phosphorylation, and catabolism.	4.0
	4.	Describe the metabolism of methionine and cysteine.	3.0
	5.	Explain the relationship between hyperhomocysteinemia and vitamin $B_{12}$ deficiency.	3.0
	6.	Identify glucogenic and ketogenic amino acids.	4.0
	7.	Explain the role of SAM, tetrahydrofolate ( $FH_4$ ) and vitamin $B_{12}$ in one carbon me	tabolism.
			4.0
	8.	Explain the cause and symptoms of Phenylketonuria (PKU).	4.0
	9.	Identify the amino acids that are precursors for the synthesis of dopamine, norepi	nephrine,
		acetylcholine, histamine, GABA, glutathione, and creatine.	3.0
	10.	Describe the major pathway of serine biosynthesis.	1.0
	11.	Describe the major pathway of glycine formation.	1.0
	12.	Explain the role of folic acid in glycine metabolism.	1.0
	13.	Relate homocysteine and cardiovascular disease.	1.0
	14.	Describe the function of tertrahydrobiopterin and dihydrobiopterin reductase in the me	etabolism
		of aromatic amino acids.	1.0
	15.	Relate tryptophan and niacin.	1.0
D.	Am	ino Acid Metabolism in Tissues	

1.	Desc	ribe	the m	netabolic	rates	of ami	no a	acids	released	from	n mus	cle	in th	e fasting state.	4.0
-	_								-			~			

- 2. Describe the pathways of amino acid oxidation in muscle in the fasting state. 4.0 4.0
- 3. Describe the Alanine-Glucose cycle and explain its function.
- 4. Describe the role of the purine-nucleotide cycle in muscle. 1.0

#### XII. **Nucleotide Metabolism**

### A. General Concepts

- 1. Differentiate between nucleoside, nucleotide, deoxynucleosides and deoxynucleotides. 3.0
- 2. Contrast the functions of ribonucleotides and deoxyribonucleotides. 3.0
- 3. Describe the importance of Pentose Phosphate Pathway (also called Hexose Monophosphate Shunt) for biosynthesis of nucleotides. 4.0
- 4. Describe the importance PRPP synthetase and its regulation in relationship to purine and pyrimidine nucleotide synthesis. 4.0
- 5. Identify structures of purines (adenine and guanine) and pyrimidines (cytosince, uracil, and thymine). 1.0
- 6. Explain the usage of purine and pyrimidine analogs in cancer treatment, viral infections, and gout.

1.0

### B. Metabolism of Purine Nucleotides

1.	Describe <i>de novo</i> synthesis of purine nucleotides.	4.0
2.	Describe salvage pathways of purine nucleotide biosynthesis.	4.0

- 3. Outline the regulatory steps of *de novo* and salvage pathways of purine nucleotide synthesis.
- 3.0 4. Describe the importance of folate in purine nucleotide biosynthesis.

4.0

	5.	Explain the conversion of ribonucleotides into deoxyribonucleotides.	4.0
	6.	Describe degradation of purine nucleotides.	4.0
	7.	Relate hyperuricemia and gout disease.	4.0
	8.	Compare the chemotherapies available for the management of gout.	3.0
	9.	Describe Lesch-Nyhan syndrome.	3.0
	10.	Explain severe combined immunodeficiency (SCID) due to adenosine deaminase	deficiency.
			1.0
	11.	Explain the classification of certain sulfanomides (also called PABA analogs) as	antibiotics.
			1.0
	12.	Explain positive and negative regulations of ribonucleotide reductase.	1.0
	13.	Explain the effect of hydroxyurea on ribonucleotide reductase.	1.0
	14.	Explain why deficiency of glucose-6-phosphatase may lead to gout.	1.0
С.	Ме	tabolism of Pyrimidine Nucleotides	
	1.	Describe the de novo synthesis pathway of pyrimidine.	4.0
	2.	Identify and describe the key regulatory step of de novo synthesis pathway of	pyrimidine.
			4.0
	3.	Explain the importance of carbamoyl phosphate synthetase II.	4.0
	4.	Differentiate between carbamoyl phosphate synthetase II and carbamoyl phosphate	synthetase
		l.	3.0
	5.	Describe thymidylate synthase and the reaction it catalyzes.	4.0
	6.	Explain the effect of folate deficiency on the activity of thymidylate synthase.	4.0
	7.	Explain the use of 5-fluorouracil (5-FU) as an anti-cancer drug.	3.0
	8.	Explain the use of methotrexate as an anti-cancer drug.	4.0
	9.	Describe orotic aciduria and its treatment.	1.0
	10.	Describe the conversion of UMP to CTP.	1.0

# XIII. <u>Heme Metabolism</u>

1.	Describe the pathway of heme synthesis.	4.0
2.	Describe heme catabolism.	3.0
3.	Describe the metabolism of bilirubin in the liver and in the gut.	3.0
4.	Distinguish between hemolytic, cholestatic and hepatocellular jaundice.	4.0
5.	Explain -ALA synthase inhibition from hemin.	1.0
6.	Explain porphyrias.	1.0
7.	Relate photosensitivity to porphyrias.	1.0
8.	Explain the effect of lead poisoning on heme synthesis.	1.0
9.	Describe the formation of urobilinogen.	1.0

# XIV. <u>Hemostasis and Blood Coagulation</u>

1.	Define:	
	a. hemostasis	4.0
	b. coagulation	4.0
2.	Describe the role of platelets in wound healing.	4.0
3.	Describe the roles of the following factors in platelet activation and aggregation:	
	a. ADP	4.0

	b.	Platelet activating factor (PAF)	4.0
	с.	Thromboxane $A_2$ (TXA <sub>2</sub> )	4.0
	d.	Prostacyclin (PGI <sub>2</sub> )	4.0
4.	Explair	the anti-platelet effect aspirin in low dosages.	4.0
5.	Describ	be the role of von Willebrand factor in coagulation.	4.0
6.	Describ	be the following pathways of coagulation:	
	a.	Tissue factor pathway (extrinsic)	4.0
	b.	Contact activation pathway (intrinsic)	4.0
	с.	Final common pathway	4.0
7.	Explair	η the importance of vitamin K-dependent $\gamma$ -carboxylation of certain glutamate re	sidues of
	factors	VII, IX, X, II and proteins C and S.	3.0
8.	Explair	the importance of vitamin K epoxide reductase (VKOR or VKORC).	3.0
9.	Descrit	be the action mechanisms of the following anticoagulants:	
	a.	Antithrombin	4.0
	b.	Heparin	4.0
	с.	Tissue factor pathway inhibitor (TFPI)	4.0
	d.	Proteins C and S	4.0
10.	Explair	the anti-coagulation action of warfarin and related coumarins.	4.0
11.	Define	fibrinolysis.	4.0
12.	Explair	the action of plasmin.	4.0
13.	Explair	the roles of the following factors in fibrinolysis:	
	a.	Tissue plasminogen activator (tPA)	4.0
	b.	Urokinase	4.0
	с.	Plasminogen	4.0
	d.	Plasminogen activator inhibitor (PAI)	4.0
	e.	$\alpha$ 2-antiplasmin and $\alpha$ 2-macroglobulin	4.0
	f.	Thrombin-activatable fibrinolysis inhibitor (TAFI)	4.0

# XV. <u>Diabetes</u>

2. Differentiate between type 1 and type 2 diabetes, including treatment of each. <b>4.0</b>
3. Explain a glucose tolerance test. 4.0
4. Discuss metabolic syndromes of type I and type II diabetes. 4.0
5. Explain non-enzymatic glycation of proteins. 4.0
6. Explain the clinical significance of HbA1 <sub>c</sub> levels. 4.0
7. Describe the polyol pathway and its role in diabetic retinopathy and neuropathy. <b>4.0</b>
8. Describe ketoacidotic, hyperosmolar, and hypoglycemic diabetic comas, including the
treatments for each. 4.0
9. Explain insulin synthesis. 4.0
10. Explain the regulation of insulin secretion.4.0
11. Explain the significance of the postprandial level of plasma C-peptide. <b>3.0</b>
12. Contextualize insulin action within blood glucose regulation.4.0
13. Identify the risk factors associated with type II diabetes.3.0
14. Explain gestational diabetes.1.0

# XVI. Free Radicals and Antioxidants

1.	Define free radicals [or reactive oxygen species (ROS)].	4.0
2.	Define antioxidant.	4.0
3.	Explain mitochondrial metabolism of ROS.	4.0
4.	Describe the synthesis of nitric oxide by nitric oxide synthase (NOS).	4.0
5.	Define <i>oxidative stress</i> .	4.0
6.	List the types of ROS-induced damages in the cell.	4.0
7.	Describe the production of free radicals during ischemic reperfusion.	3.0
8.	Describe the functions of superoxide dismutase, catalase, and glutathione peroxidase.	4.0
9.	Characterize the biological importance of glutathione.	4.0
10.	Describe glutathione reductase and its importance.	4.0
11.	Relate hemolytic anemia to G6PD deficiency.	4.0
12.	Explain the oxygen-dependent pathway of microbial killing in neutrolphils.	4.0

# XVII. <u>Nutrition</u>

В.

# A. Metabolic Fuels and Dietary Components

1.	Define <i>calories</i> .	4.0
2.	Explain resting metabolic rate (BMR), dietary reference intakes (DRI), and daily energy	
	expenditure.	4.0
3.	List the energy content (calories) of carbohydrates, alcohol, fat, and protein.	4.0
4.	Explain the glycemic index of foods.	4.0
5.	Compare and contrast proteins from wheat, corn, rice and beans against animal protein	is in
	terms of quality.	4.0
6.	Explain the protein-sparing effect of carbohydrate.	4.0
7.	Differentiate between Kwashiorkor and Marasmus.	4.0
8.	Discuss methods used for nutritional assessment.	4.0
9.	List the water and fat-soluble vitamins and the function of each.	3.0
10.	. Describe the symptoms of the following vitamin deficiencies	
	a. Niacin deficiency and Pellagra	3.0
	b. Vitamin B1 deficiency and Beri-Beri and Wernicke-Korsakoff syndromes	3.0
	c. Vitamin C deficiency and Scurvy	3.0
	d. Vitamin D deficiency and Rickets and Osteomalasia	3.0
	e. Vitamin A deficiency and night blindness and retardation of growth	3.0
	f. Vitamin K deficiency and hemorrhage	3.0
	g. Folic acid deficiency and megaloblastic anemia and birth defects	3.0
	h. Vitamin B <sub>12</sub> deficiency and pernicious anemia	3.0
	i. Vitamin B <sub>2</sub> deficiency and dermatitis	3.0
11.	. Identify and define the essential nutrients.	1.0
Th	e Fed or Absorptive State	
1.	Define fed/absorptive state.	4.0
2.	Describe the digestion and absorption of dietary carbohydrates, proteins, and fats.	4.0
3.	Describe the changes in hormone levels after a meal.	4.0
4.	Identify and compare glucose metabolism during the fed state in the following tissues:	
	a. Liver	4.0

		b.	brain and other neural tissues	4.0		
		с.	red blood cells	4.0		
		d.	muscle	4.0		
		e.	adipose tissue	4.0		
	5.	Describe	e metabolism of lipoproteins in the fed state.	4.0		
	6.	Describe	e metabolism of amino acids in the fed state.	4.0		
С.	C. Fasting and Starvation					
	1.	Define <i>f</i>	fasting state.	4.0		
	2.	Explain	the metabolism of the liver during fasting.	4.0		
	3.	Explain	the metabolism of adipose tissue during fasting.	4.0		
	4.	Explain	the effects of prolonged fasting on the body.	4.0		

5. Define prolonged fasting/starvation.4.0

6. Describe the metabolic changes in various tissues during prolonged fasting. **4.0** 

### XVIII. Integration of Metabolism

- Identify the major metabolic pathways operating in the liver, brain, red blood cell, heart and skeletal muscle, adipose tissue, and the metabolic fuels used by them.
   4.0
- Analyze the necessity of organs to work together to ensure availability of fuels in the bloodstream.
   4.0
- Describe how insulin, glucagon, and epinephrine regulate metabolic pathways via the regulation of key enzymes in various tissues.
   4.0
- Describe the alterations in metabolism that occur in the obese state and the biochemical signals regulating obesity.
   4.0
- 5. Explain the metabolic changes that occur during acute and chronic ethanol consumption.

3.0

# **EMBRYOLOGY LEARNING OBJECTIVES**

Fertilization, Implantation, and Early Development Development of the Gastrointestinal System Development of the Respiratory System Development of the Cardiovascular System Development of the Urogenital System Development of the Pharyngeal Apparatus and the Head and Neck Development of the Nervous System Development of the Musculoskeletal System Development of the Integumentary System

# I. Fertilization, Implantation, and Early Development

1.	Define:	
	a. blastomere	3.0
	b. <i>morula</i>	3.0
2.	Describe the process of fertilization.	2.0
3.	Explain the process of cleavage of the zygote.	2.0
4.	Discuss the formation of the blastocyst, including components and the products of their	
	formation.	3.0
5.	Describe the process of implantation, including the formation of the bilaminar disc.	3.0
6.	Describe the process of chorionic villi formation.	2.0
7.	Describe the process of gastrulation and the formation of the germ layers.	3.0
8.	Identify germ layers and their derivatives.	3.0
9.	Discuss the reorganization of the intraembryonic mesoderm.	3.0
10.	Describe the processes and significance of notochordal development.	3.0
11.	Describe the process of embryonic folding and the formation of the intraembryonic coel	lom.
		3.0
12.	Explain the critical nature of the fourth through eighth weeks of human development.	3.0
13.	Compare and contrast the major features of the embryonic and fetal periods of develop	ment.
		2.0
De	velopment of the Gastrointestinal System	

1.	List the derivatives and major developmental events, including common anomalies, of the	
	foregut, the midgut, and the hindgut.	3.0
2.	. Discuss the rotations, malrotations, and repositioning of the embryonic gut and gut-derivative	
	organs.	3.0
3.	Describe the process and anomalies of recanalization of the gut tube.	2.0

# III. <u>Development of the Respiratory System</u>

II.

IV.

1.	Describe the development of the respiratory system, including stages of lung development	nent.
		2.0
2.	Describe the formation of the diaphragm, including development of congenital diaphra	agmatic
	hernia (CDH).	2.0
3.	Describe the embryogenesis of tracheoesophageal atresias, stenoses, and fistulas.	2.0
Development of the Cardiovascular System		

Describe the development of the primitive cardiovascular system and blood cells.	3.0
Describe the formation of the embryonic heart tube.	3.0
Describe the development of the fetal heat from the embryonic heat tube.	3.0
Explain septation of the atria and ventricles, and discuss commonly associated defects.	3.0
Describe the development and derivatives of the aortic arches.	2.0
Discuss changes if the cardiovascular system following birth.	2.0
	Describe the development of the primitive cardiovascular system and blood cells. Describe the formation of the embryonic heart tube. Describe the development of the fetal heat from the embryonic heat tube. Explain septation of the atria and ventricles, and discuss commonly associated defects. Describe the development and derivatives of the aortic arches. Discuss changes if the cardiovascular system following birth.

### V. <u>Development of the Urogenital System</u>

1.	Describe the formation and derivatives of the pronephros, mesonephros, and metanephros.
	3.0
2.	Discuss the development of the kidneys and ureters, including repositioning and anomalies.
	2.0
3.	Describe the development of the urinary bladder and urethra. <b>2.0</b>
4.	Describe the development, including anomalies, of the male and female gonads, ducts, and
	external genitalia. 2.0

# VI. Development of the Pharyngeal Apparatus and the Head and Neck

1.	Describe the development and derivatives of the pharyngeal (brachial) apparatus and co	ommon
	anomalies.	2.0
2.	Describe the development, including common anomalies, of the face, palate, and nasal	cavities.
		2.0
3.	Discuss the development of the eye and ear.	1.0

# VII. Development of the Nervous System

1.	Explain the process of neurulation and neural crest formation, including neural tube d	efects.
		4.0
2.	List the derivatives of the neural crest.	3.0
3.	Describe cell differentiation within the neural tube.	3.0
4.	Describe the development, including anomalies, of the brain vesicles and their derivat	ives.
		3.0
5.	Describe the development, including anomalies, of the spinal cord.	4.0
6.	Discuss the formation of the peripheral nervous system and cranial nerves.	4.0

# VIII. Development of the Musculoskeletal System

1.	Discuss the three groups of cells derived from somites, including their migration and the	
	structures derived from each group.	3.0
2.	Identify the role of somatic mesoderm in muscular system development.	4.0
3.	Describe the development and derivatives of hypaxial and epaxial musculature.	3.0
4.	Describe the role of intramembranous and endochondral ossification in development of th	
	axial and appendicular skeletal systems, including common anomalies.	4.0

# IX. <u>Development of the Limbs</u>

1.	Describe the role of the apical ectodermal ridge (AER) in lower limb development.	4.0
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2.	Describe and compare hand and foot plates, and digital rays in upper and lower limb	
	development.	4.0
3.	Discuss the importance of limb axes and limb rotation.	4.0
4.	Discuss the importance of myotome and dermatome formation in limb development.	3.0
5.	Describe the development of the nerve distribution of the limbs.	4.0
6.	. Describe the anomalies in limb development (eg, amelia and meromelia, cleft foot/hand, talipe	
	equinovarus, polydactyly, and syndactyly.	4.0

# X. Development of the Integumentary System

1.	Describe the development of epidermis and dermis.	3.0
2.	Describe the development of skin appendages (eg, hair, nails, sebaceous glands and sw	eat
	glands).	3.0
3.	Explain the basis of dermatome distribution of sensory nerves.	4.0

# **MEDICAL GENETICS LEARNING OBJECTIVES**

I. Medical Knowledge

Genome Organization/Gene Regulation Gene Variation Population Genetics Inheritance Cytogenetics and Molecular Genetics Biochemical Genetics Cancer Genetics

II. Patient Care

Medical Genetics/Inheritance Genetic Testing Cancer Genetics Reporductive and Prenatal Genetics Treatment/Management Interpersonal and Communciation Skills Practice-Based Leargning and Improvement Professionalism Systems-Based Practice

# I. Medical Knowledge

# A. Genome Organization/Gene Regulation

	1.	1. Describe the organization of the human genome including the approximate number of genes	
		the number of chromosomes, and how DNA is packaged into chromatin.	3.0
	2.	Describe the organization and distribution of the mitochondrial genome.	2.0
	3.	Describe the structure and function of genes.	3.0
	4.	Describe the process and regulation of gene expression including the steps of transcript translation; the role of regulatory factors such as transcription factors and poncoding R	tion and
		the significance of heterochromatin versus such rematin	<b>2 0</b>
	F	Eveloin how errors in gone expression can result in disease	3.0 1 0
	5. 6	Explain now errors in gene expression can result in disease.	4.0
	0.	Explain now temporal and spatial patterns of gene expression vary throughout the life i	20
	-	Discuss the concernt of enigenetics	5.0
	7.	Discuss the concept of epigenetics.	4.0
	8.	Explain the role of epigenetics in regulation of gene expression, development, and disea	ase.
	~		3.0
	9.	Describe how environmental exposures can influence epigenetic modifications.	
			3.0
В.	Ge	netic Variation	
	1.	Explain the concept of genetic individuality as it applies to medicine.	4.0
	2.	Describe the types and extent of variation in the human genome including sequence an	d
		structural variation in coding and non-coding sequences (eg, single nucleotide variants,	
		insertion-deletions, copy number variants).	3.0
	3.	Define the terms mutation and polymorphism and describe their role in both normal hu	ıman
		variation and disease.	4.0
	4.	Describe missense, nonsense, frame shift, microdeletion, and splice site mutations that	lead to
		human disease and their functional consequences.	3.0
	5.	Explain the basis of genotype-phenotype correlations and how different types of mutat	ions
		influence clinical outcomes and disease severity.	4.0
	6.	Define dominant negative, loss of function, gain of function, haploinsufficiency mutatio	ns.
			3.0
	7.	Describe the role of allelic variation and its contribution to both normal and pathogenic	,
		phenotypic variation.	3.0
	8.	Describe the spectrum of genetic contribution to disease, from disease-causing mutation	ons in
		Mendelian disorders to genetic and non-genetic susceptibility factors in multifactorial d	lisease.
			3.0
	9.	Compare and contrast rare (high risk) versus common (low risk) genetic variants with re	espect to
		their contribution to human health and disease susceptibility.	1.0
	10.	Define pharmacogenetics and pharmacogenomics.	3.0
	11.	Explain how genetic variants can affect drug response in individuals.	3.0
	12.	Describe the principles of genetic linkage analysis and association studies, including the	concept
		of linkage disequilibrium, and how they are used to identify genes contributing to disea	se.
	13.	Explain the strengths and limitation of these approaches stated in #12.	1.0
	14.	Describe how understanding the pathophysiology of a specific genetic mutation could le	ead to
		more effective treatment.	3.0
	15.	Describe the etiology of common genetic diseases.	3.0

### C. Population Genetics

- Explain genetic variation with respect to geographic ancestry and evolution, and its effect on variation between populations.
   2.0
- 2. Explain basic concepts of population genetics, including founder effect and genetic drift. **1.0**
- 3. Apply the concepts of the Hardy-Weinberg law to determine genetic risk carrier frequency, gene frequency, and disease frequency. **3.0**
- Explain how carrier frequency within populations influences local health care policy and practice.
   3.0

### D. Inheritance

- 1. Compare and contrast Mendelian, monogenic, polygenic, and multifactorial inheritance. 4.0
- Describe the characteristic features of Mendelian inheritance patterns (autosomal dominant, autosomal recessive, X-linked, and Y-linked).
   4.0
- Use information in a pedigree to deduce probabilities of transmission for Mendelian traits and diseases.
   4.0
- Explain how factors such as reduced penetrance, delayed age of onset, variable expressivity, genetic heterogeneity (locus and allelic), anticipation, pleiotropy and environmental factors affect the phenotypic expression of a disease and the observed pattern of inheritance.
- Describe the how non-Mendelian inheritance, including somatic and germline mosaicism, uniparental disomy, epigenetics and genomic imprinting, unstable repeat expansion and contraction, and chromosomal rearrangements affect the phenotype and recurrence risk of genetic disorders.
   3.0
- Describe the characteristic features of mitochondrial inheritance and explain the role of maternal inheritance and heteroplasmy in mitochondrial diseases.
   **3.0**
- 7. Explain the principles of multifactorial inheritance as it applies to complex disorders. **3.0**
- Describe the threshold model and the factors that can be used as predictors of multifactorial inheritance.
   3.0

### E. Cytogenetics and Molecular Genetics

- 1. Describe the structure and function of chromosomes. Compare and contrast their segregation in mitosis and meiosis. **2.0**
- 2. Demonstrate a basic understanding of cytogenetic nomenclature. **3.0**
- Explain and contrast the uses and limitations of a G-banded karyotype, fluorescence *in situ* hybridization, and cytogenomic arrays, particularly with regard to detection of genomic copy number changes.
   2.0
- Describe the types of numerical and structural variation seen in human chromosomes (eg, translocations, inversions, deletions, and duplications).
   3.0
- Define mosaicism and explain how it affects the phenotypic expression of a chromosomal disorder.
   2.0
- 6. Compare and contrast molecular diagnostic techniques used in genetic testing, including Southern blotting, polymerase chain reaction, DNA sequencing, array comparative genomic hybridization, fluorescence *in situ* hybridization, genomic and expression array-based technologies and next generation sequencing.
   3.0

### F. Biochemical Genetics

1. Explain what is meant by an inborn error of metabolism. **3.0** 

	2.	Describe the underlying genetic defect and pathogenesis for metabolic disorders, such acid disorders, urea cycle defects, lysosomal storage diseases, fatty acid oxidation defeorganic acidurias, and carbohydrate disorders.	as amino cts, <b>4.0</b>
	3.	Describe how allelic heterogeneity, environmental factors, and modifier genes contribu	ite to
		variable presentation of metabolic diseases.	3.0
	4.	Discuss the various approaches to treatment of metabolic disorders.	2.0
G.	Са	ncer Genetics	
	1.	Describe the multistep genetic model of cancer.	3.0
	2. Describe the role of oncogenes, tumor suppressor genes and DNA repair genes in		eoplastic
		process.	4.0
3. Explain why germline mutations in these genes are associated with an increase		Explain why germline mutations in these genes are associated with an increased risk of	cancer
		and with inherited and familial cancer syndromes.	4.0
	4.	Differentiate between inherited, familial and sporadic cancers.	2.0
	5.	Compare the genetic/epigenetic basis by which cancers arise, including somatic mutation	on,
		epigenetic changes, and germline mutation.	3.0
	6.	Explain how current cytogenetic and DNA technologies are used to establish the diagno	osis,
		prognosis, treatment and long term follow up of cancer.	3.0
7. Explain how genotype of the tumor and/or patient influence		Explain how genotype of the tumor and/or patient influences rational/targeted drug de	esign and
		individualized cancer treatment.	2.0

### II. Patient Care

### A. Medical Genetics/Inheritance

1.	Recognize the indications to refer for a genetics evaluation, including family history of dis	sease,
	congenital anomalies, developmental disability, and multiple miscarriages or reproductiv	'e
	failure.	4.0

- Obtain and interpret medical, social, and family histories and physical exam findings in order to determine if a patient is at risk for a genetic disorder.
   4.0
- 3. Utilize a three-generation family history to construct a pedigree and interpret the mode of inheritance. 2.0
- 4. Assess recurrence risks for Mendelian, multifactorial, and mitochondrial disorders. **3.0**
- 5. Explain the relevance of a genetics evaluation and basic concepts of inheritance.
- Obtain appropriate information regarding management and surveillance of the disorder after genetic diagnosis is made.
   3.0
- Recognize intrinsic and extrinsic causes of congenital anomalies in isolation and/or part of a pattern.
   4.0
- Differentiate among categories of anomalies including malformation, deformation, disruption, dysplasia, syndrome, sequence, and association.
   3.0
- 9. Provide information about appropriate patient support and resources including genetics support groups, community groups, or other resources that may benefit the patient and their family.

3.0

4.0

### B. Genetic Testing

 Explain screening, diagnostic, and predictive genetic testing strategies as components in the evaluation of a patient.
 3.0

- Identify the benefits, limitations and risks of genetic tests, including the ethical concerns associated with genetic testing and the importance of the informed consent process.
   4.0
- Explain how genomic testing may be used as a component of personalized health care with a focus on prevention, assessment of disease risk, identification of pharmacogenetic variants and treatment options.
   3.0
- 4. List the indications for standard cytogenetic karyotype, FISH analysis, and cytogenomic array. **2.0**
- Interpret the results of a cytogenetic report, and recognize their clinical features, etiologies and prognoses (eg, trisomy 13, 18, 21; 47, XXY [Klinefelter syndrome]; 45,X [Turner syndrome]; del 22q; del 5p; etc).
   3.0
- Describe the clinical indications for an inborn error of metabolism that would suggest the use of biochemical tests.
   3.0
- Recognize clinical scenarios where biochemical testing strategies can provide more clinically applicable results than molecular testing results.
   1.0

### C. Cancer Genetics

- Differentiate among sporadic, familial, and hereditary cancer based on medical and family history, and identify individuals at increased personal risk for developing cancer.
   3.0
- Describe the role of genetic testing, including the benefits, limitations, and ethical implications for cancer patients and their unaffected family members.
   3.0
- 3. Describe the manifestations of common hereditary cancer syndromes. **3.0**

### D. Reproductive and Prenatal Genetics

	1.	Recognize the indications for preconception and prenatal carrier testing for genetic disc	orders
		depending on family history and specific ethnic background.	3.0
	2.	Discuss commonly used prenatal screening tests, including first and/or second trimeste	r serum
		screening, cell free fetal DNA testing, and ultrasound evaluation.	3.0
	3.	Discuss risks, benefits, and limitations of commonly used prenatal diagnostic procedure	s.
			3.0
	4.	Discuss indications for preimplantation genetic diagnosis and the process of implement	ation.
			1.0
	5.	Describe the impact of teratogenic substances on development.	4.0
Ε.	Tre	eatment/Management	
	1.	Discuss the following treatment strategies for genetic disease, including when they are utilized clinically:	best
		a. Organ transplantation, stem cell therapy and regenerative medicine	3.0
		b. Correction, enhancement, or replacement of a defective structural protein or e	nzyme
			3.0
		c. Dietary treatment	3.0
	2.	Explain the basic theories and techniques for gene therapy, and the challenges toward i	ts
		implementation.	2.0
	3.	Describe how modification of non-genetic factors, such as diet, exercise and other lifest	yle
		factors can prevent or mitigate disease in some genetically-predisposed individuals.	3.0
	4.	Explain how disease specific genetic variation and knowledge of the patient's genotype	might
		alter medical management.	3.0

5. Describe the ways in which pharmacogenetics/pharmacogenomics can inform dosing of medication, including prediction of physiological response and/or adverse drug reactions.

3.0

4.0

### F. Interpersonal Communication Skills

- Describe the role of clinical genetics professionals (eg, medical geneticists, genetic counselors, clinical laboratory directors) in patient care, and the process for making appropriate referrals for genetic evaluations.
   4.0
- Communicate with patients and families regarding genetic information in a culturally sensitive and non-judgmental manner in a way that can be understood by the patient accounting for differences in educational, socio-economic, and ethnic backgrounds.
   4.0
- Explain the medical and legal processes for diagnostic and predictive testing of adults and minors, including the risks, benefits, limitations, and implications for other family members, and obtaining informed consent.
   3.0
- Communicate family history and medical history pertinent to genetics with an interdisciplinary team of health care professionals.
   4.0

### G. Practice-Based Learning and Improvement

- 1. Use information technology to obtain current information about genetics.<sup>1</sup> **3.0**
- Demonstrate the ability to stay abreast of advances in genetics that relate to changes in medical practice.
   3.0

#### H. Professionalism

- Describe how genetic information is different from other medical information and how that difference may affect decisions of health care providers, patients, and their families.
   4.0
- 2. Identify examples of misuse of genetic/genomic information and testing results. 2.0
- 3. Recognize the need to reduce public fear and misinformation about genetics.
- Describe the potential impact of genetic information on insurance coverage and employment status.
   **3.0**
- Demonstrate effective and confidential communication regarding genetic information with patients and colleagues.
   4.0
- 6. Collaborate with genetics health professionals to provide appropriate care. **4.0**

### I. Systems-Based Practice

- Explain the implications of local, state and federal laws, including the Genetic Information Non-Discrimination Act (GINA), that affect the privacy, confidentiality and potential discrimination related to genetic information.
   4.0
- Describe the rationale for newborn screening and population-based screening, including factors for successful genetic screening programs.
   1.0
- Contrast screening versus diagnostic testing and explain why specific tests may be targeted towards a defined population.
   1.0

<sup>&</sup>lt;sup>1</sup> Gene Tests (<u>http://www.genetests.org</u>); Online Mendelian Inheritance in Man (OMIM); (<u>http://www.ncbi.nlm.nih.gov/entrez/query.fcgi?db=OMIM</u>); National Human Genome Research Institute (NIH/NHGRI): Health <u>http://www.genome.gov/Health/</u>; NIH Genetic Testing Registry: <u>http://www.ncbi.nlm.nih.gov/gtr/</u>; Genetics Home Reference <u>http://ghr.nlm.nih.gov</u>; National Organization for Rare Disorders (NORD) <u>http://www.rarediseases.org</u>

- Explain implications and limitations of direct to consumer genetic testing, and the need for involvement of a genetics healthcare professional in interpretation of result.
   3.0
- 5. Identify the challenges of including genetic information in electronic medical records, including confidentiality, insurance coverage, and other unforeseen issues. **3.0**

# **COMPOSITE HISTOLOGY LEARNING OBJECTIVES**

Tissue Preparation and Microscopy The Cell Tissue and Basic Tissue Types Epithelium and Glands **Connective Tissues** Muscle Tissue Nervous Tissue Circulatory System Lymphatic System Integument Endocrine System **Respiratory System** Urinary System Digestive System Male Reproductive System Female Reproductive System

# I. <u>Tissue Preparation and Microscopy</u>

1.	Describe tissue preparation for microscopy.	1.0
2.	Compare the appearance of cells in light microscopy (LM), scanning electron microscopy	, (SEM),
	and transmission electron microscopy (TEM)	1.0
3.	Recognize basic staining and patterns, including immunohistochemical markers	2.0

# II. <u>The Cell</u>

1.	List the major organelles and describe their primary functions.	4.0
2.	Identify the major organelles as seen using light and electron microscopy.	3.0
3.	Describe the appearance, structure, and roles of plasma membranes, including special	
	modifications.	3.0
4.	Describe the appearance and functions of the nucleus and its components as seen in lig	ht using
	both light and electron microscopy.	3.0
5.	Compare the basic structures of the major cytoskeletal elements and describe their prin	nary
	functions.	3.0
6.	Explain intracellular trafficking.	2.0
7.	Draw the cell cycle, and list the functions of each part.	2.0
8.	Identify mitotic figures.	3.0
9.	Compare and contrast mitosis and meiosis.	2.0
10.	Compare and contrast cell renewal, necrosis, and apoptosis, including their appearances	s in
	micrographs.	1.0
11.	Determine the function of a cell based on its organelle complement.	3.0

# III. <u>Tissue and Basic Tissue Types</u>

1.	Define <i>tissue</i> .	4.0
2.	Describe the characteristics of each of the primary histological tissue types and discuss the	ieir
	function.	4.0

# IV. Epithelium and Glands

1.	Define <i>epithelium</i> and describe its functions.	4.0
2.	Describe the classification of epithelia and identify types of epithelia and their functions.	4.0
3.	Identify and describe the apical, lateral, and basal domains of epithelia, including junctio	nal
	complexes and cell surface specializations.	4.0
4.	Describe the basic structure and functions of the basement membrane.	4.0
5.	Describe the classification and appearance of exocrine glands by duct system, type of se	cretion,
	and mode of secretion.	2.0
6.	Identify mucous and serous secreting glands.	3.0
7.	List where in the body, each type of epithelium is found.	4.0

# V. <u>Connective Tissues</u>

1.	Identify and describe the composition of the major classes of connective tissue.	4.0
2.	Describe the embryonic connective tissues.	1.0
3.	List the types of connective tissue, cartilage and bone and identify the location and func	tion of
	each.	4.0
4.	Identify and describe resident and wandering cells of each class of connective tissue pro	per,
	cartilage, and bone.	4.0
5.	Identify and describe & differentiate among collagen, elastic, and reticular fibers and de	termine
	locations of each.	4.0
6.	Compare the properties and location of collagen types, I, II, III, IV.	4.0
7.	Compare and contrast the composition and organization of extracellular matrix for each	class of
	connective tissue proper, cartilage and bone.	4.0
8.	Compare and contrast the general structure of lamellar versus woven bone and compac	t versus
	spongy bone.	4.0
9.	List the general process in the remodeling and repair of bone fractures.	3.0
10.	Compare and contrast intramembranous and endochondral ossification.	3.0
11.	Identify regions within the growth plate.	3.0
12.	Identify and describe the cellular and extracellular components of blood	4.0
13.	Compare the major components of serum and plasma.	3.0
14.	Explain the process of hematopoiesis.	3.5
15.	Identify and describe the cells that appear in circulating blood.	4.0

# VI. <u>Muscle Tissue</u>

1.	Describe the histological structure of the three types of muscle tissue, including innervation	tion, and
	location in the body.	4.0
2.	Describe the repair process of skeletal, cardiac, and smooth muscle.	1.0

# VII. <u>Nervous Tissue</u>

			<i>c</i>	
1	Identity and describe the	histological structu	re of a typical neuro	n and its synantic connection
÷.	identity and describe the	motorogical stracta	c of a cypical fical o	in and its synaptic connection.

		4.0
2.	Describe the structure and function of pseudo-unipolar, bipolar, and multipolar neurons	•
		3.0
3.	Describe the process of myelination in the CNS and PNS and identify cell types involved.	3.0
4.	Identify and describe peripheral nerves and ganglia.	3.0
5.	Describe the structure and function of the supporting cells of the CNS and PNS.	3.0
6.	Describe the elements that make up the blood-brain barrier.	2.0

# VIII. <u>Circulatory System</u>

1.	Describe the general features and three basic tunics of arteries, veins, and lymphatic ve	ssels.
		4.0
2.	Explain the locations and functions of endothelial cells and pericytes.	3.0

3.	Compare, Identify and describe the histological structure of elastic and muscular arteries	and
	arterioles.	3.0
4.	Compare, identify and describe the histological structure of medium and large veins and	
	venules.	3.0
5.	Describe the histological structure, organ location and function of the different types of	
	capillaries.	3.0
6.	Compare and identify the histological structure of the three layers of the heart.	3.0
7.	Identify and describe the structure and function of Purkinje fibers versus cardiac myocyte	es.
		2.0
Lvi	nphatic System	

1.	Identify and describe the locations of the cells of the lymphatic system.	3.0
2.	Describe the recirculation of lymphocytes.	3.0
3.	Describe the histological structure and function of diffuse lymphoid tissue and lymphati	ic
	nodules.	3.0
4.	Describe and compare the histological structure and function of lymph nodes, spleen, a	nd
	tonsils.	3.0
5.	Identify and describe the histological structure and function of the thymus and bone ma	arrow in
	relation to differentiation and education of lymphocytes.	3.0

### X. Integument

IX.

4.0
3.0
4.0
2.0
3.0
4.0
ds. <b>2.0</b>
3.0
ու s.

## XI. Endocrine System

- Identify and describe the histological structure and major hormone secretions of the endocrine glands: pituitary, thyroid, parathyroid, adrenal, pineal, and pancreatic islets.
   3.0
- Identify endocrine cell types, secretory products, and feed-back regulation for each of the endocrine organs.
   3.0
- 3. Describe the hypothalamo-hypophyseal portal system and the hypothalamo-hypophyseal tract.

3.0

4. Describe regeneration of neurons and nerve fibers in the peripheral and central nervous system

# XII. <u>Respiratory System</u>

1. Describe the histological structures of the nasal cavity, nasopharynx, and larynx. **2.0** 

	2. 3. 4. 5.	Compare and contrast the histological structures of the trachea, bronchi, bronchioles, respiratory bronchioles, and alveolar ducts. Identify and describe the histological structure of alveoli, including components of the in alveolar septum. Describe the components of the blood-air barrier. Describe the functions of dust cells, type I and II pneumocytes, clara cells, and K cells.	3.0 nter- 3.0 3.0 3.0
XIII.	<u>Uri</u>	nary System	
	1. 2.	Describe the general structures of the cortex, medulla and renal pelvis of the kidney. Identify and describe the histological structure and location of the components of a urin tubule.	<b>3.0</b> hiferous <b>4.0</b>
	3.	Identify and describe the components of the glomerular filtration barrier and their func-	tions.
	4. 5. 6.	Describe the location and structure of the juxtaglomerular apparatus (complex). Trace the flow of blood through the kidneys. Describe the epithelial and muscular structures of the ureter, the urinary bladder, and t urethra.	3.0 3.0 3.0 he 2.0

# XIV. Digestive System

1.	Describe the histological structure of the oral cavity, salivary glands, lips, tongue, and	
	oropharynx.	2.0
2.	Identify the histological structures of the four basic layers of the alimentary canal from t	the
	esophagus to the anus.	3.0
3.	Compare and contrast the histological structures of the four basic layers of the alimenta	ary canal
	from the esophagus to the anus.	3.0
4.	Identify and describe the function of the cell types:	
	a. parietal	3.0
	b. chief	3.0
	c. enteroendocrine	3.0
	d. paneth	3.0
	e. enterocytes	3.0
5.	Describe the histological structure of the gastrointestinal-associated glands, including the	ne liver,
	gall bladder, and exocrine pancreas.	3.0

# XV. <u>Male Reproductive System</u>

1.	Describe the general structure and function of the testis and the histological appearance of the	
	cells of the seminiferous tubules.	2.0
2.	Explain spermatogenesis and spermeiogenesis.	2.0
3.	Describe the histological structure and function of the excurrent duct system and associated	
	glands.	2.0
4.	Describe Sertoli and Leydig cell types in regards to their secretory products and feedb	back
	regulation.	2.0

# XVI. Female Reproductive System

1.	Discuss the general structure and function of the ovary.	2.0
2.	Describe the histological appearance of the cells of the ovarian follicles and the corpus	s luteum.
		2.0
3.	Explain oogenesis and follicular development.	2.0
4.	Describe the histological structure and function of the uterine tube, uterus, vagina, an	d
	genitalia.	2.0
5.	Describe the histological appearance and physiological changes of the endometrium of	ver the
	course of one menstrual cycle.	2.0
6.	Distinguish the histological features of lactating from non-lactating breast tissue.	2.0

# **PHYSIOLOGY LEARNING OBJECTIVES**

Cardiovascular Cell and Membrane Endocrine Physiology Gastrointestinal Physiology Integration and Exercise Physiology Muscle Physiology Neurophysiology Pulmonary Physiology Renal Physiology

### I. <u>Cardiovascular</u>

В.

### A. Characteristics of Cardiac Muscle

1.	Compare and contrast the duration of the action potential and the refractory period in a	a cardiac
	muscle, a skeletal muscle, and a nerve.	3.9
2.	Describe the temporal relationship between an action potential in a cardiac muscle cell	and the
	resulting contraction (twitch) of that cell and explain why cardiac muscle cannot remain	in a
	state of sustained (tetanic) contraction.	3.9
3.	Outline the steps in excitation-contraction coupling in cardiac muscle.	3.9
4.	Outline the sequence of events that occurs between the initiation of an action potential	l in a
	cardiac muscle cell and the resulting contraction and then relaxation of that cell.	3.8
5.	Explain the special role of Ca <sup>2+</sup> in the control of contraction and relaxation of cardiac mu	uscle.
		3.8
6.	Compare and contrast cardiac and skeletal muscle in terms of cell size, electrical connect	ctions
	between cells, and arrangement of myofilaments.	3.8
7.	Describe role of gap junctions in creating a functional syncytium, based upon ion perme	ability
	and electrical resistance.	3.9
8.	Describe the role of extracellular calcium in cardiac muscle contraction.	3.8
9.	Explain how intracellular calcium concentration modulates the strength of cardiac musc	le
	contraction.	3.8
10.	Identify the source of intracellular calcium that mediates excitation-contraction coupling	g.
		3.5
11.	Explain the role of Starling's Law of the Heart in keeping the output of the left and right	
4.0	ventricles equal.	4.0
12.	Differentiate between the way changes in preload and changes in contractility influence	20
10	ventricular force development.	<b>3.8</b>
13.	Compare the energetic consequences of these two separate mechanisms of force modu	
		5.8
Ele	ctrophysiology of the Heart	
1.	Describe and interpret a typical action potential in a ventricular muscle and a pacemake identifying both the voltage and time axes.	er cell,

#### 3.8

- 2. Explain how ionic currents contribute to the four phases of the cardiac action potential. **3.8**
- 3. Describe differences in shapes of the action potentials of different cardiac cells.

### 3.8

- 4. Describe the ion channels that contribute to each phase of the cardiac action potential. **3.8**
- 5. Explain how differences in channel population influence the shape of the action potential in the nodal, atrial muscle, ventricular muscle, and Purkinje fiber cardiac cells.

#### 3.8

6. Explain the basis for the long duration of the cardiac action potential and the resultant long refractory period.

3.9
	7.	Identify the advantage of the long plateau of the cardiac action potential and the long r period.	efractory 3.9
	8.	Describe the normal sequence of cardiac activation (depolarization), beginning in the SA	A node,
		conduct the impulse through any of these areas	10
	۵	Explain why the AV node is the only normal electrical nathway between the atria and the	4.0
	5.	ventricles	25
	10	Explain the functional significance of the slow conduction through the AV node includin	g factors
	10.	that influence conduction velocity through the AV node	<b>3 5</b>
	11	Explain the ionic mechanism of nacemaker automaticity and rhythmicity	3.5
	12	Identify cardiac cells that have nacemaker potential and their spontaneous rate and hur	moral
		factors that influence their rate.	3.5
	13.	Describe the significance of "overdrive suppression" and "ectopic pacemaker," including	gthe
		conditions necessary for each to occur.	2.8
	14.	Compare and contrast the sympathetic and parasympathetic nervous system influence	on heart
		rate and cardiac excitation in general.	3.9
	15.	Identify which arm of the autonomic nervous system is dominant at rest and during exe	rcise and
		describe ionic mechanisms of these effects on both working myocardium and pacemake	er cells.
			3.9
	16.	Explain how cell injury, resulting in a less negative resting potential, alters ionic events i	n
		depolarization and repolarization.	3.1
	17.	Define decremental conduction, re-entry, and circus movement.	2.9
С.	Car	rdiac Function	
	1.	Describe and interpret the length tension relationship in a single cardiac cell.	3.8
	2.	Correlate the cellular characteristics of length, tension, and velocity of shortening with t	the intact
		ventricle characteristics of end diastolic volume, pressure, and dP/dt.	3.8
	3.	Define <i>preload</i> .	3.8
	4.	Explain why ventricular end-diastolic pressure, atrial pressure, and venous pressure all pressure and venous pressure all pressure and venous pressure and venous pressure and venous pressure atrial pressure.	provide
		estimates of ventricular preload, as well as why ventricular end-diastolic pressure provi	des the
		most reliable estimate.	3.8
	5.	Define afterload.	3.6
	6.	Explain how arterial pressure influences afterload, and describe a condition when arteri	ial
	_	pressure does not provide a good estimate of afterload.	3.6
	7.	Define <i>contractility</i> .	3.0
	8.	Explain why dP/dt is a useful index of contractility and explain now the calcium transien	
	0	Differentiate between cardiac performance and cardiac contractility	3.0
	9. 10	Differentiate between cardiac performance and cardiac contractility in determining of	<b>3.4</b>
	10.	performance	<b>3 1</b>
	11	Explain how changes in sympathetic activity alter ventricular work, cardiac metabolism	oxvgen
	± ± •	consumption, and cardiac output.	3.3
	12.	Explain how the Law of LaPlace applies to ventricular function in the normal and volume	р
		overloaded (failing) ventricle.	2.5
	13.	Relate the ventricular pressure volume loop to the phases and events of the cardiac cyc	le (ECG,
		valve movement).	3.3
	14.	Differentiate between stroke volume and stroke work identifying stroke volume and str	oke work
		from a pressure-volume loop.	2.6

	15. 16. 17.	Define <i>ejection fraction</i> . Calculate ejection fraction from end diastolic volume, and/or stroke volume, and predic change in ejection fraction that would result from a change in preload, afterload, and contractility. Describe the changes in pressure volume loops that would result from changes in afterla preload, or contractility, for one cycle and the achieved new steady state.	3.3 t the 3.3 pad, 2.9
D.	Cai	rdiac Cycle	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> </ol>	Describe the basic functional anatomy of the atrioventricular and semilunar valves, and how they operate. Draw the pressure, volume, heart sound, and ECG changes in the cardiac cycle in correct temporal relationship. Identify the intervals of isovolumic contraction, rapid ejection, reduced ejection, isovolu relaxation, rapid ventricle filling, reduced ventricular filling, and atrial contraction. Identify the various phases of ventricular systole and ventricular diastole. Describe the relationship between pressure and flow into and out of the left and right v during each phase of the cardiac cycle. Explain how and why left sided and right sided events differ in their timing.	explain 2.4 4.0 mic 4.0 4.0 entricles 4.0 2.3
Ε.	Phy	vsiology of Cardiac Defects and Heart Sounds	
	1. 2. 3. 4.	Describe the factors that contribute to the formation of turbulent flow. Describe the timing and causes of the four heart sounds. Describe the expected auscultation sounds that define mitral stenosis, mitral insufficient aortic stenosis, and aortic insufficiency. Explain how these pathologic changes would affect cardiac mechanics and blood pressu	<b>3.0</b> <b>3.8</b> cy, <b>2.9</b> re.
	5. 6.	Define <i>dipole.</i> Describe the characteristics of a vector and how dipoles generated by the heart produce waveforms of the ECG.	2.9 2.3 e the 2.3
F.	The	e Normal and Abnormal Electrocardiogram (ECG)	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> </ol>	Describe the electrode conventions used to standardize ECG measurements. Identify the electrode placements and polarities for the 12 leads of a 12-lead electrocard and the standard values for pen amplitude and paper speed calibration on a diagram. Identify the components of a typical bipolar (Lead II) ECG tracing and explain the relatio between each of the waves, intervals, and segments in relation to the electrical state of heart. Explain why the ECG tracing looks different in each of the 12 leads. Identify mean electrical vector (axis) of the heart, give the normal range, and determine mean electrical axis from knowledge of the magnitude of the QRS complex in the standa leads. Describe the alteration in conduction responsible for tachycardia, bradycardia, AV block Parkinson-White (WPW) syndrome, bundle branch block, flutter, and fibrillation. Describe electrocardiographic changes associated respectively with myocardial ischemia and death.	<b>3.4</b> diogram <b>3.4</b> nship the <b>4.0</b> <b>2.9</b> the ard limb <b>2.9</b> , Wolff- <b>3.1</b> a, injury, <b>3.1</b>
	8.	Define <i>injury current</i> and describe how it is alters the S-T segment of the ECG.	3.1

9. Describe the principles underlying cardiac output measurements using the Fick, dye dilution, and thermodilution methods. **2.1** 

#### G. Cardiac Output and Venous Return

- Explain how cardiac function (output) curves are generated and how factors that cause hypereffective or hypoeffective changes (contractility) in the heart can alter the shape of cardiac function curves.
   2.3
- Describe the concept of "mean systemic pressure," its normal value, and how various factors can alter its value.
   3.3
- 3. Define venous return.
- Describe the concept of "resistance to venous return" and identify the factors determining its value, what factors are most important in practice, and how various interventions would change the resistance to venous return.
   3.9
- 5. Describe skeletal muscle pump, and thoracic (respiratory) pump.
- 6. Explain how exercise affects venous return from the foot and leg.
- Describe the changes in blood volume and pressure when a person moves from a supine to a standing position.
   3.9
- Interpret a vascular function curve and predict how changes in total peripheral resistance, blood volume, and venous compliance influence this curve.
   2.4
- Explain how the intersection point of the cardiac function and vascular function curves represents the steady-state cardiac output and central venous pressure under the conditions represented in the graph.
   2.4
- Use the intersection point of the cardiac function curve and vascular function curve to predict how interventions such as hemorrhage, heart failure, autonomic stimulation, and exercise will affect cardiac output and right atrial pressure, and predict how physiological compensatory changes would alter acute changes.

#### H. Cardiovascular Fluid Dynamics

- Describe the components of blood (cells, ions, proteins, platelets) giving their normal values and identify the relationship of the three red blood cell concentration estimates, red blood cell count, hematocrit, and hemoglobin concentration to each other.
   3.1
- 2. Identify the source, stimulus for formation, and function of the hormone erythropoietin.
- Relate the rate of red blood cell synthesis to the normal red blood cell life span and the percentage of immature reticulocytes in the blood.
   2.5
- Describe the functional consequence of the lack of a nucleus, ribosomes, and mitochondria for protein synthesis and energy production within the red blood cells.
   1.6
- Discuss the normal balance of red blood cell synthesis and destruction, including how imbalances in each lead to anemia or polycythemia.
   2.1
- Explain how red blood cell surface antigens account for typing of blood by the ABO system and rhesus factor.
   2.4
- Describe the factors that determine the total energy of the flowing blood and the relationship among these factors and the usual reference point for physiological pressure.
   1.9
- 8. Describe and differentiate between flow and velocity.
- Describe the relationship between pressure, flow, and resistance in the vasculature, calculate for one variable, if the other two are known; connect this relationship to the arteries, arterioles, capillaries, venules, and veins; and explain how blood flow to any organ is altered by changes in resistance to that organ.

3.9

3.9

3.9

4.0

	10.	Describe the factors that influence resistance to flow and the relationships among them Poiseulles' Law.	, using <b>3.0</b>
	11.	Describe the relationship between flow, velocity, and cross-sectional area.	3.0
	12.	Describe the influence vascular compliance has on flow, velocity, and cross-sectional are	ea.
			3.0
	13.	Explain how hemodynamics in blood vessels, especially microcirculation, deviate from the	heory
		due to anomalous viscosity, distensibility, and the glycocalyx.	3.0
	14.	Define resistance and conductance.	3.4
	15.	Describe the effects of adding resistance in series versus in parallel on total resistance a	nd flow. <b>3.4</b>
	16.	Relate the effects to the redistribution of flow from the aorta to the tissues during exercise	cise. <b>3.4</b>
	17.	Identify and describe the factors that shift laminar flow to turbulent flow and the relation	onship
		between velocity, viscosity, and audible events, such as murmurs and bruits.	3.4
	18.	Describe the principles of flow through collapsible tubes, the Starling resistor, and what pressure gradient determines flow for different relative values of inflow, surrounding, a	nd
		outflow pressures.	2.3
	19.	Explain how hemodynamics in blood vessels, especially microcirculation, deviates from due to anomalous viscosity, distensibility, axial streaming, and critical closing behavior.	theory <b>1.6</b>
Ι.	Art	erial Pressure and the Circulation	
	1.	Describe the organization of the circulatory system and explain how the systemic and	
		pulmonary circulations are linked physically and physiologically.	3.9
	2.	Explain blood pressure measurement with a catheter and transducer and identify the	
	_	components of blood pressure waveform.	3.8
	3.	Compare and contrast invasive measurements with indirect estimation of blood pressur	e by a
		sphygmomanometer and explain how each approach provides estimates of systolic and pressures.	diastolic <b>3.8</b>
	4.	Calculate the pulse pressure and the mean arterial pressure given systolic and diastolic	blood
		pressures.	3.8
	5.	Describe how arterial systolic, diastolic, mean, and pulse pressure are affected by chang	es in
	_	stroke volume, heart rate, arterial compliance and total peripheral resistance.	3.9
	6.	Explain why systolic arterial pressure, but not mean arterial pressure, is higher in leg art	eries
	-	than in the aorta.	3.8
	7. o	Predict the ratio of ankie-to-arm systolic arterial pressures in a healthy person.	3.8 Actional
	о.	compare and contrast pressures, oxygen saturations, velocity of blood now and cross-so	stomic
		and nulmonary circulations	<b>2</b> /
	q	Identify the cell membrane recentors and second messenger systems mediating the cor	J. <del>-</del>
	5.	of vascular smooth muscle by noreninenbrine, angiotensin II, and vasopressin	2.3
	10.	Identify the cell membrane receptors and second messenger systems mediating the relations and second messenger systems and sec	axation
		of vascular smooth muscle by nitric oxide, bradykinin, prostaglandins, and histamine.	2.0
J.	The	e Microcirculation and Lymphatics	
	1.	Explain how water and solutes traverse the capillary wall.	2.9
	2.	Use Fick's equation for diffusion to identify the factors that will affect the diffusion med	iated
		delivery of nutrients from the capillaries to the tissues.	2.9
	3.	Define and give examples of <i>diffusion-limited</i> and <i>flow-limited</i> exchange.	2.9

	4.	Explain how changes in capillary surface area affect the capacity for fluid exchange.	3.0
	5.	Describe how each component of the the Starling equation influences fluid movement a	across
		the capillary wall.	4.0
	6.	Describe the pathway for leukocyte migration across the microcirculation, including leu	kocyte
		expression of cellular adhesion molecules, and recognition sites in the vascular endothe	lial cells
			1.3
	7.	Describe the processes of angiogenesis, including the stimulus that initiates new vessel	growth,
		starting at the post-capillary venule.	0.9
	8.	Explain how smooth muscle contractile mechanisms differ from the contractile mechan	isms of
		skeletal and cardiac muscle.	3.1
	9.	Describe the involvement of G protein-coupled receptors and signal transduction pathw	/ays in
		the regulation of smooth muscle contraction.	3.1
	10.	Explain the involvement of endothelial cells in the regulation of vascular diameter and	• •
		Inflammatory responses.	3.1
	11.	Explain how altering pressure or resistance in pre- and post-capillary regions alters capi	llary
	10	pressure, and discuss the consequence of this change on transmural fluid movement.	3.1
	12.	Explain why fiuld does not usually accumulate in the interstitium of the lungs, using the	2.0
	10	components of the starting equation.	<b>2.9</b>
	13.	Explain now histamine alters the permeability of the post-capillary venues, as well as h	ow the
	11	Describe the lymphotics and explain how the structural characteristics of terminal lymp	<b>5.1</b>
	14.	allow for the reabsorption of large compounds, such as protains	<b>7 8</b>
	15	Compare and contrast the structure of lymphatic canillaries and systemic canillaries inc	<b>2.0</b>
	15.	the significance of the smooth muscle in the walls of the lymphatic vessels	<b>7</b> 1
	16	Identify critical functions of the lymphatic system in fat absorption interstitial fluid	2.1
	10.	reabsorption, and clearing large proteins from the interstitial spaces.	3.9
	17.	Describe and interpret the relationship between interstitial pressure and lymph flow, and	nd
		explain why edema does not normally develop as interstitial pressure increases.	2.1
	18.	Explain how edema develops in response to	
		a. venous obstruction;	3.6
		b. lymphatic obstruction;	3.6
		c. increased capillary permeability;	3.6
		d. heart failure;	3.6
		e. tissue injury or allergic reaction; and	3.6
		f. malnutrition.	3.6
К.	Red	gulation of Arterial Pressure	

- 1. Describe the anatomical components of the baroreceptor reflex.
- 2. Outline the sequence of events in the baroreflex that occur after an acute increase or decrease in arterial blood pressure, include receptor response, afferent nerve activity, CNS integration, efferent nerve activity to the SA node, ventricles, arterioles, venules, and hypothalamus.
  - 3.8

- Outline the sequence of events mediated by cardiopulmonary (volume) receptors that occur after an acute increase or decrease in arterial blood pressure including receptor response, afferent nerve activity, CNS integration, efferent nerve activity to the heart, kidney, hypothalamus, and vasculature.
   3.6
- 4. Outline the sequence of events mediated by cardiopulmonary (volume) receptors that occur after an acute increase or decrease in central venous pressure including receptor response,

	5. 6. 7. 8. 9.	afferent nerve activity, CNS integration, efferent nerve activity to the heart, kidney, hypothalamus, and vasculature.3Compare and contrast the sympathetic and parasympathetic nervous system control of he rate, contractility, total peripheral resistance, and venous capacitance.3Predict the cardiovascular consequence of altering sympathetic nerve activity and parasympathetic nerve activity.3Compare and contrast the relative contribution of short- and long-term mechanisms in bl pressure and blood volume regulation.3Describe the cardiovascular reflexes initiated by decreases in blood O2 and increases in bloc CO2.3Describe the release, cardiovascular target organs, and mechanisms of cardiovascular effect angiotensin, atrial natriuretic factor, bradykinin, and nitric oxide.3	<ul> <li><b>3.6</b></li> <li>eart</li> <li><b>3.9</b></li> <li>ood</li> <li><b>3.4</b></li> <li>ood</li> <li><b>3.0</b></li> <li>ects for</li> <li><b>3.1</b></li> </ul>
L.	Loc	cal Control of Blood Flow	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> </ol>	Explain autoregulation of blood flow to the brain, and distinguish between short-term and term autoregulatory responses and the mechanisms responsible for each. Explain how the theory of metabolic regulation of blood flow accounts for active hyperem reactive hyperemia. Identify the role of PO <sub>2</sub> , PCO <sub>2</sub> , pH, adenosine, and K <sup>+</sup> in the metabolic control of blood flow specific tissues. Describe the synthetic pathway for nitric oxide (EDRF, endothelial derived relaxing factor) including substrate and the interplay between endothelium and vascular smooth muscle. Describe the conditions and the mechanisms whereby humoral substances contribute to regulation of the microcirculation. Explain of intrinsic (local), neural, and humoral control mechanisms, and contrast their relative dominance in the CNS, coronary, splanchnic, renal, cutaneous, and skeletal muscle vascular beds. Describe the role of angiogenesis in providing a long-term match of tissue blood flow and metabolic need.	d long- 3.1 hia and 3.4 w to 3.9 l.6 3.5 2.8 2.8
М.	Fet	al and Neonatal Circulation	
	1. 2. 3. 4.	Describe the progressive changes in maternal blood volume, cardiac output, and peripher resistance during pregnancy and at delivery. Compare and contrast the blood flow pattern in the fetus with that of a normal neonate, including the source of oxygenated blood. Describe the function in utero of the fetal ductus venosus, foramen ovale, and ductus arte and explain the mechanisms causing closure of these structures at birth. Describe the relative differences in oxygen saturation and pressure for blood in the major vessels and cardiac chambers of the fetus, and explain how these values change at birth.	ral L. <b>0</b> eriosus, L. <b>3</b> • blood
	_	le la companya de la	

5. Explain the unfavorable consequences to the neonate if either the ductus arteriosis or the foramen ovale fails to close. **1.3** 

# N. Homeostasis and Injury

 Identify, in sequence, the enzymes and substrates and processes involved in the formation of fibrin polymers, and contrast the initiation of thrombin formation by intrinsic and extrinsic pathways.

- Compare and contrast the mechanisms of anticoagulation for heparin, EGTA c) Coumadin and identify clinical uses for each agent.
   1.5
- Describe the mechanisms of fibrinolysis by TPA (tissue plasminogen activator), streptokinase and urokinase.
   1.5
- 4. Explain the role of the platelet release reaction on clot formation and distinguish between a thrombus and an embolus. **1.9**
- Explain why the activation of the clotting cascade does not coagulate all of the blood in the body.
   1.9

## O. Hemorrhage and Shock

- Describe the direct cardiovascular consequences of the loss of 30% of the circulating blood volume on cardiac output, central venous pressure, and arterial pressure, and describe the compensatory mechanisms activated by these changes.
   3.4
- Identify positive feedback mechanisms activated during severe hemorrhage that may lead to circulatory collapse and death.
   3.1
- Compare and contrast the change in plasma electrolytes, hematocrit, proteins, and colloid osmotic pressure following resuscitation from hemorrhage using water, 0.9% NaCl, plasma, and whole blood.
   2.5

## P. Coronary and Skeletal Muscle Circulations

- Describe the phasic flow of blood to the ventricular myocardium through an entire cardiac cycle and contrast this cyclic variation in myocardial flow in the walls of the right and left ventricles in the subendocardium and subepicardium of the left ventricle.
   2.6
- Identify the area of the ventricle most susceptible to ischemic damage and explain why the risk is increased at high heart rates.
   2.6
- Explain how arteriovenous O<sub>2</sub> difference and oxygen extraction in the heart is unique when compared with other body organs.
   2.4
- Explain the mechanism whereby coronary blood flow is coupled to myocardial workload, and identify stimuli that cause increases in coronary blood flow to occur.
   2.6
- Explain how sympathetic stimulation alters heart rate, contractility, and coronary vascular resistance, as well as both directly and indirectly to change coronary blood flow and identify the relative importance of the direct and indirect CNS effects in determining coronary blood flow during exercise.
   3.4
- 6. Describe what is meant by coronary vascular reserve and the role of collateral blood vessels and describe the physiological and pathological events that decrease coronary vascular reserve.
- Compare and contrast the neural and local control of skeletal muscle blood flow at rest and during exercise.
   3.4
- 8. Compare and contrast the effect of phasic and sustained skeletal muscle contraction on extravascular compression of blood vessels and on central venous pressure.
  2.6

# Q. Cerebral, Splanchnic, and Cutaneous Circulation

- 1. Compare and contrast the local and neural control of cerebral blood flow, and describe the relative important of O<sub>2</sub>, CO<sub>2</sub>, and pH in regulating cerebral blood flow. **3.0**
- Describe the structural components of the blood brain barrier, and explain how this barrier impedes the movement of gases, proteins, and lipids from the blood to neurons.
   2.3
- 3. Identify the differences in cerebrospinal fluid and plasma relative to protein concentration. 2.3
- 4. Describe the function of cerebrospinal fluid.

2.3

- 5. Compare and contrast the mechanisms of hemorrhagic and occlusive stroke. **2.1**
- 6. Compare and contrast the local and neural control of the splanchnic circulation. **1.9**
- Explain the role of the hepatic portal system and the hepatic artery in providing flow and oxygen to the liver.

  1.9
- Describe the blood pressure in the hepatic portal vein, hepatic sinusoids, and the vena cava.
   1.5
- 9. Explain how hepatic microcirculatory fluid exchange will be altered, including the development of ascites, given an increase in central venous pressure. **1.5**
- Explain how the GI circulation is adapted for secretion and absorption, including enterohepatic circulation.
   1.5
- 11. Compare and contrast local and neural control of cutaneous blood flow.
- 12. Describe the unique characteristics of skin blood flow that are adaptive for body temperature regulation. **3.1**

## R. Exercise and Cardiovascular Physiology

- 1. Describe the cardiovascular consequences of exercise on peripheral resistance, cardiac output, AV oxygen difference, and arterial pressure. **3.0**
- Describe the redistribution of cardiac output during exercise to the CNS, coronary, splanchnic, cutaneous, and skeletal muscle vascular beds during sustained exercise (distance running), and explain the relative importance of neural and local control in each vascular bed.
   2.9
- Identify and describe adaptations to physical training on the cardiovascular system, including the mechanisms underlying each.
   2.1
- 4. Compare and contrast the effects of static versus dynamic exercise on blood pressure. 1.9

# II. <u>Cell and Membrane</u>

# A. Biological Membranes, Solutes and Solutions

- Describe the polar structure of water, and explain how the formation of hydrogen bonds permits the dissociation of salts (such as NaCl), saccharides, and other polar molecules. 1.9
- 2. Contrast the definitions of hydrophobic and hydrophilic related to water polarity. **1.9**
- Describe the three-dimensional composition of a cell membrane, and explain how the distribution of phospholipids and proteins influences the membrane permeability of ions, hydrophilic, and hydrophobic compounds.
   3.1
- Define *reflection coefficient*.
   Explain how the relative permeability of a cell to water and solutes will generate an osmotic pressure.
   3.5
- 6. Compare and contrast the osmotic pressure generated across a cell membrane by a solution of particles that freely cross the membrane with that of a solution with the same osmolality, but whose particles cannot cross the cell membrane.
   3.5
- 7. Identify the usual units used to describe concentration. **3.4**
- 8. Identify the typical value and normal range for plasma Na<sup>+</sup>, K<sup>+</sup>, H<sup>+</sup> (pH), HCO<sub>3</sub><sup>-</sup>, Cl<sup>-</sup>, Ca<sup>2+</sup>, and glucose, and the typical intracellular pH and concentrations of Na<sup>+</sup>, K<sup>+</sup>, Cl<sup>-</sup>, Ca<sup>2+</sup>, and HCO<sub>3</sub><sup>-</sup>.
- 3.49. Differentiate between osmole, osmolarity, osmolality and tonicity.3.3
- 10. Identify the typical values and normal range for plasma osmolality. **3.3**

	11.	Describe how the difference in free energy of a solute or solvent between two compon have chemical, electrical, and/or hydrostatic pressure components, and explain how, at	ents can t
		equilibrium, for a given component, the free energy difference between the two compa	artments
		is zero.	0.6
	12.	Define Donnan equilibrium and describe resulting characteristics.	1.3
	13.	Describe the linear relationship between forces and flow in the context of solutes, fluid	s and
		electricity.	2.3
	14.	Explain how changes in the concentration gradient, surface area, time, and distance will	
	4 -	Influence the diffusional movement of a compound, using Fick's Law of Diffusion.	3.6
	15.	Explain now a potential difference across a membrane will influence the distribution of	a cation
	10	and an anion, based on the principle of ionic attraction.	2.0
	10.	Define steady state.	<b>5.0</b>
	17.	content to cell solute gradients and cell volume maintenance.	<b>3.0</b>
	18.	Explain how the Nernst equation, accounts for both the chemical and electrical driving	forces
		that act on an ion.	3.0
	19.	Predict the direction that an ion will move when the membrane potential is at its equili	brium
		potential; is more negative than its equilibrium potential; or is less negative than its equ	ullibrium
	20	potential, based on the Nernst equation.	3.4
	20.	identify values in a typical non-excitable cell for the membrane potential for ENa, EK, EC	ci, and
	21	EUG.	3.4
	21.	and external ion concentrations	2 1
	22	Calculate an equilibrium notential for that ion using the Nernst equation	2 1
	22.	Compare and contrast the difference in EK (the Nernst notential for $K^+$ ) caused by a 5 m	5.1 Fa/l
	25.	increase in extracellular $K^{+}$ with the change in ENa (the Nernst potential for Na <sup>+</sup> ) caused	l hv a 5
		mEq/l increase in extracellular Na $^{+}$	<b>3</b> .1
	24	Explain how the resting membrane potential is generated.	3.0
	25.	Calculate the membrane potential by using either the Goldman-Hodgkin-Katz equation	or the
		chord conductance equation: and given an increase or decrease in the permeability of H	K. Na. or
		Cl, predict how the membrane potential would change.	3.0
	26.	Differentiate between diffusion, facilitated diffusion, secondary active transport, and p	rimary
		active transport.	3.4
	27.	Explain how transport rates of certain molecules and ions are accelerated by the preser	nce of
		specific membrane transport proteins ("carrier" and "channel" molecules).	2.9
	28.	Explain how energy from ATP hydrolysis is used to transport ions such as $Na^+$ , $K^+$ , $Ca^{2+}$ , a	and H⁺
		against their electrochemical differences.	2.8
	29.	Explain the role of ATP-binding cassette transporters in movement across membranes,	as well as
		their role in multi-drug resistance and its significance for cancer chemotherapy.	1.4
	30.	Explain how energy from the Na $^+$ and K $^+$ electrochemical gradients across the plasma m	embrane
		can be used to drive the net "uphill" (against a gradient) movement of other solutes (eg	5,
		Na <sup>+</sup> /glucose co-transport; Na <sup>+</sup> /Ca <sup>2+</sup> exchange or counter-transport), and describe how t	his
		principle can be used in therapy for secretory diarrhea.	2.9
	31.	Explain the role of water channels (aquaporins) in facilitating the movement of water a	cross
		diological membranes.	3.5
В.	Ехс	itable Cells	

1. Define *gating*, *activation*, and *inactivation*.

	2. 3.	Describe the cell properties that determine the rate of electronic conduction. Differentiate between the properties of electrotonic conduction, conduction of an actio	<b>2.0</b>
		potential, and saltatory conduction.	3.0
	4.	Identify regions of a neuron where each type of electrical activity may be found.	3.0
	5.	Compare and contrast the cell-to-cell spread of depolarization at a chemical synapse will a gap junction based on speed and fidelity (success rate).	h that at <b>3.0</b>
	6.	Describe a differentiate temporal summation and spatial summation for the chemical sy	napse.
			2.9
	7.	Describe the principle of the voltage clamp and how it is used to identify the ionic select	ivity of
		channels.	0.6
	8.	Compare and contrast the gating of ion-selective channels by extracellular ligands, intra- ligands, stretch, and voltage.	cellular 2.4
	9.	Describe the properties of voltage-gated Na <sup>+</sup> , K <sup>+</sup> , and Ca <sup>2+</sup> channels, and explain how vol	tage
		influences their gating, activation, and inactivation.	2.8
	10.	Describe how the activity of voltage-gated Na <sup>+</sup> , K <sup>+</sup> , and Ca <sup>2+</sup> channels generates an action potential, and explain the roles of those channels in each phase (depolarization, oversho	n bot,
		repolarization, hyperpolarization) of the action potential.	3.8
	11.	Describe the mechanisms by which an action potential is propagated along both nonmy	elinated
		and myelinated axons.	3.6
	12.	Predict the consequence on action potential propagation in the early and late stages of	
		demyelinating diseases, such as multiple sclerosis.	3.6
С.	Cel	ll Volume Regulation, Organelles and Intracellular pH	
	1.	Explain how regulation of the concentrations of Na+, $K^+$ , $Cl^-$ , and other solutes influence volume.	cell <b>2.6</b>
	2.	Explain how various transporters (eg, $Na^+/H^+$ exchange, $Cl/HCO_3$ exchange, $Na^+HCO_3$ co- transport, etc.) contribute to the control of intracellular pH.	2.4
	3.	Describe Ca <sup>2+</sup> accumulation in the sarcoplasmic and endoplasmic reticulum, mediated by	/ Ca <sup>2+</sup>
		ATPase.	3.0
~	Do	aulation of Coll Function	
D.	Reg	guidtion of Cen Function	
	1.	Describe how intracellular signaling pathways can influence the expression and function	of
		proteins.	1.4
	2.	Describe and provide examples of how phosphorylation/dephosphorylation of proteins channels and membrane receptors) can act as negative and positive effectors of signal	(eg <i>,</i>
		transduction.	1.6
	3.	Define agonist and antagonist as related to membrane receptor ligands.	3.5
	4.	Describe the intracellular signaling pathways for cholinergic nicotinic, cholinergic musca alpha-1 adrenergic, alpha-2 adrenergic, beta-1 adrenergic, beta-2 adrenergic, and beta-3	rinic, 3
		adrenergic receptors.	2.1
	5.	Compare and contrast the receptor location and signaling pathways of peptide and stere	oid 2.8
	6.	Describe the processes of activation, inactivation, up-regulation, down-regulation, sensi and desensitization.	<b>t</b> ization, <b>3.5</b>
Ε.	Ері	ithelial Cell	

1. Describe the movement of a compound that travels across an epithelium by a transcellular pathway and a compound that travels via a paracellular pathway. **3.5** 

	2. 3.	<ol> <li>Describe the role of the "tight" junctions in leaky and tight epithelia.</li> <li>Describe the functional significance of polarized distribution of various transport prot</li> </ol>	
		apical or the basolateral cell membrane.	2.0
	4.	Describe solute-solvent coupling in transport.	1.9
F.	Се	ll Motors	

- Explain how cell molecular motors work to generate force and to transport organelles and other cargo.
   1.3
- Describe how the mobilization of calcium initiates contractions in smooth, striated, and cardiac muscle, the sliding filament model of muscle contraction, and contrast the cellular and molecular basis of muscle contraction in smooth and striated muscle.
   3.5

## G. Transcapillary Transport

- Differentiate between osmotic pressure, oncotic pressure, and hydrostatic pressure, as they pertain to movement across the endothelium of the capillaries.
   3.3
- Predict the permeability of cardiovascular capillaries to small ions/crystalloids (eg, NaCl) and proteins (albumin) based on the capillary reflection coefficient.
   2.1
- 3. Explain how permeability, hydrostatic pressure, and oncotic pressure influence transcapillary exchange of fluid, based on the Starling hypothesis. **3.5**

## III. Endocrine Physiology

## A. General Principles

	1.	Describe the principle of negative feedback control of hormone secretion.	3.9
	2.	Describe the principles of positive feedback and feed forward control of hormone secre	tion.
			3.8
	3.	Identify the bases of hormone measurements.	1.3
	4.	Compare and contrast endocrine, paracrine, and autocrine based on the site of hormor	ne release
		and the pathway to the target tissue.	3.3
	5.	Describe major differences in mechanisms of action of peptides and amines working the	rough
		membrane receptors and steroids, vitamin D, and thyroid hormones working through n	uclear
		receptors.	3.3
	6.	Define hormone, target cell, and receptor.	4.0
	7.	Compare and contrast hormone actions that are exerted through changes in gene expre	ession
		with those exerted through changes in protein phosphorylation.	3.3
	8.	Describe the effects of plasma hormone binding proteins on access of hormones to the	ir sites of
		action and degradation and on the regulation of hormone secretion.	3.4
	9.	Describe the effects of secretion, excretion, degradation, and volume of distribution on	the
		concentration of a hormone in blood plasma.	2.5
В.	Po	sterior Pituitary	
	1.	Compare and contrast the anterior and posterior pituitary lobes with respect to cell typ	es,
		vascular supply, development, and innervation.	3.0
	2.	Identify the target organs or cell types for oxytocin, and describe its effects on each.	2.9

- 3. Identify the stimuli for oxytocin release during parturition or lactation. **2.9**
- 4. Identify the target cells for vasopressin (antidiuretic hormone).

- 5. Describe the stimuli and mechanisms that control vasopressin (ADH) secretion. **3.4**
- Identify disease states caused by over-secretion and under-secretion of vasopressin (ADH), describe the principle signs and symptoms of each, and provide a physiological basis for these.

## C. Anterior Pituitary

- 1. Describe the general structure and actions of the glycoprotein hormones FSH, LH, and TSH.
- 2. Describe the general structure, actions, and metabolism of the GH/prolactin family. **3.0**
- Describe the general structure and actions of the POMC family: ACTH, MSH, β-lipoprotein, βendorphin.
   3.0
- Identify hypothalamic factors that control the secretion of each of the anterior pituitary hormones, and describe their route of transport from the hypothalamus to the anterior pituitary.
   3.6
- Describe and interpret the short-loop and long-loop negative feedback control of anterior pituitary hormone secretion.
   3.3
- Predict the changes in secretory rates of hypothalamic, anterior pituitary, and target gland hormones caused by over-secretion or under-secretion of any of these hormones or receptor deficit for these hormones.
   3.3
- 7. Describe the importance of pulsatile and diurnal secretion. 2.5

### D. Thyroid Gland

- Outline the steps in the biosynthesis, storage, and secretion of tri-iodothyronine (T<sub>3</sub>) and thyroxine (T<sub>4</sub>) and their regulation.
   3.3
- 2. Define *iodine pool*.
- 3. Describe the distribution of iodine and the iodide metabolic pathway, and relate the distribution of radioiodide in the body to thyroid hormone synthesis, metabolism, and excretion. **2.1**
- Describe factors that control the synthesis, storage, and release of thyroid hormones and describe the importance of thyroid hormone binding in blood on free and total thyroid hormone levels.
   3.1
- Explain the significance of the conversion of T<sub>4</sub> to T<sub>3</sub> and reverse T<sub>3</sub> (rT<sub>3</sub>) in extra-thyroidal tissues.
   2.5
- 6. Outline the actions of thyroid hormones on development and metabolism.
- Discuss the causes and consequences of over-secretion and under-secretion of thyroid hormones, and explain why either condition can cause an enlargement of the thyroid gland.

#### E. Parathyroid Gland

- Describe the cells of origin for parathyroid hormone, its biosynthesis, and its transport within the blood.
   3.1
- Identify the target organs and cell types for parathyroid hormone and describe its effects on each.
   3.9
- Describe the functions of the osteoblasts and the osteoclasts in bone remodeling, and identify the factors that regulate their activities.
   3.9
- Identify the time course for the onset and duration for each of the biological actions of parathyroid hormone.
   2.6
- Describe the regulation of parathyroid hormone secretion and the role of the calcium-sensing receptor.
   3.4

3.4

3.0

2.1

3.5

	6.	Describe the causes and consequences of over-secretion and under-secretion of parathy hormone.	vroid <b>3.4</b>
	7.	Identify the sources of vitamin D, the biosynthetic pathway, and the organs involved in modifying it to the biologically active $1,25(OH_2)D_3$ (1-25 dihydroxy cholecalciferol) form.	3.5
	8.	Identify the target organs and cellular mechanisms of action for vitamin D.	• •
	٥	Describe the negative feedback relationship between the parathyroid hormone and the	3.8
	9.	biologically active form of vitamin D $[1,25(OH_2)D_3]$ .	3.0
	10.	Describe the consequences of vitamin D deficiency and vitamin D excess.	3 /
	11.	Identify the cell of origin and target organs or cell types for calcitonin.	2.8
	12.	Identify the stimuli that can promote secretion of calcitonin.	3.1
	13.	Describe the actions of calcitonin, and identify which are physiologically important.	3.2
F.	Adı	renal Gland	
	1.	Identify the functional zones, innervation, and blood supply of the adrenal glands and th	e
		principal hormones secreted from each zone.	3.3
	2.	Describe the biosynthesis of the adrenal steroid hormones (glucocorticoids, mineralocor	ticoids,
	n	and androgens) and the key features that distinguish each class.	2.5
	3. ⊿	Describe the cellular mechanism of action of adrenal cortical normones.	2.5
	4.	are produced	<b>4 0</b>
	5.	Describe the actions of glucocorticoid hormones in injury and stress.	4.0
	6.	Describe the components of the neuroendocrine axis that control glucocorticoid secretic	on and
		describe how factors in the internal and external environment influence the neuroendoo	crine
		axis.	4.0
	7.	Identify the causes and consequences of over-secretion and under-secretion of glucocor	ticoids
		and adrenal androgens.	3.5
	8.	Identify the major mineralocorticoids, as well as their biological actions and target organ	is or
	٥	ussues. Identify the physiological stimuli that promote increased mineral continuit secretion, and	<b>3.9</b>
	5.	these stimuli to regulation of sodium and potassium excretion.	<b>3.9</b>
	10.	Identify the factors can modulate the secretory response and describe how they are det	ected.
			3.9
	11.	Identify the causes and consequences of over-secretion and under-secretion of	
		mineralocorticoids.	3.6
	12.	Describe and interpret the negative feedback control of aldosterone secretion.	3.0
	13.	Identify the chemical nature of catecholamines, their biosynthesis, mechanism of transp within the blood, and explain how they are degraded and removed from the body.	ort 2.4
	14.	Identify how the structure of norepinephrine differs from epinephrine.	3.0
	15.	Describe the biological consequences of activation of the adrenal medulla, and identify t	he
		target organs or tissues for catecholamines, along with the receptor subtype that media response and the mechanism by which epinephrine and norepinephrine can produce dif	tes the ferent
		effects in the same tissues.	3.8
	16.	Describe the change in the ratio of epinephrine to norepinephrine release from the adre	enal
		medulla during sympathetic activation (fight and flight), or in prolonged food deprivation	n.
	17	Identify the low stimuli equains established an increase tion	3.8
	1/.	identity the key sumuli causing catecholamine secretion.	5.4

	18.	List the factors that can modulate the secretory response and the responses of target tis	sues.
	19.	Describe the interactions of adrenal medullary and cortical hormones in response to stre	<b>3.4</b> 255.
			3.6
	20.	Identify disease states caused by an over-secretion of adrenal catecholamines.	3.1
G.	Pai	ncreas	
	1.	Identify the major hormones secreted from the endocrine pancreas, their cells of origin, their chemical nature.	and <b>4.0</b>
	2.	List the target organs or cell types for glucagon, and describe its principal actions on eac	h.
	3. 4.	Identify the time course for the onset and duration of the biological actions of glucagon. Describe the control of glucagon secretion.	3.9 3.0 3.9
	5.	Identify the major target organs or cell types for insulin, the major effects of insulin on e the consequent changes in concentration of blood constituents.	ach, and <b>4.0</b>
	6. 7.	Identify the time course for the onset and duration for the biological actions of insulin. Describe the relationship between blood glucose concentrations and insulin secretion, a explain the roles of neural input and gastrointestinal hormones on insulin secretion.	<b>4.0</b> nd
	8. 9.	Identify the factors that modulate the secretory response of insulin. Identify disease states caused by over-secretion, under-secretion or decreased sensitivit insulin, and describe the principal signs and symptoms of each and provide a physiologic for these.	<b>4.0</b> y to cal basis <b>4.0</b>
Н.	Gro	pwth	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> </ol>	Describe the relationship between growth hormone and the insulin-like growth factors a binding proteins in the regulation of growth. Describe the regulation of growth hormone secretion, and identify the roles of hypothal factors and IGF-I. Identify the target organs or cell types for insulin-like growth factors that account for longitudinal growth. Describe how thyroid, insulin, gonadal, and adrenal hormones modulate growth. Describe the nature and actions of local growth factors.	and their 3.8 amic 3.6 3.8 3.4 3.0
Ι.	End	docrine Integration of Energy and Electrolyte Balance	
	1. 2.	Identify the normal range of plasma glucose concentrations, as well as the chemical form anatomical sites of storage pools for glucose and other metabolic substrates. Identify the hormones that promote the influx and efflux of glucose, fat, and protein inte out of energy storage pools and their impact on the uptake of glucose by tissues. Establish specific roles for insulin, glucagon, glucocorticoids, catecholamines, growth hor	ns and <b>3.6</b> 5 and <b>3.5</b>
	3.		mone,
	3. 4.	and thyroid hormone. Describe the changes in metabolic fuel utilization that occurs in long- and short-term fas in acute and sustained exercise, and describe how increases or decreases in hormone se produce these changes.	<b>3.5</b> ting and cretion <b>2.8</b>
	3. 4. 5.	and thyroid hormone. Describe the changes in metabolic fuel utilization that occurs in long- and short-term fas in acute and sustained exercise, and describe how increases or decreases in hormone se produce these changes. Describe the role of appetite and metabolic rate in the maintenance of long-term energy balance and fat storage.	<b>3.5</b> ting and cretion <b>2.8</b> / <b>2.1</b>

	7.	Identify the normal range of dietary sodium intake, sodium distribution in the body, and of sodium excretion, and describe the roles of antidiuretic hormone, aldosterone, angio	l routes tensin,
		and atrial natriuretic hormone in the regulation of sodium balance.	3.1
	8.	Identify the normal range of dietary potassium intake, potassium distribution in the boo	ly, and
		routes of potassium excretion.	3.1
	9.	Explain how acute changes in aldosterone, insulin, and acid/base concentrations affect in	the
		plasma potassium concentration and the movement of potassium into and out of the	
		intracellular compartment.	3.1
	10.	Describe the chronic regulation of body potassium balance and plasma potassium levels	s by
		aldosterone through its actions on renal excretion, intestinal excretion, and dietary	
		appetite/absorption.	3.1
	11.	Identify the normal range of dietary calcium intake, calcium distribution in the body, and	d routes
		of calcium excretion.	3.1
	12.	Describe the regulation of the plasma calcium concentration by parathyroid hormone, v	ritamin
		D, and calcitonin based on exchange with bone, renal excretion, and intestinal excretion	n and/or
		absorption.	3.1
	13.	Identify the normal range of dietary phosphate intake, phosphate distribution in the bo	dy, and
		routes of phosphate excretion.	3.0
	14.	Describe the regulation of the plasma phosphate concentration by parathyroid hormone	e <i>,</i>
		vitamin D, and calcitonin based on exchange with bone, renal excretion, intestinal excre	tion
		and/or absorption.	3.0
1	Мс	ale Reproductive Physiology	
J.	IVIC	ine neproductive r hysiology	
	1.	Describe the physiological functions of the major components of the male reproductive	tract.
			2.4
	2.	Describe spermatogenesis and the role of different cell types in this process.	2.1
	3.	Describe the endocrine regulation of testicular function: the role of the GnRH pulse gen	erator,
		FSH, LH, testosterone, and inhibin.	2.6
	4.	Identify the cell of origin for testosterone, its biosynthesis, mechanism of transport with	in the
		blood, how it is metabolized and how it is eliminated. List other physiologically produce	ed
		androgens.	2.4
	5.	Identify the target organs or cell types for testosterone and describe its effects on each.	3.1
	6.	Describe the cellular mechanisms of action for testosterone.	2.1
	7.	Identify the neural, vascular, and endocrine components of the erection and ejaculation	1
		response.	1.8
	8.	Identify the causes and consequences of over-secretion and under-secretion of testoste	rone for
		prepubescent and postpubescent males.	2.1
	9.	Compare and contrast the actions of testosterone, dihydrotestosterone, estradiol, and	
		Müllerian inhibitory factor in the development of the male and female reproductive trad	cts.
			1.8
К.	Fer	nale Reproductive Physiology	
	1	Describe opgenesis and its relationship to changes in the ovarian follicle and the roles of	fesh
	т.	LH estradiol inhibin and paracrine agents in opgenesis and follicular maturation	<b>7</b> 9
	_	Lin, esti autor, initioni, anu paracime agents in obgenesis anu toincular matulation.	2.0

- Describe ovulation, as well as the formation and decline of the corpus luteum.
   Explain the roles of pituitary hormones in the formation and decline of the corpus luteum.
  - 2.6

4.	Describe the hormonal regulation of estrogen and progesterone biosynthesis and secre the ovary.	tion by <b>2.9</b>
5.	Identify the cells responsible for their biosynthesis, the mechanism of their transport in	the
	blood, and explain how they are degraded and removed from the body.	2.9
6.	Identify the target organs or cell types for estrogen action and describe its effects on ea	ach.
		3.0
7.	Describe the cellular mechanisms of action for estrogen.	2.5
8.	Identify the principal physiological actions of progesterone, its target organs or cell type	es, and
	describe its effects on each and the importance of "estrogen priming."	3.0
9.	Describe the cellular mechanisms of action for progesterone.	2.8
10	. Describe and interepret the changes in the endometrium and the ovary during the mer	strual
	cycle and correlate these changes with changes in blood concentrations of FSH, LH, est	radiol,
	progesterone, and inhibin.	2.1
11	. Describe how the changes in ovarian steroids produce the proliferative and secretory p	hases of
	the uterine endometrium and menstruation and changes in basal body temperature du	iring the
17	menstrual cycle.	2.1
12	. Identify the pathways of sperm and egg transport that can result in rerunzation and the	10
12	Identify the protoin hormones secreted by the placental and describe the role of huma	1.9 n
13	chorionic gonadotronin (bCG) in the rescue of the cornus luteum in maintaining pregna	ncy parly
	nost-implantation	<b>2 2</b>
14	Describe the interactions between the placenta and the fetal adrenal cortex in the proc	duction of
	estrogens during pregnancy.	1.5
15	. Discuss the roles of oxytocin, relaxin, and prostaglandins in the initiation and maintena	nce of
_	parturition.	1.8
16	. Describe the role of estrogens, progesterone, placental lactogen, prolactin, and oxytoci	n in
	mammary gland development during puberty, pregnancy, and lactation.	1.6
17	. Describe the basis for the inhibition of milk secretion during pregnancy and the initiatic	on of
	lactation after parturition.	1.6
18	. Differentiate between milk secretion and milk ejection, and describe the hormonal reg	ulation of
	both during lactation, including the role of suckling.	1.6
19	. Describe the physiological bases for the antifertility actions of contraceptive steroid ho	rmones.
		1.9
20	. Describe the age-related changes in the male and female reproductive systems, includi	ng the
	mechanisms responsible for these changes: In utero development, Puberty and Senesc	ence.
		1.5
_		
Ga	astrointestinal Physiology	

## \_\_\_\_\_

IV.

# A. Functions and Regulation of GI Tract

- Describe the overall role of the gastrointestinal system with respect to the whole body balance of water, electrolytes, carbohydrates, fats, and proteins.
   3.3
- Explain the processes of digestion, absorption, metabolic production, metabolic consumption, secretion, and excretion.
   3.3
- 3. Identify appropriate metabolic waste products present in the feces. **3.3**

	4.	Differentiate between the processes of ingestion, digestion, absorption, secretion, and excretion, including the location in the GI tract where each process occurs, for carbohyd	lrates,
	5.	proteins, and fats. Identify the approximate normal volumes of fluid entering and leaving the gastrointesting and leaving the	2.9 nal tract
	6.	Describe the major characteristics of and temporally relate the cephalic, gastric, and interpreted to the second s	1.5 estinal
	7	phases of GL (regulation. Describe the four classes of luminal stimuli that trigger GL reflexes	2.1
	7. 8	Describe the histoanatomical characteristics of the enteric nervous system given either	a cross-
	0.	section or a longitudinal section of the intestine.	<b>0.6</b>
	9.	Identify and locate the myenteric and submucosal plexus, given either a cross-section of longitudinal section of the intestine.	r a <b>0.6</b>
	10.	Contrast the sympathetic and parasympathetic modulation of the enteric nervous syste the effector organs of the GI tract.	m and <b>3.0</b>
	11.	Classify the following enteric nervous system neurotransmitters as excitatory or inhibita effect: norepinephrine, acetylcholine, CCK, VIP, histamine, and somatostatin.	tory in <b>2.1</b>
	12.	Define <i>long reflex</i> and <i>short reflex</i> with respect to the GI tract.	1.8
	13.	Describe the similarities and differences in regulating gastrointestinal function by nerves	 S,
		hormones, and paracrine regulators, including receptors, proximity, and local versus glo	bal <b>1.8</b>
	14.	Identify the cell type and anatomical location of the endocrine cells secreting gastrin, se	cretin,
	15.	Identify families to which gastrin, secretin, and CCK and other (non-GI) hormones belon	<b>1.0</b> g.
	16.	Define incretins, and identify two gastrointestinal hormones that function in this manne	<b>1.0</b> r.
			1.3
	17.	Describe the function of somatostatin and histamine as paracrine regulators of acid secutive stomach.	retion in <b>2.5</b>
В.	Sal	ivary Gland	
	1.	Compare and contrast the plasma and salivary concentrations of $Na^+$ , $Cl^-$ , and $HCO_3^-$ at b and high secretion rates, and identify the principal cell types involved in each secretion	oth low
			1.3
	2.	Identify the substrates and digestion products of salivary amylase (ptyalin).	1.8
	3.	Identify the stimuli and cell types involved in GI secretion of mucous, and describe the for	unction
	4.	Identify three types of stimuli that increase salivary secretion.	1.5
	5.	Identify the components of saliva important in oral hygiene, and explain the role of saliv	vary
	-	secretions in eliminating heavy metals.	1.1
С.	Esc	pphagus	
	1.	Identify the normal resting esophageal pressure, and explain why this pressure varies w	ith the
		respiratory cycle.	1.1
	2.	Describe the origin and consequence of the high basal tone found in the upper esophag	eal
	2	sphincter (UES) and lower esophageal sphincter (LES).	1.8
	<u></u> з.	identify the stimulus that initiates the swallowing sequence, as well as the point at whic	n the
		swallowing sequence becomes automatic (independent of voluntary control).	T.0

	4.	Compare and contrast the patterns of external and internal innervations of the upper, m and lower esophagus.	niddle, <b>1.3</b>
	5.	Describe the pressure changes that occur in the esophagus as a bolus of food moves from pharynx to the stomach, including the pressures immediately oral and aboral to the bolu	m the us, and
	_	the pressures in the upper and lower esophageal sphincters.	1.3
	6.	Compare and contrast primary and secondary peristalsis based on initiating event, volur	ntary
	-	control, reflex propagation, and regions of the pharynx and esophagus involved.	1.3
	7.	Compare and contrast the lower esponageal tone, innervation, and motility defects that	1ead to
		nearcourn with those leading to achaiasia.	2.0
D.	Sto	nmach	
	1.	Explain the storage, digestion, and motility roles of the stomach.	3.4
	2.	Compare and contrast the Na $^{*}$ , K $^{*}$ , and Cl $^{-}$ concentrations of gastric secretion with that o	f plasma
		at low and at high gastric secretion rates, and identify the cell types that mediate this ch	ange.
			1.4
	3.	Identify the protein component of chief cell secretions.	2.3
	4.	Describe the generation of an "alkaline tide" in the hepatic portal venous system following	ng
		ingestion of a meal.	1.6
	5.	Describe the role of HCl in the gastric digestion of carbohydrates, proteins, and fats.	1.9
	6.	Describe the pH of the stomach in the fasted state, and outline the time course and cause	ses of
		the pH changes in the two hours after ingestion of a protein meal.	1.4
	7.	Identify the stimuli for pepsinogen release and the mechanism for activating pepsinoger	n, and
		describe the digestion products of pepsin activity.	2.0
	8.	Explain the role of the stomach in preventing pernicous anemia.	2.5
	9.	Describe the regulation of H <sup>+</sup> -K <sup>+</sup> ATPase, the stimuli for activation, and process of activat	tion,
		including vesicular fusion with the luminal plasma membrane.	1.5
	10.	Describe the mechanism of gastric H <sup>+</sup> generation and secretion, including the role of K <sup>+</sup> ,	CI-HCO₃,
		carbonic anhydrase, H <sup>+</sup> -K <sup>+</sup> ATPase and Na <sup>+</sup> -K <sup>+</sup> ATPase.	2.1
	11.	Describe the modulation of gastric acid secretion by the enterochromatin-like cell (ECL c	ell), and
		explain the control of this process (including potentiation) by vagal stimulation, gastrin,	
	4.2	histamine, and somatostatin.	2.1
	12.	Describe the pathways for the gastric absorption of electrolytes, water, lipids, amino aci	as, and
	10	Carbonyarales.	1.0
	13.	Haliobastar pulori	20
	11	Identify the stimuli that increase gastrin release and inhibit gastrin release	5.0 2 1
	14.	Identify the effects of acid, fat, and solutions of high osmolarity in the duodenum on gas	
	15.	secretion, and describe the mechanisms by which these effects regulate gastric secretion	n
		seletion, and desense the meenanisms by which these encets regulate gastile selection	23
	16	Explain recentive relaxation of the stomach and identify mechanism and consequence	2.5
	10.	Explain receptive relaxation of the scontactly and facturely meentalism and consequences	2.0
	17	Describe origin and form of electrical activity and the progression of peristaltic waves ac	ross the
	±7.	body and antrum of the stomach, including their roles in mixing and propulsion of gastri	C C
		contents.	1.6
	18.	Explain how the frequency is altered by the volume of gastric contents.	1.6
	19.	Define <i>gastroparesis</i> and explain how diabetes can cause it.	1.6
	20.	Predict the effects of meal content (osmolarity, fat content, etc.), particle size, and volu	me on
		the rate of gastric emptying, including duodenal feedback.	2.0

- 21. Identify the causes of peptic ulcer disease.
- E. Pancreas
  - 1. Identify the major ionic and peptide/protein components secreted by the exocrine pancreas.
  - Compare and contrast the plasma and pancreatic concentrations of Na<sup>+</sup>, Cl<sup>-</sup>, and HCO<sub>3</sub><sup>-</sup> at low secretion rates and at high secretion rates and the principal cell types involved in each secretion rate.
     2.6
  - 3. Describe the mechanisms by which chyme from the stomach is neutralized in the duodenum.
  - 4. Describe the mechanism by which pancreatic zymogens are activated in the small intestine.
  - 5. Identify the stimuli that release secretin and CCK and the cellular mechanisms by which these agents control pancreatic secretion, as well as any synergistic effects between CCK and secretin.
  - Describe the role of CFTR in pancreatic ductular secretion and predict the consequences of cystic fibrosis on the GI system.
     2.0
  - 7. Discuss the effects of the autonomic nerves on the pancreas. **2.1**
- F. Bile
  - Identify the water, ionic, bile salt, and bilirubin components of bile as secreted by the liver, and explain the modification of bile as it is stored in the gall bladder and the role of secretin on the hepatic production of bile.
     2.3
  - Describe the cellular mechanisms for the hepatic uptake, conjugation, and secretion of bile salts and bilirubin.
     2.0
  - Describe the role of CCK in causing release of bile from the gall bladder, including the effects on the sphincter of Oddi.
     2.4
  - Describe the amphipathic structure of bile acids, and explain how this property assists the digestion of fats.
  - 5. Differentiate between primary and secondary bile acids. **0.8**
  - Compare and contrast the physical state of an emulsion with a micellar solution, and explain the conditions for the formation of emulsifications and miceles in the duodenum.
     1.0
  - 7. Define *enterohepatic circulation*.
  - 8. Describe the mechanism of reabsorption of bile acids in the early portion of the small intestine with the mechanism found in the later part of the small intestine.
     0.9
  - Discuss the effects of an increase in hepatic portal vein bile acid concentration on the rate of bile secretion, bile acid synthesis, and diseases of the gallbladder.
     1.3

## G. Small Intestine

- 1. Describe the role of the microvilli, the unstirred layer, and tight junctions in determining the rate at which glucose, amino acids, water, lipids, and electrolytes are absorbed. **1.8**
- Identify the chemical classes of the carbohydrates entering the duodenum from the stomach, including the mechanisms mediating further digestion and absorption across the apical and basolateral membranes of the intestinal epithelia, as well as pancreatic secretions and brushborder enzymes.
   2.0
- 3. Explain the small intestine and colonic consequence of a deficiency in the enzyme lactase, and identify ethnic groups who commonly exhibit this deficiency. **2.0**

91

2.3

1.6

2.4

	4.	Identify the chemical classes of the proteins entering the duodenum from the stomach, including the mechanisms mediating further digestion and absorption across the apical a	and
		basolateral membranes of the intestinal epithelia, as well as pancreatic secretions and b	rush-
	_	border enzymes.	<b>1.8</b> .
	5.	Compare and contrast the secondary active transport of amino acids with that of di- and	tri-
	6	Identify the chemical classes of the linids entering the duodenum from the stomach incl	1.0
	0.	the mechanisms mediating further digestion and absorption across the anical and basola	ateral
		membranes of the intestinal epithelia, and explain the roles of pancreatic lipase, colipase	e. and
		micelles.	<b>2.1</b> .
	7.	Explain the role of the endoplasmic reticulum in processing lipids absorbed across the ap	pical
		membrane of enterocytes.	1.3
	8.	Describe the composition and formation of chylomicrons, their movement across the en	terocyte
		basolateral membrane, and the route of entry into the cardiovascular system.	2.1
	9.	Define steatorrhea, and explain the effects of steatorrhea on the absorption of fat-solub	le
		vitamins.	2.0
	10.	. Explain the absorption of water-soluble vitamins, including the role of intrinsic factor in t	the
	11	absorption of vitamin $B_{12}$ .	<b>2.</b> 0
	11.	the duodenum and colon, and identify the cause of this change.	<b>1.1</b>
	12.	. Describe the pathways, if any, by which sodium ions, water, iron, and calcium are absorb the small intestine and colon.	oed in <b>2.0</b>
	13.	Describe the cellular mechanisms of colonic sodium, potassium, and bicarbonate secreti	on, as
		well as the regulation of this process by aldosterone.	1.5
	14.	Define <i>dietary fiber</i> and identify sources commonly found in the US diet.	2.0
	15.	Identify substrates and products of colonic bacterial metabolism, and explain the impact	: of
	4.0	metabolites on the rate and composition of intestinal gas formation (flatus).	0.9
	16.	Describe the production and absorption of short chain fatty acids in the colon.	0.9
Н.	Inte	estinal Motility	
	1.	Describe the characteristics of the basic electrical rhythm (BER) of the small intestine, an	nd
	•	explain its relation to smooth muscle contractile activity.	2.3
	2.	Describe the role of "interstitial cells of Cajal" in generation of electrical slow waves, and	l explain
		interconsequence of the frequency gradients of electrical slow waves occurring within the	e 16
	z	Describe the functional significance of ongoing activity of enteric inhibitory motor neuro	ns to
	5.	intestinal circular muscle.	<b>1.2</b>
	4.	Define <i>ileus</i> and explain why surgery can cause it.	1.1
	5.	Compare and contrast the patterns of intestinal motility seen during the absorptive phase	se
		(segmentation) with that of the post-absorptive phase between meals.	1.6
	6.	Compare and contrast the effects of parasympathetic and sympathetic nervous activity i	n
		modulating small intestinal motility.	3.1
	7.	Describe the effects of distension on small intestinal motility.	2.1
	8.	Describe the effects of increased pressure in the ileum and cecum on the ileocecal sphin	cter,
	~	and relate to gastroileal reflex.	1.6
	9.	Compare and contrast colonic motor activity with the motor activity in the small intestin	e.
			1.3

- Compare and contrast the colonic motor activity during a mass movement with that during haustral shuttling, and explain the consequence of each type of colonic motility.
   2.3
- 11. Describe the sequence of events occurring during reflexive defecation, differentiating those movements under voluntary control and those under intrinsic control. **2.8**

# V. Integration and Exercise Physiology

## A. Thermoregulation

	1.	Describe the thermal balance for the body, including heat production (metabolism, exerci	se,			
		shivering) and heat loss (convection, conduction, radiation, and evaporation).	2.1			
	2.	Identify those mechanisms that shift from heat production to heat loss when environmen	tal			
		temperature exceeds body core temperature.	2.7			
	3.	Explain the thermoregulatory set point, and describe the negative feedback control of bo	dy core			
		temperature, including the role of the hypothalamic set point.	2.9			
	4.	Compare and contrast the stability of body core with that of skin temperature.	3.0			
	5.	Explain the role of cutaneous blood flow and sweating on skin temperature.	3.0			
	6.	Identify the mechanisms for maintaining thermal balance in the following environments:				
		a. Desert (120°F)	2.0			
		b. Snow skiing (10°F)	2.0			
		c. Falling through ice into a lake (water temp 37°F)	2.0			
		d. Snorkeling in 80°F water	2.0			
	7.	Explain how the change in core temperature that accompanies exercise differs from the c	hange in			
		core temperature produced by influenza, which alters the thermoregulatory set point.	2.1			
	8.	Identify and describe the physiological changes that occur as a result of acclimatization to	heat and			
		cold.	1.9			
B.	Fxe	ercise				
	1.	Compare and contrast the normal distribution of cardiac output with the distribution of	cardiac			
	2	output during aerobic (sustained) exercise and anaerobic (brief maximal burst) exercise. <b>3.4</b>				
	Ζ.	Explain the local regulation of blood flow and the role of capillary reserve in altering ske				
	n	Muscle blood flow.	3.4			
	3.	Define $VO_{2MAX}$ and identify situations in which it is limited by cardiac output and by pulm				
	Λ	gds excitalige.	2./			
	4.	accompanies eversise and how it can occur without any measurable change in arterial h	le lood gag			
		values	<b>7 1</b>			
	5	Values.	<b>2.4</b>			
	5.	contribute to an increase in Vo	1 <b>0</b>			
	6	Explain how muscle fatigue $V_{02MAX}$ .	1.5			
	0.	nerformance	10			
	7	Describe how chronic physical activity alters insulin sensitivity and glucose entry into ce	lls			
	7.	Desense now enrome physical activity alters insum sensitivity and glucose entry into te	2.9			
	8	Describe the health benefits of exercise training on the cardiovascular musculoskeletal	immune			
	0.	systems, and for weight control.	<b>2.7</b>			
			,			

#### VI. <u>Muscle Physiology</u>

#### A. Skeletal Muscle Structure and Mechanism of Contraction

- 1. Diagram and label a skeletal muscle at all anatomical levels, from the whole muscle to the sarcomere, including two different stages of myofilament overlap at the sarcomere level.
- 2. Explain the function and role of the heavy and light chains of myosin.
- 3. Diagram the structure of the thick and thin myofilaments and label the constituent proteins.
- 4. Describe the relationship of the myosin-thick filament bare zone to the shape of the active length:force relationship. **2.5**
- 5. Describe and interpret the sequence of chemical and mechanical steps in the cross-bridge cycle, and explain how the cross-bridge cycle results in shortening of the muscle. **3.4**

#### B. Control of Skeletal Muscle Contraction

- 1. Describe the steps in excitation-contraction coupling in skeletal muscle, and describe the roles of the sarcolemma, transverse tubules, sarcoplasmic reticulum, thin filaments, and calcium ions.
- 2. Describe the roles of ATP in skeletal muscle contraction and relaxation.
- 3. Describe the basic structure of the neuromuscular junction.
- 4. Identify the steps, in sequence, involved in neuromuscular transmission in skeletal muscle, and identify the location of each step on a diagram of the neuromuscular junction. **3.9**
- 5. Differentiate between an endplate potential and an action potential in skeletal muscle. 3.3
- 6. Identify the possible sites for blocking neuromuscular transmission in skeletal muscle, and provide an example of an agent that could cause blockage at each site.

3.6

3.5

2.3

3.0

4.0

4.0

3.8

#### C. Mechanics and Energetics of Skeletal Muscle Contraction

1. Describe the relationship of preload, afterload, and total load in the time course of an isotonic 3.3 contraction. 2. Differentiate between an isometric and isotonic contraction. 3.9 3. Differentiate between a twitch and a tetanus in skeletal muscle and explain why a twitch is smaller in amplitude than a tetanus. 4.0 4. Describe and interpret the length versus force diagram for muscle, and label the three lines that represent passive (resting), active, and total force and describe the molecular origin of these forces. 3.9 Describe the interaction of the length:force and the force:velocity relationships. 2.8 6. Describe and interpret force versus velocity relationships for two skeletal muscles of equal maximum force generating capacity but of different maximum velocities of shortening. **3.0** 7. Relate the power output of skeletal muscle to its force versus velocity relationship, using a diagram. 2.4 8. Describe the influence of skeletal muscle tendons on contractile function. 3.1 Identify the energy sources of muscle contraction and rank the sources with respect to their relative speed and capacity to supply ATP for contraction. 3.0 10. Define muscular fatigue and list some intracellular factors that can cause fatigue. 3.5

- 11. Compare and contrast the structural, enzymatic, and functional features of fast-glycolytic and<br/>slow-oxidative fiber types in skeletal muscle.2.9
- Describe the role of the myosin crossbridges acting in parallel to determine active force and the rate of crossbridge cycling to determine muscle speed of shortening and rate of ATP utilization during contraction.
   2.6
- Describe and interpret the functional consequences of the parallel and series arrangement of myofibrils in a skeletal muscle.
   2.6
- 14. Explain how the arrangement of a skeletal muscle to the skeleton can influence mechanical performance of the muscle. **2.8**
- Define *motor unit* and describe the order of recruitment of motor units during skeletal muscle contraction of varying strengths.
   **3.0**
- Describe what basic science information can be learned from an electromyographic (EMG) examination.
   4.0

### D. Smooth Muscle

	1.	Describe the differences in actomyosin regulation of smooth and skeletal muscle, and co	ompare
		and contrast their respective contractile units.	2.9
	2.	Compare and contrast the length versus force relationships in skeletal and smooth must	cle, and
		describe the functional implications of the differences observed.	2.3
	3.	Compare and contrast the force versus velocity relationships in skeletal and smooth mu	scle, and
		describe the primary basis for the observed differences in velocity of shortening.	2.3
	4.	Explain why smooth muscles can develop and maintain force with a much lower rate of	ATP
		hydrolysis than skeletal muscle.	2.9
	5.	Differentiate between muscle relaxation from the contracted state and the phenomeno	n of
		stress relaxation and give examples of each process.	1.7
	6.	Describe the intracellular pathways that control contraction and relaxation in smooth m	uscle.
			2.9
	7.	Describe the distinguishing characteristics of multi-unit and unitary smooth muscles.	2.9
Ε.	Car	rdiac Muscle	
	1.	Describe the structure of cardiac muscle cells.	3.3
	2.	Compare and contrast the structure of cardiac muscle cells with that of smooth and ske	letal
		muscle cells.	3.3
	3.	Describe the physiological consequences of the low-resistance pathways between cardi	ас
		muscle cells.	3.3
	4.	Describe and interpret the relationship between an action potential and a twitch in card	liac
		muscle, and explain why this prevents a tetanic contraction.	3.4
	5.	Describe and interpret the steps in the excitation-contraction coupling mechanism in ca	rdiac
		muscle.	3.4
	6.	Compare and contrast the excitation-contraction coupling mechanism in cardiac muscle	with
		that of skeletal muscle.	3.4
	7.	Describe and interpret the length versus force curve for cardiac muscle and skeletal mus	scle,
	_	showing the active and passive relationships.	3.1
	8.	Identify the range over which cardiac and skeletal muscle perform their respective phys	iological
	•	functions.	3.1
	9.	Describe contractility in cardiac muscle and on a length versus force diagram.	3.4
	10.	identify the pathway for an isotonic contraction of cardiac muscle, and describe how an	increase
		in contractility changes the relationship between afterload and amount of shortening.	3.4

11. Identify and describe inotropic interventions that could change cardiac contractility. 2.9

#### **Neuorophysiology** VII.

А.	Ele	ctrophysiology	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> <li>11.</li> <li>12.</li> <li>13.</li> </ol>	<ul> <li>Define:</li> <li>a. dendrites</li> <li>b. axon</li> <li>c. axon hillock</li> <li>d. soma</li> <li>e. synaptic cleft</li> <li>Identify dendrites, axon, axon hillock, soma, and synaptic cleft on a neuron diagram.</li> <li>Explain the Nernst equation, as well as the effects of altering either the intracellular or extracellular Na<sup>+</sup>, K<sup>+</sup>, Cl<sup>-</sup>, or Ca<sup>2+</sup> concentration on the equilibrium potential for that ion.</li> <li>Describe the normal distribution of Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>2+</sup>, and Cl<sup>-</sup> across the cell membrane.</li> <li>Explain how the relative permeabilities to Na<sup>+</sup>, K<sup>+</sup>, Ca<sup>2+</sup>, and Cl<sup>-</sup> create a resting membrar potential.</li> <li>Describe ionic basis of an action potential.</li> <li>Compare and contrast the generation and conduction of graded potentials with that of a potentials, identifying the area on a neuron in which each occurs.</li> <li>Describe the basis for the calculation and the role of the space constant and time constant enveronal processes.</li> <li>Define membrane capacitance and describe its role in the spread of current in myelinated demyelinated neurons.</li> <li>Compare conduction velocities in a compound nerve, identifying how the diameter and myelination lead to differences in conduction velocity, and explain the use of these differences in conduction velocity, and explain the use of these differences.</li> <li>Describe the ionic basis for inhibitory and excitatory post-synaptic potentials, and explait these changes can alter synaptic transmission.</li> <li>Describe the effects of hyperkalemia, hypercalcemia, and hypoxia on the resting membra action potential.</li> <li>Describe the effects of demyelination on action potential propagation and nerve conduction</li> </ul>	3.7 3.7 3.7 3.7 3.7 3.9 2.9 3.7 4.0 action 3.0 action 3.0 action 3.0 attion 3.0 action 3.0 3.0 action 3.0 3.0 action 3.0 action 3.0
В.	Ne	urochemistry	
	1. 2. 3.	Compare and contrast electrical and chemical synaptic transmission based on velocity o conduction, fidelity, and the possibility for neuromodulation (facilitation or inhibition). Describe chemical neurotransmission, listing in correct temporal sequence events begin with the arrival of a wave of depolarization at the presynaptic membrane and ending wi graded potential generated at the postsynaptic membrane. Identify the characteristics of a neurotransmitter.	f 3.0 ning th a 4.0 3.1
	4.	<ul> <li>Describe the synthetic pathways, inactivation mechanisms, and neurochemical anatomy mechanisms of receptor transduction for the following neurotransmitters</li> <li>a. Catecholamines (DA, NE, E)</li> <li>b. Acetylcholine (ACh)</li> <li>c. Serotonin (5-hydroxytryptamine 5-HT)</li> </ul>	1.4 1.4 1.4 1.4

c. Serotonin (5-hydroxytryptamine 5-HT)

	5. 6. 7.	d. Histamine1e. GABA (gamma-aminobutyric acid)1f. Glutamate1g. Endorphins1h. Enkephalins1i. Dynorphins1j. Substance P1Identify the major receptor classifications and representative receptor agonists and antagefor major neurotransmitters.2Describe the relationships between neurotransmitter dysfunction and neuropathology.2Diagram the adult ventricular system and relate it to its embryological development.0	.4 .4 .4 .4 .4 .4 .4 .3 .7 .9
С.	Cer 1. 2. 3. 4. 5. 6.	ebral Fluid and Blood Brain Barriers         Identify the meninges and subarachnoid spaces on a diagram.       1         Describe formation and reabsorption of cerebral spinal fluid, including the anatomy and function of the choroid plexi.       1         Describe the normal pressure, volume, and composition of the CSF.       1         Describe how CSF can vary in certain pathological conditions.       1         Describe the endothelial basis of the blood-brain barrier, and predict the consequence of the barrier for the central nervous system distribution of intravenously administered hydrophic and hydrophobic drugs.       1         Differentiate between postsynaptic inhibition and presynaptic inhibition and provide example cach.       2	.3 .0 .0 this ilic .9 nples .6
D.	<ol> <li>3pn</li> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> </ol>	Describe the anatomical location, function, and afferent neurotransmission of muscle spin and Golgi tendon organs. 3 Describe, in sequence, the neuronal activity initiated by striking the patellar tendon with a percussion hammer (the patellar tendon reflex) through contraction of a muscle. 4 Compare and contrast this reflex with the inverse myotactic reflex. 4 Describe the role of the gamma efferent system in the stretch reflex, and explain the significance of alpha-gamma co-activation. 3 Describe the properties of the flexor reflex initiated by touching a hot stove, and identify v pain is sensed, when flexor contraction occurs, and the neuronal connections and role of t crossed extensor reflex. 3 Describe the clinical tests and findings that allow a physician to distinguish between upper lower motor neuron disorders, including the Babinski sign. 3 Describe the anatomy and functions of the major ascending and descending spinal cord tra including any crossing of the midline. 2 Describe the use of dermatones, sensory deficits, and motor deficits to identify local spina lesions, and spinal cord hemisection, including the immediate and long-term consequence spinal cord transection. 3	dle .9 .0 .0 .7 when the .4 r and .7 acts, .9 l cord es of .2
Ε.	Nei	ve Conduction and EMG Studies	
	1. 2. 3.	Describe the procedure used for measuring nerve conduction velocity.2Describe the repetitive nerve stimulation procedure for assessing the integrity of the neuromuscular junction.2Compare and contrast the different EMG findings in neuropathy and myopathy.2	.0 .1 .6

4. Describe the physiological deficit and the effects with myasthenia gravis.

#### F. Autonomic Nervous System

- Compare and contrast the sympathetic and parasympathetic branches of the autonomic nervous system (ANS) based on spinal cord division of origin; length of preganglionic and postganglionic neurons; neurotransmitters and receptors at the ganglionic; and target organ synapse.
   3.3
- 2. Identify the sensory input of the ANS.
- 3. Identify the major central nervous system control centers of the ANS. 2.6
- 4. Describe the functional effects of normal and abnormal ANS activity or lack of activity. 3.4

### G. Brainstem Reflexes

- 1. Describe the function of the cardiovascular baroreceptor and respiratory stretch receptor.
- Identify the stimulus and its receptor, the afferent pathway, the brain stem nuclei involved, the efferent pathway, and the resulting effect for each brain stem reflex.
   3.0
- 3. Compare and contrast the effects of intra-axial and extra-axial brain stem lesions. **1.1**

### H. Cerebrovascular System

- 1. Describe the local factors affecting brain blood flow, and contrast their effectiveness with that of autonomic regulation of cerebral blood flow. **2.9**
- Describe cerebrovascular disorders (stroke, aneurysm, migraine headache) as to primary cause and effect, including how excitotoxic mechanisms can lead to neuronal death following stroke or injury.
   3.1

## I. Somatosensory System

- Define and contrast point localization and two-point discrimination in psychophysical and neurophysiological terms and explain why the threshold for two-point discrimination changes in different areas of the body surface.
- 2. Identify the submodalities of discriminative touch.
- Describe, including function, Pacinian corpuscles, Meissner's corpuscles, Ruffini endings, Merkle cell, A-delta and C free nerve endings, Golgi tendon organ, and muscle spindle.
   3.7
- Describe the functional organization at all levels and submodalities served by the dorsal column-medial lemniscal, and the equivalent components of the trigeminal system.
   3.7
- 5. Differentiate between feed-forward and feedback inhibition within neuronal circuits, and provide physiological examples of each. **1.9**
- Compare and contrast the proprioceptive pathways to the cerebellum with that to the cerebral cortex.
   2.3
- Differentiate the submodalities of nondiscriminative touch, temperature, and nociception based on receptor transduction mechanism; localization within the spinal gray matter; and central termination of the pathways.
   3.0
- 8. Describe the functional organization at all levels and submodalities served by the anterolateral system, and identify the equivalent components of the spinal trigeminal system. **2.0**
- 9. Describe the control of pain perception, including central processing and the role of endorphins.

3.4

3.7

2.9

2.3

	10. 11.	Describe the gating mechanism theory for control of pain transmission, and relate it to of TENS (transcutaneous electrical nerve stimulation) and spinal cord stimulation. Describe pain perception, the basis for central pain syndromes, and their roles in neuropain.	o the use 3.3 opathic 2.4
	12. 13.	Describe the peripheral and central mechanisms of primary hyperalgesia and secondal hyperalgesia, and explain their roles in neuropathic pain.2Describe the mechanism of referred pain of visceral origin.2	.9 .9
J.	Vis	ual System	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> <li>11.</li> <li>12.</li> </ol>	Describe the refraction of light as it passes through the eye to the retina, identifying the components that account for refraction of light at the center of the eye and away from center. Describe the process of accommodation, contrasting the refraction of light by the lense vision and in far vision. Describe the refractive deficits that account for myopia, hyperopia, presbyopia, and astigmatism, and explain their correction by eyeglasses or contact lenses. Describe the electrical responses produced by bipolar cells, horizontal cells, amacrine of ganglion cells, and discuss the function of each. Compare and contrast the transduction process for rods and the three types of cones, the range of spectral sensitivity, as well as the ionic basis of these responses. Describe the neuronal circuitry forming the basis for antagonist center-surround recept of retinal ganglion cells. Describe the receptive field properties of all neuron types in the visual pathway (retina geniculate to visual cortex), and explain how convergence, divergence, and afferent su inhibition affect visual neuron receptive fields. Identify the visual field defects resulting from retinal lesion, optic nerve lesion, optic cell optic tract, lateral geniculate nucleus, optic radiations, and primary visual cortex. Describe the processing of information in the visual cortex, and discuss the consequent lesion in the higher visual association areas. Identify and compare functional properties of scotopic and photopic vision. Explain the basis for the differing light sensitivities of the fovea and optic disk.	ne eye n the 1.4 in near 2.0 1.6 cells, and 1.3 including 1.0 otive fields 1.4 a to lateral irround 1.0 hiasm, 1.6 ortex, hinance. 1.3 ce of a 2.0 1.4 1.6
v	12.	explain the basis for the differing light sensitivities of the rovea and optic disk.	1.0
к.	Sm		1.0
	1. 2. 3. 4.	Describe the olfactory receptors and transduction mechanisms. Describe the olfactory pathways. Describe taste receptors and transduction mechanisms. Describe the taste pathways.	1.6 1.4 1.6 1.1
L.	Au	ditory System	
	1. 2.	Describe the function of the outer ear, middle ear, and inner ear. Outline the mechanical structures over which sound energy is transmitted to auditory	2.7 receptors. 2.7
	3. 4.	Describe the human audibility curve, and explain the changes that occur with aging. Explain the frequency analysis performed by the cochlea on the basis of its physical pro-	<b>0.9</b> operties. <b>1.1</b>

	8.	Identify conductive, central, and sensorineural deafness, and identify the tests used to assess them. <b>1.9</b>
М.	Ve	stibular System
	1.	Describe the structure, normal stimulus, mechanism of transduction at the receptor level, and function of the otolith organs. <b>2.7</b>
	2.	Describe the structure, normal stimulus, mechanism of transduction at the receptor level, and function of the semicircular canals. <b>2.7</b>
	3.	Describe the central connections of the vestibular nerve (the two targets of first order afferents and the four targets of second order afferents), and relate these to the major functions of the vestibular apparatus. <b>2.0</b>
	4.	Describe the neural mechanisms of nystagmus, past pointing, and caloric testing, and relate the direction of the nystagmus to the direction of rotation or which ear (left or right) was irrigated with cold or warm water. <b>1.9</b>
	5. 6.	Identify and describe four clinical signs of vestibular system dysfunction.2.1Describe the different kinds of gaze (voluntary) eye movements and reflex eye movements.1.7
N.	Me	edial and Lateral System Control of Movement
	1.	Describe and identify the components of the motor control systems, including cerebral cortex, basal ganglia, cerebellum, thalamus, brainstem motor nuclei, and spinal cord in relation to each other and the flow of information among these structures and, ultimately, to the alpha and gamma motor neurons. <b>3.1</b>
	2.	Identify on a cross section of the spinal cord the organization of the sensory and motor components of gray matter and the somatotopic arrangement of motor neuron pools. <b>2.9</b>
	3.	Identify the medial and lateral motor systems, their origin, pathway, and termination within the spinal cord and describe their functions in motor control. <b>3.1</b>
0	4. (p	rehellum and Basal Ganalia
0.	1. 2.	Describe the roles of the cerebellum in the regulation of skilled movement. <b>3.6</b> Identify functional divisions of the cerebellum, including the input and output connections of each. <b>3.2</b>
	3.	Differentiate the functions of the divisions of the cerebellum, and explain their integration with lateral and medial motor systems. <b>2.1</b>
	4.	Identify and describe the circuitry of the cerebellar cortex, assigning the functional role of each neuron type, its synaptic effect (excitatory/inhibitory), how this circuit functions as a timing
	5.	Based upon input-output organization, somatotopic organization, and overall function, predict the neurological disturbances that can result from disease or damage in different regions of the cerebellum.
	6.	Compare and contrast the spinal proprioceptive pathways to the cerebellum with those to the cortex. <b>2.3</b>

5. Explain how deformations of the basilar membrane are converted into action potentials in

7. Explain how pitch, loudness, and localization of sounds in space are coded by central auditory

6. Outline the auditory pathways including all central connections.

auditory nerve fibers.

neurons.

1.7 1.4

	7.	Identify and describe the major interconnections between components of the basal gan the motor cortex and the neurotransmitters influencing the flow of information in the se	glia and ystem.
			2.4
	8.	Describe the overall function of the basal ganglia in movement control and initiation in	
		association with medial and lateral motor systems.	3.6
	9.	Describe the signs of rigidity, dyskinesias, akinesia, tremor, chorea, hemiballism, and ath and assign a likely lesion site or chemical system defect for each and appropriately relat	netosis, e these
		to known clinical syndromes.	3.0
	10.	Describe the physiological basis for the rationale for treatment of Parkinsonism with	
		anticholinergic drugs, L-DOPA, or transplantation of catecholamine-producing cells.	2.9
Р.	Cer	rebral Cortex	
	1.	Identify and describe the medial-to-lateral, rostral-to-caudal, and surface-to-white matt organizations of the primary motor cortex and the premotor cortex, and locate the	er
		supplementary motor cortex.	1.7
	2.	Compare and contrast the effects of electrical stimulation of the motor and premotor co	ortex,
		relating these to the control of voluntary movement.	2.3
	3.	Describe the origin, course, and termination of the pyramidal tract.	2.4
	4.	Compare and contrast the consequences of upper motor neuron loss to lower motor ne	uron
		loss, and describe the consequences of pyramidal tract transection.	3.6
	5.	Develop, describe, and interpret a flow diagram for the brain regions involved in planning	ıg,
		initiating, and properly executing a skilled voluntary movement.	2.4
	6.	Identify Brodmann areas for visual, auditory, somatic sensory, motor, and speech areas.	3.0
	7.	Identify the cortical areas that receive projections from the ventral lateral, dorsomedial,	,
		pulvinar, medial geniculate, lateral geniculate, ventral posterolateral, and posteromedia	ıl nuclei.
			0.7
	8.	Discuss the cortical areas important for language.	2.1
	9.	Discuss the cortical areas important for spatial relations.	2.1
	10.	Describe the functions of the prefrontal association cortex.	2.1
	11.	Define and explain the physiological basis of evoked potentials and the electroencephal	ogram
		(EEG), and identify the main clinical uses of each.	1.9
	12.	Describe the primary types of rhythms that make up the EEG and the corresponding beh	navioral
		states.	1.9
	13.	Describe the origin of spontaneous electrical activity of the cerebral cortex.	1.6
	14.	Distinguish EEG activity from evoked potentials, and identify the uses of evoked potential	als.
			1.9
Q.	Sle	ep	

1. Describe the behavioral, EEG, and other characteristics of the stages of slow-wave sleep and rapid-eye-movement (REM) sleep and explain the changes in sleep stages associated with aging, drugs, and sleep deprivation. 2.6 2. Distinguish slow wave sleep and paradoxical sleep. 2.1 3. Identify and describe the neural systems important for the regulation of sleep-waking. 2.1 4. Identify and describe the neurochemical systems important for sleep and waking. 2.0 5. Describe narcolepsy and sleep apnea, and provide a pathophysiologic basis for each. 2.0 6. Describe the mechanisms important in the production of coma. 2.4 1.6

## R. Seizure Disorders

VIII.

	1. 2.	Identify typical normal and abnormal EEG records. Describe characteristics of generalized and partial seizures.	1.7 2.3	
S.	Нy	pothalamus		
	1. 2. 3.	Describe the structure of the hypothalamus, including the major hypothalamic nuclei an Describe the major functions of the hypothalamus and its nuclei/areas. Explain the role and mechanisms of the hypothalamus as it relates to thirst, hunger, temperature regulation, and the defense mechanism.	ad areas. 1.6 3.3 3.3	
Т.	Lin	nbic System		
	1. 2. 3. 4. 5. 6. 7.	Describe the major components of the limbic system. Describe the major afferent and efferent connections of the hippocampus. Describe the major afferent and efferent connections of the amygdala. Describe reinforcement functions of the limbic system. Describe the functions of the hippocampus. Describe the functions of the amygdala. Describe the role of dopamine in the limbic system in disorders of thought and disorder mood.	1.2 1.1 2.0 2.3 2.1 s of 2.0	
U.	Agi	ing of the Brain		
	1. 2. 3.	Describe the gross, histological, and biochemical changes that occur in the brain through Define <i>dementia</i> . Describe the characteristics of Alzheimer's disease.	n aging. 1.6 2.6 2.7	
V.	Ме	emory and Lateralization		
	1. 2. 3.	Identify the structural elements of the brain that appear to be involved in memory in ma and explain the proposed role of each in memory processing and storage. Describe the mechanisms proposed for short-term and long-term memory storage. Describe the major differences in hemispheric function in humans.	ammals, 1.7 1.4 2.9	
•	<u>Pul</u>	Imonary Physiology		
A.	Pulmonary Mechanics			
	4	• A state of a set of a set of a state of		

- Explain how pleural pressure, alveolar pressure, airflow, and lung volume change during a normal quiet breathing cycle.
   3.7
- Identify the onset of inspiration, cessation of inspiration, and cessation of expiration on diagram of pleural pressure, alveolar pressure, airflow, and lung volume during a normal quiet breathing cycle.
   3.7
- Explain how differences in pressure between the atmosphere and alveoli cause air to move in and out of the lungs.
   3.7
- 4. Describe and interpret a normal pulmonary pressure-volume (compliance) curve. **2.4**
- Define *compliance* and identify two common clinical conditions in which lung compliance is higher or lower than normal.
   3.3

	6.	Describe and interpret the pressure-volume (compliance) curves for the lungs, chest wa	ll, and
		respiratory system on the same set of axes.	3.3
	7.	Show and explain the significance of the resting positions for each of these three structu	ires.
	8.	Identify the forces that generate the negative intrapleural pressure when the lung is at	2.5
		functional residual capacity, and predict the direction that the lung and chest wall will m	ove if
		air is introduced into the pleural cavity (pneumothorax).	3.6
	9.	Describe and interpret a normal spirogram, identifying the four lung volumes and four	
		capacities.	3.9
	10.	List the volumes that comprise the four lung capacities.	3.9
	11.	Identify which lung volumes and capacities cannot be measured by spirometry.	3.9
	12.	Describe how changes in lung volumes occur in patients with emphysema and pulmonal	γ
		fibrosis.	3.1
	13.	Define surface tension.	3.3
	14.	Apply surface tensions to lung mechanics, including the effects of alveolar size and the r	ole of
		surfactants.	3.3
	15.	Define <i>atalectasis</i> , and explain the role of surfactants in preventing it.	3.3
	16.	Describe the principal components of pulmonary surfactant and explain the roles of eac	h.
	17	Describe the offects of simulay dispector and type lost flow on simulay resistones	1.1
	10	Describe the effects of airway diameter and turbulent flow on airway resistance.	2.7
	10.	Describe now already resistance alters dynamic lung compliance.	<b>2.3</b>
	15.	forced vital canacity (EVC) timed forced expiratory volumes (EEVs) as well as the maxim	ny the
		expiratory flow rate between 25-75% of EVC (EEE25-75%)	3.0
	20.	Describe and interpret a normal maximal effort flow-volume curve and identify the effort	rt-
	_0.	dependent and -independent regions.	1.5
	21.	Explain why each point in the effort-independent region of the curve represents a maxir	nal flow
		rate that is uniquely dependent on lung volume, based upon the concept of dynamic	
		compression or airways.	1.5
	22.	Discuss how and why the shape of the flow-volume curve is shifted in chronic obstructiv	e lung
		disease (COPD).	1.5
	23.	23. Differentiate between the two broad categories of restrictive and obstructive lung d	
		including the spirometric abnormalities associated with each category.	3.0
	24.	Describe the regional differences in alveolar ventilation in healthy and diseased lungs, a	nd
		explain the basis for these differences.	2.1
В.	Alv	veolar Ventilation	
	1.	Define:	
		a. hypoventilation	4.0
		b. hyperventilation	4.0
		c. hypercapnea	4.0
		a. eupnea	4.0
		e. nypopnea	4.0
	r	1. IIYperphea Define partial pressure and fractional concentration as they apply to gases in air	4.U 2.C
	۷. ۲	List the normal fractional concentrations and sea level partial procedures for Q _ CO_ and	<b>3.0</b> N
	٦.	List the normal fractional concentrations and sea level partial pressures for $O_2$ , $CO_2$ , and	<b>3.6</b>

	4.	Identify the normal airway, alveolar, arterial, and mixed venous $PO_2$ and $PCO_2$ values, as	$PCO_2$ values, as well as			
	_	the normal arterial and mixed venous values for $O_2$ saturation, [HCO <sub>3</sub> ], and pH.	3.6			
	5.	Differentiate between anatomic dead space, physiologic dead space, wasted (dead space	e)			
	6	Ventilation, total minute ventilation, and alveolar minute ventilation.	3.0 1 2			
	0. 7	Differentiate the relationships between alveolar ventilation and the arterial PCO <sub>2</sub> and PC	1.3 0,			
			3.7			
	8.	Describe in quantitative terms the effect of ventilation on PCO <sub>2</sub> according to the alveola	r			
		ventilation equation.	2.3			
	9.	Estimate the alveolar oxygen partial pressure (PAO <sub>2</sub> ) using the simplified form of the alv	eolar			
		gas equation and describe the relationship between deadspace and alveolar $PO_2$ .	2.9			
С.	Pulmonary Circulation					
	1.	Compare and contrast the systemic and pulmonary circulations with respect to pressure	es,			
		resistance to blood flow, and response to hypoxia.	3.4			
	2.	Describe the regional differences in pulmonary blood flow in an upright person.				
	3.	Identify and describe zones I, II, and III in the lung, with respect to pulmonary vascular pressure				
		and alveolar pressure.	1.9			
	4.	Explain now pulmonary vascular resistance changes with alterations in cardiac output o	r วา			
	5	Explain changes in nulmonary vascular resistance in terms of distension and recruitmen	z.i			
	5.	pulmonary vessels.	<b>2.1</b>			
	6.	Identify the zones in which distension and recruitment of pulmonary vessels apply.	2.1			
	7.	Explain how pulmonary vascular resistance changes with lung volume, as well as in term	ns of			
		alterations in alveolar and extra-alveolar blood vessels.	1.9			
	8.	Describe the consequence of hypoxic pulmonary vasoconstriction on the distribution of				
		pulmonary blood flow.	3.4			
	9.	Discuss the effects of inspired nitric oxide on pulmonary vascular resistance and hypoxic				
	10	Vasoconstruction.	<b>1.9</b>			
	10.	10. Explain the development of pulmonary edema by increased nydrostatic pressure, increased nydrostatic pressure, and				
		hemodilution (eg. with saline volume resuscitation).	3.4			
	11.	Describe the major functions of the bronchial circulation.	2.0			
~	<b>D</b>					
D.	Pulmonury Gus Exchange					
	1.	Identify the factors that affect diffusive transport of a gas between alveolar gas and pull	monary			
	2	Capillary blood.	3./ nillary			
	۷.	reserve time (ie, the nortion of the erythrocyte transit time in which no further diffusion	nof			
		oxygen occurs).	2.6			
	3.	Define <i>oxygen diffusing capacity</i> .	3.4			
Ε.	Ventilation Perfusion Relationship					
	1 Describe how the ventilation (perfusion ( $V/O$ ) ratio of an alveolar-capillary lung unit determines					
	<b>-</b> .	the PO <sub>2</sub> and PCO <sub>2</sub> of the blood emerging from that lung unit.	<b>2.9</b>			
	2.	Identify the average V/Q ratio in a normal lung.	2.7			
	3.	Explain how V/Q is affected by the vertical distribution of ventilation and perfusion in the	ie			
		healthy lung.	2.7			

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	4.	Describe the normal relative differences from the apex to the base of the lung in alveolar arterial $PO_2$ , $PCO_2$ , pH, and oxygen and carbon dioxide exchange.	ar and <b>1.9</b>				
	5.	Explain how the presence of abnormally low and high V/Q ratios in a person's lungs will arterial $PQ_2$ and $PCQ_2$ .	affect 2.7				
	6.	Describe two causes of abnormal V/Q distribution.	2.7				
F.	Ga	s Transport					
	1	1 Define right-to-left shunts, anatomic and physiological shunts, and physiologic dead space					
	1.	(wasted ventilation).	3.0				
	2.	Describe the consequences of right-to-left shunts, anatomic and physiological shunts, a	nd				
		physiologic dead space for pulmonary gas exchange.	3.0				
	3.	Describe the airway and vascular control mechanisms that help maintain a normal					
		ventilation/perfusion ratio.	2.4				
	4.	Identify two compensatory reflexes for V/Q inequality.	2.4				
	5.	Calculate the alveolar to arterial $PO_2$ difference, (A-a) $DO_2$ .	2.4				
	6.	Describe the normal value for (A-a) $DO_2$ and the significance of an elevated (A-a) $DO_2$ .	2.7				
	7.	Identify five causes of hypoxemia.	3.1				
G.	Oxy	ygen and Carbon Dioxide Transport					
	1.	Define oxygen partial pressure (tension), oxygen content, and percent hemoglobin satur	ration as				
		they pertain to blood.	4.0				
	2.	Outline the information that can be received from the following tests/devices:					
		a. spirometer	4.0				
		b. arterial blood gas (ABG) analysis	4.0				
		c. pulse oximeter	4.0				
		d. transcutaneous oximeter	4.0				
	•	e. hyperspectral imaging	4.0				
	3. Describe and interpret an oxyhemoglobin dissociation curve (hemoglobin oxygen e						
		blood owgen content	2 c				
	Λ	Draw the relationship between PO, and dissolved plasma $\Omega_{\rm c}$ content (Henry's Law)	3.0				
	ч. 5	Compare the relative amounts of $\Omega_2$ carried bound to be moglobin with that carried in t	be				
	5.	dissolved form.	3.6				
	6.	Describe how the shape of the oxyhemoglobin dissociation curve influences the uptake	and				
		delivery of oxygen.	3.3				
	7.	Define <i>P50</i> and describe its physiological significance.	2.3				
	8.	Describe how the oxyhemoglobin dissociation curve is affected by changes in blood					
		temperature, pH, PCO <sub>2</sub> , and 2, 3-DPG, and describe a situation where such changes hav	e				
		important physiological consequences.	4.0				
	9.	Describe how anemia and carbon monoxide poisoning affect the shape of the oxyhemo	globin				
		dissociation curve, $PaO_2$ , and $SaO_2$ .	3.0				
	10.	identify the forms in which carbon dioxide is carried in the blood, as well as the percent	age of				
	11	total $U_2$ transported as each form.	3.9 2.6				
	11. 17	Explain the importance of the childred shift in the transport of $CO_2$ by the blood.	<b>2.0</b>				
	12.	location	<b>3</b> 7				
		iocation.	J./				

- Describe and interpret the carbon dioxide dissociation curves for oxy- and deoxyhemoglobin, and explain the interplay between CO<sub>2</sub> and O<sub>2</sub> binding on hemoglobin that causes the Haldane effect.
   2.7
- 14. Explain why the total gas pressure of the venous blood is subatmospheric and why this situation is accentuated when breathing  $100\% O_2$ . **2.9**
- 15. Explain how breathing 100%  $O_2$  can result in further arterial  $O_2$  desaturation in hypoxemic patients who develop mucous plugging of their airways (absorption atelectasis). **2.9**
- 16. Define respiratory acidosis and alkalosis.
- 17. Identify clinical examples of respiratory acidosis and alkalosis.
- 18. Describe the mechanism and function of respiratory acid base compensations. **4.0**

## H. Respiratory Control

- Identify the regions in the central nervous system that play important roles in the generation and control of cyclic breathing.
   3.4
- Identify examples of reflexes involving pulmonary receptors that influence breathing frequency and tidal volume, including the receptors and neural pathways involved.
   2.7
- Identify the anatomical locations of chemoreceptors sensitive to changes in arterial PO<sub>2</sub>, PCO<sub>2</sub>, and pH that participate in the control of ventilation and the relative importance of each in sensing alterations in blood gases.
   3.3
- Describe how changes in arterial PO<sub>2</sub> and PCO<sub>2</sub> alter alveolar ventilation, including the synergistic effects when PO<sub>2</sub> and PCO<sub>2</sub> both change.
   3.7
- Describe the respiratory drive in a COPD patient, and predict the change in respiratory drive when oxygen is given to a COPD patient.
   3.3

## I. Environmental Influences

- Describe the mechanisms for the shift in alveolar ventilation that occur immediately upon ascent to high altitude, after remaining at altitude for two weeks, and immediately upon return to sea level.
   2.4
- 2. Describe the physiological basis of shallow water blackout during a breath-hold dive. **0.7**
- 3. Describe the significance of the feed-forward control of ventilation (central command) during exercise, and the effects of exercise on arterial and mixed venous PCO<sub>2</sub>, PO<sub>2</sub>, and pH. **2.0**

# J. Age Effects and Nonrespiratory Lung Functions

- Describe the effect of aging on lung volumes, lung and chest wall compliance, blood gases, and respiratory control.
   2.3
- 2. Identify the mechanism by which particles are cleared from the airways. **3.0**
- Describe mechanisms for clearance of vasoactive substances from the blood during passage through the lung.
   2.0
- 4. Identify a substance that is almost completely cleared from the blood during passage through the lung, as well as one that is not cleared to any significant extent. **2.0**

# IX. <u>Renal Physiology</u>

A. Body Fluids

4.0

- Identify the normal extracellular and interstitial fluid (plasma) osmolarity and concentrations of Na<sup>+</sup>, K<sup>+</sup>, Cl<sup>-</sup>, HCO<sub>3</sub><sup>-</sup>, proteins, creatinine, and urea; and contrast these values with those for intracellular fluids.
   3.1
- Estimate the total body water, lean body mass, extracellular fluid volume, interstitial fluid volume, intracellular fluid volume, blood volume, and plasma volume, given the body weight and percentage of body fat in an individual.
   3.1
- Compare and contrast the movement between intracellular and extracellular compartments, including the interstitial compartment, caused by increases or decreases in extracellular fluid osmolality.
   3.4
- Given the composition and osmolality of a fluid, identify it as hypertonic, isotonic, or hypotonic and predict the change in transcellular fluid exchange that would be caused by placing a red blood cell in solutions with varying tonicities.
   3.9
- 5. Identify major routes and normal ranges for water intake and loss, and predict how changes in intake and loss affect the distribution of total body water. **3.6**
- Describe and demonstrate the use of the indicator dilution principle to measure plasma volume, blood volume, extracellular fluid volume, and total body water; and identify compounds used to measure each volume.
   2.1
- Predict the general change in extracellular volume, extracellular osmolality, intracellular volume, and intracellular osmolality caused by infusion of three liters of 0.9% NaCl, lactated Ringer's solution, 0.45% NaCl, and 7.5% NaCl.
   3.1
- Identify the site of erythropoietin production, the adequate stimulus for erythropoietin release, and the target tissue for erythropoietin action.
   3.1

## B. Structure of Kidney, Nephron and Bladder

- Identify the renal cortex, renal medulla, renal calycies, medullary pyramids, renal pelvic space, renal artery, renal vein, and ureter, given a cross section of a kidney.
   3.2
- Outline the tubular segments through which ultrafiltrate flows after it is formed at Bowman's capsule to when it enters the renal pelvis, and identify each structure as being located in the renal cortex or renal medulla.
   2.8
- Distinguish between cortical and juxtamedullary nephrons, based on the glomerulus location and the length of the loop of Henle.
   2.8
- 4. Outline the blood vessels through which blood flows from the renal artery to the renal vein.
- Identify and describe the afferent and efferent arterioles, glomerular capillary network, mesangium, Bowman's capsule, and the juxtaglomerular apparatus (including the specialized juxtaglomerular arteriole cells and the macula densa); and describe the three layers comprising the glomerular filtration barrier.
   2.8

# C. Micturition

- 1. Explain the role of somatic, (pudendal) sympathetic, and parasympathetic nerves in the micturition reflex and in urination.
- Explain the roles of spinal cord reflex centers, micturation center in brain stem, and cortical and subcortical centers in micturation.
   2.3
- 3. Explain the role of detrussor muscle, internal renal sphincter, and external urethral sphincter in micturation. 2.3
- D. Renal Clearance

3.0

- 1. Explain the clearance principle and use the clearance equation and an appropriate compound to estimate the glomerular filtration rate, renal plasma flow, and renal blood flow. **3.9**
- Differentiate between the use of inulin and creatinine clearances as measures of the glomerular filtration rate.
   3.0
- Calculate the filtered load, tubular transport, excretion rate, and clearance of inulin, creatinine, para-amino hippuric acid (PAH), glucose, and penicillin, given the plasma and urine concentrations and the urine flow rate.
   3.0
- 4. Predict how changes in filtration, reabsorption, and secretion will affect renal excretion of inulin, creatinine, para-amino hippuric acid (PAH), glucose, and penicillin. **3.0**
- Identify the tubular load, tubular transport maximum (T<sub>max</sub>), and splay for each substance, using a graph of the urinary excretion of glucose, creatinine, PAH, penicillin and inulin.
   1.9

### E. Glomerular Filtration Rate and Renal Hemodynamics

- Identify the filtration barriers that impede the filtration of H<sub>2</sub>O, Na<sup>+</sup>, inulin, albumin, and red blood cells.
   Define *renal blood flow, renal plasma flow, glomerular filtration rate,* and *filtration fraction,* and list typical values for each.
   3.8
- Identify the filtration coefficient at the glomerular capillary, describe the membrane properties that contribute to it, and explain its role in determining GFR.
   2.5
- Calculate the net filtration force at the glomerular capillaries, given the capillary and Bowman's capsule hydrostatic and oncotic pressures.
   **3.0**
- 5. Predict the changes in glomerular filtration caused by increases or decreases in the capillary and Bowman's capsule hydrostatic and oncotic pressures. **3.0**
- Describe the relative resistances of the afferent and efferent arterioles and the effects on renal blood flow and GFR of selective changes in each.
   3.1
- Describe the myogenic and tubuloglomerular feedback mechanisms that mediate the autoregulation of renal plasma flow and glomerular filtration rate.
   3.2
- Predict the change in renal blood flow and glomerular filtration rate caused by an increase in renal sympathetic nerve activity.
   3.1
- Predict the change in renal blood flow and glomerular filtration caused by increased synthesis of angiotensin II, increased release of atrial natriuretic peptide, increased prostaglandin formation, and increased nitric oxide formation.
   2.8
- 10. Identify components of the filtration barrier whose dysfunction would result in hematuria and proteinuria. **2.5**
- Predict the changes in net filtration force that occur as blood travels along the glomerular capillary where hydrostatic pressure falls and colloid osmotic pressure increases, based on Starling's forces.
   2.3
- Predict the change in renal blood flow and GFR caused by urinary tract obstruction, hypoalbuminemia, and diabetic nephropathy.
   3.1
- 13. Compare blood flow to, and oxygen consumption by, the kidneys with that of resting skeletal and cardiac muscle.2.4
- 14. Describe the effects of changes in peritubular capillary hydrostatic and colloid osmotic pressures on net proximal tubular fluid reabsorption.3.0
- 15. Contrast the transcellular and paracellular pathways for movement across proximal tubular epithelia, using glucose, para-amino hippuric acid (PAH), water, and Cl<sup>-</sup>. **2.3**
- F. Transport Properties of Nephron Segments
- Differentiate between active (primary and secondary) transport, facilitated diffusion, and passive diffusion based on energy source and carrier protein involvement.
   3.4
- Describe the contribution of the major nephron segments to the reabsorption of the filtered load of solute and water.
   3.8
- Describe the cellular mechanisms for the transport of Na<sup>+</sup>, Cl<sup>-</sup>, K<sup>+</sup>, HCO<sub>3</sub><sup>-</sup>, Ca<sup>2+</sup>, phosphate, organic solutes (eg, glucose, amino acids, and urea), and water by the major tubular segments.
   3.0
- 4. Describe the function of the following renal transporters and their predominant localization along the tubules with regard to nephron segment and apical versus basolateral membranes
  - a. transport ATPases (Na<sup>+</sup>/K<sup>+</sup>-ATPase, H<sup>+</sup>/K<sup>+</sup>-ATPase, H<sup>+</sup>-ATPase, and Ca<sup>2+</sup>-ATPase) **3.0**
  - b. ion and water channels ( $K^{+}$ , ENaC, Cl<sup>\*</sup>, Ca<sup>2+</sup>, aquaporins)
  - c. coupled transporters (Na<sup>+</sup>-glucose, Na<sup>+</sup>/H<sup>+</sup>-antiporter, Na<sup>+</sup>-K<sup>+</sup>-2Cl<sup>-</sup>-symporter, Na<sup>+</sup>-phosphate symporter, Na<sup>+</sup>-Cl<sup>-</sup>-symporter, Na<sup>+</sup>-HCO<sub>3</sub><sup>-</sup>symporters, Cl<sup>-</sup>/HCO<sub>3</sub><sup>-</sup>-antiporter)
- Describe the nephron sites and molecular mechanisms of action of the following classes of diuretics (osmotic, carbonic anhydrase inhibitors, loop, thiazide, K<sup>+</sup>-sparing).
   **3.0**
- 6. Identify and describe clinical syndromes related to defects in specific renal transporters. **1.3**
- 7. Describe the effects of reductions in GFR on plasma creatinine concentrations. **3.6**

#### G. Urine Concentration and Dilution

- 1. Predict how changes in body fluid volume and osmolality caused by a net water loss or gain in the body would alter the rate of urine production and the osmotic composition of the urine. **3.3**
- Predict how changes in body fluid volume and osmolality caused by a net NaCl loss or gain in the body would alter the rate of urine production and the osmotic composition of the urine.
   3.1
- Identify the two most powerful stimuli promoting ADH release, and describe the negative feedback control mechanisms for each.
   3.9
- Describe the role of the ascending limb of the loop of Henle in producing a high renal interstitial fluid osmolality.
   3.6
- Compare and contrast, beginning with the loop of Henle, the tubular fluid and interstitial fluid osmolality changes that allow either a dilute or a concentrated urine to be produced and excreted.
   3.6
- Predict the consequence on urine concentrating ability if the medullary osmotic gradient is disrupted, and describe how the osmotic gradient would be re-established.
   3.0
- Identify and describe the tubular section and cellular mechanisms by which ADH increases permeability to water and urea, and explain the role of these to produce either dilute or concentrated urine.
   3.6
- Calculate osmolar and free water clearance and estimate expected free water clearance for an individual producing either dilute or concentrated urine, given urine and plasma osmolarities and urine volume.
   1.8
- Describe the actions of the different classes of diuretics on the ability of the kidneys to maximally concentrate and dilute urine.
   2.4
- 10. Differentiate between central and nephrogenic diabetes insipidus based on plasma ADH levels<br/>and the response to an injection of ADH.**3.0**
- H. Na<sup>+</sup> Balance and Regulation of Extracellular Fluid Balance
  - Identify the major routes of Na<sup>+</sup> loss from the body and describe the role of Na<sup>+</sup> in maintaining extracellular fluid volume.
     3.3

3.0

	2.	Calculate the normal filtered load of Na <sup>+</sup> and identify the tubular sites of Na <sup>+</sup> reabsorption alterations in Na <sup>+</sup> reabsorption in conditions of euvolemia, volume depletion, and volume	on, the ne
		expansion.	2.9
	3.	Describe the receptors involved in the monitoring of ECF volume (eg, high-pressure	
		baroreceptors and low-pressure cardiopulmonary stretch receptors), and diagram the n	eural
		reflex regulation of renal Na $^{+}$ and water excretion.	2.9
	4.	Describe and interpret the formation and generation of angiotensin II, beginning with re	ennin
		and identify the factors that can promote renin release.	3.0
	5.	Describe the regulation of $Na^+$ reabsorption along the nephron, including the effects of	
		sympathetic nerves, angiotensin II, aldosterone, and atrial natriuretic peptide.	3.3
	6.	Describe the actions of the different classes of diuretics on Na <sup>+</sup> handling by the kidneys	and ECF
		volume regulation.	2.8
	7.	Explain the contribution of the kidneys to progression of and/or the compensation for t	he
		altered fluid volume regulation characteristic of congestive heart failure and hepatic circ	rhosis.
			2.6
	8.	Describe the regulation of proximal tubule reabsorption that underlies the phenomenon	n of
		glomerulotubular balance.	2.4
	9.	Describe the role of the renin-angiotensin-aldosterone system in the regulation of syste	mic
		arterial blood pressure in volume-replete and volume-depleted states and in secondary	forms of
		hypertension.	3.4
,	$\nu^+$	Delanco	
1.	ĸ	вашисе	
	1.	Identify the major routes of $K^{+}$ loss from the body.	3.3
	2.	Explain the role of extracellular $K^{\dagger}$ in maintaining normal nerve and muscle function. <b>3.3</b>	}
	3.	Describe $K^*$ distribution within the body, extrarenal $K^*$ homeostasis, the role insulin,	
		epinephrine, and aldosterone in the movement of $K^{*}$ between intracellular and extracel	lular
		pools and describe the $K^{\star}$ shift caused by acidosis.	3.3
	4.	Calculate the normal filtered load of $K^+$ .	2.6
	5.	Identify the tubular sites of $K^{\star}$ reabsorption and secretion.	3.0
	6.	Describe the factors that regulate $K^{+}$ secretion in the collecting duct (eg, aldosterone, pl	asma
		$K^{*}$ ), and distinguish these from factors that alter $K^{*}$ secretion at this site (eg, luminal flui	d flow
		rate, acid-base disturbances, anion delivery).	3.3
	7.	Contrast the tubular sites of action of K <sup>+</sup> wasting and K <sup>+</sup> sparing diuretics.	2.6
J.	Ca	$^{+}$ and Phosphate Balance	
	1.	Identify the major storage pools of Ca and phosphate, as well as major routes of Ca <sup>-</sup> a	ind
	2	phosphate loss from the body.	3.1
	2.	Describe the regulation of plasma Ca by calcitonin and phosphate by parathyroid norn	none.
	2		3.1
	3.	Calculate the normal filtered load of Ca <sup>2+</sup> .	2.5
		a. Identify the tubular sites of Ca <sup>+</sup> reabsorption.	2.5
		b. Calculate the normal filtered load of phosphate.	2.5
	-	c. Identify the tubular sites of phosphate reabsorption.	2.5
	4.	Describe the renal regulation of Ca <sup>+</sup> and phosphate transport by PTH, calcitonin, and	
		1,25-dihydroxy vitamin D (calcitriol), and distinguish from other factors that alter their t	ransport
	_	(ECF volume, acid-base disorders).	3.4
	5.	Describe the role of the kidney in the production of 1,25-dihydroxy vitamin D (calcitriol)	

- Describe the effects of diuretics on Ca<sup>2+</sup> and phosphate excretion, especially noting the effect of thiazides to decrease Ca<sup>2+</sup> excretion and loop diuretics to increase Ca<sup>2+</sup> excretion.
   2.1
- K. Acid-Base Balance
  - 1. Identify the normal range of pH values, and the upper and lower limits compatible with life.
  - 2. Describe the role of buffers in maintaining pH, including the roles of the lungs and kidneys.
  - Describe the respiratory and renal regulation of the CO<sub>2</sub>/HCO<sub>3</sub><sup>-</sup> buffer system, which allows a buffer with a pK<sub>a</sub> of 6.1 to be physiologically important in the maintenance of the normal plasma pH of 7.4.
     3.9
  - Differentiate between CO<sub>2</sub>-derived (volatile acid) and nonvolatile acid, the relative amounts produced each day through dietary intake and cellular metabolism, and the normal routes of loss from the body.
     2.8
  - Calculate the filtered load of HCO<sub>3</sub><sup>-</sup>, and identify the major sites of reabsorption (and secretion) along the nephron, emphasizing the importance of H<sup>+</sup> secretory mechanisms in this process.
  - 6. Describe the cellular mechanisms responsible for net transepithelial movement of  $HCO_3^{-1}$ .
  - Describe the adjustments in filtered load and HCO<sub>3</sub><sup>-</sup> reabsorption (H<sup>+</sup> secretion) by alterations in systemic acid-base balance and distinguish these from factors that alter this process (eg, ECF volume, aldosterone, and angiotensin II).
     2.9
  - Describe net acid excretion by the kidneys, titratable acid, the importance of urinary buffers, and the production and excretion of ammonia.
     3.1
  - Differentiate between the reclamation of filtered bicarbonate and the formation of new bicarbonate.
     3.1
  - Identify the magnitude and the time course of the compensations that act to minimize change in pH of the body fluids, including buffers, respiratory adjustments, and renal adjustment, given a sudden increase or decrease in pH.
     3.4
  - Identify simple and mixed metabolic and respiratory acid-base disturbances and distinguish between increased and normal anion gap metabolic acidosis, chloride-sensitive and chlorideresistant metabolic alkalosis, and acute and chronic respiratory disturbances, based upon blood values.
     3.1
  - 12. Explain processes that lead to acid-base disturbances and list common causes of these processes.
  - 13. Describe the effects of carbonic anhydrase inhibitors and other classes of diuretics on acid-base<br/>balance and the reabsorption of  $HCO_3^-$  by the nephron.**3.0**
- L. Integrative and Pathophysiological Aspects
  - Describe the relationships between sodium balance and plasma volume as they contribute to cardiovascular hemodynamics and arterial pressure.
     3.9
  - Explain the role of the renin-angiotensin-aldosterone systems in the regulation of sodium balance and arterial pressure, with emphasis on the actions of angiotensin II on various target organs and tissues.
     3.9
  - 3. Describe pressure natriuresis and the mechanisms mediating and modulating this process.

3.1

3.5

3.6

3.6

3.3

	<ol> <li>Describe how impairments in renal function and pressure natriuresis contribute to the term regulation of arterial pressure, as well as the development and maintenance of hypertension.</li> </ol>		ong- <b>3.1</b>
М.	Uri	ine and Metabolite Elimination	
	1. 2. 3.	Describe the renal handling of uric acid (urate). Explain how renally-acting drugs affect hyperuricemia. Describe the metabolic sources and elimination of ammonia, uric acid, and creatinine.	4.0 3.0 3.5

# **MICROBIOLOGY/IMMUNOLOGY**

# **LEARNING OBJECTIVES**

Antimicrobial Agents and Control of Microbes Basic Bacteriology Basic Concepts in Immunology Basic Mycology and Parasitology Basic Virology Cardiac Infections Clinical Immunology Genitourinary Infections and STDs Gastrointestinal Infections Infectious Pathogenesis Skin, Soft Tissue, and Bone Infections Nervous System Infections Respiratory Tract Infections Zoonotic and Opportunistic Infections

# I. Antimicrobial Agents and Control of Microbes

1.	Define:		
	a.	Antiseptic	4.0
	b.	aseptic	4.0
	с.	bactericidal	4.0
	d.	bacteriostatic	4.0
	e.	disinfectant	4.0
	f.	germicide	4.0
	g.	sepsis	4.0
	h.	sterilization	4.0
2.	Describe	the general effects chemical and physical agents have on membranes, proteins	s, and
	nucleic a	cids that are Lethal to cells.	3.5
3.	Describe	the differential effect that dry heat and moist heat have on cells.	3.0
4.	Compare	e and contrast using boiling versus autoclaving to control microbial growth.	3.0
5.	Identify	when filtration is most appropriate for sterilization.	3.0
6.	Describe	the effects of ionizing and nonionizing radiation on microbes.	3.0
7.	Describe	thymine dimer formation and its effect on cell function.	2.0
8.	Different	iate between, and provide examples of, cationic and anionic detergents.	2.0
9.	Identify 1	the effects of surfactants on bacteria.	2.0
10.	Identify 1	the mechanism of action and uses of the following in controlling microbial grow	/th:
	a.	quaternary ammonium compounds	3.0
	b.	phenol and derivatives of phenol	3.0
	с.	alcohols	3.0
	d.	halogens	3.0
	e.	iodine	3.0
	f.	tincture of iodine	3.0
	g.	iodophor	3.0
	h.	chlorine and its various forms	3.0
	i.	hydrogen peroxide	3.0
	j.	metals	3.0
	k.	alkylating agents	3.0
	Ι.	formaldehyde	3.0
	m.	glutaraldehyde	3.0
	n.	ethylene oxide	3.0
	0.	beta-propiolactone	3.0
	р.	mineral acids	3.0
	q.	organic acids	3.0
	r.	alkalis	3.0
11.	Identify 1	the basis on which antimicrobials are selected for patients.	3.0
12.	Describe	important side effects of antimicrobial agents and describe how each would be	2
	recogniz	ed in a patient.	4.0
13.	Identify 1	the purpose of antibiotic susceptibility testing.	4.0
14.	Describe	basic procedures used to perform antimicrobial susceptibility testing and to in	terpret the
	test resu	Its of:	
	a.	broth dilution	3.5
	b.	agar plate dilution	3.5
	с.	agar disk diffusion (Kirby-Bauer)	3.5

d. gradient diffusion (E-test)	3.5
e. colorimetric (chromogenic)	3.5
Identify the pros and cons of each method of susceptibility testing:	
a. broth dilution	3.5
b. agar plate dilution	3.5
c. agar disk diffusion (Kirby-Bauer)	3.5
d. gradient diffusion (E-test)	3.5
e. colorimetric (chromogenic) 3.5	
Define <i>MIC</i> and <i>MBC</i> .	4.0
Define broad-spectrum, narrow-spectrum, and expanded-spectrum as they apply to	antimicrobial
agents.	3.0
Describe major classes of antimicrobial agents based on their mechanisms of action.	4.0
Define and provide examples of bactericidal and bacteriostatic drugs.	4.0
Characterize cell wall and membrane active agents as bactericidal.	3.0
Identify the primary mode of action, mechanisms of bacterial resistance, spectrum c	of activity, and
any unique characteristics of the following antimicrobial agents, and list examples w	hen applicable
a. Sulfonamides	4.0
b. Trimethoprim	4.0
c. Dapsone	4.0
d. Daptomycin	4.0
e. Isoniazid	4.0
f. Ethambutol	4.0
g. Pyrazinamide	4.0
h. beta-lactams	4.0
I. cephalosporins	4.0
J. Imipenem	4.0
k. aztreonam	4.0
I. Vancomycin	4.0
m. pacitracin	4.0
n. polymyxin	4.0
o. quinoiones	4.0
p. mampin a prinoglycosidos	4.0
q. annogiyeosides	4.0
r. chloramphonicol	4.0
t macrolides	4.0
	4.0
v strentogramins and oxazolidinones	4.0
w metronidazole	4.0
22 Identify the essential features of a beta-lactam antibiotic	3.0
23. Explain how beta-lactamase works.	4.0
24. Explain the usage of the sulfamethoxazole-trimethoprim combination	3.0
25. Identify the most significant adverse effects associated with the use of chloramph	enicol. <b>3.0</b>
26. Explain what clavulanic acid, tazobactam, and sulbactam have in common and wh	at they are
used for in clinical medicine.	4.0
27. Explain why some antimicrobial agents are most effective against rapidly growing	cells while
other agents are active against both rapidly growing and resting cells.	3.0
	<ul> <li>d. gradient diffusion (E-test)</li> <li>e. colorimetric (chromogenic)</li> <li>Identify the pros and cons of each method of susceptibility testing: <ul> <li>a. broth dilution</li> <li>b. agar plate dilution</li> <li>c. agar disk diffusion (Kirby-Bauer)</li> <li>d. gradient diffusion (E-test)</li> <li>e. colorimetric (chromogenic)</li> </ul> </li> <li>3.5 Define <i>MIC</i> and <i>MBC</i>.</li> <li>Define broad-spectrum, narrow-spectrum, and expanded-spectrum as they apply to agents.</li> <li>Describe major classes of antimicrobial agents based on their mechanisms of action.</li> <li>Define and provide examples of bactericidal and bacteriostatic drugs.</li> <li>Characterize cell wall and membrane active agents as bactericidal.</li> <li>Identify the primary mode of action, mechanisms of bacterial resistance, spectrum of any unique characteristics of the following antimicrobial agents, and list examples were a. Sulfonamides</li> <li>b. Trimethoprim</li> <li>c. Dapsone</li> <li>d. Daptomycin</li> <li>e. Isoniazid</li> <li>f. Ethambutol</li> <li>g. Pyrazinamide</li> <li>h. beta-lactams</li> <li>i. cephalosporins</li> <li>j. imipenem</li> <li>k. aztreonam</li> <li>l. vancomycin</li> <li>m. bacitracin</li> <li>n. polymyxin</li> <li>quinolones</li> <li>p. rifampin</li> <li>q. aminoglycosides</li> <li>r. tetracyclines</li> <li>s. chloramphenicol</li> <li>t. macrolides</li> <li>u. clindamycin</li> <li>v. streptogramins and oxazolidinones</li> <li>w. metronidazole</li> </ul> 22. Identify the essential features of a beta-lactam antibiotic. 23. Suplain how beta-lactamase works. 24. Identify the most significant adverse effects associated with the use of chloramphe 26. Explain what clavulanic acid, tazobactam, and sulbactam have in common and who used for in clinical medicine. 27. Explain what clavulanic acid, tazobactam, and sulbactam have in common and who used for in clinical medicine. 28. Explain what clavulanic acid, tazobactam, and sulbactam have in common and who used for in clinica

28. Explain the mechanisms of the following inherent resistances to antimicrobial a mycoplasma resistance to cell wall active antibiotics, anaerobe resistance to ar	agents: ninoglycosides.
aerobic resistance to metronidazole, and gram-negative resistance to vancomy	cin. <b>3.0</b>
29 Describe the mechanism of action clinical use and provide examples of azole	s and fungins
	3 0
30 Describe the mechanism of action and clinical use of terbinafine flucytosine a	riseofulvin
tolnaftate and notassium indide	3 0
31 Identify the targets of attack for current antifungal agents	4.0
22 Describe the mechanism of action and clinical use of acyclovir, famciclovir, val	acyclovir
sz. Describe the mechanism of action and chinical use of acyclovit, familiciovit, val	acyciovii, mivir zanamivir
riboviria adefovir diaivovil enterovir and interferen alaba	<b>1</b> 1111VII, 2d11d1111VII,
ribavirin, aderovir dipivoxii, entecavir, and interferon-alpha.	<b>3.</b> 0
33. Describe the mechanism of action and clinical use of HAART therapies, nucleos	ide reverse
transcriptase inhibitors, non-nucleoside reverse transcriptase inhibitors, protea	ase inhibitors,
fusion inhibitors, integrase inhibitors and maturation inhibitors.	4.0
34. Identify the steps in the process of viral pathogenesis that are targets of antivir	al agents. <b>3.5</b>
35. Identify a group of natural antiviral compounds.	1.0
36. Describe the clinical uses and antiviral effects of the Type I and Type II interfere	ons. <b>3.0</b>
37. Explain the limitation in choice of antiparasitic drugs.	2.0
38. Explain the mode of action of antibacterial agents in parasitic diseases.	1.0
39. Explain the origin of the term "vaccination".	1.0
40. Describe different types of vaccines and differentiate their levels of effectivene	ess. <b>4.0</b>
41. Identify two inactivated viral vaccines currently in use.	3.0
42. Identify the live attenuated viral vaccines in current use.	3.0
43. Outline advantages and limitations of inactivated versus attenuated vaccines.	3.0
44. Compare and contrast different types of vaccines, including advantage and disa	advantages of
each.	3.0

#### II. Basic Bacteriology

1.	Compare and contrast prokaryotic and eukaryotic cells, particularly with respect to cell w	vall
	structure, nuclear membranes, DNA structure, plasmids, and ribosomes.	4.0
2.	Describe the morphology and arrangements of bacterial cells.	3.5
3.	Explain the use and significance of both Gram and Acid-fast stains and describe how eac	h
	staining procedure works.	3.5
4.	Describe the structure, arrangements, and functions of prokaryotic flagella.	2.0
5.	Identify the connection between flagellar proteins and H-antigens.	2.0
6.	Describe the structure and functions of pili/fimbriae.	3.0
7.	Explain antigenic variation of pili/other cell surface proteins and identify its clinical signif	icance.
		3.0
8.	Describe the structure of bacterial capsules.	3.0
9.	Describe the role of bacterial capsules in pathogenicity.	4.0
10.	Describe terms used to describe capsules (eg, K antigen, slime layer).	2.0
11.	Describe the quelling reaction.	1.0
12.	Describe the formation and importance of bacterial biofilms.	3.0
13.	Compare and contrast the structure of Gram-positive and Gram-negative cell envelopes.	4.0
14.	Describe the role of peptidoglycan in bacteria.	3.0
15.	Explain the importance of peptidoglycan as a target for antibiotics.	4.0

16.	Define <i>lysozyme</i> and explain where it is found, as well as its biological activity.	3.0
17.	Describe teichoic acids in terms of importance and where they are located.	3.0
18.	Describe the components and functions of the outer membrane of Gram-negative bacter	eria. <b>3.5</b>
19.	Describe porins found in Gram-negative bacterial cell walls and their importance.	3.0
20.	Discuss the structure and biological activities of lipopolysaccharide.	4.0
21.	Describe the bacterial secretion systems, including where they are found and their impo	ortance
	to pathogenicity.	3.0
22.	Explain the uniqueness of mycoplasmas amongst bacteria.	2.0
23.	Describe the structure and functions of cytoplasmic membranes in bacteria.	3.0
24.	Explain the term "penicillin-binding proteins" and give examples of PBPs.	3.0
25.	Explain the function of penicillin-binding proteins in bacteria.	3.0
26.	Identify and describe the major contents of bacterial cytoplasm.	3.0
27.	Describe the structure and functions of endospores.	4.0
28.	Identify the two major genera of clinically-relevant bacteria that produce endospores ar	nd
	describe the similarities and differences between them.	4.0
29.	Explain the principles used to classify bacteria taxonomically.	3.0
30.	Describe the methods used to identify bacteria from a clinical isolate in the clinical labor	ratory.
	·	3.5 <sup>°</sup>
31.	Explain the functions of siderophores and their role in pathogenicity.	2.0
32.	Contextualize the term "fastidious" with respect to bacterial nutrition.	2.0
33.	Classify bacteria based upon oxygen and temperature requirements and list examples o	f each
	classification.	3.0
34.	Explain the importance of proper pH and proper osmotic pressure for microbial growth.	2.0
35.	Explain the importance of growing in a high salt concentration with respect to Staphyloc	coccus
	and Enterococcus.	2.0
36.	Explain the term "generation time" and the factors that affect it.	3.0
37.	Describe the four growth phases of bacteria and explain the importance of each.	3.0
38.	Explain "quorum sensing" and its importance.	3.0
39.	Explain how to obtain a pure culture of bacteria and explain its significance in diagnosis.	3.5
40.	Describe the various microscopic methods used to observe microbial pathogens.	3.0
41.	Differentiate between nonselective, selective, and differential growth media and list con	nmon
	examples of each.	3.0
42.	Define glycolysis, fermentation, aerobic respiration, and anaerobic respiration.	3.0
43.	Explain how metabolic capabilities of bacteria relate to bacterial identification and to	
	pathogenicity.	2.0
44.	Identify the active and passive transport mechanisms used by bacteria.	2.0
45.	Describe the differences between bacterial and eukaryotic transcription and translation	. 3.0
46.	Define mutation, base substitution mutation, frame-shift mutation, genotype, and phene	otype.
		3.5
47.	Describe an operon and its regulation mechanisms.	3.0
48.	Describe DNA repair mechanisms in bacteria.	3.0
49.	Explain transformation as it occurs in bacteria.	3.0
50.	Define transfection, homologous recombination, nonhomologous recombination, donor,	
	recipient, and transformant.	3.0
51.	Describe conjugation as it typically occurs in Gram-negative bacteria when the donor is:	F+, Hfr,
	or F'.	3.0
52.	Define male and female bacteria, F factor, plasmid, sex pilus, Hfr cell, and episome.	3.0
53.	Describe resistance transfer factors and discuss their significance to human medicine.	3.5

54.	. Describe the environmental pressures that favor the development of antibiotic-resistant				
	bacteria	Э. 	3.0		
55.	Describ	e pathogenicity islands.	3.0		
56.	Define	insertion sequence and transposon and explain their importance to virulence and	disease.		
	ا ما م مع : 4	a cleative processing that any load to antihistic resistance	3.5		
57. E0	Describ	a the assential features of hasterial viruses, defining hasterianhage, cancid, and	3.0		
58.	cansom	e the essential realures of bacterial viruses, defining bacteriophage, capsid, and iere.	3.0		
59	Describ	e the lytic and lysogenic cycles as they occur in bacteriophage-infected bacteria.	3.0		
60.	Define	lytic, virulent, and temperate phages and what is meant by "prophage".	3.0		
61.	Define	<i>Insogenic conversion</i> and describe its significance in a clinical environment.	3.0		
62.	Describ	e transduction as it occurs in bacteria, and differentiate between generalized and	d		
	speciali	zed transduction.	3.0		
63.	Explain	Koch's Postulates and its limitations.	3.0		
64.	Explain	how pathogenic microbes can evade the non-specific first-line defenses of the be	ody. <b>3.5</b>		
65.	Describ	e the components of the non-specific second-line defenses of the body and their			
	functio	n as a barrier to disease.	4.0		
66.	Compai	re and contrast true pathogens versus opportunistic pathogens.	4.0		
67.	Differer	ntiate between a toxigenic and an invasive pathogen.	3.0		
68.	Compai	re and contrast exotoxins and endotoxins.	4.0		
69.	Describ	e the source and function of the toxic shock syndrome toxin.	3.5		
70.	Describ	e AB toxin structure and function.	3.0		
71.	1. Explain the attributes of a microbe that contribute to invasiveness.				
72.	2. Explain the role of the body's normal flora in in health and disease.				
73.	. Describe the major normal flora microbes, where they are found, and which are important				
	opportu	unistic pathogens/their disease associations.	3.0		
74.	Describ	e the major mechanisms of transmission of infectious diseases.	4.0		
75.	Define	the following terms:			
	a.	bacteremia	4.0		
	b.	carrier	4.0		
	с.	communicable disease	4.0		
	d.	endemic	4.0		
	e.	endotoxin	4.0		
	f.	enterotoxin	4.0		
	g.	epidemic	4.0		
	h.	exotoxin	4.0		
	i.	fomite	4.0		
	J.	infectious dose	4.0		
	K.	latent infection	4.0		
	Ι.	microbiome	4.0		
	m.	opportunistic pathogen	4.0		
	n. c	punueninc pulliogenicity	4.0		
	0. 2	pyennu	4.0		
	р. ~	pycyclic	4.0		
	q. r	pyrogenic senticemia	4.U / 0		
	۱. د	subclinical infection	4.0		
	5. +	superinfection	<del>.</del> // 0		
	ι.	superingection	4.0		

u. systemic infection	4.0
v. toxoid	4.0
w. virulence	4.0
x. zoonosis	4.0
76. Describe proper specimen collection from various anatomical sites.	3.0

# III. Basic Concepts in Immunology

1.	Explain the function of follicular dendritic cells.	3.0
2.	Differentiate between self-antigen and foreign-antigen.	4.0
3.	Characterize active and passive immunity.	4.0
4.	Compare and contrast innate and adaptive immunity.	4.0
5.	Identify the cells of the innate immune response and describe their general function in	terms of
	recognition of microbes, production of cytokines, and destruction of microbes.	4.0
6.	Identify physical and physiological barriers to infection.	4.0
7.	Describe the role that complement plays in innate immunity.	4.0
8.	Describe the phagocytic barrier to infection.	4.0
9.	Explain the concept of innate pattern recognition of microbes by phagocytic cells.	4.0
10.	Generalize the major components of the inflammatory response.	4.0
11.	Describe the local and systemic effects of the innate immune response as they relate to	o TNF-
	alpha, IL-1, and IL-6.	3.0
12.	Describe the overlap between innate and adaptive immunity.	3.5
13.	Describe the essential characteristics of humoral and cell-mediated immunity.	4.0
14.	List and describe the essential features of the adaptive immune response – specificity,	diversity,
	specialization, self limitation, and memory.	4.0
15.	Explain the essential role of gene families in the evolution of antigen recognition in the	immune
	system.	2.0
16.	Explain the theory of clonal selection.	3.0
17.	Describe the cells involved in adaptive immunity (T cells, B cells, and antigen presenting	g cells).
		4.0
18.	Describe the phases of the adaptive immune response.	3.5
19.	Describe the basic aspects of T and B cell activation and the role of antigen presenting	cells in
	this process.	4.0
20.	Describe the basic effector function of T and B cells in an immune response.	4.0
21.	Generalize the development of white blood cells from stem cells to progenitor cells to	mature
	cells.	3.0
22.	Describe the maturational stages of B and T cells and the cytokine signals that direct	
	differentiation of the cell lines.	3.0
23.	Describe the markers used to distinguish different lineages, subsets, and maturational	stages of
	lymphocytes.	3.0
24.	Describe the functional significance and/or cellular distribution of the following:	
	a. CD 3	3.0
	b. CD 4	3.0
	c. CD 8	3.0
	d. CD 19	3.0
	e. CD 20	3.0
	t. CD 40	3.0

	g. TCR	3.0
	h. BCR	3.0
25.	Describe the characteristics and functions of monocytes and macrophages.	3.5
26.	Describe the characteristics and functions of the granulocytic cells, mast cells, den	dritic cells,
	and natural killer cells.	4.0
27.	Explain the role of the bone marrow in lymphocyte origin.	3.5
28.	Explain the role of the thymus in maturation and selection of T lymphocytes.	3.5
29.	Describe the developmental pathway of T cells in the thymus.	3.0
30.	Describe the functionality of the thymus as the major site of selection and matura	tion of both
	helper T cells and CTLs.	3.5
31.	Explain the processes of positive and negative selection in the thymus.	3.5
32.	Describe the process of programmed cell death (apoptosis) and its role in the thyn	nus. <b>3.5</b>
33.	Describe the overall structure of the TCR (alpha/beta and gamma/delta) and assoc	ciated
	polypeptides.	3.0
34.	Describe the development of TCR and CD4/CD8 expression in maturing T cells.	3.0
35.	Describe the sequence of events in the B cell developmental pathway from stem c	ell to
	immature B cell in the bone marrow.	3.0
36.	Describe the order of rearrangement and expression of immunoglobin heavy and	light chain
~ 7	genes during development of the B cell.	3.0
37.	Describe the structure of the BCR and associated polypeptides.	3.0
38.	Identify the antigen-independent and antigen-dependent phases in B cell ontogen	y. <b>2.0</b>
39.	Explain the induced tolerance of two basic mechanisms: clonal deletion and clonal	anergy (or
40	functional inactivation).	3.5
40.	Describe the major T cell effector populations in the periphery.	4.0
41.	response	nune 20
12	Identify and describe peripheral B cell subpopulations)	3.0
42. 12	Explain the role of the germinal center in R cell responses to antigen	2.0
чэ. ЛЛ	Explain the role of the lymphatic system in the transport of antigen and immune c	ells in the
	hody	35
45	Describe the distribution of lymph nodes in the body	3.0
46	Describe the function of the secondary lymphoid organs in tranning and processin	g of antigens
10.		<b>3.0</b>
47.	Explain the function of different regions of the spleen and lymph nodes in adaptive	e immune
	responses.	3.0
48.	Explain the location and function of specialized lymphoid tissues, such as the much	osal-
	associated lymphoid tissues.	3.0
49.	. Describe lymphocyte recirculation and the role of adhesion molecules in lymphocy	/te trafficking.
		3.0
50.	Explain the role of physical barriers in innate immunity.	3.0
51.	Explain the role of physiological barriers in innate immunity.	3.0
52.	Explain the role of the acute phase response and associated soluble effector prote	ins in the
	innate immune response.	3.0
53.	Describe the inflammatory process and its role in innate immunity.	3.5
54.	Identify the key inflammatory cytokines and their local, as well as systemic, roles i	n innate
	immunity.	3.0
55.	Describe the basic components of complement system.	3.5
56.	Describe the complement receptors, expression pattern and function.	3.0

- 57. Differentiate between the three complement pathways: classical, lectin, and alternative.
  - 3.5
- 58. Describe the effector molecules of complement activation and their biologic functions. **3.5**
- 59. Describe the mediators and protective functions of each of the following materials and activities generated by complement activation:

b.       anaphylatoxins       4         c.       clearace of immune complexes       4         d.       B cell activation       4         e.       opsonization       4         f.       enhancement of phagocytosis       4         g.       respiratory burst       4         h.       cytokine production       4         60.       Explain the role of complement in bacterial clearance and lysis.       3         61.       Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.       1         62.       Describe the regulation of the complement system.       3         63.       Identify and describe the function of the phagocytic cells in the body.       4         64.       Define the functional role of cell adhesion molecules including selectins, integrins immunoglobulin superfamily members, and accessory molecules.       3         65.       Describe the role of endothelial cell activation in leukocyte recruitment.       3         66.       Describe the steps involved in phagocytic cell recruitment and migration into sites of inflammation: rolling, activation, tight adhesion, and transendothelial migration.       3         67.       Describe the stages of phagocytosis.       3         68.       Explain the role of full-like receptors in recognition of pathogen associated molecular path (PAMPS) and the a		a.	chemotaxis	4.0
c.       clearance of immune complexes       4         d.       B cell activation       4         e.       opsonization       4         f.       enhancement of phagocytosis       4         g.       respiratory burst       4         h.       cytokine production       4         60.       Explain the role of complement in bacterial clearance and lysis.       3         61.       Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.       1         62.       Describe the regulation of the complement system.       3         63.       Identify and describe the function of the phagocytic cells in the body.       4         64.       Define the functional role of cell adhesion molecules including selectins, integrins immunoglobulin superfamily members, and accessory molecules.       3         65.       Describe the chemotactic factors involved in the recruitment of various inflammatory cell.       3         66.       Explain the role of endothelial cell activation in leukocyte recruitment.       3         67.       Describe the steps involved in phagocytic cell recruitment and migration into sites of inflammation: rolling, activation, tight adhesion, and transendothelial migration.       3         68.       Explain the role of foll-like receptors in recognition of pathogen associated molecular patt (PAMPS) and the activation of innate immune cee		b.	anaphylatoxins	4.0
d.       B cell activation       4         e.       opsonization       4         f.       enhancement of phagocytosis       4         g.       respiratory burst       4         h.       cytokine production       4         60.       Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.       3         61.       Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.       3         62.       Describe the regulation of the complement system.       3         63.       Identify and describe the function of the phagocytic cells in the body.       4         64.       Define the functional role of cell adhesion molecules including selectins, integrins immunoglobulin superfamily members, and accessory molecules.       3         65.       Describe the role of endothelial cell activation in leukocyte recruitment.       3         66.       Describe the steps involved in phagocytic cell recruitment and migration into sites of inflammation: rolling, activation, tight adhesion, and transendothelial migration.       3         67.       Describe the steps involved in phagocytic cell recruitment and migration into sites of inflammation: rolling, activation of inate immune cells.       3         68.       Explain the role of toll-like receptors in recognition of pathogen associated molecular path (PAMPS) and the activation of inate immune cells.       3 <td></td> <td>с.</td> <td>clearance of immune complexes</td> <td>4.0</td>		с.	clearance of immune complexes	4.0
<ul> <li>e. opsonization</li> <li>f. enhancement of phagocytosis</li> <li>g. respiratory burst</li> <li>h. cytokine production</li> <li>60. Explain the role of complement in bacterial clearance and lysis.</li> <li>31. Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.</li> <li>12. Describe the regulation of the complement system.</li> <li>33. Identify and describe the function of the phagocytic cells in the body.</li> <li>44. Define the functional role of cell adhesion molecules including selectins, integrins immunoglobulin superfamily members, and accessory molecules.</li> <li>35. Describe the role of endothelial cell activation in leukocyte recruitment.</li> <li>36. Describe the the chemotactic factors involved in the recruitment of various inflammatory cells inflammation: rolling, activation, tight adhesion, and transendothelial migration.</li> <li>37. Describe the steps involved in phagocytic cell recruitment and migration into sites of inflammation: rolling, activation, tight adhesion, and transendothelial migration.</li> <li>38. Explain the role of toll-like receptors in recognition of pathogen associated molecular patt (PAMPS) and the activation of innate immune cells.</li> <li>39. Explain the role of FC receptors and complement receptors in opsonization, phagocytosis, activation of phagocytic cells.</li> <li>31. Identify and describe the stages of phagocytosis.</li> <li>32. Identify and describe the stages of phagocytosis.</li> <li>33. Identify the pathways involved in reactive oxygen burst and the formation of reactive oxygen burst and the formation of reactive oxygen burst and the formation.</li> <li>33. Explain the role of natural killer (NK) cells in mediating antiviral immunity and the role of activating and inhibitory receptors in the control of their function.</li> <li>34. Explain the fundamental difference between B cell and T cell epitopes.</li> <li>35. Explain the fundamental difference between B cell and T cell epitopes.</li> <li>36. Explain the fundamental</li></ul>		d.	B cell activation	4.0
f.       enhancement of phagocytosis       4         g.       respiratory burst       4         h.       cytokine production       4         60.       Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.       1         61.       Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.       1         62.       Describe the regulation of the complement system.       3         63.       Identify and describe the function of the phagocytic cells in the body.       4         64.       Define the functional role of cell adhesion molecules including selectins, integrins immunoglobulin superfamily members, and accessory molecules.       3         65.       Describe the chemotactic factors involved in the recruitment of various inflammatory cell.       3         66.       Describe the steps involved in phagocytic cell recruitment and migration into sites of inflammation: rolling, activation, tight adhesion, and transendothelial migration.       3         87.       Explain the role of foll-like receptors in recognition of pathogen associated molecular patt (PAMPS) and the activation of innate immune cells.       4         98.       Explain the role of macrophages in antigen processing and presentation.       4         91.       Explain the role of natural killer (NK) cells in mediating antiviral immunity and the role of activating and inhibitory receptors in the control of their function.		e.	opsonization	4.0
g.       respiratory burst       4         h.       cytokine production       4         60.       Explain the role of complement in bacterial clearance and lysis.       3         61.       Explain the use of plasma CH50 and AH50 levels in the assessment of disease processes.       1         62.       Describe the regulation of the complement system.       3         63.       Identify and describe the function of the phagocytic cells in the body.       4         64.       Define the functional role of cell adhesion molecules including selectins, integrins immunoglobulin superfamily members, and accessory molecules.       3         65.       Describe the role of endothelial cell activation in leukocyte recruitment.       3         66.       Describe the steps involved in phagocytic cell recruitment of various inflammatory cell.         67.       Describe the steps involved in phagocytic cell recruitment and migration into sites of inflammation: rolling, activation, tight adhesion, and transendothelial migration.       3         68.       Explain the role of toll-like receptors in recognition of pathogen associated molecular patt (PAMPS) and the activation of innate immune cells.       3         69.       Explain the role of macrophages in antigen processing and presentation.       4         71.       Identify and describe the stages of phagocytosis.       3       3         72.       Identify the pathways involved		f.	enhancement of phagocytosis	4.0
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<ul> <li>70. Compare and contrast antigenetty and immunogenetty.</li> <li>77. Define antigen, antigenic determinant, epitope, and hapten, and give examples of each.</li> <li>78. Explain the fundamental difference between B cell and T cell epitopes.</li> <li>79. Describe antigen-antibody interaction as a subset of receptor-ligand type interactions.</li> <li>80. Define affinity and avidity, and explain their roles in immune processes.</li> <li>81. Differentiate between soluble and insoluble immune complexes.</li> <li>82. Describe the basic structure of the immunoglobulin (Ig) molecule.</li> <li>83. Describe the overall chain structure of the major classes and subclasses of immunoglobuli</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	76	Compa	re and contrast antigenicity and immunogenicity	2.0
<ul> <li>78. Explain the fundamental difference between B cell and T cell epitopes.</li> <li>79. Describe antigen-antibody interaction as a subset of receptor-ligand type interactions.</li> <li>80. Define <i>affinity</i> and <i>avidity</i>, and explain their roles in immune processes.</li> <li>81. Differentiate between soluble and insoluble immune complexes.</li> <li>82. Describe the basic structure of the immunoglobulin (Ig) molecule.</li> <li>83. Describe the overall chain structure of the major classes and subclasses of immunoglobuli</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	70.	Define	antigen antigenic determinant enitone and hanten and give examples of each	3.0
<ul> <li>79. Describe antigen-antibody interaction as a subset of receptor-ligand type interactions.</li> <li>80. Define <i>affinity</i> and <i>avidity</i>, and explain their roles in immune processes.</li> <li>81. Differentiate between soluble and insoluble immune complexes.</li> <li>82. Describe the basic structure of the immunoglobulin (Ig) molecule.</li> <li>83. Describe the overall chain structure of the major classes and subclasses of immunoglobuli</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	78	Evolain	the fundamental difference between B cell and T cell enitones	3.0
<ul> <li>80. Define <i>affinity</i> and <i>avidity</i>, and explain their roles in immune processes.</li> <li>81. Differentiate between soluble and insoluble immune complexes.</li> <li>82. Describe the basic structure of the immunoglobulin (Ig) molecule.</li> <li>83. Describe the overall chain structure of the major classes and subclasses of immunoglobuli</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	70. 79	Describ	e antigen-antibody interaction as a subset of recentor-ligand type interactions	3.0
<ul> <li>81. Differentiate between soluble and insoluble immune complexes.</li> <li>82. Describe the basic structure of the immunoglobulin (Ig) molecule.</li> <li>83. Describe the overall chain structure of the major classes and subclasses of immunoglobuli</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	80	Define	affinity and avidity and explain their roles in immune processes	3.0
<ul> <li>82. Describe the basic structure of the immunoglobulin (Ig) molecule.</li> <li>83. Describe the overall chain structure of the major classes and subclasses of immunoglobuli</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	81	Differer	ntiate between soluble and insoluble immune complexes.	3.0
<ul> <li>83. Describe the overall chain structure of the major classes and subclasses of immunoglobuli</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	82.	Describ	e the basic structure of the immunoglobulin (Ig) molecule.	4.0
<ul> <li>3</li> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>	83.	Describ	e the overall chain structure of the major classes and subclasses of immunoglobu	ilins.
<ul> <li>84. Identify the different types of light chains.</li> <li>85. Differentiate between isotype, allotype, and idiotype.</li> </ul>				3.5
85. Differentiate between isotype, allotype, and idiotype. 2	84.	Identifv	the different types of light chains.	3.0
	85.	, Differer	ntiate between isotype, allotype, and idiotype.	2.0

86.	Describe the overall structure of the major Ig fragments, as well as the enzymatic digest to obtain these fragments.	ion usec <b>3.0</b>
87.	Describe the basic domain structure of the Ig molecule and the essential features of the structure.	tertiary <b>4.0</b>
88.	Describe constant, variable and hypervariable regions with respect to antibody structure	e. <b>3.5</b>
89.	Explain the specialized functions of the human Ig isotypes.	4.0
90.	Identify examples of the specific role of different Ig isotypes in host defense (eg, IgA may	y
	neutralize toxins in the gut).	4.0
91.	Describe the composition and function of secretory immunoglobulins.	3.5
92.	Explain the process by which IgA crosses the epithelium and identify the role of the poly	′-lg
	receptor in IgA secretion.	3.0
93.	Explain molecular genetic mechanisms involved in the generation of antibody diversity (	eg,
	multiple V region gene elements, variable recombination, junctional diversity, etc.).	3.0
94.	Define linkage disequilibrium.	3.0
95.	Explain allelic exclusion with respect to immunoglobulin gene expression.	2.0
96.	Describe the genetic mechanism used to produce membrane-bound and secreted forms	s of Ig.
~-		3.0
97.	Explain isotype switching and its functional significance.	3.5
98.	Describe the mechanism used to regulate expression of IgD.	1.0
99.	Describe somatic hypermutation and explain its functional significance.	3.0
100.	Describe the overall structure of the TCR molecule.	3.0
101.	Differentiale between the two types of TCR.	3.0
102.	Compare the gone organization of the TCP loci with that of the PCP loci	3.0
103.	Describe the function of MHC molecules in antigen presentation and in cell cell interact	<b>2.0</b>
104.	the immune system	
105	Describe the genetic organization of the $HIA$ complex	<del>7</del> .0 2 0
105.	Describe the major structural features of the MHC gene products (eg. Class I molecules :	are two
100.	chains a heavy chain and a heta-2 microglobulin chain)	30
107	Identify the tissue distribution of class L and class II MHC	4.0
108.	Identify examples of MHC/disease correlations and provide a hypothesis to account for	this
	correlation.	3.0
109.	Explain MHC polymorphism and estimate the selective advantage of such a system.	3.0
110.	Explain MHC restriction and provide examples of the functional consequences of the	
	"restriction" of T cell recognition.	3.0
111.	Define haplotypes, genotypes, phenotypes, and alleles.	3.0
112.	Describe a proposed model of MHC-Ag-TCR interaction.	3.5
113.	Explain how T lymphocytes recognize antigen bound to MHC molecules.	3.0
114.	Explain how T lymphocytes primarily recognize protein antigens and in fact recognize lin	near
	determinants as opposed to the conformational determinants recognized by B cells.	3.0
115.	Explain how T lymphocytes recognize antigen on the surface of other cells (antigen-pres	enting-
	cells (APCs) or target cells).	4.0
116.	List and describe the functions of each type of APC.	4.0
117.	Describe the phenomenon of MHC-restricted antigen recognition, as well as the recogni	ition
	process for both helper and cytotoxic T lymphocytes.	3.5
118.	Describe the pathway of processing of both exogenous and endogenous protein antiger	ıs.
		3.0

119.	Compare and contrast the presentation of exogenous and endogenous antigens to T	
	lymphocytes.	4.0
120.	Identify and explain the steps involved in lymphocyte activation.	3.0
121.	Describe the overall structure of the B cell antigen receptor, as well as how the B cell anti	tigen
	receptor recognizes a wide range of antigens.	3.5
122.	Describe the BCR complex.	3.0
123.	Identify the functional significance of Immunoreceptor Tyrosine-based Activation Motifs	
	(ITAMs).	2.0
124.	Describe the overall structure of the TCR molecule.	3.0
125.	Describe the composition and function of the CD3 complex.	3.0
126.	Explain the activation of T cells (eg, the interactions between APCs and T cells leading to	T cell
	activation).	4.0
127.	Summarize the biochemical events triggered in T and B cells by antigen recognition.	3.0
128.	Explain the functional role of the T cell accessory protein CD4 and CD8 in recognition of a	antigen
	and T cell activation.	4.0
129.	Identify examples of cell adhesion molecules, (eg, ICAM, LFA-1) and describe their role in	n T cell
	activation.	2.0
130.	Describe the mechanism of superantigen activation of T cells.	3.0
131.	Describe the mechanism of antigen induced B lymphocyte activation.	3.0
132.	Compare and contrast the effects of T-independent and T-dependent antigens on B cell	
	activation.	3.0
133.	Describe the mechanism of TH-B cell collaboration and explain the observation known a	s the
	"hapten-carrier effect."	2.0
134.	Diagram the cell-cell interactions in a humoral immune response to a protein antigen (eg	g, TH,
	cytokines, APCs interact with T cells, activated T cells interact with antigen-specific B cell	s, etc.).
		3.0
135.	Explain the two-signal model of T cell activation and the role of costimulatory CD28 mole	ecules.
		3.0
136.	Describe how most B cell responses require CD40-dependent signals in addition to that p	provided
	by antigen alone and identify the functional role of CD40 signaling.	2.0
137.	Describe how antigen-dependent signaling in the absence of costimulation leads to indu	ction of
	anergy in T and B cells and explain what this means.	3.5
138.	Describe the concept of central tolerance as it pertains to T cell development in the thyn	nus.
		3.0
139.	Define the roles played by mechanisms leading to apoptosis and anergy in the regulation	n of T
	cell development in the thymus.	3.0
140.	Differentiate between positive and negative selection of thymocytes.	3.0
141.	Explain central tolerance as it pertains to B cell development in the bone marrow.	3.0
142.	Explain the role that receptor editing plays in B cell selection.	2.0
143.	Identify the factors that control T and B cell selection and tolerance including avidity and	affinity
	of interactions between the antigen receptor and antigen.	3.0
144.	Explain peripheral tolerance induction for T and B cells.	3.0
145.	Explain the role of regulatory T cells in mediating peripheral tolerance.	3.0
146.	Relate activation-induced cell death to feedback control of T cell activation.	2.0
147.	Explain the role of Fas and Fas ligand in mediating apoptosis of activated T and B cells.	2.0
148.	Describe the major pathways that lead to apoptosis in lymphocytes.	2.0
149.	Explain the role of CTLA-4 in attenuation of T cell activation.	2.0

150. Explain the process by which B cell coreceptors modulate antigen receptor signaling th recruitment of effector proteins to ITIMs and ITAMs.	rough the 1.5
151. Describe the process of antibody-dependent feedback in negative regulation of B cells	and
explain the role played by the FcR-gamma-IIb. ITIM.	2.0
152. Explain the role of CD19 in positively regulating activation of B cells.	2.0
153. Explain the basis of cytokine nomenclature and identify the major classifications of cyto	okines.
	3.0
154. Describe the general functions of cytokines (IL-1, 2, 3, 4, 5, 6, 7, 8, 10, 12, 17, IFNs, GM-	-CSF, G-
CSF, MCSF, TNF-alpha, TGF-beta).	4.0
155. Compare and contrast the individual effect of cytokines, including IL- 1, TNF-alpha, IL-6	and
interferons, on innate immunity.	3.0
156. Describe the lipopolysaccharide (LPS)-induced cytokine cascade.	3.0
157. Describe the cytokines involved in the acute phase response.	3.0
158. Describe the role of several cytokines (including IFNs, lymphotoxin, IL-5, IL-12) as regula	ators of
immune-mediated inflammation.	3.0
159. Describe the role of cytokines in regulation of Ig class switch recombination.	3.0
160. Explain the role of cytokines and cytokine receptors, including IL-2, IL-4, IL-6, TGF-beta,	, in the
activation, growth and differentiation of lymphocytes.	3.0
161. Explain the role of cytokines in T helper cell differentiation into Th1, Th2, or Th17 cells	and
describe the production of cytokines by these distinct T helper cell subsets.	3.0
162. Explain the role of chemokines and chemokine receptors in regulation of immune cell t	rafficking
and localization within immune organs.	3.0
163. Identify the populations of effector T cells and explain their activation requirements.	4.0
164. Explain the process whereby effector CTLs are generated from CTL precursors.	3.0
165. Explain the process by which effector CTLs recognize target cells.	3.0
166. Explain the role of Fas and Fas ligand in CTL-mediated lysis of target cells.	2.0
167. Describe the process of CTL-mediated cell lysis.	3.0
168. Describe the functions of NK cells.	3.0
169. Describe the roles of CD4 Th-1 and CD4 Th-2 lymphocytes in the immune response.	4.0
170. Describe what antibody dependent cell-mediated cytotoxicity (ADCC) is.	3.5
171. Identify cell-mediated immune responses induced by NK responses, ADCC, LAK, DTH, a	nd
provide clinical examples of each.	2.0

### IV. Basic Mycology and Parasitology

Define:		
a.	hyphae	3.0
b.	septate	3.0
с.	nonseptate	3.0
d.	pseudohyphae	3.0
e.	mycelium	3.0
f.	zygospores	3.0
g.	ascospores	3.0
h.	ascus	3.0
i.	basidiospores	3.0
j.	conidia	3.0
k.	arthroconidia	3.0

	I. chlamydoconidi	3.0
	m. <i>blastospores</i>	3.0
	n. sporangiospores	3.0
	o. macroconidia	3.0
	p. microconidia	3.0
	q. dimorphism	3.0
2.	Compare the structure of fungal cells to other eukaryotic cells and to bacteria.	4.0
3.	Compare and contrast yeasts, molds, and dimorphic fungi.	4.0
4.	Explain the basis for fungal taxonomy.	3.0
5.	Describe the major attributes of Deuteromycetes (fungi imperfecta), Zygomycetes,	
	Ascomycetes, Archiascomycetes (Pneumocystis), and Basidiomycetes.	2.0
6.	Describe the laboratory identification of fungi.	3.0
7.	Define KOH preparation and Sabouraud's agar.	4.0
8.	Define cyst, trophozoite, oocyst, schizogony, vector, intermediate host, and definitive ho	ost.
		3.0
9.	Describe the classification of protozoa.	3.0
10.	Describe the classification of helminths.	3.0
11.	Define <i>mycotoxicosis</i> .	3.0
12.	Differentiate among endothrix, exothrix, and favic fungal infection of hair.	4.0
13.	Identify the primary genera that causes cutaneous mycoses (dermatomycoses).	4.0
14.	Differentiate among anthropophilic, zoophilic, and geophilic dermatophytes, and descri	be the
	clinical significance of associating the mycotic agent with its source.	3.0
15.	Describe the use of the Wood's lamp in diagnosing mycotic infections.	3.5
16.	Describe the basis of the "Id reaction."	3.5
17.	Describe the use of macroconidia and microconidia identification in in the determinatio	n of
	dermatophytes.	3.5
18.	Differentiate between eumycotic and actinomycotic mycetomas.	3.5
19.	Identify the causative agent, method of acquisition, geographic distribution, primary system	mptoms,
	and treatment for sporotrichosis, chromoblastomycosis, histoplasmosis, blastomycosis,	
	coccidiomycosis, and candidiasis.	3.5
20.	Differentiate among superficial, cutaneous, subcutaneous, systemic and opportunistic r	nycoses
	and give one example of each.	4.0
21.	Describe the agents, method of transmission, clinical manifestations, and geographic	
	distribution of malaria, giardiasis, schistomiasis, larva migrans, and toxoplasmosis.	3.0

#### V. <u>Basic Virology</u>

1. Define:	
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a.	conditional mutants	3.5
b.	recombination	3.5
с.	reassortment	3.5
d.	complementation	3.5
e.	phenotypic mixing	3.5
f.	cell culture	3.0
g.	one-step growth experiment	3.0
h.	cytopathic effect	3.0
i.	syncytia	3.0

	j. plaque 3 k. hemagglutination assay 3	8.0 8.0
2.	Identify and describe the size, shape, nucleic acid, capsid, capsomere, nucleocapsid, capsi	d
	symmetry, icosahedral, helical, envelope, and peplomer of viruses. 4	1.0
3.	Explain virus classification. 3	8.0
4.	Describe DNA virus families, including whether they are enveloped or naked; the DNA stru	ucture
	(dsDNA, ssDNA, linear, clrcular); and replication site (cytoplasm or nucleus).	3.0
5.	Describe the RNA virus families, including whether they are enveloped or naked; the RNA	
	structure (dsRNA, ssRNA, linear, clrcular); sense (positive, negative, or ambisense); capsid	
	symmetry; and replication site (cytoplasm or nucleus).	3.0
6.	Explain defective virus and prior, including their replication cycle.	3.0
7.	Describe virus multiplication, including adsorption, entry, naked viruses, enveloped viruse	es,
	uncoating site, role of reverse transcriptase, viral protein synthesis, assembly, release (lys	is or
	budding), and virus load. 4	1.0
8.	Differentiate between antigenic drift and antigenic shift.	1.0
9.	Describe the cultivation of viruses in the laboratory.	3.0
10.	Explain the use of viruses in gene therapy. 2	2.0
11.	Discuss malignant transformation and oncogenes.	8.0

## VI. <u>Cardiac Infections</u>

1.	Identify the organisms that commonly cause endocarditis.	3.5
2.	Explain the epidemiologic factors underlying etiologies in particular patients.	3.0
3.	Describe "vegetative" lesions associated with endocarditis and explain the contribution	to the
	diagnosis and effect on therapeutic options.	2.0
4.	Explain laboratory procedures that distinguish between the organisms causing endocard	litis.
		3.0
5.	Identify the clinical sample that would be used, and which lab procedures, selective and	
	differential media, and biochemical assays would be necessary to distinguish between the	ne
	pathogens responsible for endocarditis.	2.0
6.	Describe the important virulence factors for pathogens causing endocarditis and discuss	how
	these factors contribute to the virulence of these organisms.	3.0
7.	Identify the most common infectious causes of myocarditis.	2.0
8.	Explain the epidemiology and pathogenesis of Coxsackie virus infections and the reason	that
	most Coxsackie virus infections are subclinical.	2.0
9.	Discuss the protective acquired immune response that prevents disease in most people	infected
	with Coxsackie viruses, and how the timing of this immune response correlates with	
	symptomatic versus asymptomatic infection.	2.0

# VII. <u>Clinical Immunology</u>

1.	Describe the basis of classification of hypersensitivity reactions into hypersensitivity type	es.
		3.0
2.	Describe the pathophysiologic mechanisms associated with Type I (IgE)-mediated injury.	4.0
3.	Explain the process of mast cell degranulation.	4.0
4.	Describe the primary effector mediators released by mast cells.	4.0

5.	Describe the pathologic changes in tissues during anaphylactic reactions.	4.0
6.	Compare and contrast the acute phase reaction with the late phase reaction in anaphyla	actic
	reactions.	4.0
7.	Explain the modulator role of eosinophils in allergic and anaphylactic reactions.	3.0
8.	Correlate the effect of mediators on target organs with clinical expression of allergic rea	ctions.
		3.0
9.	Identify therapeutic modulation of type I hypersensitivity.	4.0
10.	Describe the clinical expression of anaphylactic reactions and diagnosis via skin tests, an	d the
	immunoassays RIST and RAST.	4.0
11.	Describe the clinical symptoms and basis of the symptoms of allergic asthma.	3.0
12.	Describe the bronchial wall changes that occur in asthma.	3.0
13.	Identify the treatment considerations of the various forms of asthma.	3.0
14.	Differentiate between type II and type III hypersensitivity reactions.	4.0
15.	Compare complement mediated cell lysis with antibody dependent cell cytotoxicity.	3.0
16.	Compare immunopathology of Goodpasture's syndrome with Lupus.	3.0
17.	Explain the pathogenesis of drug-induced type I and II hypersensitivity.	3.0
18.	Describe the basis for erythroblastosis fetalis.	2.0
19.	Describe the mechanism and histopathology of Arthus reaction.	3.0
20.	Describe type IV cell mediated hypersensitivities.	4.0
21.	Identify the basis for and examples of contact hypersensitivity.	4.0
22.	Identify the mechanisms involved in and manifestations of a positive tuberculin reaction	۱.
		4.0
23.	Describe the granulomatous response.	4.0
24.	Identify and describe autoimmune diseases associated with specific organs.	3.0
25.	Identify autoimmune diseases that are systemic in nature.	3.0
26.	Explain the role that gender, genetics, environment, and infectious disease play in the	
	development of autoimmunity.	3.0
27.	Describe the mechanisms that help to explain antiself-responses (eg, immunological cr	OSS-
	reaction or molecular mimicry).	3.0
28.	Explain the role of MHC genes in autoimmunity.	3.0
29.	Describe the basic types of therapeutic intervention used to treat autoimmune disease.	3.0
30.	Describe the immunologic basis of graft rejection.	4.0
31.	Define autograft, isograft, allograft, and xenograft.	4.0
32.	Explain the role of CD4 and CD8 T cells in graft rejection.	4.0
33.	Explain why the non-self MHC molecules are the major molecular targets in graft rejecti	on.
		3.0
34.	Differentiate between major and minor MHC molecules.	3.0
35.	Describe the overall molecular structure of the MHC Class I and II molecules.	3.0
36.	Describe hyperacute, acute, and chronic rejection.	4.0
37.	Compare and contrast the immunological reactions occurring in hyperacute, acute, and	chronic
	rejection events resulting from a foreign graft.	3.0
38.	List tests used to measure tissue histocompatibility.	3.0
39.	Describe approaches to prolonging graft survival (eg, immunosuppressive drugs, mAbs,	and
	immune modulators).	3.0
40.	Describe the special immunological complexities that can be associated with bone married	ow
	transplantation.	2.0
41.	Differentiate congenital versus acquired immunodeficiency.	3.0
42.	Identify the basic classification of congenital immunodeficiencies.	2.0

43.	Describe the clinical presentation and pathophysiology associated with severe con immunodeficiencies.	nbined <b>3.0</b>
44.	Describe the signs and symptoms and developmental abnormality associated with syndrome.	DiGeorge 2.0
45.	Describe the basic B cell defect associated with X-linked agammaglobulinemia, Hy	per-IgM
46	syndrome, common variable immunodeficiency, and selective IgA deficiency.	3.0 ase leukocyte
10.	adhesion deficiencies, and Chediak-Higashi syndrome.	<b>3.0</b>
47.	Describe the basic immunologic defect associated with Wiscott-Aldrich syndrome,	Ataxia-
	telangiectasia, and IFN-gamma/IL-12 receptor deficiencies.	2.0
48.	Explain the effects of specific complement deficiencies on patients.	3.0
49.	Identify basic therapeutic approaches for treatment of SCID, B cell deficiencies, an	d phagocytic
50	Cell deficiencies.	
50.	induced radiation induced)	<b>4 0</b>
51.	Identify the immunological abnormalities associated with HIV infection.	4.0
52.	Describe the concept of immunosurveillance.	3.0
53.	Explain the principle of tumor-specific antigens and what role they might have clin	ically. <b>3.0</b>
54.	Describe the roles of antibody, T cells, NK cells, and macrophages in tumor immun	ity. <b>3.0</b>
55.	Explain the involvement of MHC molecules in tumor immunity (eg, the effect of vin	ally induced
	low MHC expression).	3.0
56.	Explain ways that tumors evade immune recognition.	3.0
57.	Describe approaches to tumor immunotherapy.	3.0
58.	Identify potential causes of lymphoproliferative disorders.	3.0
59.	Describe the immune response to both intracellular and extracellular bacterial infe	ections.
60	Evaluin the mode of action of adjuncents	3.5
61	Describe delayed type hypersensitivity as it relates to host responses against intra-	<b>5.0</b>
01.	hacteria	2 5
62.	Describe the host immune response to parasitic infection.	3.0
63.	Identify mechanisms of immune evasion.	3.0
64.	Explain the basis for inactivated, attenuated, recombinant, and DNA vaccines.	3.5
65.	Differentiate between active and passive immunity to microbes.	3.5
66.	Differentiate between primary and secondary immune responses to vaccines and	microbes.
		3.5
67.	Describe the use of monoclonal antibodies to modulate immune cell function or to	remove
	specific immune cells from the body.	3.0
68.	Describe the use of immunosuppressive drugs for the treatment of autoimmune d	isease or to
<u> </u>	prevent transplant rejection.	3.5
69.	immunodoficionerios or concor	2.0
70	Describe the use of IVIG in the treatment of autoimmune disease and congenital	2.0
70.	immunodeficiencies	3.0
71	Identify the potential therapeutic roles of cytokines or antibodies specific for cytok	kines and/or
	their receptors in:	
	a sepsis	3.0
	b. inflammatory bowel disease	3.0
	c. rheumatoid arthritis	3.0

	d. graft-versus-host Disease	3.0
72.	Describe the basis of ELISA, Western blotting, flow cytometry, immunofluores	cence staining
	and RIA tests.	3.0
73.	Differentiate between immune tolerance and immune deficiency.	3.0

# VIII. Genitourinary Infections and STDs

1.	Define cystitis and pyelonephritis.	3.0
2.	Distinguish acute from chronic pyelonephritis.	3.0
3.	Describe the most common causes of community-acquired versus nosocomial urinary to	ract
	infections (UTIs).	3.0
4.	Explain the routes of transmission of agents of UTIs.	3.0
5.	Identify the major host defenses that protect against infection by UTI-causing bacteria.	3.0
6.	Identify factors that predispose patients to UTIs.	3.0
7.	Explain the prevalence of bacterial UTIs in females.	3.0
8.	Describe diagnostic methods for bacterial UTIs.	3.0
9.	Identify viral and parasitic agents of UTIs.	2.0
10.	Discuss the common causes of UTIs, including:	
	a. Uropathogenic <i>E. coli</i>	3.0
	b. P. aeruginosa	3.0
	c. Klebsiella sp	3.0
	d. Proteus spp	3.0
	e. Staphylococcus saprophyticus	3.0
	f. Enterococcus sp	3.0
11.	Describe structural and cultural characteristics of Treponema pallidum.	3.0
12.	Describe the epidemiology and pathogenesis of syphilis, including primary, secondary, a	ind
	tertiary manifestations of disease.	3.5
13.	Describe congenital syphilis and describe its manifestations and prevention.	3.0
14.	Describe neurosyphilis and describe its manifestations.	3.0
15.	Explain the mode of transmission of <i>T. pallidum</i> .	4.0
16.	Describe methods for the diagnosis of syphilis.	4.0
17.	Explain the difference between non-specific and specific serological tests for syphilis an	d the
	pattern of the immune response vis-à-vis these tests in treated and untreated cases.	3.5
18.	Identify antibiotics of choice in treating syphilis.	3.0
19.	Describe structural and cultural characteristics of Neisseria gonorrhoeae.	3.0
20.	List the virulence factors associated with <i>N. gonorrhoeae</i> .	3.0
21.	Describe modes of transmission of gonorrhea.	3.5
22.	Explain the diagnosis and treatment of gonorrhea.	3.0
23.	Distinguish between gonococcal and non-gonococcal urethritis.	3.0
24.	Describe disseminated gonococcal infections and distinguish them from gonococcal infe	ections of
	the eyes and throat.	3.5
25.	Describe the mechanisms of acquired penicillin resistance by Neisseria gonorrhoeae and	t
	alternative drugs for treating resistant strains.	3.0
26.	Explain the importance of phase and antigenic variation in pathogenesis of <i>N. gonorrho</i>	eae.
		3.0
27.	Describe the correlation between <i>N. gonorrhoeae</i> cervicitis and pelvic inflammatory dis	ease
	(PID) in women.	3.5

28. Identify the causative agents of non-gonococcal urethritis.	3.0
29. Describe the life cycle and unique properties of <i>Chlamydia trachomatis</i> .	3.0
30. Describe structural and cultural characteristics of Ureaplasma urealyticum and Myc	oplasma
genitalium.	2.0
31. Describe the diagnosis and treatment of non-gonococcal urethritis (NGU).	3.0
32. Describe how NGU can lead to PID in women.	3.0
33. Describe the characteristics and causative agent of lymphogranuloma venereum (LC	GV). <b>2.0</b>
34. Describe the characteristics and pathogenesis caused by Klebsiella (Calymmatobact	erium)
granulomatis.	1.0
35. Describe the characteristics and pathogenesis of Haemophilus ducreyi.	2.0
36. Describe how symptoms of chancroid can be confused with those of primary syphili	s, LGV,
granuloma inguinale, or genital herpes.	2.0
37. Describe characteristics and pathogenesis of the protozoan <i>Trichomonas vaginalis</i> .	2.0
38. Describe the signs associated with non-specific vaginitis and bacterial vaginosis.	2.0
39. Describe the characteristics and pathogenesis of Candida albicans.	3.5
40. Explain how <i>Candida</i> can cause disease as a member of normal human flora.	3.5
41. Describe the diagnosis and treatment of vulvovaginal candidiasis.	3.0
42. Describe the virion and genome structure of herpes simplex type 2 (HSV-2).	3.0
43. Describe the transmission and pathogenesis of HSV-2 infections.	3.0
44. Describe the concept of viral latency/reactivity and its significance with respect to g	enital herpes
infections.	3.5
45. Identify and explain current strategies for preventing and treating HSV-2 infections.	3.0
46. Describe the virion and genome structure of human papillomavirus (HPV).	4.0
47. Explain the transmission and pathogenesis of HPV.	4.0
48. Explain the association of cervical cancer with certain types of HPV infections.	4.0
49. Describe methods for detection, treatment, and prevention of HPV infections.	4.0
50. Describe the virion and structure of human cytomegalovirus (CMV).	3.0
51. Describe the epidemiology and pathogenesis of CMV.	3.0
52. Explain why primary CNV intection in a healthy individual is clinically unapparent, b	
Immunocompromised adults can lead to a mononucleosis syndrome.	<b>3.U</b>
53. Explain why Civiv causes the most common intrautenine viral infection and now cyto	
forming organs, and/or ponyous system	
FA Describe the general nethogenesis and transmission of henetitic Rivirus	5.0
54. Describe the genome, pathogenesis, and transmission of hepatitis B virus.	4.0
55. Explain the transmission and me cycle of the numan inimunouenciency virus (HIV).	4.0
Gastrointestinal Infections	
1. Define <i>diarrhea</i> .	3.0

т.	Define didrifted.	5.0
2.	Differentiate between gastroenteritis and enterocolitis.	2.0
3.	Identify the five most common cause of diarrhea in infants.	2.0
4.	Describe clinical findings in acute gastroenteritis.	3.0
5.	Differentiate between an invasive infection and a toxin-mediated illness based on clinica	al
	findings.	3.0
6.	Describe the two main modes for transmitting infectious agents that cause gastroentering	tis and
	diarrhea.	3.0
7.	Describe the pathogenesis of bacterial diarrhea.	3.0

IX.

8.	Explain the mechanisms of damage from enterotoxins, cytotoxins, and invasive organism	ms.
_		3.0
9.	Describe the clinical and diagnostic techniques used to identify organisms causing	• •
10	gastroenteritis.	3.0
10.	Explain the recommended treatment for gastroenteritis.	3.0
11.	Describe the characteristics of the major bacterial, viral, and parasitic organisms causing	5
		2 E
	a. E. Coll b. Shigella spp	3.5 2 5
	c. V cholera	3.5
	d V narahemolyticus	3.5
	e. C. difficile	3.5
	f. Salmonella spp.	3.5
	g. Y. enterocolitica	3.5
	h. <i>C. perfringens</i>	3.5
	i. Norovirus	3.5
	j. Rotavirus	3.5
	k. E. histolytica	3.5
	I. G. lamblia	3.5
	m. <i>C. parvum</i>	3.5
	n. <i>Taenia</i> spp.	3.5
12.	Define <i>hepatitis</i> .	3.0
13.	Define <i>jaundice</i> .	3.5
14.	Describe the symptoms and laboratory findings present in acute hepatitis.	4.0
15.	Explain the mechanism of liver damage in hepatitis.	3.0
16.	Identify the potential long-term sequelae of chronic hepatitis.	4.0
17.	Identify external factors that greatly accelerate microbe-induced liver damage.	3.0
18.	Identify the fatality rate of fulminant hepatitis.	2.0
19. Describe the basic viral properties, principal routes of infection, global prevalence, po		
	establish chronic infections, clinical symptoms, means of diagnosis (including serologic i	markers),
20	Identify the viral hensitic infections that can be provented by immunization	4.0
20.	Identify additional virus (other than Henstitis $\Lambda_{-}$ E and Yellow fever virus) that may tak	4.U
21.	liver	30
22	Identify two spirochetes that may target the liver	2.0
23.	Identify two parasites that may target the liver.	2.0
24.	Describe the characteristics and clinical manifestations of	
	a. Typhoid fever;	3.0
	b. <i>Campylobacter jejuni</i> infection;	3.0
	c. Botulism;	3.0
	d. infant botulism;	3.0
	e. S. aureus infections/intoxications; and	3.0
	f. Helicobacter spp.	3.0
25.	Describe the characteristics of Helicobacter pylori and explain the inflammatory condition	ons of
	the GI tract with which it is associated.	3.5
26.	Differentiate among the conditions caused by the ETEC, EPEC, and EHEC strain designat	ions of
	Escherichia coli.	3.0

- 27. Identify the bacteria that are associated with causing food intoxications, denote approximate time between ingestion of the bacterial toxin and the appearance of symptoms for each.
  - 3.0

28.	Describe the epidemiology	and pathogenesis of C. difficile infe	ctions. 3.5

- 29. Describe the oral diseases and pathogenesis caused by Candida, HSV, HPV, Actinomyces israelii, viridians-group streptococci, Histoplasma, and Coxsackieviruses. 3.5
- 30. Describe the infections caused by oral normal flora in other parts of the body. 3.5

#### **Infectious Pathogenesis** Χ.

1.	Differentiate between endogenous (ie, normal flora) and exogenous sources of infection	n.
		4.0
2.	Explain how normal flora on skin or mucosal membranes can cause disease when introc	luced
	into deeper tissues.	4.0
3.	Explain how exogenous infections are a result of encounters with organisms in the envir	ronment.
		4.0
4.	Discuss the following common mechanisms of microbial transmission:	
	a. direct skin or mucosal contact	4.0
	b. inhalation	4.0
	c. ingestion	4.0
	d. vertical transmission (congenital; mother to baby)	4.0
_	e. vector-born transmission	4.0
5.	Explain the ways that anatomical sites exposed to the environment serve as portals of n	nicrobial
~	entry.	4.0
6.	Discuss how entry may or may not involve the crossing of epithelial barrier (eg, inhalatio	on versus
_	the carrying of microorganisms into deeper tissues by macrophages, or insect bites).	4.0
7.	Explain the significance of microbial adhesion as a component of the establishment of a	n
0	Intection.	4.0
8. 0	Explain which microbial surface structures can function as adhesins.	4.0
9. 10	Differentiate between bacterial fillipital and animpital adhesins.	2.0
11	Identify the best cell surface components that can act as recentors	3.0 2.0
12	Discuss the function of neutralizing antibodies in preventing microbial attachment	2.0
13	Explain how attachment helps microorganisms to remain at a particular location/evade	innate
13.	defense mechanisms	30
14.	. Describe antimicrobial compounds and the targets that are used to interfere with attac	hment.
		2.0
15.	Describe the action of invasins.	2.0
16.	Describe the role of secreted enzymes in invasiveness of bacteria.	2.0
17.	. Describe the advantage of encapsulation for bacteria and give examples of encapsulate	d
	organisms.	3.5
18.	Define hemolysin and cytolysin and give an example of each.	3.0
19.	Explain the mechanisms of action for the pore-forming and phospholipase cytolysins.	3.0
20.	Discuss the streptococcal hemolysins in terms of their mechanisms of action.	3.0
21.	. Explain how hemolysis patterns on blood agar can help with species differentiation and	disease
	diagnosis.	3.0
22.	Discuss the advantages of intracellular growth from a microbial perspective.	3.0

23.	Contrast mechanisms of bacterial entry into a phagocytic versus a non-phagocytic cell.	3.0
24.	Identify bacteria that rearrange actin to enable their entry and identify the basic steps in	n the
	process.	1.0
25.	Characterize the following intracellular survival mechanisms, giving specific microbial ex	amples:
	a. escape from phagolysosome	3.0
	b. prevention of phagolysosome fusion	3.0
	c. evasion/neutralization of lysosomal contents	3.0
	d. alteration of phagolysosomal environment	3.0
26.	Describe the adaptations/virulence factors utilized by extracellular bacteria to evade the	e host's
	antimicrobial defenses.	3.0
27.	Assess the significance of intracellular growth when selecting an appropriate antimicrob	ial
	agent.	3.0
28.	Explain the significance of tissue tropism in microbial pathogenesis.	3.0
29.	Identify the factors, both host and microbial, that influence the colonization of a particu	lar site
	by a microorganism.	2.0
30.	Describe how the adhesin-receptor interaction determines the tissue tropism of a	
	microorganism.	3.0
31.	Define commensalism, parasitism, colonization, and mutualism.	3.5
32.	Explain the benefits of microorganism colonization to the host.	3.5
33.	Identify factors that predispose to the development of disease when a host encounters	а
	microorganism.	3.5
34.	Identify mechanisms of host cell damage.	3.0
35.	Explain the genetic control of bacterial toxin production.	1.0
36.	Differentiate between exotoxins and endotoxins.	3.0
37.	Distinguish the determining factor(s) of the cell to which an exotoxin binds.	3.0
38.	Identify the different types of toxins and describe their mechanisms of action.	3.0
39.	Identify the source of endotoxins.	3.0
40.	Explain pathogenesis of septic shock produced by endotoxins.	3.5
41.	Explain infectious pathogenesis of disseminated intravascular coagulation.	3.0
42.	Identify the major mechanism of tissue damage caused by fungi.	3.0
43.	Describe the morphologic growth patterns of fungi and identify which are advantageous	for
	allowing invasion of host tissue.	3.5
44.	Identify the host cell surface molecule that is a receptor for several bacteria and viruses.	2.0
45.	Describe the process by which a virus enters a host cell and elicits cell death in a lytic inf	ection.
		3.5
46.	Describe the changes in the host cell seen as a result of viral infection.	4.0
47.	Explain occurrences in a virally-infected cell that result in persistent or latent infection.	3.0
48.	Describe the changes in a cell that is transformed by viral infection.	3.0
49.	Identify bacterial components that are active in eliciting a host immune response.	4.0
50.	Describe the elicitation of the cytokine response to microbial infection of the host.	4.0
51.	Explain the immune response involved in the development of lesions characteristic of	
	Mycobacterium tuberculosis.	3.5
52.	Describe the mechanism of damage to the host that may occur from virus-antibody imm	nune
	complexes.	3.5
53.	Describe the mechanism of damage to the host that may occur from the cell-mediated r	esponse
	to a virus.	3.5
54.	Explain the damage that may occur with autoimmune sequelae of an infection.	3.5

55. Describe how each of the following factors facilitates evasion of the host immun (innate and/or adaptive):	ie response
a. polysaccharide capsule	3.5
b. pili/fimbriae	3.5
c. IgA protease	3.5
d. leukocidin	3.5
e. coagulase	3.5
f. protein A	3.5
g. M protein	3.5
h. lipoteichoic acid	3.5
56. Describe mechanisms used by bacteria to evade the degradative enzymes inside	phagocytic cells
(polymorphonuclear cells, macrophages, or monocytes) and survive intracellula	rly. <b>3.0</b>
57. Explain the resilience of bacteria in a biofilm to antimicrobials and to host immu	ne responses.
	4.0
58. Explain how antigenic variation facilitates evasion of the host immune response	by pathogens,
and how this affects host and therapeutic/prophylactic mechanisms to prevent	reinfection. <b>4.0</b>
59. Describe several mechanisms used by viruses to evade the antiviral interferon re	esponse. <b>2.0</b>
60. Explain how HIV- and CMV-mediated down regulation of MHC class I expression	enhances their
ability to evade the nost immune response.	1.0
61. Identify and describe viruses that produce syncytia and explain the mechanism (	of cell-to-cell
Spread that enhances their ability to evade the nost immune response.	Z.U
infoctious agents	
62 Explain what is meant by "immune privileged" sites in the body and describe vir	<b>5.U</b> Succe that ovhibit
a transm for these sites	
64 Describe mechanisms used by viruses to produce persistent infections	3.0
65. Describe the mechanism through which hernes viruses produce a latent infection	n in their host
and the contribution to the ability to evade the host immune response.	<b>4.0</b>
66. Describe the development of "immune tolerance" in neonates infected with her	patitis B virus.
rubella virus, or CMV, and discuss the effects on the infected infant.	2.0
67. Compare and contrast the mechanisms of persistence for HBV and HCV.	2.0
68. Explain the lack of host immune response in prion diseases.	3.0
69. Explain the contribution of antigenic shift and antigenic drift to the ability of infl	uenza virus to
evade the host immune response.	3.5
70. Explain the generation of viral "quasi-species" and the contribution to the ability	of some viruses
to evade the host immune response.	1.0
71. Describe and give examples of the following modes of transmission:	
a. person-to-person	4.0
b. nosocomial/hospital-acquired	4.0
c. endogenous infection	4.0
d. percutaneous	4.0
e. fomites	4.0
f. soil	4.0
g. vertical transmission	4.0
h. horizontal transmission	4.0
i. aerosols	4.0
J. food/water	4.0
k. zoonotic	4.0

l. vector-borne	4.0
m. sexual contact	4.0
n. fecal-oral	4.0
72. Describe structural features of viruses that often affect their stability in the environment	t and
mode of transmission.	3.0
73. Identify the major sites of entry for infectious agents into the body and the barriers they	/ must
overcome at these sites to survive.	3.5
74. Describe conditions that enhance the transmission of infectious agents from person-to-p	person
via non-sexual modes.	3.0
75. Define reservoir and vector in the context of zoonoses.	3.5
76. Differentiate between self-limited infection, resolution of infection, and chronic infection	n.
	3.0
77. Describe the steps that occur in an acute, self-limiting infection with respect to the pathe	ogen,
pathogenesis, and host immune response.	3.0
78. Describe the role of international travel, exotic pets, and exotic food sources in the sprea	ad of
emerging infectious diseases.	3.0
79. Compare and contrast the major characteristics of a chronic viral infection versus those	of a
latent viral infection.	2.0
80. Differentiate the roles of humoral versus cell-mediated immune responses in mediating	
clearance of viruses.	3.0
81. Explain the term "chronic carrier."	3.0
82. Explain the term "slow virus infection."	1.0

# XI. Skin, Soft Tissue, and Bone Infections

1.	. Define:		
	a. abscess	4.0	
	b. <i>boil</i>	4.0	
	c. carbuncle	4.0	
	d. <i>furuncle</i>	4.0	
	e. <i>folliculitis</i>	4.0	
	f. pyoderma (impetigo)	4.0	
	g. erysipelas	4.0	
	h. <i>cellulitis</i>	4.0	
	i. <i>macule</i>	3.5	
	j. papule	3.5	
	k. plaque	3.5	
	I. pustule	3.5	
	m. <i>vesicle</i>	3.5	
	n. <i>bulla</i>	3.5	
2.	. Identify the most common infectious caus	e of myositis. 3.0	
3.	. Describe the way that coagulase helps Sta	<i>phylococcus aureus</i> evade host immunity. <b>2.0</b>	
4.	Describe the other important virulence factors for Staphylococcus aureus and describe the		
	contribution of these factors to the virule	nce of the organism. <b>4.0</b>	
5.	. Identify the most common causative agen	ts of necrotizing fasciitis. 4.0	
6.	. Explain the pathogenesis of necrotizing fa	sciitis and virulence factors that affect this	
	pathogenesis.	4.0	

	7.	Explain why surgery, even amputation, is often necessary in the treatment of necrotizing	B
		fasciitis.	4.0
	8.	Identify the most important infectious causes of osteomyelitis.	4.0
	9.	Describe the important virulence factors for all organisms associated with osteomyelitis	and the
		contribution of each to the pathogenesis.	4.0
	10.	Describe the routes by which various microbes gain access to bone and explain why the	se
		lesions are often polymicrobial.	4.0
	11.	Explain the use of surgical debridement and prolonged bactericidal antibiotic therapy in	chronic
		osteomyelitis.	4.0
	12.	Explain how laboratory procedures could distinguish among the causative agents of	
		osteomyelitis.	3.0
	13.	Identify the most important infectious cause of gas gangrene.	4.0
	14.	Explain the identification of <i>Clostridium perfringens</i> from gangrenous tissue and describ-	e the
		diagnosis and treatment of the disease.	3.5
	15.	Identify the important virulence factors for C. perfringens and how these factors contrib	ute to
		the virulence of the organism.	3.5
	16.	Explain gas gangrene's infrequency despite the presence of the organism in human inter	stines
		and in soil.	2.0
	17.	Explain the role of wounds in the pathogenesis of gas gangrene.	3.0
18. Explain why anaerobic or necrotic wounds are typically necessary for the develop		of	
		tetanus.	3.5
	19.	Explain the prevention of tetanus.	4.0
	20.	Identify the condition in infants that has been associated with the ingestion of raw or	
		unpasteurized honey.	3.0
	21.	Describe the important virulence factors for Clostridium tetani and the contribution of t	hese
		factors to the pathogenesis of tetanus.	3.5
А.	Pat	thogens	
[Ap	ply t	the following learning objectives to each of the pathogens that follow]	

	- · ·		
1.	Descrit	be the clinical case setting in which each disease would be found.	3.5
2.	Descrit	be the microbial pathogens known to cause the disease.	4.0
3.	Descrit	be the pertinent microbial structures related to virulence, including toxins.	3.5
4.	Descrit	be the pertinent biochemical pathways related to pathogen virulence.	2.0
5.	Describ	be the epidemiology of each disease.	4.0
6.	Describ	be the etiology/pathogenesis of each disease.	4.0
7.	Describ	be the clinical aspects of each disease.	4.0
8.	Describ	be the immune response to each pathogen.	3.0
9.	Describ	be methods of diagnosis of each disease.	3.5
10.	Describ	be current therapy (and antibiotic resistance) for each disease.	3.0
11.	Describ	be methods of prevention of each disease.	3.5
	a.	Staphylococcus aureus: Scalded skin syndrome, carbuncle, furuncle, folliculitis,	impetigo,
		wound infection, toxic shock syndrome	4.0
	b.	Streptococcus pyogenes: Impetigo, erysipelas, cellulitis, necrotizing fasciitis, gas	5
		gangrene, scarlet fever, toxic shock syndrome	4.0
	с.	Clostridium perfringens: Gas gangrene	4.0
	d.	Clostridium tetani: Tetanus	4.0
	e.	Propionibacterium acnes: Acne	3.0
	f.	Mycobacterium leprae: Leprosy	2.0

g.	Treponema pallidum: Syphilis	3.0
h.	Treponema sp.: Yaws, pinta	1.0
i.	Borrelia burgdorferi: Lyme disease	4.0
j.	Rickettsia rickettsii: Rocky Mountain spotted fever	4.0
k.	Rickettsia typhi: Endemic typhus	2.0
Ι.	Rickettsia prowazekii: Epidemic typhus	2.0
m.	Erysipelothrix rhusiopathiae: Erysipeloid	2.0
n.	Nocardia sp.: Cutaneous nocardiosis	3.0
о.	Malassezia furfur: Tinea versicolor	3.0
p.	Microsporum, Trichophyton, and Epidermophyton: Tinea corporis, tinea pedis, ti	nea
	cruris, tinea nigra, onychomycosis	4.0
q.	Sporothrix schenckii: Sporotrichosis	3.5
r.	Phialophora and Cladosporum: Chromomycosis	3.0
s.	Petriellidium and Madurella: Mycetoma	2.0
t.	Coccidioides immitis: Coccidiodomycosis	3.5
u.	Cryptococcus neoformans: Cryptococcosis	3.0
ν.	Blastomyces dermatiditis: Blastomycosis	3.5
w.	Leishmania tropica: Cutaneous leishmaniasis	2.0
х.	Leishmania braziliensis: Mucocutaneous leishmaniasis	2.0
у.	Hookworms (Ancylostoma and Necator): Cutaneous larval migrans	3.0
z.	Onchocerca volvulus: Onchocerciasis	1.0
aa.	Papillomaviruses: Warts	4.0
bb.	Poxviruses – Molluscum contagiosum (fleshy papules) and Smallpox	3.5
cc.	Herpes Simplex, Coxsackievirus: Vesicles	4.0
dd.	Measles, Rubella, Dengue, Parvovirus B19: Maculopapular rash	3.0
ee.	Candida spp.: thrush	3.5
ff.	Trichinella spiralisis: trichinosis	2.0

#### XII. <u>Nervous System Infections</u>

1.	Differentiate meningitis from encephalitis.	3.5	
2.	Identify the organisms that cause 80% of cases of bacterial meningitis beyond the neonatal		
	period.	3.0	
3.	Identify the common causes of bacterial meningitis in infants less than 1 month of age.	3.0	
4.	Describe host factors that may increase the risk for bacterial meningitis.	3.0	
5.	Define aseptic meningitis.	3.5	
6.	Describe the pathogenesis, clinical signs and symptoms, and diagnostic techniques that	allow for	
	differentiation among bacterial, viral, and fungal meningitis and encephalitis.	4.0	
7.	. Describe the pathogenesis, clinical signs and symptoms, diagnostic techniques, and treatments		
	of S. pneumoniae, N. meningitidis, H. influenzae, Group B strep, E. coli, L. monocytogene	s, and	
	M. tuberculosis as bacterial causes of meningitis/encephalitis.	3.5	
8.	Describe the pathogenesis, clinical signs and symptoms, diagnostic techniques, and trea	tments	
	for enteroviruses, Mumps, arboviruses, LCMV, herpesviruses, influenza viruses, HIV, CMV, and		
	Rubella as viral causes of meningitis/encephalitis.	3.0	
9.	Describe the pathogenesis, clinical signs and symptoms, diagnostic techniques, and trea	tments	
	of C. albicans, C. neoformans, C. immitis, and H. capsulatum as fungal causes of		
	meningitis/encephalitis.	3.0	

10.	Describe the pathogenesis, clinical signs and symptoms, diagnostic techniques, and	treatments
	ofof <i>T. gondii</i> and <i>P. falciparum</i> as parasitic causes of meningitis/encephalitis.	3.0

- 11. Describe the pathophysiology of subacute sclerosing panencephalitis and progressive multifocal leukoencephalopathy. **2.0**
- 12. Describe the pathogenesis, inheritance pattern, and course of disease for both Creutzfeldt-Jakob disease (CJD) and variant CJD. **3.0**
- Describe the risks to the fetus associated with Group B streptococci carriage as part of the normal vaginal flora in a pregnant woman.
   **3.0**

#### XIII. <u>Respiratory Tract Infections</u>

1.	Define <i>rhinitis</i> .	3.0	
2.	Identify the types of viruses that cause most cases of rhinitis.	3.0	
3.	Describe the means by which rhinitis viruses are spread.	3.0	
4.	Identify the major host defenses preventing infections by rhinitis viruses.	3.0	
5.	Identify the recommended treatment for rhinitis.	2.0	
6.	Define <i>pharyngitis</i> .	3.5	
7.	Identify the characteristics and describe the means of spread of viruses causing pharyngitis:		
	a. Rhinoviruses	3.5	
	b. Adenoviruses	3.5	
	c. Coronaviruses	3.5	
	d. Epstein-Barr virus	3.5	
8.	Describe the treatment for viral pharyngitis.	2.0	
9.	Identify the virulence factors, normal reservoirs, and mode of transmission of the bacter	rial	
	causes of pharyngitis:		
	a. Streptococcus pyogenes	3.0	
	b. Corynebacterium diphtheria	3.0	
	c. Neisseria gonorrhoeae	3.0	
10.	Describe the method of diagnosing bacterial pharyngitis.	3.0	
11.	Identify complications of infection by <i>Streptococcus pyogenes</i> and describe the events that lead		
	to the complications.	4.0	
12.	Identify the antibiotics used to treat bacterial pharyngitis.	3.0	
13.	Define <i>sinusitis</i> .	3.0	
14.	Identify the major bacterial causes of sinusitis and identify characteristics, normal reserv	/oirs,	
	and virulence factors associated with each.	3.0	
15.	Identify the major host defenses that protect against sinusitis-causing bacteria.	2.0	
16.	Identify the factors that predispose a patient to sinusitis.	2.0	
17.	Identify the major complication of sinusitis.	2.0	
18.	Describe recommended treatment recommended of sinusitis.	2.0	
19.	Define otitis media.	3.0	
20.	Identify the major bacterial causes of otitis media and identify characteristics, normal re	servoirs,	
	and virulence factors associated with each.	3.0	
21.	Identify the major host defenses that protect against bacteria that cause otitis media.	2.0	
22.	Identify the factors that predispose a patient to otitis media.	2.0	
23.	Identify the major complication of otitis media.	3.0	
24.	Describe recommended treatment of otitis media.	3.0	
25.	Define <i>bronchitis</i> .	2.0	

26. Identify the types of infectious agents that are involved in most cases of bronchitis a bronchiolitis.	nd <b>3.0</b>
27. Identify the clinical presentation associated with bronchitis- and bronchiolitis-causin	g agents. <b>3.0</b>
28 Identify the characteristics, attachment mechanisms, major virulence factors, and m	echanisms
of pathogenesis for each etiologic agent of bronchitis and bronchiolitis.	2.0
29. Describe the means by which the etiologic agents of bronchitis and bronchiolitis are	spread.
, , ,	3.0
30. Identify the major host defenses preventing infection by agents causing bronchitis a	nd
bronchiolitis.	2.0
31. Describe the method of diagnosing bronchitis and bronchiolitis.	2.0
32. Describe recommended treatments of bronchitis and bronchiolitis.	2.0
33. Define <i>pneumonia</i> .	3.5
34. Differentiate between acute and chronic pneumonia.	3.0
35. Name the major bacterial agents causing pneumonia and describe the clinical preser	ntations
associated with each.	3.5
36. Describe the normal reservoir of each bacterial agent of pneumonia.	3.0
37. Identify the major fungal agents causing pneumonia and describe the clinical presen	tations
associated with each.	3.5
38. Describe the normal reservoir of each fungal agent of pneumonia.	3.0
39. Identify the major viral agents causing pneumonia and describe the clinical presenta	tion
associated with each.	3.0
40. Describe the normal reservoir of each viral agent of pneumonia.	3.0
41. Identify pneumonia agents suggested by environmental history.	3.0
42. Discuss the differential diagnosis of cavitary lesion on chest radiograph.	2.0
43. Identify the characteristics and attachment mechanisms of each etiologic agent of pl	neumonia.
44. Describe the major viewlance factors and machanisms of astherappacie of each infact	1.0
44. Describe the major virulence factors and mechanisms of pathogenesis of each mect	
OF prieumonia.	3.0
45. Describe the major bost defenses preventing infection by these agents causing pneur	<b>0.c</b> nonia
40. Identify the major host defenses preventing infection by these agents causing pried	3 0
17 Describe recommended treatment of pneumonia	3.0
48. Describe the nathogenesis clinical signs and symptoms courses of clinical disease d	liagnosis
and treatment of pulmonary tuberculosis.	3.5
49. Define <i>miliary tuberculosis</i> and describe its clinical manifestations.	3.0
50. Describe the pathogenesis of primary respiratory infection caused by the influenza v	irus
followed by bacterial pneumonia.	3.5

# XIV. Zoonotic and Opportunistic Infections

1.	Describe the disease(s) caused by, the animal reservoir(s) of, and the mode of transmission for
	the following etiologic agents:

a.	Bacillus anthracis	3.5
b.	Yersinia pestis	3.5
c.	Pasteurella multocida	3.5
d.	Francisella tularensis	3.5

	e.	Brucella spp.	3.5
	f.	Borrelia spp.	3.5
	g.	Rabies virus	3.5
	h.	Viral hemorrhagic fever viruses	3.5
2.	Describ	be the following opportunistic infections	
	a.	Enterobacter	3.0
	b.	Vibrio vulnificus	3.0
	с.	Haemophilus influenzae (nontypeable)	3.0
	d.	Eikenella corrodens	3.0
	e.	Pseudomonas aeruginosa	3.0
	f.	Actinomyces	3.0
	g.	Bacteroides	3.0
	h.	Fusobacterium	3.0
	i.	Prevotella	3.0
	j.	Porphyromonas	3.0
	k.	Peptostreptococcus	3.0

# **PATHOLOGY, PART I LEARNING OBJECTIVES**

Cell Adaptation, Injury, and Death Inflammation Control of Cell Growth and Repair Fluid and Hemodynamics Coagulation Genetics Immunity Neoplasia Infectious Disease Environmental Pathology Nutritional Disease Principles of Laboratory Testing

#### I. <u>Cell Adaptation, Injury, and Death</u>

1.	Define and use in proper context:	
	a anthrocosis	4.0
	h anontosis	4.0
	c. atrophy	4.0
	d. autopsis	4.0
	e. autophasy	4.0
	f. bilirubin	4.0
	g. cellular swelling	4.0
	h. free radicals	4.0
	i. ganarene	4.0
	i. heat-shock protein	4.0
	k. hemosiderin	4.0
	I. hemosiderosis	4.0
	m. heterophagy	4.0
	n. homeostasis	4.0
	o. hyaline	4.0
	p. hyperplasia	4.0
	q. hypertrophy	4.0
	r. hypoxia	4.0
	s. infarct	4.0
	t. ischemia	4.0
	u. <i>karyolysis</i>	4.0
	v. karyorrhexis	4.0
	w. lipofuscin	4.0
	x. melanin	4.0
	y. metaokasua	4.0
	z. necrosis	4.0
	aa. neoplasia	4.0
	bb. <i>pyknosis</i>	4.0
	cc. reperfusion injury	4.0
	dd. steatosis	4.0
	ee. telomeres	4.0
2.	Compare cell and tissue adaptation, reversible cell injury, and irreversible cell injury	(cell death)
	in terms of etiology, pathogenesis, and morphology (gross, histologic, and ultrastruct	tural).
		3.3
3.	Understand the biochemical mechanisms associated with both reversible and irrever injury, including:	sible cell
	a. depletion of ATP:	3.3
	b. mitochondrial damage;	3.3
	c. influx of calcium and loss of calcium homeostasis:	3.3
	d. damage to DNA; and	3.3
	e. defects in membrane permeability.	3.3
4.	Explain free radicals, including their generation, their roles in cell injury, and their rel	noval.
		3.0

5. Discuss the mechanisms for the following types of injury:

	a.	hypoxic	3.0
	b.	ischemic	3.0
	с.	reperfusion injury	3.0
	d.	chemical (toxic) injury	3.0
6.	Explair	hypertrophy, hyperplasia, atrophy, and metaplasia in terms of physiologic vers	sus
	pathol	ogic, etiologies and the mechanisms of their development.	2.7
7.	Compa	are and contrast the morphologic differences in the following types of necrosis,	including
	comm	on sites or tissues where the processes occur as well as causative mechanisms	
	a.	Coagulative	3.0
	b.	Liquefactive	3.0
	с.	Gangrenous	3.0
	d.	caseous	3.0
	e.	fat	3.0
	f.	fibrinoid	3.0
8.	Explair	n the process of apoptosis, including physiologic and pathologic apoptosis, morp	bhology,
	and m	echanisms.	2.7
9.	Discus	s the significance of intracellular accumulations and the mechanisms for the acc	cumulation
	of		
	a.	lipids;	3.0
	b.	proteins;	3.0
	с.	glycogen;	3.0
	d.	endogenous pigments;	3.0
	e.	exogenous pigments; and	3.0
	f.	hyaline change.	3.0
10	. Compa	re and contrast dystrophic and metastatic calcification in terms of etiology/pat	hogenesis,
	morph	ologic appearance, and clinical significance.	3.3
11	. Explair	n mechanisms of cellular aging, including telomere shortening, environmental in	sults, DNA
	repair	defects, and abnormal growth factor signaling.	1.7
Inf	lamma	tion	

1. Define and use in proper context:

Π.

-		
a.	abcess	3.7
b.	activation	3.7
c.	adhesion	3.7
d.	anaphylactoxin	3.7
e.	cellulitis	3.7
f.	chemotaxis	3.7
g.	cytokine	3.7
h.	edema	3.7
i.	effusion	3.7
j.	emigration	3.7
k.	erosion	3.7
I.	exudates	3.7
m.	fibrosis	3.7
n.	granuloma	3.7
0.	inflammation	3.7
p.	integrins	3.7

	q.	margination	3.7	
	r.	opsonin	3.7	
	s.	organization	3.7	
	t.	phagocytosis	3.7	
	u.	purulent	3.7	
	v.	pus	3.7	
	w.	pyogenic	3.7	
	х.	resolution	3.7	
	v.	rollina	3.7	
	, Z.	selectins	3.7	
	aa.	serosanauineous	3.7	
	bb.	serous	3.7	
	сс.	sunnurative	3.7	
	bh bh	transudate	3.7	
	60. 60	ulcer	3.7	
2	Identify	stimuli that trigger an acute inflammatory reaction	2.0	
2. 2	Describ	the classic vascular changes and cellular events of acute inflammation	2.0	
ג. ⊿	Discuss	the five cardinal signs of inflammation in terms of nathogenesis and merphole	<b>3.</b> /	
4.	Discuss	the five calcular signs of final infation in terms of pathogenesis and morpholo	ву. <b>Э Э</b>	
-	Decerit		<b>3.</b> /	
5.	Describ	e the mechanisms responsible for increased vascular permeability in acute init	ammation.	
c		the following chamical mediators of inflormation in terms of their origin (call	3.2	
6.	placma) interrelationships, and their chief functions.			
	piasma	), interrelationships, and their chief functions:		
	a.	arachidonic acid metabolites	3.2	
	b.	chemokines	3.2	
	с.	coagulation cascade	3.2	
	d.	complement cascade	3.2	
	e.	cytokines	3.2	
	f.	kinin system	3.2	
	g.	lysosomal granule contents	3.2	
	h.	neuropeptides	3.2	
	i.	nitric oxide	3.2	
	j.	oxygen-derived free radicals	3.2	
	k.	platelet activating factor	3.2	
	١.	vasoactive amines	3.2	
7.	Discuss	Discuss each of the following in terms of the role it plays in the development of either the acute		
	or chro	nic inflammatory reaction:		
	a.	adhesion molecules	3.5	
	b.	endothelial cells	3.5	
	с.	eosinophils	3.5	
	d.	fibroblasts	3.5	
	e.	giant cells	3.5	
	f.	lymphocytes	3.5	
	g.	mast cells/basophils	3.5	
	h.	monocyte/macrophage	3.5	
	i.	neutrophils	3.5	
	i.	plasma cells	3.5	
	, k.	platelets	3.5	
		•		
agent by	8. Describe the steps involved in the isolation and destruction of an infectious ag	8.		
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3.0	neutrophils.			
3.0	9. Describe important related extracellular and intracellular factors.	9.		
, pathogenesis,	10. Compare and contrast acute and chronic inflammation in terms of etiology, pa	10.		
3.5	morphology, laboratory findings, outcomes, and systemic effects.			
ination of an	11. Compare and contrast resolution and organization with respect to the termina	11.		
2.8	inflammatory response.			
nd pathogenesis.	12. Compare and contrast lymphangitis and lympadenitis in terms of etiology and	12.		
3.3				
yte adhesion;	13. Discuss defects in leukocyte function, especially inherited defects in leukocyte	13.		
dal activity; and	inherited defects in phagolysosome function; inherited defects in microbicidal			
2.8	acquired deficiencies.			

14. Discuss the systemic effects of inflammation, including pathogenesis, laboratory values, and clinical signs and symptoms. 3.3

#### **Control of Cell Growth & Repair Objectives** III.

1. Define and use in proper context:

	a.	angiogenesis	4.0
	b.	autocrine	4.0
	с.	contact inhibition	4.0
	d.	contraction	4.0
	e.	continuously dividing cells	4.0
	f.	contracture	4.0
	g.	dehiscence	4.0
	h.	endocrine	4.0
	i.	fibrosis	4.0
	j.	granulation tissue	4.0
	k.	keloid	4.0
	١.	metalloproteinase	4.0
	m.	nondividing cells	4.0
	n.	organization	4.0
	0.	paracrine	4.0
	р.	pluripotent	4.0
	q.	proud flesh	4.0
	r.	quiescent cells	4.0
	s.	regeneration	4.0
	t.	repair	4.0
	u.	scar	4.0
	٧.	stem cells	4.0
2.	Disting	uish between continuously dividing cells (labile cells), quiescent cells (stable), an	d
	nondiv	ding cells (permanent) and categorize cells accordingly.	3.0
3.	Describ	e the role of stem cells in tissue regeneration and maintenance.	2.8
4.	Compa	re and contrast embryonic stem cells and somatic stem cells.	2.0
5.	Describ	e the cell cycle and define the following abbreviations (M, G <sub>0</sub> , G <sub>1</sub> , S, and G <sub>2</sub> ).	2.3
6.	Discuss	the actions of epidermal growth factor, transforming growth factor, fibroblast g	growth

factor 1 and 2, transforming growth factor, platelet derived growth factor, and vascular

		tions.
7	Discuss the relate of exterines, specifically TNF and U.1. in repair	3.5
7. 0	Explain the role of recenters, specifically five and IC-1, in repair.	<b>5.5</b>
ο.	explain the fole of receptors, signal transouction pathways, and transcription factors in	<b>1</b> 10
٥	Define the following terms and describe their role in tissue repair and regeneration:	2.3
9.	cadharing	2.0
	a. cuurernis b. elastic fibers	2.0
	b. Elastic Jibers	2.0
	d elastin	2.0
	u. elastin	2.0
	e. Integrins f collagen type I	3.0
	a collagen type I	2.0
	g. collagen type ll	2.0
	i. collagen lV	2.0
	i fibrillin	2.0
	j. jibrinin k laminin	2.0
	K. Minimi	2.0
	m proteoglycans	2.0
	n bengrin sulfate	2.0
۱n	Discuss the mechanisms of angiogenesis including the growth factors important to the	<b>5.0</b>
10.		<b>3.3</b>
11.	Compare and contrast healing by first intention (primary union) and second intention	
	(secondary union).	10
		4.0
L2.	. Describe the local and systemic factors that influence wound healing, including whethe	r each of
12.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> </ul>	er each of <b>4.0</b>
12. 13.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula</li> </ul>	r each of 4.0 tion
12. 13.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> </ul>	4.0 r each of 4.0 tion 4.0
12. 13. 14.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> </ul>	4.0 r each of 4.0 tion 4.0 4.0
12. 13. 14. <b>-Iu</b>	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> </ul>	4.0 r each of 4.0 ation 4.0 4.0
12. 13. 14. <b>Flu</b> 1.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> </ul>	4.0 r each of 4.0 tion 4.0 4.0 hanisms
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12. 13. 14. <u>F<b>I</b>U</u>	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>hid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechand be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> </ul> </li> </ul>	4.0 r each of 4.0 4.0 4.0 4.0 4.0 4.0 4.0
12. 13. L4. <b><u>=</u>IU</b>	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>iid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechand be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> </ul> </li> </ul>	4.0 r each of 4.0 4.0 4.0 4.0 4.0 4.0 4.0
12. 13. 14. <u>Flu</u>	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>iid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechand be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> <li>e. inflammation</li> </ul> </li> </ul>	4.0 r each of 4.0 tion 4.0 4.0 4.0 4.0 4.0 4.0 4.0
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12. 13. 14. 1.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>tid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechanism and be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> <li>e. inflammation</li> </ul> </li> <li>Compare edema of the following on the basis of pathogenesis morphologic changes, ar effects: <ul> <li>a. subcutaneous tissue:</li> </ul> </li> </ul>	4.0 r each of 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 d.0 d.0 d.0
12. 13. 14. <b>:1</b> u	<ul> <li>Describe the local and systemic factors that influence wound healing, including whether these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>tid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechand be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> <li>e. inflammation</li> </ul> </li> <li>Compare edema of the following on the basis of pathogenesis morphologic changes, ar effects: <ul> <li>a. subcutaneous tissue:</li> <li>i. dependent edema</li> </ul> </li> </ul>	4.0 r each of 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0
12. 13. 14.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>tid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechand be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> <li>e. inflammation</li> </ul> </li> <li>Compare edema of the following on the basis of pathogenesis morphologic changes, ar effects: <ul> <li>a. subcutaneous tissue:</li> <li>i. dependent edema</li> <li>ii. pitting edema</li> </ul> </li> </ul>	4.0 r each of 4.0 ation 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0
12. 13. 14. <b>∃IU</b> 1.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whether these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>tid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechand be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> <li>e. inflammation</li> </ul> </li> <li>Compare edema of the following on the basis of pathogenesis morphologic changes, ar effects: <ul> <li>a. subcutaneous tissue:</li> <li>i. dependent edema</li> <li>ii. pitting edema</li> </ul> </li> </ul>	4.0 r each of 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 d clinical 4.0 4.0
12. 13. 14. 11.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whether these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>tid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mechanism and be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> <li>e. inflammation</li> </ul> </li> <li>Compare edema of the following on the basis of pathogenesis morphologic changes, ar effects: <ul> <li>a. subcutaneous tissue:</li> <li>i. dependent edema</li> <li>ii. pitting edema</li> </ul> </li> </ul>	4.0 r each of 4.0 ation 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0
12. 13. 14. <u>F<b>I</b>U</u> 1. 2.	<ul> <li>Describe the local and systemic factors that influence wound healing, including whethe these accelerates or delays the rate of healing.</li> <li>Discuss the pathologic aspects of repair including contracture, keloid, excessive granula (proudflesh), ulceration, fibrosis, wound dehiscence, and hypertrophic scar.</li> <li>Describe aspects of cutaneous wound healing as it relates to wound strength.</li> <li><b>tid and Hemodynamics</b></li> <li>Discuss the pathogenesis of edema, giving examples associated with the following mecland be able to describe it as being systemic or localized <ul> <li>a. reduced plasma oncotic pressure</li> <li>b. increased hydrostatic pressure</li> <li>c. sodium retention</li> <li>d. lymphatic obstruction</li> <li>e. inflammation</li> </ul> </li> <li>Compare edema of the following on the basis of pathogenesis morphologic changes, ar effects: <ul> <li>a. subcutaneous tissue:</li> <li>i. dependent edema</li> <li>ii. pitting edema</li> </ul> </li> <li>b. lungs</li> <li>c. brain</li> </ul>	4.0 r each of 4.0 ation 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0

IV.

4.	escribe chronic passive congestion of the lungs, liver, kidneys, and spleen, in terr	ms of			
5.	compare acute and chronic hemorrhage in terms of common causes, clinical man	ifestations,			
	nd compensatory mechanisms.	3.0			
6.	Describe the following stages of shock, in terms of pathophysiology, morphologic changes,				
	rognosis:				
	a. non-progessive (compensated)	3.0			
	<ul> <li>progressive (decompensated)</li> </ul>	3.0			
	c. irreversible	3.0			
7.	compare and contrast the following types of shock in terms of pathogenic mechan	nism, common			
	auses, structural changes, functional changes, and clinical features and prognose	s:			
	a. Neurogenic	3.3			
	b. Hypovolemic	3.3			
	c. Hemorrhagic	3.3			
	d. Septic	3.3			
	e. Cardiogenic	3.3			
	f. Anaphylactic	3.3			
8.	ist the morphologic changes and functional effects of shock on the lungs, kidneys	s, adrenals,			
	rain, and gastrointestinal tract.	2.7			
9.	escribe thrombi in terms of factors conditioning the development of thrombi and	d possible fate			
	f thrombi.	3.3			
10.	Distinguish between venous thrombi and arterial thrombi on the basis of:				
	a. etiologic and precipitating factors	3.3			
	b. common sites of occurrence	3.3			
	c. type and size of vessel involved	3.3			
	d. morphologic appearance	3.3			
	e. organs commonly involved	3.3			
	f. local and distant effects	3.3			
	g. fate of lesions and prognosis	3.3			
	h. clinical and laboratory features	3.3			
11.	compare and contrast the following types of emboli with emphasis on defining m	orphologic			
	eatures, etiologic/precipitating factors, organs commonly involved, type and size	of vessels			
	volved, complications, and clinical manifestations:				
	a. pulmonary	3.3			
	b. systemic	3.3			
	c. fat	3.3			
	d. air	3.3			
	e. amniotic fluid	3.3			
12.	compare and contrast arterial and venous infarcts on the basis of location, pathog	genesis,			
	norphology, and clinical manifestations.	3.0			

### V. <u>Coagulation</u>

1. Outline the process of normal hemostasis, in terms of the following and describe the role and interaction of each of the following elements of hemostasis:

a.	intrinsic pathway	3.0
b.	extrinsic pathway	3.0
c.	final common pathway	3.0

	d.	fibrin formation and fibrinolysis	3.0
	e. f	rolo of platelets	5.U 2.0
	ו. מ	role of vascular integrity	3.0
	g. h	events in dissolution of a thrombus	3.0
2	Discus	s thrombocytopolesis in terms of morphology of megakanyocytes: fate of m	ogakarvorvtes
۷.	life sna	an of platelets: factors which influence thrombocytopolesis: and abnormal n	nornhologic
	forms	of platelets and megakaryocytes	<b>2.2</b>
З	Discus	s thrombocytopenia in terms of differential diagnosis clinical features bone	marrow
5.	mornh	ology and laboratory findings	2.4
4	Compa	are and contrast bleeding due to vascular defect (localized or generalized), n	latelet defect.
	and co	agulation defect, in terms of:	
	a.	etiologic/precipitating factors	2.6
	b.	genetics	2.6
	c.	common sites of occurrence	2.6
	d.	organs affected	2.6
	e.	type and size of vessels involved	2.6
	f.	results, complications, and fate of lesions	2.6
	g.	clinical features	2.6
	h.	laboratory findings	2.6
5.	Discus	s thrombocytosis in terms of diagnosis and differential diagnosis.	2.2
6.	Outlin	e the processes for stepwise evaluations of bleeding patients, patients with	suspected
	platele	t disorder, and patients with suspected hypercoagulability.	3.2
7.	List an	d discuss the laboratory diagnostic procedures used to approach patients w	ith bleeding
	disord	ers and thrombotic disorders.	3.2
8.	Compa	are and contrast bleeding disorders due to the following, in terms of etiology	, genetics,
	pathog	genesis, clinical presentation, laboratory diagnosis, and clinical course:	
	a.	factor VII deficiency (hemophilia A)	2.8
	b.	factor IX deficiency (hemophilia B)	2.8
	с.	factor XI deficiency (hemophilia C)	2.8
	d.	von Willebrand disease	2.8
	e.	vitamin K deficiency	2.8
	f.	liver disease	2.8
9.	Discus	s disseminated intravascular coagulopathy (DIC) in terms of etiology, pathog	genesis,
	morph	ologic features, clinical presentation and course, laboratory diagnosis, and c	complications
	and pr	ognosis.	2.6
10.	Explair	n the hypercoagulable state in terms of Virchow's triad.	3.4
11.	Descri	be the mechanism(s) by which aspirin, NSAIDs, coumadin (warfarin), and he	parin affect
	hemos	tasis, and discuss the methods by which each is monitored.	3.5
Ge	netics		
1	Define	the following and use in proper context:	
÷.	Cunc		

a.	agenesis	3.3
b.	aneuploid	3.3
c.	aplasia	3.3
d.	autosomal dominant	3.3
e.	autosomal recessive	3.3

VI.

f.	balanced translocation	3.3
g.	carrier	3.3
h.	chromosomal disorders	3.3
i.	chromosome	3.3
j.	codon	3.3
k.	congenital abnormality	3.3
١.	congenital disease	3.3
m.	deletion	3.3
n.	diploid	3.3
0.	DNA	3.3
p.	dominant	3.3
а.	double minute	3.3
r.	euploid	3.3
s.	expressivity	3.3
t.	familial disease	3.3
U.	appe	3.3
v.	genetic disease	3.3
w	genetic heterogeneity	3.3
v	genetic heterogenety	2 3
л. v	hanloid	2.2
y. 7	hereditary disease	2.2
2.	heterozyanus	2.2
hh	homozygous	2.2
00. CC	insertions	2.2
dd	inversion	2.2
uu.	kanyotyne	22
ff	linkage	2.2
11. aa	luan hunathasis	3.3 2 2
gg.	nyounasis	3.3 2 2
::	maigania	3.3 2.2
	mitosis	3.3
]]. L.L.		3.3
КК.	monosomy	3.3
	mosaicism	3.3
mn	n. <i>MRNA</i>	3.3
nn.	multifactorial inneritance	3.3
00.	mutation	3.3
pp.	neonatal	3.3
qq.	nondisjunction	3.3
rr.	operator gene	3.3
SS.	operon	3.3
tt.	phenotype	3.3
uu.	polysomy	3.3
vv.	pleiotropy	3.3
ww	n. recessive	3.3
XX.	regulatory gene	3.3
уу.	ring chromosome	3.3
ZZ.	RNA	3.3
aaa	n. rRNA	3.3

	bbb. sex-linked	3.3
	ccc. structural gene	3.3
	ddd. teratogens	3.3
	eee. transcription	3.3
	fff. translocation	3.3
	ggg. trinucleotide-repeat mutation	3.3
	hhh. <i>Trisomy</i>	3.3
	iii. variable expressivity	3.3
	jjj. X-linked disorders	3.3
	kkk. Penetrance	3.3
2.	Compare and contrast congenital and familial abnormalities, and provide two examples	of each.
	Emphasis should be placed on demonstrating an understanding of etiology, morphology	,
	laboratory finding, and clinical features.	3.0
3.	Describe each of the following genetic diseases and provide examples of each:	
	a. Simple autosomal dominant	3.0
	b. Simple autosomal recessive	3.0
	c. X-linked recessive	3.0
4.	Given the mode of inheritance for a family history involving a disease with classic mende	elian
	inheritance, predict the likelihood of various phenotypes and genotypes in family memb	ers.
		2.0
5.	Given a family history or pedigree, indicate the most likely mode of inheritance	
	a. autosomal dominant;	3.0
	b. autosomal recessive;	3.0
	c. sex-linked dominant; and	3.0
	d. sex–linked recessive.	3.0
6.	Give three examples of diseases with multifactorial inheritance emphasizing the pathog	enesis,
	morphology, laboratory studies and clinical presentation.	2.0
7.	Give three examples of teratogenic agents and compare and contrast their methods of a	action.
		2.0
8.	List five common genetic abnormalities and describe in terms of pathogenesis, common	
_	features, and classification.	2.0
9.	Describe lysosomal storage diseases, citing mode of inheritance and major defect and cl	inical
	symptoms.	2.0
10.	Describe two major disorders involving the sex chromosomes.	1.7
11.	Describe Trisomy 21, including pathogenesis, morphology, clinical presentation, clinical	course,
	and complications.	2.0
12.	Describe the following in terms of methodology of performance of test, appropriateness	sin
	various types of clinical situations, and clinical implications:	
	a. Karyotyping	3.0
	b. KFLP	3.0
		3.0
4.2	a. DNA sequencing	3.0
13.	Describe the modes of inheritance of mitochondrial disorders and give two examples of	diseases
	associated with them.	2.0

### VII. <u>Immunity</u>

1. Define and use in proper context:

	a.	acute serum sickness	3.0
	b.	allergen	3.0
	с.	amyloid	3.0
	d.	anaphylaxis	3.0
	e.	anergy	3.0
	f.	antibody	3.0
	g.	anti-nuclear antibodies (ANA)	3.0
	h.	antigen	3.0
	i.	arthus reaction	3.0
	j.	atopy	3.0
	k.	autoimmunity	3.0
	Ι.	B lymphocytes	3.0
	m.	cellular rejection	3.0
	n.	dendritic cells	3.0
	0.	graft-versus-host disease	3.0
	p.	immunity, adaptive	3.0
	q.	immunity, cellular	3.0
	r.	immunity, humoral	3.0
	s.	immunity, innate	3.0
	t.	macrophages	3.0
	u.	MHC (major histocompatibility complex)	3.0
	٧.	natural killer cells	3.0
	w.	β-pleated sheet	3.0
	х.	rejection, acute	3.0
	у.	rejection, chronic	3.0
	Ζ.	rejection, hyperacute	3.0
	aa.	rheumatoid factor	3.0
	bb.	T lymphocytes	3.0
	CC.	tolerance, central	3.0
	dd.	tolerance, peripheral	3.0
	ee.	tolerance, self	
2.	Discuss	and classify the MHC (Major Histocompatabilit	y Complex) molecules as class I or II. 2.0
3.	Compa	re and contrast the four types of hypersensitivit	y reactions in terms of type of reaction,
	prototy	pic disorder, immune mechanisms, mediators,	pathologic lesions, and clinical disorders.
			3.0
4.	Compa	re and contrast hyperacute, acute, and chronic	transplant rejection in terms of etiology,
	pathog	enesis, and morphology.	3.0
5.	Define	immunologic tolerance and discuss the mechan	isms of both central and peripheral
	toleran	ce.	1.7
6.	Discuss	the mechanisms of autoimmune diseases in ter	rms of the breakdown of self-tolerance,
	enviror	imental triggers, and genetics.	3.0
7	Correla	te the following autoantibodies with the major	autoimmune disease(s) it is associated

7. Correlate the following autoantibodies with the major autoimmune disease(s) it is associated with and provide the diagnostic significance

a.	antinuclear (ANA)	3.0
b.	anti-double-stranded DNA	3.0
c.	SS-A (Ro)	3.0
d.	Nuclear RNP	3.0
e.	Anticentromere	3.0

		f.	anti-Smith (Sm)	3.0
		g.	antihistone	3.0
		h.	SS-B (La)	3.0
		i.	ScI-70	3.0
		j.	Jo-1	3.0
	8.	Discuss	and describe the genetics, etiology, immunologic basis, clinical presentation,	
		morph	biogy, and complications of the following primary immunodeficiencies	
		a.	x-linked agammaglobulinemia of Bruton	1./
		D.	common variable immunodeficiency	1./
		C.	DiGeorge syndrome (thymic hypoplasia)	1./
		d.	severe combined immunodeficiency syndrome	1./
	0	e. Diaguas	Wiskoul-Aldrich Syndrome	1./
	9.	Discuss	secondary immunodeficiencies (chemotherapy, diabetic, steroids) in terms of	atiologies. <b>3.0</b>
	10.	Discuss diagno	acquired immunodeficiency syndrome (HIV infection, AIDS) in terms of epidem stic criteria, incidence, risk factors, pathogenesis, immunologic defects, associa	niology, ted
		infectio	ons and neoplasms, morphology, and clinical presentation.	3.0
VIII.	<u>Ne</u>	oplasia		
	1.	Define		
		a.	cell proliferation	3.5
		b.	cell differentiation	3.5
		с.	adenoma	3.5
		d.	polyp	3.5
		e.	papilloma	3.5
	2.	Describ	e the principles of carcinogenesis, including fundamental genetic changes, unre	egulated
		cell pro	liferation, monoclonal nature of tumor cells, and loss of apoptosis.	3.0
	3.	Differe	ntiate between benign and malignant tumors.	3.8
	4.	Discuss	; the following terminology applied to tumors and explain how it reflects the tis	sue of
		origin:		
		a.	benign tumors (-oma suffix)	3.0
		b.	carcinoma (epithelial)	3.0
		С.	sarcoma (mesenchymal)	3.0
		d.	lymphatic	3.0
	5.	Compa	re and contrast anaplasia, dysplasia, and carcinoma in situ.	3.0
	6.	Compa	re and contrast features that characterize well differentiated tumors from poor	·ly
	_	differe	ntiated tumors.	3.5
	7.	Compa	re and contrast tumor invasion and tumor metastasis.	3.5
	8.	Descrit	e the three pathways by which tumors metastasize and give a common examp	le of each. 3.3
	9.	List the	e most common metastatic sites involved in breast, colon, lung, and prostate ca	ncer.
				3.3
	10.	List the	e steps involved in hematogenous spread of a tumor.	2.3
	11.	List the	e steps involved in invasion of ECM.	2.3
	12.	Describ	be the function of E cathedrins and their significance in invasion of ECM.	1.5
	13.	Briefly	describe the role of the enyme family of proteases in tumor metastasis includir	ig tumor
		, locatio	n, vascular drainage recognizing that carcinogenesis is a multistep process.	1.5

14. Discuss the factors which determine the site of metastasis.	2.5
15. Describe the metastasis of malignant tumors to regional lymp	oh nodes with emphasis on the
term "sentinel lymph node".	3.0
16. List the most frequent genetic mutations occurring in malign following functions:	ancies and categorize as to the
a. oncogenes	3.0
b. tumor suppressor genes	3.0
c. genes regulating apoptosis	3.0
d. DNA repair genes	3.0
17. Describe molecular changes leading to progression from nor	mal epithelium to carcinoma.
	3.0
18. Define <i>cahcexia</i> and explain why it is encountered in cancer	patients. <b>3.0</b>
19. Describe paraneoplastic syndrome and discuss its clinical sign	nificance. <b>3.0</b>
20. Compare and contrast <i>staging</i> and <i>grading</i> of malignant tum	ors. <b>3.3</b>
21. Discuss the different diagnostic procedures and laboratory m	ethods used in the diagnosis of
malignancies.	3.2
22. Discuss the following tumor markers and the significant asso	ciated cancer:
a. PSA	2.8
h CEA	2.8
c a-fetoprotein	2.8
d Estrogen	2.8
e. Progesterone	2.8
f Δlkolin nhosnhatase	2.8
g Beta HCG	2.8
23 Discuss the use of molecular techniques in assessing program	is and hereditary predisposition in
the diagnosis of cancer	1 8
24 Identify important tumor antigens and describe their known	11505 1 5
25. Discuss the function of immune cells involved in immunosury	veillance 20
26. Explain the mechanisms by which tymor cells escape immun	osurveillance 18
27. Discuss the role of gonder age, diet, and environment in the	development of malignancy
27. Discuss the role of gender, age, diet, and environment in the	
29 Define the terms profession and beterogeneity as they relate	to the behavior of tumor cells
28. Define the terms projession and neterogeneity as they relate	
Infactious Disease	3.0
<u>Infectious Disease</u>	
1. Discuss body defense mechanisms including nonspecific and	specific barriers to infection.
	4.0
2. List factors for predisposition to infection.	4.0
3. Describe tissue damage caused by infection.	4.0
4. Compare and contrast infectious organisms, transmission par	tterns and mechanisms of disease.
	4.0
5. Classify bacterial infections through morphology, aerobic/and	aerobic, gram positive/ negative,
gram stain, culture and sensitivity, and present of endo-/exo	toxins. 4.0
6. Identify and describe the following bacterial organisms:	
a. Bacillis anthracis	3.0
b. Bordetella pertussis	3.0
c. Borelia	3.0
d Campylobacter	3.0
a. Cumpyrobucter	5.0

IX.

	e.	Chlamydia	3.0
	f.	Clostridium	3.0
	g.	Diphtheria	3.0
	h.	E. coli	3.0
	i.	Enterobacter	3.0
	i.	Listeria	3.0
	j. k.	Mycobacterium	3.0
	1	Yconlasma	3.0
	m	Neisseria	3.0
	n.	Pseudomonas	3.0
		Salmonolla	2.0
	0. n	Shinolla	3.U 2 0
	μ. α	Staphylococcus	5.0
	q.	Staphylococcus	3.0
	r.	-	3.0
	S.	Ireponema	3.0
	t.	Vibrio cholera	3.0
	u.	Yersinia	3.0
7	<ol> <li>Classify</li> </ol>	morphology of viral organism, the method of diagnosis of viral infections titers	, and
	intrace	Ilular reproduction.	3.0
8	<ol> <li>Identify</li> </ol>	and describe the following viral diseases:	
	a.	Arbovirus	3.0
	b.	Enterovirus	3.0
	с.	Hanta Virus	3.0
	d.	Hepatitis viruses	3.0
	e.	Herpesvirus family	3.0
	f.	HIV	3.0
	g.	HPV	3.0
	h.	Influenza virus	3.0
	i.	Measles virus	3.0
	i.	Molluscum contagiosum virus	3.0
	k.	Mumps virus	3.0
	L	Rhinovirus	3.0
	m	RSV	3.0
	n	Rubella virus	3.0
	0		3.0
٩	Classify m	pornhology of infectious fungal organisms and diagnosis of infections through Ki	OH slides
5.	cultures	and snore identification with emphasis differentiating between deen and superf	icial
	fungal inf	and spore identification with emphasis unreferitiating between deep and super-	2 5
10	Idoptify a	nd describe the following fungal diseases:	3.5
10.		Asporaillus	2 E
	d. h	Asperginus Plastomycoc dormatitidic	3.3 3 E
	D.	Bidstoffiytes definiditions	3.5
	(.		3.5
	α.		3.5 2.5
	e.	cryptococcus neotormans	3.5
	t.	Epidermophyton	3.5
	g.	Histoplasma capsulatum	3.5
	h.	Microsporum	3.5
	i.	Pneumocystis	3.5

9.

j	. Sporothrix schenckii	3.5
k	k. Trichophyton	3.5
I.	. Zygomycosis	3.5
11. Ident	tify and describe the following prion infections:	
a	a. Kuru	1.5
k	<ol> <li>Creutzfeldt-Jakob disease</li> </ol>	1.5
C	c. Mad Cow	1.5

### X. Environmental Pathology

XI.

1.	Discuss the effects of tobacco abuse and pneumoconioses, with special emphasis on morphology, clinical effects, and comorbidities.	2.0
2.	Discuss ethanol, fetal alcohol syndrome, methanol, and ethylene glycol, in terms of mor	phology,
3.	Discuss the abuse of cocaine, amphetamines, narcotics, and marijuana in terms of morp	hology,
	clinical effects, and comorbidities.	3.3
4.	Discuss arsenic, lead, mercury, and organophosphate insecticides as heavy mental toxic with special emphasis on morphology, clinical effects, and comorbidities.	agents, <b>3.3</b>
5.	Discuss estrogen oral contraceptives (OCTs), NSAIDs, and acetaminophen as therapeutic	drugs,
	in terms of morphology, clinical effects, and comorbidities.	3.3
6.	Describe the following mechanical injuries in terms of cause and physical appearance:	
	a. Abrasion	4.0
	b. avulsion	4.0
	c. contusion	4.0
	d. incision	4.0
	e. laceration	4.0
	f. puncture wound	4.0
	g. stab wound	4.0
	h. asphyxia	4.0
	i. pattern injury recognition	4.0
	j. gunshot wounds (shotgun versus gunshot)	4.0
7.	Discuss the effects of systemic hyperthermia and frostbite in terms of pathophysiologic	
	mechanisms, morphology, clinical effects, and comorbidities.	3.8
8.	Differentiate full thickness burns versus partial thickness burns, specifically in terms of	
	pathophysiologic mechanisms, morphology, clinical effects, and comorbidities.	3.8
9.	Discuss the effects of electrical injuries, in terms of pathophysiologic mechanisms, morp	hology,
	clinical effects, and comorbidities.	3.8
10.	Discuss the effects of radiation injuries—both whole body and localized—in terms of	
	pathophysiologic mechanisms, morphology, clinical effects, and comorbidities.	2.6
Nu	tritional Disease	
1.	Compare and contrast primary and secondary dietary insufficiencies.	2.5
۷.	compare and contrast the following protein-energy mainutrition:	2.0
		2.0
	D. Marasmus	2.0
	c. secondary protein energy malnutrition	2.0

_		
3.	Describe fat soluble vitamin deficiencies and excesses for vitamins A, D, E, and K with resp	ect to
	morphology and clinical effects. 2	2.4
4.	Describe the water soluble vitamin deficiencies for vitamins B1, B2, B6, B12, C, niacin, fola	ite,
	panthothenic acid, and biotin, with respect to morphology and clinical effects. 2	2.4
5.	Describe the effects of the following mineral deficiencies: zinc, iron, iodine, copper, fluorid	de, and
	selenium. 2	2.0
6.	Describe the effects of obesity on hypertension, atherosclerosis, diabetes mellitus, and ca	ncer
	with emphasis on morphology, clinical effects, and co-morbidities. 3	3.2
7.	Describe the effects of Anorexia nervosa and bulimia, with emphasis on morphology, clinic	cal
	effects, and co-morbidities. 2	2.3
8.	Describe the effects of diet on cancer, with emphasis on morphology, clinical effects, and	со-
	morbidities. 2	2.0
9.	Describe the effects of diet on atherosclerosis, with emphasis on morphology, clinical effe	ects,
	and co-morbidities. 3	<b>.</b> 0

### XII. <u>Principles of Laboratory Testing</u>

3.

1.	Identify	units of measure used in basic laboratory principles and discuss collection and	
	transpo	ortation of specimens.	3.0
2.	Define	and use in proper context	
	a.	accuracy	3.0
	b.	analytic variable	3.0
	с.	anatomic pathology	3.0
	d.	autopsy	3.0
	e.	biopsy	3.0
	f.	clinical pathology	3.0
	g.	coefficient of variation	3.0
	h.	false negative	3.0
	i.	false positive	3.0
	j.	fine needle aspiration	3.0
	k.	frozen section	3.0
	I.	histopathology	3.0
	m.	incidence	3.0
	n.	postanalytic variable	3.0
	0.	precision	3.0
	p.	predictive value	3.0
	q.	prevalence	3.0
	r.	reference range	3.0
	s.	screening test	3.0
	t.	sensitivity	3.0
	u.	specificity	3.0
	٧.	standard deviation	3.0
	w.	surgical pathology	3.0
	х.	true negative	3.0
	у.	true positive	3.0
	Ζ.	turnaround time	3.0
D	escribe	the appropriate uses of clinical laboratories, surgical pathology, frozen sections,	
С	ytopath	ology, and autopsies.	3.0

4.	Calculate sensitivity and specificity, given raw data.	2.3
5.	Compare and contrast precision and accuracy.	3.0
6.	Explain the concept of quality assurance and its role in the clinical laboratory.	2.0
7.	Define reference range and understand its role in the diagnosis of disease.	3.0
8.	Explain and provide examples of the use of decision levels in clinical medicine.	3.0
9.	Describe the use of laboratory tests to screen for and to monitor disease.	3.3
10.	Discuss the effects of sample handling on laboratory results, including turnaround time, t	type of
	tube used for blood collection, timing of collection, transport, and storage.	2.0

# PATHOLOGY, PART II

# **SYSTEMIC DISEASE LEARNING OBJECTIVES**

Vascular Disease Cardiac Disease Chemistry of Cardiac Disease Hematopoietic System Disorders Myeloid Neoplasms Lymphoid Neoplasms **Pulmonary Disease** Gastrointestinal Disease Pathology of the Liver and Extrahepatic Biliary System Pancreatic Disease Genitourinary Disease Renal Disease **Renal Function Tests** Breast Disease **Endocrine Disorders** Diabetes Dermatopathology Joint Disease Bone Disease Soft Tissue Disease Head, Neck, and Special Sensory Organ Pathology Neuromuscular Disease Central Nervous System Disease

# I. Vascular Disease

II.

1.	Discuss the effects of age, sex, geographic location, and risk factors on the pathogenes prevalence of atherosclerosis	is and <b>4.0</b>
2	Outline the development of the atherosclerotic lesion with respect to pathogenic med	hanisms
	morphology, clinical manifestations, and complications.	4.0
3.	Discuss the following forms of vasculitis in terms of incidence, etiology, pathogenesis,	
	morphology, and clinical features, complications, and prognoses:	
	a. infectious vasculitis	3.0
	h. giant cell arteritis	3.0
	c. polyarteritis nodosa	3.0
	d hypersensitivity vasculitis	3.0
	e. thromboangijtis obliterans (Buerger disease)	3.0
4	Compare and contrast the following disorders in terms of etiology nathogenesis, type	and
	distribution of vessels involved, clinical features, and complications and prognoses:	unu
	a. atherosclerotic aneurysm	3.0
	b. svphilitic aneurysm	3.0
	c. aortic (dissecting) aneurysm	3.0
	d. cvstic medial necrosis	3.0
5.	Discuss the following disorders in terms of etiology, complications, and clinical feature	s and
	prognoses:	
	a. varicose veins	4.0
	b. thrombophlebitis	4.0
	c. lymphangiitis	4.0
	d. lymphedema	4.0
6.	Compare and contrast the pathophysiologic, morphologic, and clinical differences betw	veen
	atherosclerosis, arteriolosclerosis, and medial calcinosis.	3.0
7.	Discuss the differences between primary and secondary Raynaud phenomenon, with e	mphasis
	on the pathophysiology and clinical presentation.	3.5
<u>Ca</u>	rdiac Disease	
4		2.0
1.	List the most common forms of heart disease in the United States.	3.0
2.	Compare and contrast the following congestive heart failure, left-sided heart failure, a	na right-
	sided heart failure, in terms of etiology, pathogenesis, compensatory mechanisms, and	1
2	morphology.	3.0
3.	Compare and contrast the following items relied to congestive heart failure and be abl	e to use
	in proper context the following items: backward failure, foreward failure, and high-ou	
4	Tallure.	3.0
4.	Discuss congenital near disease in terms of ient-to-right and right-to-left shufts, with s	special
	attention to the most common forms of congenital heart disease (ventricular septal de	1 <b>7</b>
F	Discuss and acarditis muccarditis and paricarditis in terms of classification, anidamial	1./
э.	otiology/pathogenesis, merchology, clinical features, and progracis	<sup>ν</sup> εγ,
c	Compare and contrast acute require four and chronic requires is the set discourse in the	J.U uding
0.	compare and contrast acute meananchever and chronic meananchever and cutre condi-	uung
	patriogenesis, diagnostic criteria (Jones criteria), morphology (cardiac and extracardiac	·), <b>20</b>
	complications, and clinical leatures.	3.0

	etiology, pathogenesis, morphology, clinical features, morphology, clinical feature	es,
	complications, and prognosis:	
	a. calcific aortic stenosis	3.0
	b. aortic insufficiency	3.0
	c. mitral stenosis/insufficiency	3.0
	d. mitral valve prolapse	3.0
	e. mitral annular calcification	3.0
	f. tricuspid insufficiency	3.0
	g. pulmonic insufficiency	3.0
8.	Compare and contrast dilated (congestive) cardiomyopathy, hypertronic cardiom	yopathy, and
	restrictive cardiomyopathy in terms of etiology, pathogenesis, morphology, and c	linical course.
		3.0
9.	Describe coronary artery disease in terms of epidemiology, risk factors, etiologic	factors,
	pathogenesis, and complications.	4.0
10.	Describe myocardial infarct in terms of etiologic factors; risk factors; pathogenesi	s; morphology;
	clinical, laboratory, and electrocardiographic findings; complications; and progno	sis. <b>3.3</b>
11.	Compare and contrast right-sided and left-sided hypertensive heart disease in ter	ms of
	a. etiologic factors;	3.0
	b. pathogenesis;	3.0
	c. morphology;	3.0
	d. clinical features; and	3.0
	e. prognosis.	3.0
12.	Discuss sudden cardiac death in terms of cause, relationship to arrhythmias, and	cardiac
	morphology.	3.0
13.	Discuss the following cardiac tumors:	
	a. myxoma	1.0
	b. rhabdomyoma	1.0

### III. <u>Chemistry of Cardiovascular Disease</u>

IV.

1.	Describe the origin, function, and disease states seen with elevations of serum creatinin phosphokinase (CPK), cardiac troponing, and myoglobin.	e 4.0
2.	Describe the way that C-reactive protein (CRP), homocysteine, beta natriuretic peptide, lipds (triglycerides and HDL and LDL cholesterol) serve as markers for an increased risk of	and of
	cardiovascular disease.	3.5
3.	Describe familial hypercholesterolemia with emphasis on genetics, pathophysiology,	
	morphology, and clinical presentation.	3.5
4.	Compare and contrast causes of secondary hyperlipidemias.	3.0
He	matopoietic System	
1.	Discuss the significance of a bone marrow smear on each of the following:	

a.	Pronormoblast	1.6
b.	Normoblast	1.6
c.	Megaloblast	1.6
d.	Myeloblast	1.6

	e. Promyelocyte	1.6
	f. Myelocyte	1.6
	a Metamyelocyte	1.6
	g. Metallyclocyte	1.0
	h. band form	1.6
	i. neutrophil	1.6
	i basophil	1.6
	j, osopeniu	1.0
	k. eosmophil	1.0
	I. plasma cell	1.6
	m. lymphocyte	1.6
	n. megakarvocyte	1.6
2	Define and state the significance of the following red cell parameters:	
۷.	Denne and state the significance of the following red cell parameters.	2 2
	a. Herioyiobili	5.2
	b. Hematocrit	3.2
	c. mean corpuscular volume (MCV)	3.2
	d. mean corpuscular hemoalobin (MCH)	3.2
	e mean corpuscular hemoglohin concentration (MCHC)	3.2
	f red cell distribution width	2.2
	1. rea cell distribution width	3.2
	g. reticulocyte count	3.2
	h. <i>anemia</i>	3.2
3.	Compare and contrast anemia secondary to the following, in terms of incidence, eti	ology and
0.	nathogenesis marrow and nerinheral blood mornhology laboratory diagnostic crite	ria and
	pathogenesis, marrow and peripheral blood morphology, laboratory diagnostic crite	ina, anu
	clinical features and course:	
	a. acute versus chronic blood loss	3.2
	b. increased rate of destruction (hemolytic anemias)	3.2
	c impaired red cell production (erythropoiesis)	3.2
л	Compare and contract homelutic anomias in terms of stiples, nother species labora	ton
4.	Compare and contrast nemory ic anemias in terms of ecology, pathogenesis, labora	lory
	diagnosis, and clinical findings and course, according to	
	<ul> <li>a. hereditary versus acquired;</li> </ul>	3.0
	b. intravascular versus extravascular hemolysis; and	3.0
	c intrinsic (hereditary spherocytosis and G-6-PD deficiency) versus extrinsic RI	RC
	(antibady mediated mechanical trayma and chamical injury) defects	20
_	(antibouy-ineulateu, mechanical trauma, anu chemical mjury) delects.	5.0
5.	Discuss the following types of anemia in terms of etiology, marrow and peripheral b	lood
	morphology, laboratory diagnostic criteria, and clinical features and course:	
	a. iron deficiency anemia	3.0
	h megalohlastic anemia	3.0
	s. felate deficiency enemie	3.0
	c. Totate deficiency anemia	3.0
	d. pernicious anemia	3.0
	e. anemia of chronic disease	3.0
	f. aplastic anemia	3.0
6	Discuss iron in terms of requirements sources GL absorption storage and transport	forms
0.	intermentation of test results, and altered levels and assorption, storage and transport	20
	interpretation of test results, and altered levels and association with disease.	3.8
7.	Compare and contrast the following types of hemoglobinopathies in terms of etiological sectors of the sectors o	gy,
	geneotype, morphology on peripheral smear, clinical symptoms, and laboratory diag	gnostic
	criteria:	-
	a Sickle cell disease	26
		2.0
	D. Hemoglobin C disease	2.6
	c. Hemoglobin SC disease	2.6
-		

8. Compare and contrast alpha and beta thalassemias in terms of

a.	major versus minor types;	2.6
b.	morphology on peripheral smear;	2.6
с.	laboratory diagnostic criteria;	2.6
d.	clinical symptoms; and	2.6
e.	genotypes.	2.6

# V. <u>Myeloid Neoplasms</u>

VI.

1.	Discuss the pathogenesis, morphology, immunophenotype, laboratory findings, and clir	nical
	features of acute myeloid leukemia (AML).	3.0
2.	Explain the WHO classification system for acute myeloid leukemia with emphasis on the	e four
	major classes and their prognostic implications.	2.3
3.	Compare and contrast the general features common to the myeloproliferative disorder	S
	(chronic myelogenous leukemia, polycythemia vera, and primary myelofibrosis), in term	ns of
	clinical presentation, laboratory findings, morphology, clinical presentation, and risk of	
	transformation.	3.0
4.	Define the following:	
	a. the Philadelphia chromosome	2.4
	b. BCR-ABL fusion gene	2.4
	c. hyperviscosity syndrome	2.4
	d. erythroid/myeloid ratio	2.4
	e. extramedullary hematopoiesis	2.4
5.	Discuss polycythemia vera (PV) in terms of pathogenesis, morphology, laboratory studie	es,
	clinical presentation, and complications with emphasis on the following:	
	a. Hyperviscosity syndrome	2.2
	b. Spent phase	2.2
	c. Blast crisis	2.2
6.	Compare and contrast the pathogenesis of the different myeloproliferative disorders.	2.5
7.	Describe myelodysplastic syndromes in terms of pathogenesis, morphology and clinical	course.
		2.0
8.	Distinguish between leukemia and leukemoid reactions in terms of pathogenesis, etiolo	ogy, and
	laboratory data.	2.0
Ly	mphoid Neoplasms	
1.	Differentiate lymphoma and leukemia.	2.4
2.	Discuss the general features of non-Hodgkin lymphoma in terms of incidence: principle	of
	classification, grading, and staging: laboratory methods of diagnosis; clinical features; p	rognosis:
	and extralymphatic organs involved.	2.4
3.	Discuss Hodgkin's lymphoma in terms of incidence, classification, laboratory diagnosis.	clinical
	features, and prognosis.	2.4
4.	Compare and contrast non-Hodgkin lymphoma and Hodgkin lymphoma in terms of clini	cal
	features and methods of staging.	2.4
5.	Discuss multiple myeloma in terms of clinical presentation, etiology, diagnosis, morpho	logy and
	sites of lesions. laboratory findings. clinical course. complications. and prognosis.	<b>2.4</b>
6.	Discuss acute lymphoblastic leukemia (ALL) in terms of incidence, age distribution, cyto	genetics.
	morphology (bone marrow and peripheral blood), immunophenotyping, laboratory dia	gnosis,
	clinical features, and prognosis.	2.4

7.	Discuss hairy cell leukemia in terms of incidence, age distribution, morphology, labo	ratory
	diagnosis, clinical features and prognosis.	2.4
8.	Discuss chronic lymphocytic leukemia/small lymphocytic lymphoma in terms of incid	lence,
	pathogenesis, morphology, and clinical presenation.	2.4
9.	Explain the WHO classification system for lymphoid neoplasms and classify lymphoid	d neoplasms
	into five broad categories based on their cell of origin.	2.4

### VII. <u>Pulmonary Disease</u>

1.

2.

3.

Define ar	nd use in proper context:	
a.	acute interstitial pneumonia	3.0
b.	acute lung injury (ALI)	3.0
с.	acute respiratory distress syndrome	3.0
d.	adenocarcinoma	3.0
e.	allergic bronchopulmonary aspergillosis	3.0
f.	asthma	3.0
g.	atelectasis	3.0
h.	atopic asthma	3.0
i.	cor pulmonale	3.0
j.	dyspnea	3.0
k.	emphysema	3.0
Ι.	етруета	3.0
m.	Goodpasture syndrome	3.0
n.	gray hepatization	3.0
0.	hyaline membranes	3.0
р.	hyperseneitivity pneumonitis	3.0
q.	idiopathic Pulmonary Fibrosis (IPF)	3.0
r.	large cell carcinoma	3.0
s.	lobar pneumonia	3.0
t.	malignant mesothelioma	3.0
u.	non-atopic asthma	3.0
٧.	noncaseating granulomas	3.0
w.	nonspecific interstitial pneumonia	3.0
х.	restrictive pulmonary disease	3.0
у.	sarcoidosis	3.0
Ζ.	small cell carcinoma	3.0
aa.	squamous cell carcinoma	3.0
bb	status asthmaticus	3.0
cc.	Tachypnea	3.0
dd	a1-antitrypsin (a1-AT)	3.0
Compare contracti	and contrast resorption (obstruction) atelectasis, compression atelectasis, and on atelectasis, in terms of etiology, pathogenesis, morphology, and clinical featu	res.
C		3.0
pathophy	rand contrast the two major causes of pulmonary edema with special emphasis or rsiology and morphology.	on 3.0

Discuss acute lung injury (ALI) and acute respiratory distress syndrome (ARDS) in terms of pathogenesis, morphology, and clinical course.
 3.0

5.	Compare and contrast the major differences between obstructive and restrictive pulmo	nary 2 5
6	Describe the overlapping features of the different chronic obstructive lung diseases, incl	J.J.
0.	emphysema, chronic bronchitis, and brinchiectasis	<b>4 0</b>
7	Discuss the use of the term COPD (chronic obstructive nulmonary disease)	4.0
7. 8	Compare and contrast the following obstructive nulmonary diseases in terms of etiology	, ,
0.	nathogenesis morphology clinical course, and complications:	,
	a. emphysema	4.0
	b. chronic bronchitis	4.0
	c. asthma	4.0
	d. bronchiectasis	4.0
9.	Compare and contrast idiopathic pulmonary fibrosis and pulmonary involvement in con	nective
	tissue diseases (rheumatoid arthritis, SLE, PSS, and sarcoidosis), in terms of etiology,	
	pathogenesis, morphology, clinical course, and complications.	4.0
10.	Discuss pulmonary embolism, hemorrhage, and infarction in terms of etiology, pathogen	nesis,
	morphology, clinical course, and complications.	4.0
11.	Discuss pulmonary hypertension in terms of etiology, pathogenesis, morphology, clinica	l course,
	and complications.	3.0
12.	Discuss Goodpasture syndrome in terms of pathogenesis, morphology, and clinical course	se.
		3.0
13.	Discuss the pulmonary features of cystic fibrosis (CF) in terms of lung involvement,	
	pathogenesis, genetics, morphology, clinical manifestations, pulmonary complications,	
	treatment, and prognosis.	2.0
14.	Compare and contrast bronchopneumonia, lobar pneumonia, lung abcess, and chronis	
	pneumonia in terms of etiologic organisms, pathogenesis, morphology, and clinical cour	se.
4 -		2.5
15.	Compare and contrast the following lung tumors in terms of epidemiology, etiology,	
	pathogenesis, morphology, clinical course, and prognoses:	2.0
	a. Adenocarcinoma	3.0
	b. Calcinola turnol	2.0
	d metastatic tumor	3.0
	e mucinous adenocarcinoma (mucinous bronchioloalveolar)	3.0
	f. small cell carcinoma	3.0
	g. squamous cell carcinoma	3.0
16.	Discuss inflammatory pleural effusions, noninflammatory pleural effusions, and pneumo	othorax
-	in terms of etiology, pathogenesis, morphology, and clinical course.	2.0
17.	Compare and contrast solitary fibrous tumor and malignant mesothelioma in terms of e	tiology,
	pathogenesis, morphology, clinical course, and prognoses.	3.0
<b>C</b> -	studiate stine   Disease	

# VIII. <u>Gastrointestinal Disease</u>

1.	Define	the following, and use in proper context:
	a.	Achalasia
	b.	appendicitis, acute
	с.	atresia
	d.	Barrett esophagus
	e.	carcinoid syndrome

3.0 3.0 3.0 3.0 3.0

	f.	carcinoid tumor	3.0
	g.	chronic gastritis	3.0
	h.	Crohn's disease	3.0
	i.	diarrhea	3.0
	j.	diverticulumdysphagia	3.0
	k.	erosion	3.0
	I.	esophageal varices	3.0
	m.	esophagitis	3.0
	n.	familial adenomatous polyposis	3.0
	0.	gastric ulcer	3.0
	p.	gastritis, acute	3.0
	q.	gastritis, atrophic	3.0
	r.	gastritis, autoimmune	3.0
	s.	gastroesophageal reflux	3.0
	t.	Heliobacter pylori	3.0
	u.	Hematemesis	3.0
	v.	Hemorrhoids	3.0
	w.	Hernia	3.0
	х.	Hirschspruna disease	3.0
	v.	Hyperplasic polyp	3.0
	, Z.	inflammation. transmural	3.0
	aa.	inflammatory polyp	3.0
	bb.	intestinal metaplasia	3.0
	cc.	intussusceptions	3.0
	dd.	iuvenile polyp	3.0
	ee.	linitis plastic	3.0
	ff.	lymphomas of MALT	3.0
	gg.	malabsorption	3.0
	hh.	Mallorv-Weiss syndrome	3.0
	ii.	meacolon	3.0
	ii.	meckel diverticulum	3.0
	kk.	melena	3.0
		mucocele	3.0
	mn	n. napkin rina lesion	3.0
	nn.	peptic ulcer	3.0
	00.	Peutz-Jeaher syndrome	3.0
		polyps, neoplastic	3.0
	00. 29.	nseudomembranous colitis	3.0
	rr.	Sprue (celiac, tronical, nontronical)	3.0
	55.	ulcerative colitis	3.0
	tt.	volvulus	3.0
2	Describ	e the following abnormalities in terms of etiology, nathogenesis, clinical feature	sand
	morph	ologic features:	o, ana
	a	Atresia	1.6
	h.	Diaphragmatic hernia	1.6
	р. С	Duplications	1.6
	d.	Ectopia	1.6
	е.	Eistulae	1.6
	<b>.</b>		

	f.	Hirschsprung disease	1.6
	g.	Meckel diverticulum	1.6
	h.	Omphacele	1.6
	i.	Pyloric stenosis	1.6
3.	Discuss	s diverticula, stenosis, esophageal mucosal webs, esophageal rings, and achalasia	as
	causes	of esophageal obstruction, in terms of anatomic location, morphology, and clinic	al
	feature	25.	1.4
4.	Compa	re and contrast the following causes of esophagitis: lacerations (including Mallor	y-Weiss
	tears),	chemical and infectious esophagitis, reflux esophagitis (GERD), hiatal hernia, and	
	eosino	philic esophatitis.	2.4
5.	Discuss	s esophageal varices, including pathogenesis and clinical course.	2.4
6.	Discuss	s Barrett esophagus, in terms of pathogenesis, morphologic findings, clinical cours	se, and
_	compli	cations.	1.6
7.	Discuss	s the etiology, pathogenesis, morphology, clinical course, and prognosis for esoph	ageal
~	carcino	omas.	1.6
8.	Compa	re and contrast acute, autoimmune, atrophic, and chronic gastritis in terms of et	lology,
0	pathog	enesis, morphology, and clinical features.	2.0
9.	DISCUSS	s acute gastric ulceration in terms of etiology, pathogenesis, morphology, and cilr	
10	Teature	25. A shuania sastuitis is tarma of sticlose (with any hosis on Uslichastar mulari and	2.4
10.	Discuss	s chronic gastritis) nother species more belongy (with emphasis on Heliobacter pylori and	2.4
11	Discuss	imune gastritis), pathogenesis, morphology, and clinical reatures.	<b>2.4</b>
11.	Discuss	a the complications of chronic gastricis, including peptic licer disease, mucosal at	<b>7 1</b>
12	Compo	re and contract gastric polyne, inflammatory polyne, hyperplacic polyne, and gast	<b>2.4</b>
12.	adenor	ne and contrast gastric polyps, initialititatory polyps, hyperplasic polyps, and gast	12
13	Discuss	s gastric carcinoma in terms of enidemiology nathogenesis mornhology and clin	ical
15.	feature		16
14	Discuss	s carcinoid tumor in terms of morphology and clinical features	1.4
15	Identify	v and describe causes of intestinal obstruction, including hernias, adhesions volvi	ulus, and
	intussu	sception.	1.8
16.	Discuss	s ischemic bowel disease in terms of pathogenesis, morphology, and clinical featu	ires.
			1.8
17.	Compa	re and contrast cystic fibrosis, celiac disease, and lactase deficiency in terms of	
	pathog	enesis, morphology, and clinical features.	2.2
18.	Describ	be morphologic changes and clinical features of irritable bowel syndrome.	2.0
19.	Compa	re and contrast Crohn's disease and ulcerative colitis in terms of pathogenesis,	
	morphe	ology, clinical features, and complications.	2.4
20.	Discuss	s sigmoid diverticular disease in terms of pathogenesis, morphology, clinical featu	ires, and
	compli	cations.	1.8
21.	Compa	re and contrast inflammatory, hamartomatous, hyperplastic, and neoplastic poly	ps in
	terms o	of morphology and complications.	1.8
22.	Compa	re and contrast familial adenomatous polyposis and hereditary nonpolyposis colo	prectal
	cancer	in terms of genetics and clinical features.	1.4
23.	Discuss	s adenocarcinoma of the colon in terms of epidemiology, pathogenesis, morpholo	ogy, and
_	clinical	features.	1.8
24.	Discuss	s hemorrhoids in terms of pathogenesis, morphology, and clinical features.	2.0
25.	Discuss	s acute appendicitis in terms of pathogenesis, morphology, and clinical features.	1.8

# IX. <u>Pathology of the Liver and Extrahepatic Biliary System</u>

1.	Define:		
	a.	Budd-Chiari syndrome	1.8
	b.	centrilobular hemorrhagic necrosis	1.8
	с.	passive congestion	1.8
2.	Identify	and describe histologically intracellular accumulations, necrosis, inflammation,	and
	cirrhos	is as patterns of hepatic injury.	1.4
3.	List the	common manifestations of hepatic dysfunctions and be able to explain the mec	hanism
	of the f	ollowing manifestations:	
	a.	ascites	2.8
	b.	caput medusa	2.8
	с.	esophageal varices	2.8
	d.	gynecomastia	2.8
	e.	hemorrhoids	2.8
	f.	hepatic encephalopathy	2.8
	g.	hypoalbuminemia	2.8
	h.	hypogonadism	2.8
	i.	jaundice	2.8
	j.	palmar erythema	2.8
	k.	spider angioma	2.8
	I.	splenomegaly	2.8
4.	Interpr	et the values of different components of the liver function tests.	3.4
5.	Differe	ntiate between acute and chronic liver dysfunction.	2.8
6.	Differe	ntiate between primary renal dysfunction and renal dysfunction due to hepatore	nal
	syndro	me.	2.4
7.	Explain	the major three mechanisms that contribute to the development of cirrhosis.	3.0
8.	Discuss	the mechanisms of both unconjugated and conjugated hyperbilirubinemia.	2.8
9.	List the	three different patterns of alcoholic liver injury and their key morphological find	ings.
			2.6
10.	Apprec	iate how the various forms of alcoholic liver diseases interrelate to each other.	2.6
11.	List the	main drugs which cause damage to the liver.	3.6
12.	Unders	tand the mechanisms by which alcohol, drugs and iron damage the hepatocytes.	2.8
13.	Compa	re and contrast the different forms of viral hepatitis (A, B, C, D, and E) with emph	asis on
	etiolog	y, morphology, laboratory findings, clinical course, and complications.	2.8
14.	Discuss	the pathogenesis, morphology (including possible extrahepatic manifestations),	and
	clinical	course for the following: Wilson disease, hemochromatosis, $\alpha$ 1-antitrypsin defic	liency,
	and Re	ye syndrome.	2.2
15.	Discuss	the different causes of jaundice with emphasis on whether the pathophysiologic	3
	mechai	nism produces predominately unconjugated hyperbilirubinemia or predominatel	y ac
4.0	conjuga	ated nyperbilirubinemia.	2.6
16.	Differe	ntiate between extranepatic atresia andphysiological jaundice as causes of neon	atal
4 7	cnolest	dSIS.	1.4
1/.	Differe	nuate bwiween primary billary cirriosis and primary scierosing cholangitis, in ter	ins of
	epidem	nology, radiographic infulligs, associated conditions, morphology, laboratory find	1 <b>0</b>
10		III.dl LUUISE. matastatic locions as the most common tune of malignant tumers in the liver	1.0 7 C
10. 10	Discuss	a metastatic resions as the most common type of manghant tumors in the liver.	2.0
19.	nuentiny	and describe conditions associated with hepatocenular carcinolita.	2.0

	20.	Identify hepatocellular carcinoma histologically.	
			1.4
	21.	Identify fibrolamellar carcinoma histologically and describe in terms of characteristic fea	atures,
		age, association (or lack thereof) with cirrhosis, and its hard consistency.	1.4
	22.	Discuss autoimmune hepatitis in terms of etiology, morphology, and clinical features.	2.0
	23.	Discuss the pathogenesis of gallstones.	3.0
	24.	Compare and contrast the different types of cholecystitis (acute calculous, acute acalcul	ous, and
	25.	chronic) with emphasis on clinical presentation, laboratory findings and morphology. Discuss disorders of extrahepatic bile ducts with emphasis on choledocholithiasis and	3.0
		cholangitis.	2.5
	26.	Discuss carcinoma of the gallbladder with emphasis on morphology and clinical features	5. <b>2.4</b>
Х.	Pa	ncreatic Disease	
	1.	Describe the following congenital anomalies of the pancreas:	
		a. agenesis	1.8
		b. pancreas divisum	1.8
		c. annular pancreas	1.8
		d. ectopic pancreas	1.8
	2.	Compare and contrast acute pancreatitis and chronic pancreatitis with emphasis on etic	ology,
		pathogenesis, morphology, laboratory studies, clinical features, and complications.	2.6
	3.	Discuss cystic fibrosis in terms of genetics, primary defect, morphologic findings, laborat	tory
		findings, and clinical course.	2.2
	4.	Discuss non-neoplastic pancreatic cysts with emphasis on congenital cysts and pseudocy	ysts,
		specifically in regards to etiology and morphology.	1.6
	5.	Discuss neoplastic cysts of the pancreas, including serous cystadenomas, mucinous	
		cystadenomas, and intraductal papillary mucinous neoplasms.	1.6
	6.	Discuss pancreatic carcinoma in terms of precursor lesions, molecular carcinogenesis,	
		epidemiology, etiology, pathogenesis, morphology, and clinical features.	2.0
	7.	Discuss the following pancreatic endocrine neoplasms, including hyperinsulinism and Zo Ellison syndrome in terms of clinical presentation, laboratory findings, and morphology.	ollinger- <b>2.0</b>
VI	Go	nitourinary Disease Objectives	
Λι.	00	Intournary Disease Objectives	
	1.	Compare and contrast infectious and interstitial cystitis, in terms of etiology, pathogene	sis,
		clinical course, and complications.	1.3
	2.	Discuss urothelial cancinoma, squamous cell carcinoma, and adenocarcinoma, in terms	of
		epidemiology, etiology, and clinical and pathological features.	1.3
	3.	Describe and discuss hypospadias and esispadias.	1.3
	4.	Discuss squamous cell carcinoma of the penis and scrotum; adenocarcinoma of the pros	state;
		germ cell tumors of the testis; sec-cord tumors of the testis; and malignant lymphoma of	of the
		testis in terms of incidence, risk factors, clinical symptoms, main pathological features, a	and
		prognosis.	2.0
	5.	Compare and contrast prostatitis, orchitis, and torsion of the spermatic cord in terms of	
		etiology, pathogenesis, clinical course, and complications.	1.3
	6.	Discuss nodular hyperplasia of the prostate in terms of incidence, clinical symptoms, and	d
	_	morphology.	2.0
	7.	Discuss cryptorchidism in terms of incidence, morphology and complications.	2.0

8.	Discuss infections of the lower genital tract (vulva, vagina, and cervix) in terms of comm	on
	etiologic agents, and clinical symptoms.	2.0
9.	Discuss pelvic inflammatory disease in terms of common etiologic agents, clinical sympt	oms,
	and prognosis.	1.3
10.	Discuss vaginal adenosis and vaginal adenocarcinoma in terms of epidemiology, etiology	Ι,
	pathogenesis, and clinical significance.	1.3
11.	Describe cervical polyps in terms of clinical symptoms and pathogenesis.	1.3
12.	Discuss carcinoma of the cervix in terms of incidence, risk factors, precursor lesions, clin	ical
	features, pathogenesis, and prognosis.	1.3
13.	Discuss endometriosis in terms of incidence, clinical presentation, pathogenesis, and	
1 1	complications.	1.3
14.	Discuss endometrial hyperplasia in terms of etiology, classification, and relationship to	1 2
1 5	Discuss andometrial carsinoma in terms of risk factors, nathology, clinical symptoms, and	1.5 d
13.	prognosis	u 12
16	progression. Discuss leiomyoma in terms of incidence, nathology, and clinical symptoms	1.3 1.2
17	Compare and contrast surface enithelial tumors sex cord-strongl tumors germ cell tum	nors and
۲/.	metastatic malignancy to ovary in terms of incidence age predilection morphology ho	monal
	effects, clinical features, and prognosis.	1.3
Re	nal Disease	
1.	Explain the general histologic pattern of glomerular injury with emphasis on the terms	
	a. sclerosis;	1.2
	b. proliferative;	1.2
	c. focal, segmental; and	1.2
	d. diffuse.	1.2
2.	Discuss the patterns of immunofluorescence (granular and diffuse) and correlate them w	with the
	different types of glomerulonephritis.	1.4
3.	Describe the immunological mechanisms of glomerular diseases and give examples for e	each
	mechanism.	1.8
1.	Compare and contrast nephrotic syndrome and nephritic syndrome, and correlate them	with
	the renal disease.	2.6
5.	For each of the following glomerulonephritides (acute proliferative, rapidly progressive,	
	membranous, minimal-change disease, focal segmental, and membranoproliferative) di	scuss the
	pathogenesis, morphology (light microscopy, electron microscopy, and immunofluoresc	ence),
~	laboratory findings, and clinical features.	2.6
ь.	Describe the morphology of glomeruli in the following systemic diseases:	
	a. SLE	2.2
	D. Glabetes mellitus	2.2
	c. amyloid deposition	2.2
	U. HIV	2.2
	e. nypertension	2.2
,	Information in terms of national and laboratory	<b>Z.Z</b>
· •	Discuss preprenar azotenna, renar aztonna, in terms or pathophysiology and laboratory	<b>a</b> <i>A</i>
8	Describe the nathogenesis and clinical findings of acute tubular necrosis	2. <del>4</del> 2.8
9.	List the causes of urinary tract obstruction.	2.4

XII.

	10.	Describe the characteristic features of simple cysts, dialysis-associated cysts, and adult	
		polycystic diseases of the kidney.	1.8
	11.	Describe the pathogenesis and common causes of chronic renal failure.	3.2
	12.	Discuss renal cell carcinoma with respect to epidemiology, classification, morphology, la	boratory
		findings, and clinical features.	1.4
	13.	Compare and contrast acute and chronic pyelonephritis with emphasis on pathophysiol	ogy,
		laboratory findings, morphology, and clinical course.	2.0
	14.	Discuss the conditions commonly associated with renal papillary necrosis.	1.8
	15.	Discuss hemolytic uremic syndrome with emphasis on associated organisms, laboratory	findings,
		and clinical course.	1.8
	16.	Discuss the tubular and interstitial damage in glomerular diseases with emphasis on	
		pathophysiology, morphology, and clinical features.	2.0
	17.	Compare and contrast benign nephrosclerosis (arterionephrosclerosis) and accelerated	
		nephrosclerosis (malignant nephrosclerosis) with emphasis on pathophysiology, morpho	ology,
		and clinical features.	1.6
	18.	Explain the mechanism and describe the morphological changes occurring in the kidney	tor
		thrombotic microangloathy, polyarteritis nodosa, and Wegener granulomatosis.	2.0
XIII.	<u>Re</u>	nal Function Tests	
	1.	Discuss serum creatinine and its relationship to renal function, including factors contribution	uting to
		its serum level; creatinine clearance and glomerular filtration rate and uses; and disease	S
		associated with increased and decreased serum creatinine.	3.2
	2.	Discuss blood urea nitrogen and its relationship to renal function, including factors cont	ributing
		to its serum level and diseases associated with increased and decreased blood urea nitro	ogen.
			3.2
	3.	Describe the components of a macroscopic/dipstick urinalysis and disorders associated	with
		abnormal values.	3.2
	4.	Describe the components of a microscopic urinalysis and disorders associated with abno	ormal
		values.	3.2
	5.	Discuss the use of quantitative protein urinalysis and the conditions associated with abn	ormal
		values.	3.2
	6.	Discuss urolithiasis in terms of:	
		<ul> <li>a. composition and relative incidence of various types of stones;</li> </ul>	3.2
		b. pathophysiological abnormalities associated with the common types of stones;	3.2
		c. etiology and pathogenesis of stone formation;	3.2
		d. effect of location of stones on clinical and anatomic findings; and	3.2
		e. clinical course and complications.	3.2
XIV.	Bre	east Disease	
	1.	Review the normal anatomy and histology of the breast.	1.0
	2.	Describe the following disorders of breast development:	
		a. milkline remnants, including supernumerary nipples	0.8
		b. accessory axillary breast tissue	0.8
		c. congenital nipple inversion	0.8
	3.	Discuss the following inflammatory disorders of the breast, including epidemiology, clini	ical
		presentation, and morphology	

	a.	acute mastitis	1.0
	b.	periductal mastitis	1.0
	с.	mammary duct ectasia	1.0
	d.	fat necrosis	1.0
4.	Classify	and discuss the epidemiology, morphology, clinical features, and risk of progres	sion to
	cancer	of the following fibrocystic changes:	
	a.	nonproliferative changes	3.0
	b.	proliferative changes without a typia	3.0
	с.	proliferative changes with a typia	3.0
5.	Compa	re and contrast the following with respect to morphology and clinic features:	
	a.	adenosis	2.0
	b.	apocrine metaplasia	2.0
	с.	atypical ductal hyperplasia	2.0
	d.	atypical lobular hyperplasia	2.0
	e.	complex sclerosing lesion	2.0
	f.	cysts	2.0
	g.	epithelial hyperplasia	2.0
	h.	fibrosis	2.0
	i.	papillomas	2.0
	j.	sclerosing adenosis	2.0
6.	Discuss	the incidence and epidemiology of breast cancer.	2.0
7.	Discuss	the significance of the following risk factors for the development of breast cance	er:
	a.	age	3.0
	b.	age at first live birth	3.0
	с.	age at menarche	3.0
	d.	atypical hyperplasia	3.0
	e.	breast density	3.0
	f.	breastfeeding	3.0
	g.	carcinoma of contralateral breast	3.0
	h.	carcinoma of endometrium	3.0
	i.	diet	3.0
	j.	environmental toxins	3.0
	k.	estrogen exposure	3.0
	١.	exercise	3.0
	m.	first-degree relatives with breast cancer	3.0
	n.	geographic influence	3.0
	0.	obesity	3.0
	р.	race/ethnicity	3.0
	q.	radiation exposure	3.0
	r.	tobacco	3.0
8.	Describ	be the role of genetics in the development of breast cancer with emphasis on BRC	CA-1 and
	BRCA-2		2.0
9.	Discuss	the role of hormone exposure in the development of breast cancer.	1.4
10.	Unders	tand the role of mammography in screening for breast cancer.	2.2
11.	Compa	re and contrast the epidemiology, morphology, clinical features, clinical course, a	and
	progno	sis of ductal carcinoma in situ (DCIS), including its architectural subtypes	
	(comed	locarcinoma, solid, cribiform, papillary, and micropapillary); and lobular carcinon	na in situ
	(LCIS).		2.0

12. Compa	are and contrast the following types of invasive breast cancer, in terms of epidem	iology,
morph	ology, and prognosis:	
a.	invasive ductal carcinoma, no special type (NST)	2.0
b.	invasive lobular carcinoma	2.0
с.	medullary carcinoma	2.0
d.	mucinous (colloid)carcinoma	2.0
e.	tubular carcinoma	2.0
f.	Paget disease of breast	2.0
13. Discus	s the importance of the following prognostic and predictive factors (major and n	ninor):
a.	invasive carcinoma versus in situ disease	1.8
b.	distant metastases	1.8
с.	lymph node metastases	1.8
d.	tumor size	1.8
e.	locally advanced disease	1.8
f.	inflammatory carcinoma	1.8
g.	histologic subtype	1.8
h.	nistologic grade	1.8
i.	estrogen and progesterone receptor positivity	1.8
j.	HER2/neu	1.8
k.	lymphovascular invasion	1.8
I.	proliferative rate	1.8
m.	DNA content	1.8
n.	response to neoadjuvant therapy	1.8
0.	gene expression profiling	1.8
14. Compa	are and contrast fibroadenoma and phyllodes tumor in terms of incidence, clinica	l I
presen	tation, morphology, and clinical course.	1.2
15. Descril	pe gynecomastia and carcinoma in terms of etiology/pathogenesis, clinical featur	es, and
progno	osis.	1.2

### XV. Endocrine Disorders

1.	Describe all the feedback mechanisms of the endocrine system.	3.0
2.	Differentiate between primary and secondary hyperfunction and hypofunction.	3.0
3.	Describe the normal physiology and anatomy of the pituitary gland.	3.0
4.	Discuss the clinical manifestations of pituitary disease caused by local mass effects, inclu	ıding
	visual field disturbances, increased intracranial pressure, and pituitary apoplexy.	2.0
5.	Discuss hyperpituitarism, including causes and the classification system for adenomas a	nd
	genetic abnormalities associated with pituitary adenomas.	2.0
6.	Define microadenoma and macroadenoma.	1.8
7.	Describe prolactinomas, growth hormone cell (somatotroph) adenomas, and ACTH cell	
	(corticotroph) adenoma in terms of etiology, morphology, laboratory studies, and clinica	al
	presentation.	3.0
8.	Discuss the etiology of hypopituitarism including tumors (adenomas), traumatic brain in	jury,
	subarachnoid hemorrhage, pituitary surgery or radiation, pituitary apoplexy, ischemic n	ecrosis
	of the pituitary, empty sella syndrome, and hypothalamic lesions.	2.0
9.	Discuss the clinical presentation and laboratory studies associated with hypopituitarism	3.0
10.	Compare and contrast diabetes insipidus and the syndrome of inappropriate ADH secret	tion,
	including etiology, pathogenesis, laboratory findings and clinical signs and symptoms.	2.0

11.	Describe the normal anatomy and physiology of the thyroid gland and its hormones.	3.0
12.	Define and interpret the following laboratory studies:	
	a. Free T4 (FT4)	3.0
	b. Free T3 (FT3)	3.0
	c. <i>T</i> 3	3.0
	d. <i>TRH</i>	3.0
	e. TSH (thyroid stimulating hormone)	3.0
	f. <i>T</i> 4	3.0
13.	Discuss hyperthyroidism, in terms of	
	a. etiology;	3.0
	b. pathogenesis; and	3.0
	c. clinical course, with emphasis on	3.0
	i. cardiac,	
	ii. neuromuscular system,	
	iii. ocular changes,	
	iv. Gl system,	
	v. skeletal system. and	
	vi, thyroid storm.	
14.	Discuss hypothyroidism, in terms of etiology and pathogenesis, with an emphasis on ph	vsical
	appearance, age, skeletal manifestation, CNS/cognitive defects, and clinical presentation	n.
		3.0
15.	Define Cretinism and Myxedema	3.0
16	Compare and contrast Hashimoto thyroiditis, subacute (granulomatous) thyroiditis, and	4
10.	subacute lymphocytic (nainless) thyroiditis with emphasis on etiology nathogenesis	A
	mornhology clinical course and laboratory studies	3.0
17	Discuss Graves disease with emphasis on etiology nathogenesis mornhology clinical	5.0
17.	presentation (Pretibial Myzedema, Exonhthalmos), clinical course, and laboratory studi	ec
		3.0
18	Compare and contrast diffuse nontoxic (simple) and multipodular goiter with emphasis	0n
10.	etiology nathogenesis mornhology clinical presentation clinical course and laborator	N N
	ctudies	, , , ,
10	Describe thyroid adenomas in terms of nathogenesis mornhology and clinical features	1 5
20	Compare and contrast papillary follicular apaplastic and medullary thyroid carcinoma	s in torms
20.	of genetics, nothogensis, environmental factors, mornhology, clinical course, and progr	
	of genetics, pathogensis, environmental factors, morphology, chinical course, and progr	1 E
21	Discuss calcium homoostatic mochanisms	2.0
21.	Compare and contract primary secondary and tertiary hyperparathyraidism with omr	<b>5.U</b>
22.	compare and contrast primary, secondary, and tertiary hyperparatityrolusin, with emp	
	etudios	y 20
<b>n</b> n	Studies.	<b>3.0</b>
23.	Discuss hypoparathyroidism, with emphasis on etiology, pathogenesis, morphology, cill	
24	Course, driv laboratory studies.	2.5
24.	Describe the normal physiology of the adrenal gland (cortex and medula).	3.0
25.	Discuss hyperconticolism (cushing syndrome), with emphasis on pathogenesis, morpho	ougγ,
20	clinical course, and laboratory studies.	3.0
26.	Discuss the use of the dexamethasone suppression test.	2.0
27.	compare and contrast primary and secondary hyperaldosteronism with emphasis on effective secondary hyperaldosteronism with emphasis of effective secondary hyperaldosteronism with emphasis on effecti	liology,
	pathogenesis, morphology, clinical course, and laboratory studies.	2.0

28.	Compare and contrast the different forms of 21-hydroxylase deficiency, with emphasis o	n
	etiology, pathogenesis, morphology, clinical course, and laboratory studies.	1.5

- 29. Compare and contrast the differences between primary and secondary adrenocortical insufficiency, with emphasis on etiology, pathogenesis, morphology, clinical course, and laboratory studies.
   3.0
- 30. Explain Waterhouse-Friderichsen Syndrome, Addison Disease, Autoimmune polyendocrine syndrome type 1 (APS1), and autoimmune polyendocrine syndrome type 2 (APS2). **2.3**
- 31. Compare and contrast adrenocortical adenoma and adrenocortical carcinoma with emphasis on etiology, morphology, clinical course, and laboratory studies. **1.8**
- 32. Discuss pheochromocytoma in terms of etiology, pathogenesis, morphology, clinical course, and laboratory studies. 2.5
- 33. Compare and contrast the different multiple endocrine neoplasia syndromes (MEN syndromes), with emphasis on etiology, pathogenesis, genetics, morphology, and clinical course. **1.5**

#### XVI. Diabetes

1.	Describ	e the normal structure and function/physiology of the endocrine pancreas.	2.0
2.	Define and use in proper context:		
	а.	Advance Glycosylation End Product (AGE's) HgbAlc	4.0
	b.	albuminuria	4.0
	С.	C-peptide	4.0
	d.	dawn phenomenon	4.0
	е.	diabetes mellitus	4.0
	<i>f</i> .	gestational diabetes	4.0
	<i>g</i> .	glycosuria	4.0
	h.	glycosylated hemoglobin	4.0
	<i>i</i> .	glycosylation (glycation)	4.0
	<i>j</i> .	HgbA1c	4.0
	k.	hyperglycemia	4.0
	Ι.	hyperinsulinemia	4.0
	т.	hypoglycemia	4.0
	n.	impaired fasting glucose	4.0
	0.	impaired glucose tolerance	4.0
	р.	insulin	4.0
	q.	insulin resistance	4.0
	r.	insulitis	4.0
	<i>s.</i>	ketosis	4.0
	t.	metabolic syndrome	4.0
	и.	microalbuminuria	4.0
	ν.	microangiopathy	4.0
	W.	MODY (maturity onset diabetes of youth)	4.0
	х.	polydipsia	4.0
	у.	polyphagia	4.0
	Ζ.	polyuria	4.0
	aa.	pre-diabetes	4.0
	bb.	primary diabetes	4.0
	CC.	secondaty diabests	4.0
	dd.	Somogyi phenomenon (rebound phenomenon)	4.0

	ee. type 1 diabetes	4.0
2	π. type 2 diabetes	4.0
3.	Classify and define <i>diabetes mellitus</i> and list the distinguisning features of type 1, t	ype 2, and
	gestational diabetes in terms of etiology and pathogenesis; role of inheritance or e	nvironmentai
	factors; age and frequency; mode of onset; clinical and morphological manifestatio	ns; and
4	Insulin requirements.	4.0
4.	Discuss the pathogenesis of complications of type 1 and type 2 diabetes, including	nonenzymatic
	give gives a structure of matterial linear C	and
_	activation of protein kinase C.	4.0
5.	Compare and contrast the acute complications in terms of pathogenesis, laborator	y findings and
	clinical presentation:	4.0
	a. diadetic ketoacidosis	4.0
	b. nyperosmolar nonketotic coma	4.0
~	c. hypoglycemia	4.0
6.	Discuss the following chronic complications in terms of pathogenesis, morphology,	laboratory
	findings and clinical presentation and role in mortality:	
	a. proliferative retinopathy	3.0
	b. non-proliferative retinopathy	3.0
	c. nephropathy (diabetic renal disease)	3.0
	d. diabetic neuropathy	3.0
_	e. vascular complications (both microvascular and macro-vascular)	3.0
7.	Discuss the use of laboratory tests for screening, diagnosing and monitoring patien	ts with pre-
_	diabetes, gestational diabetes, and diagnosed diabetes.	4.0
8.	Explain the role fasting glucose, random glucose, glucose tolerance test, glycosylate	ed
	hemoglobin level (HbA1C), and urine glucoe ketones in screening, diagnosing, and	monitoring
-	patients with pre-diabetes, gestational diabetes, and diagnosed diabetes.	4.0
9.	Discuss the diabetes control and complications trial (DCCT), including the effects of	tight
	glycemic control on the development of diabetic complications.	4.0

# XVII. <u>Dermatopathology</u>

1.	Define:		
	a.	excoriation	3.5
	b.	lichenification	3.5
	с.	macule	3.5
	d.	onycholysis	3.5
	e.	papule	3.5
	f.	plaque	3.5
	g.	pustule	3.5
	h.	scale	3.5
	i.	vesicle	3.5
	ј.	wheal	3.5
	k.	acantholysis	3.5
	١.	acanthosis	3.5
	m.	dyskeratosis	3.5
	n.	erosion	3.5
	0.	exocytosis	3.5
	p.	hydropic swelling	3.5

	q. hypergranulosis	3.5
	r. hyperkeratosis	3.5
	s. ientiginous	3.5
	t. papillomatosis	3.5
	u. parakeratosis	3.5 2 F
	v. spongiosi	3.5 2 F
	w. uncertainon	3.3 2 F
		3.3 2 F
r	y. eczernu Compare and contract contact dermatitic and atopic dermatitic with emphasis on clinica	3.5
Ζ.	compare and contrast contact dermatitis and atopic dermatitis with emphasis on clinical	l n
	mannestation of lesions, previous antigen exposure, and type of hypersensitivity reactio	25
2	Describe the morphologic characteristics of acute subacute, and chronic eczema	3.5
J. ⊿	Discuss lichen simpley chronicus with emphasis on etiology morphology and clinical fac	
ч.	Discuss lichen simplex enrollicus with emphasis on etiology, morphology, and enricar rea	36
5	Discuss psoriasis with emphasis on pathogenesis morphology and clinical features	3.0
5. 6	Discuss perindis with emphasis on pathogenesis, morphology, and elinical reactives.	J.J atures
0.	Discuss pempingus vulguns with empirasis on patriogenesis, morphology, and emired rec	<b>3 5</b>
7	Discuss hullous nemphigoid with emphasis on nathogenesis, morphology, and clinical fe	atures
<i>.</i>	biscuss buildus peripringola with erripriusis on pathogenesis, morphology, and errited re	<b>3 5</b>
8	Discuss dermatitis herpetiformis with emphasis on pathogenesis morphology clinical fe	oatures
0.	and disease associations.	<b>3.5</b>
9.	Discuss erythema multiforme with emphasis on pathogenesis, morphology, and clinical	features.
0.		3.5
10.	Define the term <i>taraet lesion</i> .	3.5
11.	Discuss albinism with emphasis on the pathogenesis, morphology, and clinical features.	3.5
12.	Discuss vitiligo with emphasis on the pathogenesis, morphology, and clinical features.	3.5
13.	Compare and contrast lentigo and ephelis with emphasis on pathogenesis morphology a	ind
	clinical features.	3.5
14.	Discuss nevocellular nevus with emphasis on pathogenesis, morphology, and clinical fea	tures.
		3.5
15.	Compare and contrast the difference between junctional, compound, intradermal, cong	enital,
	acquired, spitz, and dysplastic nevi.	3.5
16.	Discuss molluscum contagiosum with emphasis on etiology, morphology, and clinical fea	atures.
		3.5
17.	Discuss verruca vulgaris with emphasis on etiologic agent, classification, morphology, an	d
	clinical features.	3.5
18.	Describe varicella, including etiology and gross and microscopic manifestations.	3.0
19.	Discuss acrochordon with emphasis on gross and microscopic morphology.	3.5
20.	Discuss epidermal inclusion cysts with emphasis on pathogenesis, morphology, and clini	cal
	features.	3.5
21.	Discuss dermatofibroma with emphasis on pathogenesis, morphology, and clinical featu	res.
		3.5
22.	Discuss seborrheic keratosis with emphasis on morphology and disease associations.	3.5
23.	Discuss keratoacanthoma with emphasis on morphology and clinical features.	3.5
24.	Discuss actinic keratosis with emphasis on pathogenesis, morphology, and clinical course	е.
		3.5
25.	Discuss acanthosis nigricans focusing on morphology and disease associations.	3.6

26. Discuss xanthoma, focusing on morphology and disease associations.	3.8
27. Discuss the morphology and clinical manifestations of hemangioma.	3.8
28. Discuss Pyogenic Granuloma with emphasis on pathogenesis and clinical features.	3.8
29. Define <i>keloid</i> .	3.6
30. Explain the gross and microscopic manifestations of keloids.	3.6
31. Discuss squamous cell carcinoma with emphasis on pathogenesis, morphology, clinical	features,
and prognosis.	4.0
32. Discuss basal cell carcinoma with emphasis on pathogenesis, morphology, clinical featu	res, and
prognosis.	4.0
33. Discuss malignant melanoma with emphasis on risk factors, pathogenesis, and morpho	logy,
including staging and gross appearance, prognostic factors, and clinical features.	4.0
34. Describe acral-lentiginous melanoma and discuss its prognosis.	4.0

### XVIII. Joint Disease

3.

4.

5.

1.	Discuss the makeup and function of skeletal system and soft tissue attachments.	3.8
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2. Classify the different types of arthritis based on their primary pathological processes, radiologic appearance, gross and microscopic appearance, laboratory findings and clinical presentation into the following classifications system:

a.	non-inflammatory	4.0
b.	inflammatory	4.0
с.	infectious	4.0
d.	crystal induced	4.0
e.	hemorrhagic	4.0
Discuss	the following terms associated with osteoarthritis(degenerative joint disease):	
a.	Bouchard's nodes	4.0
b.	chondroitin sulfate	4.0
с.	crepitus	4.0
d.	eburnation	4.0
e.	exotosis	4.0
f.	fibrillation	4.0
g.	Heberden nodes	4.0
h.	joint space incongruity	4.0
i.	osteophyte	4.0
j.	sclerosis	4.0
k.	sublaxation/dislocation	4.0
١.	synovial fluid viscosity	4.0
Describ	e osteoarthritis in terms of age and sex incidence, etiology, pathogenesis, labora	atory
finding	s, and clinical findings and course.	4.0
Decribe	e the following disorders in terms of etiology, incidence and prevalence, genetic	factors,
age and	d sex associations, pathogenesis, morphology, associated disorders, laboratory fi	ndings,
clinical	course, and prognosos:	

age and sex associations, pathogenesis, morphology, associated as	nacis, laboratory mang.
clinical course, and prognoses:	
a. rheumatoid arthritis	4.0
b. seronegative spondyloarthopathies	4.0
i. ankylosing spondylitis	

- ii. reactive arthritis/Reiter's syndrome
- iii. psoriatic arthritis
- iv. enteropathic arthritis

c. scleroderma (progressive sys	scleroderma (progressive systemic sclerosis)			
d. Sjogren syndrome	d. Sjogren syndrome			
e. lupus erythematosis		4.0		
i. discoid				
ii. systemic				
iii. drug induced				
f. mixed connective tissue disea	ase	4.0		
g. polyarteritis				
h. fibromyalgia		4.0		
i. juvenile arthritis		4.0		
Compare and contrast gout and pseu	dogout/calcium pyrophosphate dehydrate (CCPD)			
arthropathy, in terms of pathogenesi	s, clinical presentation, complications, laboratory	studies,		
primary versus secondary, and acute	versus chronic.	4.0		
Discuss infectious arthritis including p	oathogenesis, organisms, radiographic findings, lat	oratory		
findings, and clinical course with emp	phasis on supportive arthritis and lyme arthritis.	4.0 <sup>′</sup>		
Discuss tenosynovial giant-cell tumor	(pigmented villonodular synovitis and giant-cell ti	umor of		
tendon sheath), in terms of both loca	lized and diffuse.	4.0		
- -				

### XIX. Bone Disease

6.

7.

8.

- 1. Examine the gross and microscopic structure, osteogenesis and remodeling of bone. **3.4**
- Identify developmental abnormalities in bone cells, matrix, and structure causing disease with emphasis on achrondroplasia, Marfan syndrome, mucopolysaccharidoses, multiple osteochondroma, osteogenesis imperfect, osteopetrosis, and Paget disease (of bone). **3.0**
- Identify diseases associated with decreased bone mass, osteoclast dysfunction, and those associated with abnormal mineral homeostasis.
   3.6

4.	Describe the types, repairs, and complications of fractures.	4.0
5.	Discuss osteonecrosis (avascular necrosis).	3.8
6.	Identify tumors of bone, cartilage, and bone marrow.	3.8
7.	Explain the disease process involved in osteomyelitis.	4.0

### XX. Soft Tissue Disease

1. Discuss the following tumors (masses) of joint and soft tissue in terms of biology (neoplastic versus non-neoplastic, benign versus malignant); epidemiology; etiology; pathogenesis; cell type and site of origin; and clinical course:

a.	benign fibrous histiocytoma	4.0
b.	Dupytren's contracture	4.0
c.	Fibroma	4.0
d.	Fibrosarcoma	4.0
e.	Ganglion	4.0
f.	leiomyoma	4.0
g.	leiomyosarcoma	4.0
h.	lipoma	4.0
i.	liposarcoma	4.0
j.	malignant fibrous histiocytoma	4.0
k.	myositis ossificans	4.0
I.	Neurofibroma	4.0

m.	Neuroma	4.0
n.	perineural fibrosis	4.0
0.	plantar fibromatosis	4.0
p.	plexiform neurofibroma	4.0
q.	rhabdomyosarcoma	4.0
r.	schwannoma	4.0
s.	synovial cyst	4.0
t.	synovial sarcoma	4.0

### XXI. Head, Neck, and Special Sensory Organs Pathology

1.	Discuss the following oral lesions in terms of etiology, pathogenesis, morphology and o			
	feature	es:		
	a.	Leukoplakia	2.5	
	b.	Erythroplakia	2.5	
	с.	Carcinoma	2.5	
2.	. Discuss the following inflammatory conditions of the upper airways in terms of etio pathogenesis, morphology, and clinical features:			
	a.	allergic rhinitis	2.5	
	b.	infectious rhinitis	2.5	
	с.	chronic rhinitis	2.5	
	d.	nasal polyps	2.5	
3.	Discuss	s nasopharyngeal carcinoma with emphasis on etiology, morphology, and c	linical course.	
			1.3	
4.	Compa	are and contrast reactive nodules of the vocal cords, squamous papilloma, a	nd	
	papillo	omatosis.	1.3	
5.	Discuss	s carcinoma of the larynx with emphasis on etiology, pathogenesis, morpho	logy, and	
	clinical	l course.	2.5	
6.	6. Identify and describe the functional anatomy of the eye.			
7.	Define	cataract and discuss in terms of its formation, clinical presentation, and its	association	
	with ce	ertain systemic diseases.	3.0	
8.	Compa	are and contrast the open-angle glaucoma and angle-closure glaucoma, in to	erms of	
	etiology, pathogenesis, morphology, and clinical course. 2			
9.	Discuss	s the retinal vascular changes associated with hypertension and malignant h	nypertension	
	with er	mphasis on ophthalmoscopic findings.	3.3	
10. Discuss the effects of diabetes mellitus on the eye including:				
	а.	cataract formation;	4.0	
	b.	glaucoma; and	4.0	
	с.	retinal changes;	4.0	
		i. background retinopathy		
		a) microaneurysms		
		b) macular edema		
		c) retinal edema		
		d) nard exudates		
		e) nemorrnages		
		II. proliferative retinopathy		
		a) neuvascularization b) vitropus homorrhages		
		b) vicieous nemormages		

- c) fibrosis
- d) retinal detachment
- 11. Discuss age-related macular degeneration, in terms of type, etiology, pathogenesis, morphology, and clinical course. **3.8**

### XXII. <u>Neuromuscular Disease</u>

1.	Describ	e the st	ructure of normal skeletal muscle and peripheral nervous tissue, including	g gross,
	microso	copic, el	ectron microscopic, and physiologic properties.	3.2
2.	Describ	e the re	actions of the motor unit, including demyelination, axonal degeneration,	muscle
	fiber at	rophy, r	nerve regeneration, and reinnervation of muscle.	3.6
3.	Describe diseases of the peripheral nerve, including:			
	a.	inflamr	natory neuropathies (immune-mediated);	3.4
		i.	Guillain-Barre syndrome	
		ii.	Demyelinating polyradiculoneuropathy	
	b.	infectio	ous Polyneuropathies;	3.4
		i.	leprosy (Hansen disease)	
		ii.	diphtheria	
		iii.	varicella-zoster virus	
	с.	heredit	ary motor and sensory neuropathies;	3.4
		i.	Charcot-Marie-Tooth disease	
		ii.	Dejerine-Sottas disease	
	d.	genetic	metabolic diseases: leukodystrophies;	3.4
	e.	acquire	ed metabolic and toxic neuropathies: diabetic neuropathy;	3.4
	f.	trauma	tic neuropathies: traumatic, compression, Morton's neuroma; and	3.4
	g.	tumors	of peripheral nerve.	3.4
4.	Discuss	disease	s of skeletal muscle, including:	
	a.	Denerv	ation atrophy (Spinal Muscular Atrophy)	3.2
	b.	Muscul	ar dystrophies	3.2
		i.	Duchenne, Becker and other muscular dystrophies	
		ii.	Myotonic dystrophy	
	с.	lon cha	nnel myopathies (channelopathies)	3.2
d. Congenital myopathies		nital myopathies	3.2	
	e. Genetic myopathies of metabolism		c myopathies of metabolism	3.2
		i.	lipid myopathies	
		ii.	mitochondrial myopathies (oxidative phosphorylation diseases)	
	f.	Inflamr	natory myopathies (noninfectious)	3.2
		i.	dermatomyositis	
		ii.	polymyositis	
	g.	Toxic m	hyopathies	3.2
		i.	thyrotoxic	
		ii.	ethanol	
		iii.	drug induced	
	h.	Disease	es of the neuromuscular junction	3.2
		i.	myasthenia gravis	
		ii.	Lambert-Eaton myasthenic syndrome	
	i.	Tumors	s of skeletal muscle	3.2
		i.	rhabdomyoma, rhabdomyosarcoma	
- ii. nodular fasciitisj. Trauma: myositis ossificans
- k. Infectious
  - i. AIDS associated myopathy
  - ii. viral myositis

## XXIII. <u>Central Nervous System Disease</u>

1. Describe the following cells of the central nervous system (CNS), with special atter		to	
	morphology, function, and location:		
	a. Astrocytes	3.0	
	b. chorolds plexus epithelial cells	3.0	
	c. ependymai cells	3.0	
	d. microglia	3.0	
	e. neurons	3.0	
	f. oligodendrocytes	3.0	
2	g. schwann cells	3.0	
2.	Explain the reactions of cells of the CNS to injury (neurons, astrocytes, other glial cells).	4.0	
3.	Discuss features unique to the CNS that affect clinical presentation of diseases, complication	ate	
	outcomes, and affect therapy, including		
	a. blood-brain barrier;	4.0	
	b. CSF;	4.0	
	c. localization of function;	4.0	
	d. selective vulnerability;	4.0	
	e. skull; and	4.0	
	f. vascular supply.	4.0	
4.	Compare and contrast the causes of cerebral edema, including vasogenic edema, cytoto	DXIC	
_	edema, and interstitial edema.	3.0	
5.	Discuss communicating and noncommunicating hydrocephalus, with emphasis on etiology	ogy,	
_	morphology, and clinical course.	2.0	
6.	Compare and contrast the subfalcine, trashtentorial, and tonsillar herniations of the bra	in, in	
_	terms of pathogenesis, morphology, clinical findings, and sequelae.	3.0	
7.	Discuss the following malformations in terms of relative frequency, etiology, pathogenesis,		
	morphology, and clinical features:	~ ~	
	a. agenesis of corpus callosum	3.0	
	b. anencephaiy	3.0	
	c. Chiari type I malformation	3.0	
	a. Chiari type II (Arnoid-Chiari) malformation	3.0	
	e. Dandy-Walker malformation	3.0	
	f. Encephalocele	3.0	
	g. Hydromyelia	3.0	
	h. Meningomyelocele	3.0	
	I. Spina bifida	3.0	
_	J. syringomyelia	3.0	
8.	Discuss perinatal brain injury including cerebral palsy, intraparenchymal hemorrhage, a	nd	
~	infarcts in terms of pathogenesis, morphology, and clinical presentation and course.	3.0	
9.	Identify and describe the types of skull fractures (displaced, diastatic) and discuss the cl	inical	
	relevance of each.	4.0	

3.2

3.2

10.	Define concussion and describe the etiology, morphology, and clinical significance of the	2
	syndrome.	4.0
11.	Discuss direct parenchymal injury in the brain, including contusion, laceration, coup inju	ry,
	contrecoup injury, and hyperextension of the neck, in terms of etiology, pathogenesis,	
	morphology, and clincail presentation and course.	3.0
12.	Discuss diffuse axonal injury in terms of pathogenesis, morphology, and clinical course.	3.0
13.	Discuss epidural hematoma and subdural hematoma (acute and chronic), in terms of	
	pathogenesis, morphology, clinical presentation, and clinical course.	3.0
14.	Explain the sequelae of brain trauma with emphasis on post-traumatic hydrocephalus, p	ost-
	traumatic dementia, dementia pugilistica, post-traumatic epilepsy, tumors, infectious di	seases,
	and psychiatric disorders	
	a. dementia pugilistica;	3.0
	b. infectious diseases;	3.0
	c. post-traumatic dementia;	3.0
	d. post-traumatic epilepsy;	3.0
	e. post-traumatic hydrocephalus;	3.0
	f. psychiatric disorders; and	3.0
	g. tumors.	3.0
15.	Explain the sequelae of spinal cord trauma with emphasis on sensory deficits, gait abnor	malities,
	and paralysis.	3.0
16.	Explain cerebrovascular disease and compare and contrast global cerebral ischemia and	focal
	cerebral ischemia, in terms of pathogenesis, morphology, and clinical features.	4.0
17.	Describe the etiology and pathogenesis of spinal cord infarction.	4.0
18.	List the important effects of hypertension on the brain, including lacunar infarcts, slit	
	hemorrhages, hypertensive encephalopathy, Charcot-Bouchard aneruysms, and intrace	rebral
	hemorrhage and describe the morphologic appearance of each.	3.0
19.	Explain causes of non-traumatic intraparenchymal hemorrhage, such as cerebral amyloi	d
	angiopathy.	2.0
20.	Discuss the pathogenesis and morphology of saccular aneurysms and describe its role in	the
	development of subarachnoid hemorrhage.	3.0
21.	Discuss the clinical signs and symptoms of subarachnoid hemorrhage and list the possib	le
	complications and causes.	4.0
22.	Classify and discuss the clinical features of arteriovenous malformations, cavernous	
	malformation, capillary telangiectasias, and venous angiomas.	3.0
23.	Compare and contrast the pathogenesis, causative organisms, laboratory findings, and c	linical
	presentation and course for acute pyogenic (bacterial) meningitis, acute aseptic (viral)	
~ .	meningitis, and chronic bacterial meningitis (tuberculosis, neurosyphilis, neuroborrelios	is). <b>4.0</b>
24.	Discuss the etiology, pathogenesis, morphology, and clinical course of progressive multi	focal
	leukoenephalopathy and subacute sclerosing panencephalitis.	3.0
25.	Compare and contrast Creutzfeldt-Jakob disease and variant Ceutzfeldt-Jakob disease, in	ncluding
•	etiology, pathogenesis, morphology, and clinical presentation.	3.0
26.	Describe multiple sclerosis (MS) in terms of geographic distribution, etiology, pathogene	esis,
~-	morphology, laboratory findings, and clinical course.	4.0
27.	Compare and contrast the following degenerative diseases with special attention to	
	patnogenesis, morphology, and clinical features:	4.0
	a. Alzneimer disease	4.0
	D. Corticobasal degeneration	4.0
	C. Dementia with Lewy Bodies	4.0

	d.	Friedreich ataxia	4.0
	e.	Huntington disease	4.0
	f.	Parkinsonism / Parkinson disease	4.0
	g.	Pick disease	4.0
	h.	Spinocerebellar ataxias	4.0
	i.	Vascular dementia	4.0
28.	Discuss	amyotrophic lateral sclerosis (ALS) in terms of pathogenesis, morphology, and	clinical
	course.		3.0
29.	Compa	re and contrast the following neoplasms in terms of epidemiology, genetics,	
	pathog	enesis, morphology, clinical features, and prognosis:	
	a.	astrocytoma, infiltrating, all grades	2.0
	b.	ependymoma	2.0
	с.	ganglioneuroma	2.0
	d.	medulloblastoma	2.0
	e.	meningioma	2.0
	f.	metastatic tumor	2.0
	g.	oligodendroglioma	2.0
	h.	pilocytic astrocytoma	2.0
	i.	primary CNS lymphoma	2.0

# PHARMACOLOGY LEARNING OBJECTIVES

General Principles Automonic Nervous System Drugs Cardiovascular and Respiratory Pharmacology Renal Drugs Pulmonary Drugs Gastrointestinal Drugs Drugs Acting on the Central Nervous System Endocrine Pharmacology Hemostasis and Blood Forming Organs Toxicology and Therapy of Intoxication Chemotherapy Herbal Medicine Vitamins

## I. <u>General Principles</u>

1. Define:

		a. pharmacology	
		b. pharmacokinetics	
		c. pharmacodynamics	1.5
	2.	Define what is meant by the term "drug."	2.5
	3.	Explain what constitute drug receptors.	4.0
	4.	Explain the concepts of agonist (full, partial, inverse), and antagonist (competitive and r	ion-
		competitive) drugs.	4.0
	5.	Explain affinity, intrinsic activity, efficacy, and potency as applied to drug receptor intera	actions.
	6.	Explain graded and quantal dose-response relationships.	4.0
	7.	Explain the long-term effects of drugs, including tolerance and regulation of gene expre	ssion.
			4.0
А.	Ph	armacokinetics - Chemical Aspects	
	1.	Discuss weak acids and bases, the Henderson-Hasselbalch equation, and the relationshi	α
		between pH and ionization of drugs.	3.5
	2.	Discuss the effect of lipid solubility of drug species, polar, and nonpolar drugs.	4.0
	3.	Identify the properties of biological membranes, mechanisms of drug movement across	
		membranes, and differentiate between which are active and which are passive process	es.
			3.5
	4.	Explain ion trapping of drugs, with emphasis on stomach contents and urine.	3.0
	5.	Explain the concept of chirality.	2.0
В.	Ab	sorption	
	4	ne per el construction de l'activit de la della	
	1.	Relate absorption to lipid solubility, blood flow, and site of drug placement.	3.0
	2.	Explain the effects of pH on absorption and the concept of first pass metabolism.	4.0
	3.	Identify factors affecting absorption.	4.0
	4.	Identify routes of absorption.	3.0
	5. c	Identify special sites of absorption.	3.0
	ю. 7	Explain the systemic absorption of drugs applied for local effects.	3.0
	7.	explain the concept of bloavallability as a function of absorption and first pass metaboli	311. Э Е
	0	Discuss developmental age related and disease related shanges in drug absorption	2.5
	ŏ.	Discuss developmental, age-related, and disease-related thanges in drug absorption.	2.5
С.	Dis	stribution	
	1.	Explain the effects of plasma protein binding on drug distribution.	2.5
	2.	Describe tissue perfusion, ease of access, tissue binding and solubility coefficients, pKa,	and
		partition coefficient as factors that affect distribution.	3.0
	3.	Explain the concept of distribution ("redistribution") as a mode of termination of drug a	ction.
			2.0
	4.	Describe the distribution of drugs into special compartments, with respect to the blood	-brain
		barrier, tight endothelial junctions, and placenta.	2.0
	5.	Discuss the importance of membrane transporters for both entry and efflux of drugs.	2.5
	6.	Explain the concept of apparent volumes of distribution in relationship to physiological	volumes.
		1.0	

7. Discuss developmental, age-related, and disease related changes in drug distribution. **3.0** 

## D. Metabolism

	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> </ol>	<ul> <li>Explain the importance of drug metabolism for excretion.</li> <li>Explain mechanisms of biotransformation.</li> <li>Identify and describe the major pathways of metabolism, including Phase I versus Phase general properties, oxidation, reduction, hydrolysis and conjugation: glucuronides, glyci sulfate esters, acetylation, glutathione, mercapturic acids.</li> <li>Explain the cytochrome P450 system in liver and other tissues. Know the major P4.050s in drug metabolism: CYP1A2, CYP2B6, CYP2Cs, CYP2D6, CYP2E1, CYP3A4, and CYP3A5.</li> <li>Explain enzyme induction, including mechanisms, time course, clinical implications, and examples of common inducers.</li> <li>Describe the clinical implications of enzyme inhibition.</li> </ul>	4.0 3.5 II, ne, 3.0 involved 4.0 4.0
	7.	Explain developmental, age-related, and disease-related changes in drug metabolism.	3.5
Ε.	Ехс	cretion	
	1. 2. 3. 4.	Explain the concept of excretion as the loss of drug molecules from the body. Differentiate between excretion of parent drug versus excretion of metabolites. Identify the major sites of drug excretion. Identify and describe concepts important for renal excretion, including role of filtration, secretion and reabsorption, molecular size, polarity, weak acids/bases, urine pH, and	1.5 2.5 4.0
	5.	transporters, as well as the importance of plasma protein binding. Explain biliary/alimentary excretion, including biliary transport, direct secretion of drugs blood to intestine, importance of plasma protein binding, molecular size, polarity, weak and weak bases	<b>4.0</b> from acids, <b>2.0</b>
	6. 7. 8. 9.	Explain the consequences of enterohepatic circulation. Explain the concept of clearance and the Cockcroft-Gault equation. Identify the formula relating organ clearance, extraction ratio and blood flow. Explain high and low extraction ratios, as well as the effects of changes in blood flow and protein binding.	4.0 2.5 1.0 d plasma 3.0
F.	Qu	antitative Pharmacokinetics	
	<ol> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> </ol>	Explain one and two compartment systems, as well as the noncompartmental model an clinical utility. Describe the distribution and elimination phases when plotting log C versus time. Identify the pharmacokinetic parameters that determine and can be estimated from the versus time plot, and explain their interrelationships to $V_{d1}$ , $Vd_{extrap}$ , $Vd_{area}$ , AUC, ke, elim $t_{1/2}$ , and Cl. Explain the effect of ka, ke, and dose on Cmax, tmax, and AUC. Estimate bioavailability from ratio of AUCs. Define <i>steady state</i> , and explain the plateau principle: Css = IR/Cl. Explain the time to steady state as a function of half-life, as well as the effects of stoppir infusion or changing infusion rate. Identify the calculation for loading dose, and explain repeated dosing in a one compartm model, including drug accumulation and plateau principle: Cssav = DxF/T x Cl, independence peak to trough variation as a function of dose, F, $t_{1/2}$ , dosing interval (T), and ka:ke ratio	4.0 4.0 2.0 2.0 2.0 1.0 4.0 1.0 1.0 1.0 4.0 1.0 1.0 1.0 4.0 1.0 1.0 1.0 1.0 1.0 1.0 1.0 1
	10.	Discuss the implications of saturation of plasma protein binding.	1.0

## G. Pharmacodynamics

	1. 2	Explain the concept of receptor occupancy: $EA/EM = [A]/([A] + KA)$ .	<b>4.0</b>
	۷.	curves	<b>2</b> .5
	3.	Discuss the relationship of potency (ED <sub>50</sub> and EC <sub>50</sub> ) to affinity ( $K_{A}$ ).	1.5
	4.	Explain the concepts of intrinsic activity and efficacy.	1.5
	5.	Describe the effects of partial and inverse agonists.	3.0
	6.	Identify the mechanisms of antagonists, and differentiate the concepts of competitive v	ersus
		noncompetitive and reversible versus irreversible antagonists.	4.0
	7.	Explain the concept of receptor reserve.	1.5
Н.	Qu	antal Dose Response Relationships	
	1.	Explain the concepts of ED <sub>50</sub> (potency) versus LD <sub>50</sub> or TD <sub>50.</sub>	1.5
	2.	Explain therapeutic indices in terms of calculation and meaning.	4.0
Ι.	Red	ceptors	
	1.	Identify the different types and subtypes of receptors.	3.0
	2.	Explain ligand-gated ion channels, including the nicotinic ACh receptor and GABA <sub>A</sub> receptor	otor.
			4.0
	3.	Explain G Protein-coupled receptors, including muscarinic ACh receptors, and adrenergi	C
		receptors (alpha <sub>1</sub> , alpha <sub>2</sub> , beta <sub>1</sub> , beta <sub>2</sub> , beta <sub>3</sub> ).	4.0
	4. r	Describe tyrosine kinase receptors (insulin) and receptors for steroid hormones.	3.5
	5.	Explain the concepts of receptor down-regulation and desensitization, and as well as the	a inverse
	c	Final construction and receptor levels.	3.0
	0. 7	Explain the concepts of receptor up-regulation and sensitization.	<b>5.0</b>
	7.	$M\Delta\Omega$ nucleic acids (actinomycin D) or target uniqueness as a basis for selective chemic	therany
		(nenicillin)	3.0
			0.0
J.	Ph	armacogenetics/genomics	
	1.	Define pharmacogenetics and pharmacogenomics, and explain their clinical importance	3.0
	2.	Explain genetic polymorphisms in terms of single nucleotide polymorphisms (SNPs), gen	e
		deletions, and gene amplifications that determine protein structure, configuration, and,	′or
		concentration.	1.5
	3.	Differentiate haplotype, genotype, and phenotype and discuss methods to determine	
		phenotype and genotype.	1.5
	4.	Identify pharmacogenetic polymorphisms that affect drug response, as well as drug disp	osition
		and toxicity.	2.0
	5.	Discuss monogenic pharmacogenetic traits that often discriminate populations into disc	rete
		phenotypes (polymorphic distribution), as well as polygenic pharmacogenetic traits that	usually
	_	provide monomorphic distributions.	1.0
	6.	Explain that the frequency of pharmacogenetic polymorphisms often differs with ethnic	ity.
	-	Fundational in the fundamental of the second state of the second s	2.0
	7.	Explain the influence of pharmacogenetics on the effects of drugs, including NAT2 (isoni	aziū,
		(warfarin), corum chalinactorasa (suscinulchalina), ducasa (chashbata dabudrasanasa	E
		(wananin), serum cholmesterase (succinyicholine), glucose-o-phosphate denydrogenase	

(analgesics; antimalarials); thiopurine-S-methyltransferase (6-mercaptopurine); beta<sub>2</sub> adrenergic receptors, (albuterol); dopamine receptors (antipsychotics); malignant hyperthermia (inhalation anesthetics); UGTA1 (irinotecan); and ABCB1 (corticosteroids). **2.0** 

## K. Drug Interactions

- 1. Describe the pharmacokinetic and pharmacodynamic drug interactions. **4.0**
- 2. Explain the concepts of additivity, synergy, potentiation, and antagonism. **3.0**
- 3. Identify and describe drug-food interactions and drug interferences with diagnostic tests. 3.5

## II. <u>Autonomic Drugs</u>

## A. Neuronal Drugs

1. Discuss the following with regard to subsequent objectives:

a.	botulinum toxin	4.0
b.	reserpine	3.0
c.	cocaine	3.0
d.	metyrosine	2.0

## Physiology and pathophysiology

Describe the anatomical projections of the sympathetic and parasympathetic nervous system.
 4.0
 Discuss the evidence for the development of neurotransmitters, cotransmitters, and end-organ specificity.
 Explain homeostasis, fight-or-flight, and rest-and-repair, with regards to the autonomic nervous system.
 Identify and explain the central control of the autonomic nervous system.
 Explain the responses of end organs to activation of each divison of the autonomic nervous system.
 Define *dominant tone.*

## **Mechanism of Action**

8.	Identify drugs that block the uptake of choline into cholinergic neurons.	2.0
9.	Identify drugs that inhibit catechol-o-methyl transferase peripherally.	1.0
10.	Identify drugs that block the storage vesicle transport system.	1.0
11.	Identify drugs that inhibit reuptake of norepinephrine (NE) into adrenergic neurons.	3.0
12.	Identify drugs that deplete NE by interfering with synthesis.	2.0

## B. Cholinergic Agonists

1. Discuss the following with regard to subsequent objectives4.0a. acetylcholine4.0b. bethanechol4.0c. pilocarpine4.0d. neostigmine4.0e. physostimine3.0f. pyridostigmine2.0

	<ul> <li>g. echothiophate</li> <li>h. edrophonium</li> <li>i. malathion</li> <li>j. parathion</li> <li>k. pralidoxime</li> <li>l. sarin</li> <li>m. nicotine</li> <li>n. varenicline</li> </ul>	3.0 4.0 2.0 2.0 4.0 3.0 4.0 3.0
Phy	ysiology and pathophysiology	
2. 3. 4. 5. 6.	Discuss the synthesis, storage, release, and inactivation of cholinergic agonists. Identify drugs that affect the synthesis, storage, release, and inactivation of acetylcholin Locate nicotinic and muscarinic receptors. Differentiate between stimulation of nicotinic and muscarinic receptors. Discuss acetylcholinesterase (AChE) and butyrylcholinesterase (BuChE), in terms of anato locations, sites of synthesis, and function.	4.0 e. 4.0 4.0 d.0 omical 1.0
Me	chanism of action	
<ol> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> </ol>	Explain the mechanism of action, including 2 <sup>nd</sup> messenger systems, of acetylcholine and drugs. Differentiate between groups of anticholinesterases, in terms of onset and duration of a and route of administration. Explain the chemical makeup of the active site of AChE (anionic and esteratic) as to attra attachment, and rates of breakdown of substrates and inhibitors. Identify and describe the mechanism by which pralidoxime reactivates phosphorylated a	related 4.0 action 3.0 action, 2.0 AChE. 4.0
Act	ions on organ systems	
11. 12.	Explain the responses to activation of muscarinic and nicotinic receptors. Identify the reason that anticholinesterases are either reversible or irreversible.	4.0 4.0
Pha	armacokinetics	
13. 14. 15.	Classify anticholinesterases as reversible or irreversible. Discuss variations in pharmacokinetics of cholinergic drugs. Explain the onset of action of anticholinesterases, route of administration, and duration action of anticholinesterases as the correlate to sites a type of attachment in the enzym	<b>2.0</b> <b>2.0</b> of e. <b>3.0</b>
16.	Explain the role of enzyme aging in the enzyme-inhibitor interaction.	4.0
Adv	verse Effects, Drug Interactions and Contraindications	
17. 18.	Discuss the adverse effects of cholinergic drugs. Identify and explain contraindications for cholinergic drugs.	4.0 4.0
The	erapeutic Uses	
19. 20.	Describe therapeutic uses of cholinergic agonists. Explain the effects of accumulated acetylcholine at muscarinic and nicotinic receptors in periphery and in the central nervous system.	<b>4.0</b> the <b>4.0</b>

	21.	Describe the therapeutic uses of anticholinesterase agents as insecticides (malathion, pa and chemical warfare agents (sarin)	athion), <b>4 0</b>
	22.	Discuss the use of anticholinesterase agents as insecticides (malathion, parathion) and c	hemical
		warfare agents (sarin).	3.0
	23.	Explain pralidozime as ineffective in reactivating all phophorylated AChE.	4.0
	24.	Explain differential toxicity of malathion and parathion in different species.	2.0
	25.	Discuss nicotine in terms of its historical, social, and toxicological significance, and expla	in the
		reasoning for its lack of clinical use, except as a smoking deterrent.	3.0
	26.	Discuss therapeutic uses and side of effects of varenicline.	2.5
С.	Cho	olinergic Antagonist	
	1.	Discuss the following with regard to subsequent objectives:	
		a. atropine	4.0
		b. ipratropium	4.0
		c. tiotropium	3.0
		d. scopolamine	4.0
		e. tolterodine	4.0
		f. oxybutynin	2.0
		g. glycopyrrolate	3.0
		h. dicyclomine	3.0
		i. nicotine	4.0
		j. succinylcholine	4.0
		k. d-Tubocurarine	4.0
		I. cistracurium	3.0
		m. hexamethonium	2.0
		n. mecamylamine	1.0
	Phy	ysiology and pathophysiology	
	2.	Differentiate between muscarinic and nicotinic receptors, including location of each.	4.0
	3.	Rationalize the original use of cholinergic antagonists in the treatment of hypertension a	
		autonomic hyperrenexia.	2.0
	Me	echanism of action	
	4.	Discuss the mechanism of action of cholinergic antagonists.	4.0
	5.	Discuss nicotine's agonist and antagonist properties.	4.0
		Actions on organ systems	
	5.	Compare and contrast the depolarizing and nondepolarizing neuromuscular junction (NI	VJ)
		blocking drugs.	
			4.0
	Ad	verse Effects, Drug Interactions and Contraindications	
	6.	Explain why muscarinic antagonists cause xerostomia, blurred vision, photophobia, tach	ycardia,
		difficulty in micturition, hyperthermia, glaucoma, and mental confusion in the elderly.	4.0
	7.	Explain the contraindication of muscarinic antagonists in glaucoma, obstructive disease	of the
		gastrointestinal tract or urinary tract, and intestinal atony.	4.0

8. Discuss the adverse side effects of competitive antagonists at the NMJ. **4.0** 

Rationalize the use of anticholinergics in bronchoconstriction, excessive salivation, and motion sickness.
 4.0

## **Therapeutic uses**

- Rationalize the therapeutic use of muscarinic receptor blockers to produce mydriasis and cyclplegia.
   4.0
- 11. Identify the uses, side effects, and genetic differences associated with the use of succinylcholine.

3.0

## D. Alpha Agonists and Antagonists

1. Discuss the following with regard to subsequent objectives:

a.	epinephrine	4.0
b.	norepinephrine	4.0
c.	phenylephrine	4.0
d.	dopamine	3.0
e.	clonidine	4.0
f.	brimonidine	2.0
g.	methyldopa	2.0
h.	phenoxybenzamine	2.0
i.	phentolamine	3.0
j.	prazosin	4.0
k.	terazosin	2.0
I.	doxazosin	2.0
m.	tamsulosin	3.0
n.	amphetamine	4.0
о.	ephedrine	2.0
p.	tyramine	3.0
q.	methamphetamine	3.0

## Physiology and pathophysiology

- Identify the steps in the synthesis, storage, release, and inactivation of norepinephrine and epinephrine.
   Explain the types and subtypes of adrenergic recentors, and identify their locations and
- 3. Explain the types and subtypes of adrenergic receptors, and identify their locations and physiologic response to activation.
- Discuss receptor selectivity of norepinephrine and epinephrine.
- 5. Differentiate between direct- and indirect-acting adrenergic drugs, in that direct agonists bind to receptors with intrinsic activity, indirect agonists release neurotransmitters from the neuron.

4.0

4.0

4.0

## **Mechanism of action**

- Differentiate the properties of drugs that bind directly to adrenergic receptors in contrast to those that act by increasing release or inhibiting reuptake of neurotransmitters.
   4.0
- 7. Understand the effects of antagonists with no intrinsic activity-binding to adrenergic receptors.

4.0

4.0

## Actions on organ systems

8. Explain the use of alpha<sub>1</sub> adrenergic antagonists for hypertension and benign prostatic hypertrophy.

<ol> <li>Discuss the importance of alpha<sub>1</sub> adrenergic agonists in the treatment of nasal conget hypotension, and paroxysmal atrial tachycardia, as well their effects to cause mydrias vasoconstriction.</li> <li>Describe the mechanism for the use of alpha<sub>2</sub> adrenergic agonists in the treatment of hypertension, as well as for the topical treatment of glaucoma.</li> </ol>	stion, sis and <b>4.0</b> f <b>4.0</b>
Adverse Effects, Drug Interactions and Contraindications	
<ol> <li>Identify the adverse side effects of alpha<sub>1</sub> and alpha<sub>2</sub> agonists.</li> <li>Explain the interactions of alpha<sub>1</sub> and alpha<sub>2</sub> agonists with oxytocic drugs and monoa</li> </ol>	<b>4.0</b> mine
oxidase inhibitors. 13 Identify the contraindications for alpha, adrenergic agonists	2.0 4.0
14. Identify the adverse side effects of nonselective alpha and selective alpha adrenergic	-110
antagonists.	4.0

## Therapeutic uses

15. Explain the limitations of the use of nonselective alpha<sub>1</sub> and alpha<sub>2</sub> adrenergic antagonists in the treatment of hypertension.
 4.0

## E. Beta Agonists and Antagonists

1. Discuss the following with regard to subsequent objectives:

a.	isoproterenol	4.0
b.	albuterol	4.0
c.	salmeterol	4.0
d.	dobutamine	3.0
e.	propranolol	4.0
f.	timolol	4.0
g.	metoprolol	4.0
h.	atenolol	4.0
i.	carvedilol	4.0
j.	labetalol	4.0

## Mechanism of action

2.	Compare and contrast the pharmacology of epinephrine and isoproterenol.	4.0
3.	Compare and contrast the pharmacology of the beta selective adrenergic agonists isopr albuterol, salmeterol, and dobutamine.	oterenol, <b>4.0</b>
4. 5.	Compare and contrast the pharmacology of proporanolol, metaprolol, and atenolol. Compare and contrast the pharmacology of the nonselective alpha and beta blocking du carvedilol and labetalol with selective beta-blocking drugs.	<b>4.0</b> rugs <b>4.0</b>
Ad	verse Effects, Drug Interactions and Contraindications	
6. 7.	Describe the adverse side effects of $beta_2$ adrenergic agonists. Describe the adverse side effects of non-selective beta adrenergic antagonists and com those of $beta_1$ selective antagonists.	<b>4.0</b> pare with <b>4.0</b>
Th	erapeutic uses	
8.	Explain the mechanism for the use of selective beta-adrenergic agonists in diseases such	h as

cardiac decompensation, asthma, premature labor, bronchospasm, and emphysema. **4.0** 

## III. Cardiovascular and Respiratory Pharmacology

## A. Introduction to Cardiovascular Drugs

1.	Describe the properties of the heart, including contractility (eg, excitation-contraction and electrical activity (eg, the action potential, automaticity, excitability, refractory pe	coupling) riod,
	conduction and the relationship to the electrocardiogram).	2.0
2.	Explain the concepts of inotropism, chronotropism, dromotropism, and lusitropism as	they
h	pertain to mechanism of action of commonly used drugs.	3.0
3. ⊿	Discuss the neuroendocrine both the response and output properties of the neart.	3.0
4. 5.	Explain the intrinsic and extrinsic regulation of the cardiovascular system.	2.0 4.0
Sp	ecific Drugs for Management of Cardiac Arrhythmias	
1.	Discuss the following with regard to subsequent objectives:	
	a. quinidine	2.0
	b. procainamide	2.0
	c. disopyramide	1.0
	d. lidocaine	4.0
	e. beta blocking agents	4.0
	i. propranolol	
	ii. sotalol	
	III. acebutolol	
	IV. esmolol	
	v. metoprotot	4.0
	a calcium channel blockers	4.0
	i diltiazem	5.0
	ii. verapamil	
	h. adenosine	3.0
	i. atropine	3.0
Ph	ysiology and pathophysiology	
2.	Explain the ionic basis of the cardiac action potential.	4.0
3.	Describe the role of specific ions and ionic conductances in the production and propage the cardiac action potential with emphasis on fast (sodium dependent) and slow (calci	ation of
	dependent) responses and explain their relevance to specific cardiac tissue types	4.0
4.	Discuss the electrophysiological differences between normal atrial and ventricular card	diac
5.	Explain the alteration of cardiac electrical activity in the production of cardiac arrhythr	nias.
_		2.5
6.	Explain the relationship between cellular cardiac electrical activity and the electrocard	iogram.
-	Identify the nother hyperical and a second	3.5
1.	triggered routing, reaptrant routing and abnormal impulse conduction)	сі(у, Э ғ
Q	Explain the pharmacogenomics of long OT Syndrome and the relationship of genetics t	o drug
ο.	selection	1.5

9. Discuss two forms of long QT Syndrome and explain the ion channels responsible for each.

### 1.5

3.5

## **Mechanism of action**

- 10. Classify antiarrhythmic drugs according to the Vaughn-Williams classification and recognize the<br/>limitations of this classification system.**4.0**
- 11. Identify the mechanism of action of each drug in each drug class. **3.0**

12.	Explain electrophysiologic actions of antiarrhythmic drugs in normal and abnormal myocardial
	and conduction tissue, and describe their effect on the phases of the cardiac action potential.

Describe the alteration of slow (calcium-dependent) and fast (sodium-dependent) responses by antiarrhythmic drugs and explain how that relates to the use of specific agents in arrhythmias of different origins (ventricular versus supraventricular).
 3.5

14. Explain the indirect autonomic actions of antiarrhythmic drugs. **3.5** 

- 15. Describe the effect of age on fast and slow channels and on the agents affecting these channels. **1.0**
- 16. Identify the relevant extracardiac actions of antiarrhythmic drugs, with special reference to the actions of amiodarone. **2.5**

## Pharmacokinetics

- 17. Identify the routes of administration, biotransformation, and excretion of selected antiarrhythmic drugs.3.0
- Discuss the pharmacokinetics and time-course of the cardiac actions of antiarrhythmic drugs (onset and duration of action).
   2.0
- 19. Explain the impact of reduced cardiac output due to myocardial infarction and cardiomyopathy<br/>on drug half-life and pharmacodynamics.2.0
- 20. Explain the influence of age on pharmacokinetic parameters, including liver metabolism (lidocaine, procainamide, and propranolol) and elimination through kidney (digoxin and sotalol).

2.0

## Adverse Effects, Drug Interactions and Contraindications

21.	21. Describe the cardiac and extracardiac manifestations of toxicity from antiarrhythmic drugs.		
		3.0	
22.	Discuss the beneficial and adverse interactions among antiarrhythmic drugs and between	1	
	antiarrhythmic drugs and cardiac glyscosides.	3.0	
23.	Explain the significance of electrolyte and acid-base imbalance in arrhythmia generation a	and	
	their influence on antiarrhythmic drug action.	4.0	
24.	Identify the possible contraindications of antiarrhythmic drugs in the presence of heart bl congestive heart failure, as well as the precautions and contraindications in other conditions	lock or ons. <b>3.5</b>	
25.	Discuss the classes of drugs (both antiarrhythmic and non-antiarrhythmic) that can produ	ce	
	acquired long QT Syndrome (LQTS).	3.0	
The	erapeutic uses		
26.	Explain the use of antiarrhythmic drugs in supraventricular arrhythmias.	4.0	
27.	Explain the use of antiarrhythmic drugs in ventricular arrhythmias.	4.0	
28.	Explain the utility of antiarrhythmic drugs in combination with electrical cardioversion or		
	implantable cardioverter-defibrillators.	2.0	

## B. Drugs for Management of Heart Failure

1.	Discuss a.	the foll ACE Ini	owing with regard to subsequent objectives: nibitors	4.0
		i. 	captopril	
	6	II.	enalapril	
	D.	angiote	ensin receptor blockers	4.0
		l. Jaan di	losartan	2.0
	ι.	100p 01	furecomide	3.0
		I. ;;	athospupic acid	
	Ь	n. thiazid	e diuretics	2.0
	u.	i	bydrochlorothiazide	5.0
	P	Reta Bl	ockers	4.0
	f.	Digoxir		3.0
	g.	Dobuta	amine	2.0
	h.	Dopam	ine	2.0
	i.	Milrino	ne	1.0
	j.	Hydrala	azine	2.0
	k.	Nitropr	russide	1.0
	I.	Isosorb	vide Nitrate	1.0
	m.	Nitrogl	ycerin	3.0
Phy	ysiology	y and Pa	athophysiology	
2. 3.	Discuss Describ	the acu e the ba	ite inotropic, dromotropic, and chronotropic effects of catecholamines. asic pathophysiology of heart failure, and identify the cardiac and extraca	<b>4.0</b> rdiac
	compe	nsatory	mechanisms that are activated.	4.0
4.	Discuss	current	recommendations for management of acute and chronic heart failure.	3.0
5.	Explain	the role	e of genetics and ethnicity in the physiology of heart failure and in the reg	gulation
	of resp	onsiven	ess to agents used in heart failure.	1.5
Me	chanis	n of Ac	tion	
6.	Discuss	the effe	ects of digoxin on myocardial contractility.	4.0

7.	Explain the ionic basis for the mechanism of action of digoxin and the cardiac glycosides	as a
	class of agents.	2.5
8.	Discuss the roles of Na <sup>+</sup> , K <sup>+</sup> -ATPase inhibition and the Na <sup>+</sup> /Ca <sup>2+</sup> exchanger.	3.0
9.	Describe the electrophysiologic effects of digoxin on atrial and ventricular muscle and	
	specialized conducting tissue.	3.0
10.	Explain the significance of direct and indirect (autonomic) actions of digoxin.	3.0
11.	Discuss the positive inotropic effects of the $\beta$ -adrenoceptor-agonists and phosphodieste	rase
	inhibitors.	3.5
12.	Discuss the effects of adrenoceptor antagonists and ACE-inhibitors on cardiac function a	nd
	ventricular remodeling in the setting of heart failure.	3.5
Ac	tions on organ systems	
13.	Describe the hemodynamic actions of digoxin in the failing heart.	3.0
14.	Outline the extracardiac actions of digoxin.	2.5

15. Discuss the effects of vasodilators on preload and afterload. **3.5** 

	16.	Descrit phosph	be the extracardiac actions of the adrenoceptor agonists, adrenoceptor antagonis hodiesterase inhibitors, and ACE-inhibitors.	sts, <b>3.0</b>
	Pha	armaco	okinetics	
	17. 18.	Identif routes Descrik	fy the routes of administration, the extent of oral absorption and bioavailability, a of elimination and extent of biotransformation of drugs used in heart failure. be the concept of digitalization (loading dose) and maintenance therapy.	nd the 2.0 2.0
	Adv	verse E	Effects, Drug Interactions and Contraindications	
<ol> <li>Describe the cardiac (delayed depolarizations and arrhythmias) and extracardiac m of digoxin toxicity (digoxin levels &gt; 2.0 ng/ml are associated with toxicity).</li> <li>Explain the significance of changes in serum electrolyte levels (potassium, sodium, magnesium) with regard to digoxin toxicity.</li> <li>Discuss potential adverse effects with concomitant use of diuretics (both potassiur potassium depleting) in the elderly or in patients with congestive heart failure, hyp and renal disease.</li> <li>Identify interactions of digoxin and quinidine, verapamil, and other relevant drugs.</li> <li>Discuss the cardiac and extracardiac side effects and limitations of the antagonist a vasodilators, phosphodiesterase inhibitors. and ACE-inhibitors.</li> </ol>			be the cardiac (delayed depolarizations and arrhythmias) and extracardiac manife oxin toxicity (digoxin levels > 2.0 ng/ml are associated with toxicity). In the significance of changes in serum electrolyte levels (potassium, sodium, calci esium) with regard to digoxin toxicity. Is potential adverse effects with concomitant use of diuretics (both potassium-spa- sium depleting) in the elderly or in patients with congestive heart failure, hypothy inal disease. Fy interactions of digoxin and quinidine, verapamil, and other relevant drugs. Is the cardiac and extracardiac side effects and limitations of the antagonist agent ilators, phosphodiesterase inhibitors, and ACE-inhibitors.	estations <b>3.0</b> um, <b>4.0</b> uring and roidism <b>4.0</b> <b>2.0</b> s, <b>3.5</b>
	The	erapeu	itic uses	
	24. 25. 26.	Describ and AC Explain metallo other a	be the role of adrenoceptor agonists, adrenoceptor antagonists, vasodilators, diu CE-inhibitors in the treatment of acute and chronic heart failure. In the use of atrial natriuretic peptide agonists, endothelial receptor antagonists, a oprotease inhibitors in the management of acute severe heart failure unresponsi agents.	4.0 retics, 4.0 ind ve to 2.0
С.	Dru	igs for	Management of Hypertension	
	1.	Discuss a.	s the following with regard to subsequent objectives: ACE Inhibitors i. captopril ii. epalapril	4.0
		b.	Angiotensin Receptor Blockers i. losartan ii. valsartan iii. candasartan	4.0
		C.	Thiazide Diuretics	4.0
		d.	<ul> <li>Nydrochlorothlazide</li> <li>Non-Selective Beta Blockers <ol> <li>propranolol</li> <li>pindolol</li> <li>timolol</li> <li>nadolol</li> </ol> </li> </ul>	3.0
		e.	Beta <sub>1</sub> -Selective Blockers i. metoprolol ii. atenolol	4.0

iii. nebivolol

f.	Alpha and Beta Blockers	3.5
	i. labetalol	
	ii. carvedilol	
g.	Alpha <sub>1</sub> Blockers	3.0
	i. prazosin	
	ii. terazosin	
	iii. doxazosin	
h.	Calcium Channel Blockers	3.0
	i. diltiazem	
	ii. verapamil	
i.	Dihydropyridines	3.0
	i. nifedipine	
	ii. nicardipine	
	iii. amlodipine	
J.	Alpha <sub>2</sub> Agonists	3.0
	i. clonidine	
	ii. methyldopa	
k.	Phentolamine	2.0
I.	Phenoxybenzamine	2.0
m.	Nitrates/Nitroglycerin/ Nitroprusside	3.0
n.	Hydralazine	2.0
0.	Minoxidil	2.0
p.	Diazoxide	1.0
q.	Fenoldopam	1.0
r.	Furosemide	3.0

## Introduction to the Vascular System and its Regulation

2.	Identify the determinants of systemic arterial blood pressure including the role of the autonomic
	nervous system, the regulation of fluid volume and the renin-angiotensin aldosterone system.

		4.0	
3.	Explain the role of the central nervous system in the regulation of blood pressure.	3.0	
4.	Explain the role of vascular endothelium and locally released regulators of vascular ton	e in the	
	maintenance of blood pressure.	4.0	
5.	Identify the types of hypertension and the relative prevalence of each.	3.0	
6.	Discuss current views for the etiology of essential hypertension.	2.5	
Mechanism of Action			

- Identify the mechanism of action of each of the several classes of agents used to manage hypertension according to the site of action within the pathogenesis of hypertension.
   4.0
- Discuss the end organ effects of untreated hypertension and the beneficial effects achieved by therapeutic management of the disease.
   3.0
- 9. Explain the actions of antihypertensive drugs on the heart, renal blood flow and renal function.

3.0

10. Explain the relevant actions of antihypertensive drugs in other organ systems (eg, CNS). 2.0

## Pharmacokinetics

	11.	Describe the time-course of antihypertensive activity (onset and duration of action) for class of agents.	each <b>2.0</b>
	Ad	verse Effects, Drug Interactions and Contraindications	
	12. 13.	<ul> <li>Discuss the cardiac and extracardiac side effects of antihypertensive drugs, including references.</li> <li>Identify both beneficial and adverse interactions between antihypertensive drugs, as we between antihypertensive drugs and other therapeutic agents.</li> </ul>	flex <b>3.0</b> ell as <b>2.0</b>
	Th	erapeutic Uses	
	14. 15. 16. 17.	<ul> <li>Explain the role of nonpharmacological treatment modalities in the management of hypertension.</li> <li>Explain the use of antihypertensive drugs in hypertensive emergencies and in pregnance eclampsia).</li> <li>Explain the use of antihypertensive drugs in pheochromocytoma.</li> <li>Identify subgroups with special antihypertensive drug considerations.</li> </ul>	3.0 y (eg, 3.0 3.0 3.0
D.	Dru	ugs for the Management of Angina and Coronary Artery Disease	
	1.	Discuss the following with regard to subsequent objectives: a. Non-Selective Beta Blockers i. propranolol	3.0
		ii. timolol b. Beta <sub>1</sub> -Selective Blockers i. metoprolol ii. atenolol	3.0
		c. Calcium Channel Blockers i. diltiazem ii. verapamil iii. nifedipine iv. nicardipine	3.0
	ا مع	d. Nitrates	4.0
	<ol> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> </ol>	Explain the normal regulation of coronary blood flow and the relationship to the events cardiac cycle. Identify the normal determinants of cardiac oxygen consumption and supply. Describe the basic pathophysiology of myocardial ischemia. Explain the significance of atherosclerotic coronary artery disease and coronary artery s (Prinzmetal's) in the production of myocardial ischemia and angina pectoris.	of the 3.0 4.0 4.0 pasm 4.0
	Me	echanism of Action	
	6. 7.	Explain the hemodynamic actions of antianginal drugs, including their coronary and per vasodilator actions. Discuss the effects of each antianginal drug or drug class on the determinants of myoca oxygen consumption (heart rate, myocardial wall tension, etc.) and/or oxygen supply (creblood flow).	ipheral <b>4.0</b> rdial oronary <b>4.0</b>

8. Discuss the effects of the antianginal drugs at the cellular level. 2.0

## Actions on Organ Systems

	9. 10.	Describe the cardiac actions of antianginal drugs (electrophysiologic, coronary vasodilator, inotropic actions). <b>3.5</b> Discuss the actions of antianginal drugs on the peripheral circulation (arterial, venous), as well as their effects on ventricular preload and afterload. <b>3.5</b>			
	Pha	armaco	okinetics		
	<ol> <li>Explain the significance of a "first-pass effect" for orally administered antianginal dru rationale underlying sublingual, intranasal and transdermal administration of nitrates</li> <li>Identify the time-course of antianginal activity (onset and duration of action).</li> <li>Explain the problem of dose intervals and tolerance development with the nitrates.</li> </ol>				
	Adv	verse E	Effects, Drug Interactions and Contraindications		
	14. 15. 16. 17.	Discuss the inte Discuss antiang Explain angina Explain contex	s the cardiac and extra-cardiac side effects of antianginal drugs with special in ceraction with drugs used to treat erectile dysfunction. Is the beneficial and adverse interactions between antianginal drugs, as well a ginal drugs and other cardiovascular drugs. In the use of antianginal drugs in classic (effort-related) angina pectoris and va- pectoris. In the concept of "myocardial preservation" and discuss the use of antiangina at of acute myocardial infarction with particular emphasis on adrenoceptor a	reference to <b>3.0</b> as between <b>3.0</b> asospastic <b>3.5</b> I drugs in the ntagonists. <b>3.0</b>	
Ε.	Drι	igs for	the Management of Hyperlipidemias		
	1.	Discuss a.	s the following with regard to subsequent objectives: Statins i. lovastatin ii. atorvastatin iii. simvastatin	4.0	
		b.	Resins i. cholestyramine ii. colesevalam	2.0	
		с.	Gemfibrozil	4.0	
		d.	Ezetimibe	2.0	
		e.	Omega-3 Fatty Acids	3.0	
		ι.		3.0	

## Physiology and Pathophysiology: Lipid Interactions with the Cardiovascular System

Explain cholesterol synthesis, transport, export, excretion, and receptor mediated cellular		
uptake.	3.0	
Identify reference intervals for lipid levels.	4.0	
Discuss the relevant hypotheses regarding the etiology of hyperlipidemias.	0.5	
Describe the basic pathophysiology of atherosclerotic vascular disease and its relation	nship to	
the hyperlipidemias ("cholesterol" or "infectious agent").	3.0	
Describe the types of hyperlipidemias (I, II, III, IV, and V), as well as the alterations in s	erum	
lipids in each type (triglycerides, cholesterol, LDL, HDL, LDL, lipoproteins).	3.0	
	Explain cholesterol synthesis, transport, export, excretion, and receptor mediated cell uptake. Identify reference intervals for lipid levels. Discuss the relevant hypotheses regarding the etiology of hyperlipidemias. Describe the basic pathophysiology of atherosclerotic vascular disease and its relation the hyperlipidemias ("cholesterol" or "infectious agent"). Describe the types of hyperlipidemias (I, II, III, IV, and V), as well as the alterations in s lipids in each type (triglycerides, cholesterol, LDL, HDL, LDL, lipoproteins).	

7. Identify the lipid profile characteristic of insulin-resistant diabetics.

## **Mechanisms of action**

- Explain the actions of each drug class on serum lipids, and compare and contrast the mechanism of each of these actions. 4.0
- 9. Identify the advantages of combinations of agents in the management of hyperlipidemia.

### Actions on organ systems

- 10. Describe alterations in plasma lipids due to other drugs (eg, protease inhibitor-induced hyperlipidemia; estrogen-induced hypolipidemia). 2.0
- 11. Explain the role of the HMG CoA reductase inhibitors in preventing acute coronary events and stroke and as adjuncts in the management of dementia and other pathological disorders.

3.0

### **Pharmacokinetics**

12. Explain the absorption, distribution, metabolism, and excretion of lipid lowering drugs. 2.0

## Adverse Effects, Drug Interactions and Contraindications

- 13. Discuss the cardiovascular and other systemic side effects of these drugs with special reference to the muscle and liver toxicities. 3.0
- 14. Describe the beneficial and adverse interactions associated with these drugs. 2.0
- 15. Recommend nonpharmacological management of hyperlipidemia (ie, life style modifications).

2.0

#### IV. **Renal Drugs**

A. Drugs Affecting Renal Function, Water and Electrolyte Metabolism

	1.	Discuss the following with regard to subsequent objectives : a. Desmopressin (dDAVP) b. Conivaptan	2.0 1.0		
		c. Vasopressin	3.0		
		d. Demeclocycline	1.0		
	Ph	ysiology and Pathophysiology			
	2. 3.	Explain the mechanisms through which the kidney makes concentrated or dilute urine. Describe the roles of vasopressin, aquaporins, V1 and V2 receptors, cyclic AMP, and	4.0		
		prostaglandins in regulating renal epithelial water permeability.	2.5		
Mechanisms of Action					
	4.	Explain how drugs can mimic or interfere with the cellular mechanisms of vasopressin.	3.0		
Actions on Organ Systems					
	5.	Summarize the renal and extrarenal effects of vasopressin and desmopressin.	2.5		
	Pharmacokinetics				

### 3.0

3.0

	6.	Explain how altering the structure of vasopressin affects its pharmacokinetics and pharmacodynamics.	1.0	
Adverse Effects, Drug Interactions and Contraindications				
	<ol> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> <li>11.</li> </ol>	Explain the mechanism of vasoconstriction produced by vasopressin. Explain how NSAIDs and clonidine can alter water reabsorption by the kidney. Discuss the possible toxicity of correcting hyponatremia with vasopressin antagonists. Explain how drugs such as clonidine, chlorpropamide, demeclocycline, lithium, and NSAI modify the action of vasopressin. Explain the alteration of ACTH secretion by blocking the V1 receptor.	3.0 4.0 1.0 Ds can 2.5 2.0	
	The	erapeutic Uses		
	12. 13. 14.	Describe the therapy of central and nephrogenic diabetes insipidus. Outline the pharmacological treatment of the syndrome of inappropriate ADH secretion (SIADH). Explain the mechanisms of demeclocycline and lithium carbonate interference with rena reabsorption.	3.0 3.0 al water 1.0	
В.	Diu	retic Drugs		
	1.	Discuss the following with regard to subsequent objectives: a. Acetazolamide b. Bumetanide c. Ethacrynic Acid d. Eplerenone e. Amiloride f. Mannitol g. Furosemide h. Thiazides i. subhydrochloria thiazide j. Triamterine	1.0 3.0 2.0 3.0 1.0 4.0 4.0 3.0 3.0	
	Phy	siology and Pathophysiology		
	2. 3. 4.	Describe the location and function of major ion transporters and channels on renal epith membranes. Explain the influence of sodium transport on the reabsorption of other ions and water in kidney. Explain hypertension or edema caused by abnormal renal function.	nelial 3.0 1 the 3.0 3.0	
	Me	chanism of Action		
	5. 6. 7. <b>Act</b>	Outline the changes that occur with electrolyte transport, water reabsorption and hemodynamics when specific diuretics inhibit kidney function. Differentiate the effects of K <sup>+</sup> - sparing diuretics. Identify the hypokalemic action of some diuretics and use of supplemental therapeutics prevent hypokalemia. <b>:ions on Organ Systems</b>	4.0 4.0 to 4.0	

	8. Explain the hemodynamic, ion transport, and excretory effects of different classes of diu drugs.			iuretic <b>4.0</b>	
	Pha	armaco	okinetics		
	9.	Explair of diur	n the importance of the organic anion transporters and protein binding to the re etics.	nal action <b>3.0</b>	
	10.	Describ	be how other drugs or diseases can interfere with the effects of diuretics.	3.0	
	Ad	verse E	ffects, Drug Interactions and Contraindications		
	<ol> <li>11.</li> <li>12.</li> <li>13.</li> <li>14.</li> <li>15.</li> <li>16.</li> </ol>	<ol> <li>Explain how thiazides and loop diuretics can cause a metabolic alkalosis.</li> <li>Relate hyponatremia to diuretic therapy.</li> <li>Relate metabolic imbalances with diuretic therapy to glucose, urate, lipids, calcium, magnesic and potassium.</li> <li>Explained the underlying mechanisms involved in metabolic imbalances with diuretic therapy especially in relation to glucose, uric acid, lipids, calcium, magnesium, and potassium.</li> <li>Identify the clinical consequences of interactions between diuretics and drugs such as cardiac glycosides, oral hypoglycemics, uricosurics, aminoglycosides, amphotericin B, NSAIDs, and angiotensin inhibitors.</li> </ol>			
	Th	aranou		0.0	
	<ol> <li>Identify the renal and extrarenal mechanisms through which diuretics are useful in tree hypertension and edema.</li> <li>Explain reduced toxic performative through osmotic drugs</li> </ol>				
/.	<u>Pu</u>	lmonar	y Drugs		
А.	Dru	ugs for	Management of Respiratory Diseases		
	1.	Discuss a.	s the following with regard to subsequent objectives: Inhaled Corticosteriods i. beclomethasone ii. fluticasone	4.0	
		b.	Cromolyn	2.0	
		с.	Omalizumab	1.0	
		d.	Leukotriene Inhibitors i. zafirlukast ii. montelukast iii. pranlukast iv. zileuton	4.0	
		e.	Beta-2 Agonists i. albuterol ii. pirbuterol iii. terbutaline iv. salmeterol	4.0	
		f.	Formterol Theophylline	3.0	
		g.	Ipratropium	3.0	

v.

h. Tiotropium

## Physiology and Pathophysiology: Introduction to Respiratory Physiology

<ol> <li>Identify the endogenous chemical mediators and receptors that function to regulate bronch smooth muscle tone.</li> <li>3.5</li> </ol>	ial
<ol> <li>Explain the role of cyclic AMP, leukotrienes and nitric oxide in regulation of bronchiolar smormuscle and pulmonary vasculature.</li> <li>3.5</li> </ol>	oth
4. Explain the role of phosphodiesterases and the various isoenzymes of PDE in the function of	:
bronchiolar smooth muscle and in the inflammatory process. <b>2.0</b> 5 Relate bronchial smooth muscle reactivity to the nathogenesis of asthma <b>4.0</b>	
<ol> <li>Explain the role of the inflammatory process in the pathogenesis of asthma and chronic</li> </ol>	
obstructive pulmonary disease (COPD). <b>4.0</b>	
<ol> <li>Discuss the similarities and differences between asthma, allergic rhinitis, and COPD, as well a the treatments for each disorder.</li> <li>3.0</li> </ol>	as
Mechanisms of Action	
8. Identify the mechanism of action of each of the major classes of agents relative to the component of pathogenesis to distinguish between agents that modify the disease process	
versus those that relieve symptoms. 4.0	
10. Explain the use of agents to treat acute episodes of asthma and in the treatment of exercise	-
induced asthma. 4.0	
11. Explain the use of various agents in the treatment of COPD.3.0	
Actions on Organ Systems	
12. Describe the relevant actions of these drugs on other physiological systems. <b>3.0</b>	
Pharmacokinetics	
<ol> <li>13. Identify the factors that influence the plasma levels of theophylline.</li> <li>14. Identify the appropriate route of administration of the various bronchodilators relative to the physico-chemical characteristics and the pharmacological action of the drug.</li> <li>15. Discuss the relative merits of inhalant administration versus oral or parenteral administration for the management of both episodic and chronic asthma.</li> </ol>	n n
Adverse Effects, Drug Interactions and Contraindications	
<ul> <li>16. Discuss the adverse effects and contraindications for each class of agents.</li> <li>3.5</li> <li>17. Explain the potential for allergic reactions to ipratropium in patients allergic to soy or peanur products.</li> </ul>	t
Therapeutic Uses	
<ol> <li>Recommend management of acute and chronic asthma and chronic obstructive pulmonary disease.</li> <li>3.5</li> </ol>	
Gastrointestinal Drugs	

A. Drugs Used for Treatment of Peptic Ulcer Disease

VI.

1. Discuss the following with regard to subsequent objectives:

	a. Histamine	4.0
	b. Cimetidine	4.0
	c. Ranitidine	4.0
	d. Famotidine	3.0
	e. Nizatidine	3.0
	Physiology and Pathophysiology	
	2. Explain the neurohumoral control of H <sup>+</sup> secretion by gastri	c parietal cells. <b>4.0</b>
	3. Explain the role of histamine in the different phases $H^+$ see	cretion. <b>2.0</b>
	<ol> <li>Identify the causes of H<sup>+</sup> hypersecretion.</li> </ol>	2.0
	Mechanism of Action	
	5. Describe the molecular mechanism of action of the H2 rec	eptor antagonists. <b>4.0</b>
	Actions on Organ Systems	
	6. Discuss the pharmacological effects of H2 antagonists on t	he stomach. <b>3.5</b>
	7. Discuss the effects of H2 antagonists on other organ syste	ms. <b>1.5</b>
	Pharmacokinetics	
	8. Discuss the pharmacokinetics of the H2 receptor antagoni	sts. <b>3.0</b>
	Adverse Effects, Drug Interactions and Contraindications	
	9. Describe the principal adverse effects of each t H2 receptor	or antagonist. <b>3.0</b>
	10. Identify the clinically important drug interactions of H2 red	ceptor antagonists. <b>3.5</b>
	11. Identify the principal contraindications of H2 receptor ant	agonists. <b>3.0</b>
	Therapeutic Uses	
	12. Identify and describe the disorders treated with H2 recept	or antagonists. <b>4.0</b>
В.	3. Proton Pump Inhibitors	
	1. Discuss the following with regard to subsequent objectives:	
	a. Omeprazole	4.0
	b. Esomeprazole	4.0
	c. Lansoprazole	3.0
	d. Rabeprazole	3.0
	e. Pantoprazole	3.0
	Physiology and Pathophysiology	
	2. Describe the mechanism of $H^{+}$ production by the parietal cell $H^{+}_{1}$	/K <sup>+</sup> ATPase. <b>3.0</b>
	Mechanism of Action	
	<ol> <li>Describe the mechanism of action of proton pump inhibitors and parietal cell proton pump.</li> </ol>	d why they are selective for the <b>4.0</b>
	Actions on Organ Systems	
	4. Discuss the pharmacological effects of the drugs on gastric funct	ion. <b>2.5</b>
	Pharmacokinetics	

	5.	Discuss the pharmacokinetics of proton pump inhibitor.	3.0			
	Adverse Effects, Drug Interactions and Contraindications					
	6. 7. 8.	Describe the principal adverse effects of proton pump inhibitors. Identify the clinically important drug interactions of proton pump inhibitors. Identify the principal contraindications of proton pump inhibitors.	3.0 2.5 2.0			
	Th	erapeutic Uses				
	9.	Identify and describe the principal disorders treated using proton pump inhibitors.	3.0			
С.	An	tacid Preparations				
	1.	<ul> <li>Discuss the following with regard to subsequent objectives:</li> <li>a. Calcium carbonate</li> <li>b. Magnesium hydroxide</li> <li>c. Aluminum hydroxide</li> <li>d. Sodium Bicarbonate</li> <li>e. Magnesium hydroxide/aluminum hydroxide</li> </ul>	3.0 3.0 3.0 2.0 3.0			
	Ph	ysiology and Pathophysiology				
	2.	Describe the mechanisms of $H^{+}$ secretion in the stomach.	3.0			
	M	echanism of Action				
	3. 4.	Describe the mechanism of action of antacid medications. Differentiate between the onset and duration of action of each antacid preparation.	3.0 2.5			
	Actions on Organ Systems					
	5.	Discuss the pharmacological effects of antacid drugs in each class on the stomach.	2.5			
	Pharmacokinetics					
	6.	Explain the absorption and systemic actions of antacid preparations.	3.0			
	Ad	verse Effects, Drug Interactions and Contraindications				
	7. 8. 9.	Describe the principal adverse effects of each antacid preparation. Identify the clinically important drug interactions with antacids. Identify the principal contraindications of antacids.	3.0 3.5 2.5			
	Therapeutic Uses					
	10	. Identify the primary indication for antacid use.	3.0			
D.	Cy	toprotectant Agents				
	1.	<ul> <li>Discuss the following with regard to subsequent objectives:</li> <li>a. PGE₂</li> <li>b. Misoprostol</li> <li>c. Sucralfate</li> </ul>	3.5 4.0 2.5			
	Physiology and Pathophysiology					
	2. 3.	Describe the mechanisms for production of the gastric cytoprotective barrier. Identify and explain causes for disruption of the cytoprotective barrier.	4.0 4.0			

	Mechanism of action				
	4.	Describe the mechanism of action of cytoprotectant drugs.	3.0		
	Actions on Organ Systems				
	5. Describe the pharmacological effect of cytoprotectant drugs on the cytoprotective barrie				
	Ph	armacokinetics			
	<ol> <li>Discuss the absorption, distribution metabolism, and excretion of cytoprotectant agents.</li> <li>2</li> </ol>				
Adverse Effects, Drug Interactions and Contraindications					
	7. 8.	Describe the principal adverse effects of cytoprotectant drugs. Identify clinically important drug interactions of the cytoprotectant drugs in each class.	3.5 3.0		
	Th	erapeutic Uses			
	9.	Identify the primary indication for use of cytoprotectant drugs.	3.0		
Ε.	Dr	ugs for Helicobacter Pylori Eradication			
	1.	<ul> <li>Discuss the following with regard to subsequent objectives:</li> <li>a. Clarithromycin</li> <li>b. Metronidazole</li> <li>c. Amoxicillin</li> <li>d. Tetracycline</li> <li>e. Proton Pump Inhibitors</li> <li>f. H2 blocker</li> <li>g. Bismuth subsalicylate</li> </ul>	4.0 4.0 2.0 4.0 4.0 2.5		
	Ph	ysiology and Pathophysiology			
	2. 3.	Explain the role of <i>H. pylori</i> in peptic ulcer disease Describe tests for evaluating <i>H. pylori</i> infection.	4.0 2.5		
	Th	erapeutic Uses			
	4.	Discuss the use of triple and quadruple therapy regimens used for <i>H. pylori</i> eradication.	4.0		
	Me	echanism of Action			
	5.	Explain the contribution of each agent in triple or quadruple therapy regimens in <i>H. pylc</i> eradication.	ori <b>2.5</b>		
	Dr	ug Interactions			
	6. 7.	Identify potential interactions of drugs used for <i>H. pylori</i> eradication. Discuss the potential for antibiotic resistant strains of <i>H. pylori</i> .	3.0 3.0		
F.	Pro	okinetic Drugs			
	1.	Define the following: a. <i>Erythromycin</i>	3.5		

b.	Metoclopramide	2.5
с.	Cisapride off market	0.5
d.	Domperidone	1.0
e.	Tegaserod	0.5
f.	Lubiprostone	4.0
g.	Alvimopan	3.5
h.	Neostigmine	1.0
i.	Bethanechol	1.5

## G. Laxatives

Н.

1	Discuss the f	allowingwith	rogard to	cubco au ont	abiantivary
1.	DISCUSS LITE I	onowing with	regard to :	subsequent	objectives:

a.	Psylium	2.5
b.	Methylcellulose	2.5
c.	Sodium phosphate	2.5
d.	Sodium citrate	2.5
e.	Lactulose	3.0
f.	Castor oil	2.0
g.	Bisacodyl	2.0
h.	Senna	2.0
i.	Cascara	2.0
j.	Mineral oil	2.0

## Physiology and Pathophysiology

2.	. Describe the neural and hormonal mechanisms controlling stomach and intestinal motility.	
		3.0
3.	Explain the changes in neural and hormonal control of stomach and intestinal motility th to delayed gastric emptying or accommodation.	at lead <b>2.5</b>
Me	echanisms of Action	
4.	Explain the molecular mechanism of action of prokinetic drugs in each drug class.	3.0
Act	tions on Organ Systems	
5.	Discuss why some drugs are selective for upper GI motility disorders and why others are selective for lower GI motility disorders.	2.0
Pha	armacokinetics	
6.	Identify the relevant pharmacokinetic features of prokinetic drugs in each drug class.	2.0
Ad	verse Effects, Drug Interactions and Contraindications	
7.	Describe the principal adverse effects of the drugs of each class.	3.0
8.	Describe the clinically important drug interactions of the drugs of each class.	2.0
9.	Identify the principal contraindications of the drugs of each class.	2.0
The	erapeutic Uses	
10.	Explain with the main therapeutic uses of the drugs of each class.	2.0
An	ti-diarrheal Drugs	

1. Discuss the following with regard to subsequent objectives:

	4.0
b. Diphenoxylate	3.0
c. Alosetron	1.5
d. Clansetron	2.0
e. Cionidine f Bifidobastorium infantis	3.U 0 E
$\pi$ Bismuth subsolicylate	0.5
g. Districti subsalicylate	2.5
Physiology and Pathophysiology	
2. Discuss the neural mechanisms controlling colonic motility and water and electrolyte a	bsorption
and secretion.	3.0
3. Identify and describe the conditions under which neural mechanisms controlling coloni	С
motility, as well as water and electrolyte absorption and secretion are impaired.	3.0
4. Discuss the neural mechanisms of visceral sensation and visceral pain.	2.0
5. Discuss the importance of maintaining normal gut flora and how disruption can lead to	altered
motility and absorption and secretion in the colon.	2.5
Mechanisms of Action	
6 Describe the molecular mechanism of action of each anti-diarrheal drug in each drug cl	200
	<b>3.0</b>
	5.0
Actions on Organ Systems	
7. Explain the effects of each anti-diarrheal drug on the colon and also on other organ sys	tems.
	3.0
Pharmacokinetics	
8. Discuss the absorption, distribution, metabolism, and secretion of each anti-diarrheal c	Irug.
	2.0
Adverse Effects, Drug Interactions and Contraindications	
9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.	2.0
<ol> <li>Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> </ol>	<b>2.0</b> heals.
<ol> <li>Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> </ol>	<b>2.0</b> heals. <b>2.0</b>
<ol> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> </ol>	2.0 heals. 2.0 iarrheal.
<ol> <li>Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> </ol>	2.0 heals. 2.0 liarrheal. 2.0
<ol> <li>Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> </ol>	2.0 heals. 2.0 liarrheal. 2.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> </ul>	2.0 Theals. 2.0 Jiarrheal. 2.0 2.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> </ul>	2.0 Theals. 2.0 Jiarrheal. 2.0 2.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> <li>1. Discuss the following with regard to subsequent objectives:</li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> <li>1. Discuss the following with regard to subsequent objectives: <ul> <li>a. Sulfapyridine</li> <li>b. Sulfapyridine</li> </ul> </li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0 3.5 2.5
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> <li>1. Discuss the following with regard to subsequent objectives: <ul> <li>a. Sulfapyridine</li> <li>b. Sulfasalazine</li> <li>c. S-amino solicylic acid</li> </ul> </li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0 3.5 3.5 3.5 3.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-diarr</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> <li>1. Discuss the following with regard to subsequent objectives: <ul> <li>a. Sulfapyridine</li> <li>b. Sulfasalazine</li> <li>c. 5-amino salicylic acid</li> <li>d. Hydrocertisone</li> </ul> </li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0 3.5 3.5 3.0 4.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> <li>1. Discuss the following with regard to subsequent objectives: <ul> <li>a. Sulfapyridine</li> <li>b. Sulfasalazine</li> <li>c. 5-amino salicylic acid</li> <li>d. Hydrocortisone</li> <li>e. Prednisone</li> </ul> </li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0 3.5 3.5 3.5 3.0 4.0 4.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> <li>1. Discuss the following with regard to subsequent objectives: <ul> <li>a. Sulfapyridine</li> <li>b. Sulfasalazine</li> <li>c. 5-amino salicylic acid</li> <li>d. Hydrocortisone</li> <li>e. Prednisone</li> <li>f. Prednisolone</li> </ul> </li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0 3.5 3.5 3.0 4.0 4.0 4.0 4.0
<ul> <li>9. Explain principal adverse effects of the drugs of each class of anti-diarrheals.</li> <li>10. Discuss the clinically important drug interactions of the drugs of each class of anti-diarr</li> <li>11. Identify and desrcibe the principal contraindications of the drugs of each class of anti-d</li> <li>Therapeutic Uses</li> <li>12. Discuss with the specific therapeutic applications of each class of drug.</li> <li>Drugs Used for Treatment of Inflammatory Bowel Disease</li> <li>1. Discuss the following with regard to subsequent objectives: <ul> <li>a. Sulfapyridine</li> <li>b. Sulfasalazine</li> <li>c. 5-amino salicylic acid</li> <li>d. Hydrocortisone</li> <li>e. Prednisone</li> <li>f. Prednisolone</li> <li>g. Methotrexate</li> </ul> </li> </ul>	2.0 heals. 2.0 liarrheal. 2.0 2.0 3.5 3.5 3.0 4.0 4.0 4.0 4.0 4.0

Ι.

h.	6-mercaptopurine	2.5
i.	Azathioprine	2.5
j.	Cyclosporine	2.5
k.	Infliximab	4.0
I.	Metronidazole	3.0
m.	Ciprofloxacin	2.5
n.	Clarithromycin	3.5
0.	Lactobacillus	1.5

## Pathophysiology

disease.

2.	Identify the mechanisms responsible of intestinal and extra-intestinal symptoms of	
	inflammatory bowel disease.	2.0
3.	Discuss the contribution of intestinal bacteria to the pathophysiology of inflammatory b	owel

## **Mechanism of Action**

 Identify and describe the mechanism of action of each of the major classes of drugs used to treat inflammatory bowel disease.
 3.0

## Pharmacokinetics

5.	Explain the routes of administration of drugs in each class used to treat inflammatory bo	owel
	disease.	2.5

- 6. Discuss the absorption and distribution of each class of drug used to treat inflammatory bowel disease and identify the impact on the choice of the route of administration. **2.5**
- Describe the mechanisms for bioactivation of the salicylates and identify the impact treatment of inflammatory bowel disease.
   2.5

## Adverse Effects, Drug Interactions and Contraindications

8.	Describe the main adverse effects of the drugs of each class used to treat inflammatory b	owel
	disease.	3.0
9.	Explain the clinically important drug interactions of the drugs of each class used to treat	

inflammatory bowel disease. **3.0** 10. Discuss the principal contraindications or precautions of the drugs of each class used to treat inflammatory bowel disease. **3.0** 

### Therapeutic Uses

11. Discuss the selective use of each class of drug for the treatment of ulcerative colitis versus Crohn's disease. **2.0** 

## J. Drugs Used to Induce or Treat Nausea and Vomiting

1. Discuss the following with regard to subsequent objectives:

a.	Apomorphine	2.0
b.	Syrup of ipecac	2.0
c.	Metoclopramide	3.5
d.	Chlorpromazine	3.0
e.	Haloperidol	1.5
f.	Ondansetron	4.0
g.	Granisetron	3.0

2.0

		h. Palonosetron	3.0
		i. Dolasetron	3.0
		j. Ramosetron	3.0
		k. Dronabinol	4.0
		I. Diphenhydramine	4.0
		m. Cyclizine	2.5
		n. Hydroxyzine	2.5
		0. Prometnazine	3.0
		p. Aprepitant	3.U 2 E
		q. Dexamethasone	2.5
		s Alprazolam	4.0
		t Sconolamine	3.5
	Phy	ysiology and Pathophysiology	010
	2.	Discuss the central and peripheral nervous system mechanisms responsible for nausea	and
		vomiting.	3.0
	Me	echanisms of Action	
	3.	Describe the mechanism of action of emetic and anti-emetic drugs.	3.5
	4.	Identify the cellular and molecular mechanisms of action of each emetic drug class.	3.0
	5.	Explain the use of multi-drug treatment of nausea and vomiting.	3.0
	Act	tions on Organ Systems	
	6.	Describe the pharmacological effects of each emetic/anti-emetic drug in each class.	3.0
	Pha	armacokinetics	
	7.	Identify the absorption, distribution, metabolism, and excretion of each emetic/anti-em class.	netic drug <b>2.0</b>
	Ad	verse Effects, Drug Interactions and Contraindications	
	8.	Describe the principal adverse effects of the emetic/anti-emetic drugs of each class.	2.0
	9.	Identify the clinically important drug interactions of the emetic/anti-emetic drugs of eac	ch class.
			2.0
	10.	Identify the principal contraindications of the emetic/anti-emetic drugs of each class.	2.0
	The	erapeutic Uses	
	11.	Describe the appropriate uses of emetic drugs.	3.0
	12.	Discuss the use of anti-emetic drugs in the treatment of chemotherapy-induced nausea vomiting versus those used for motion sickness.	and <b>3.5</b>
	<u>Drı</u>	ugs Acting on the Central Nervous System	
Δ	Fn	dogenous Compounds	
д.	4		
	1.	Denne me tonowing:	2.0
		a. Auenosine Imphosphale (ATP) b. Donamine (DA)	2.0
		c Gamma-Aminohuturic Acid (GABA)	4.U 3.0
			5.0

VII.

d.	Norepinephrine (NE)	2.0
e.	Adenosine	1.0
f.	Beta-Amyloid	1.0
g.	Bradykinin	1.0
h.	Dynorphins	3.0
i.	Glycine	3.0
j.	Nitric Oxide (NO)	3.0
k.	Acetylcholine (ACh)	4.0
١.	5-Hydroxytryptamine (5-HT)	4.0
m.	Glutamate	4.0
n.	Substance P	2.0
0.	Aspartate	1.0
р.	Beta-Endorphin	3.0
q.	Brain Derived Neurotrophic Factor (BDNF)	1.0
r.	Enkephalins	3.0
s.	Histamine	3.0

## B. Drugs Used in General Anesthesia

	5		
1.	Discuss the following with regard to subsequent objectives:		
	a.	Desflurane	1.0
	b.	Nitrous Oxide (N <sub>2</sub> 0)	4.0
	с.	Halothane	3.0
	d.	Fentanyl	4.0
	e.	Midazolam	4.0
	f.	Thiopental	2.0
	g.	Sufentanil	1.0
	h.	Isoflurane	4.0
	i.	Sevoflurane	3.0
	j.	Etomidate	1.0
	k.	Ketamine	4.0
	I.	Morphine	4.0
	m.	Alfentanil	2.0
	n.	Remifentanil	1.0

## Neurotransmitters, Neuromodulators, and Receptors

2.	Identify the major neurotransmitters in the brain, their predominant anatomical pathw their associated relevant disorders.	ays, and <b>4.0</b>
3.	Compare and contrast G protein coupled receptors and ligand-gated ion channels, and	describe
	the major effector systems coupled to various G-proteins.	4.0
4.	Explain how synaptic function changes in response to chronic administration of agonist	S,
	antagonists, and uptake blockers.	4.0
5.	Describe the processes of receptor sensitization and desensitization and provide example	oles of
	how these processes may be induced.	4.0
6.	Identify the molecular, cellular, and biochemical sites where drugs can act to affect neu	uronal
	function.	4.0
7.	Describe the blood brain barrier and list the considerations that determine whether a c	lrug will
	gain access to the central nervous system.	4.0

## Physiology, Pathophysiology, and Therapeutic Actions

Define *general anesthesia* and *dissociative anesthesia*.
 Discuss the objectives of general anesthesia and characteristics of an ideal anesthetic, and identify the stages of general anesthesia.
 **3.0**

## **Mechanism of Action**

 Explain the current theories of the mechanisms of action of inhalation anesthetics and of intravenous anesthetics.
 3.0

## Pharmacokinetics

- Explain the concept of the blood gas dissociation constant and how it affects rate of induction of anesthetic.
   4.0
- Explain how the physical properties of inhalation anesthetics influence the rate of equilibration of anesthetic in the inspired air to anesthetic in alveoli, blood, brain, muscle and fat; and how this information is related to onset and recovery from inhalation anesthesia.
- 13. Compare and contrast commonly used intravenous induction agents.
- 14. Describe relative roles of distribution and metabolism in determining duration of action in, and how duration of action may change with repeated administration of intravenous induction agents.
   3.5

### Adverse Effects, Drug Interactions and Contraindications

- Describe the complications that may ensue with the use of nitrous oxide as a direct result of the high concentrations at which it is administered and its solubility in blood relative to that of nitrogen.
   4.0
- 16. Define *malignant hyperthermia*, list some common triggering agents, and discuss its prevention and treatment.4.0
- 17. Explain the utility and adverse effects of drugs commonly used as pre-anesthetic medications or in combination with inhalation anesthetics to create a "complete or balanced anesthetic," including opioids, benzodiazepines, neuromuscular blocking agents, and antimuscarinic drugs.
- Explain the pharmacological effects of the drugs in each class on pulmonary, cardiovascular, endocrine, renal, and CNS function (aside from anesthesia).
   3.0

## **Therapeutic Uses**

- Compare and contrast commonly used intravenous induction agents, in terms of their speed of onset and duration of action.
   4.0
- 20. Explain the relative roles of distribution and metabolism in determining duration of action and how duration of action may change with repeated administration of an intravenous anesthetic.

4.0

4.0

3.0

- Define MAC (minimal alveolar concentration), name the physical property of an inhalation anesthetic that correlates best with its MAC, and explain how the concept of MAC is used in anesthesiology.
   4.0
- 22. Describe the factors involved in choosing an anesthetic protocol, including the relative advantages and disadvantages of inhalation and intravenous anesthesia.2.0

## C. Local Anesthetics

1. Discuss the following with regard to subsequent objectives:

ters	
Procaine	3.0
Benzocaine	3.0
Cocaine	2.0
Tetracaine	1.0
nides	
Lidocaine	4.0
Bupivacaine	4.0
Ropivacaine	3.0
Prilocaine	1.0
Ropivacaine	
1	ters Procaine Benzocaine Cocaine Tetracaine nides Lidocaine Bupivacaine Ropivacaine Prilocaine

## Physiology, Pathophysiology, and Therapeutic Actions

2.	Explain how the actions of clinically used anesthetics might be influenced by the frequency impulse transmission in peripheral nerves, size and class of the peripheral axons, pH, and b	of y
	vascularity of the injected area. 4.	0
3.	Explain the ionic basis of the action potential and the mechanism of action of local anesthe <b>3.0</b>	tics

## Adverse Effects, Drug Interactions and Contraindications

4.	Identify the common adverse effects of local anesthetics and indicate appropriate treat	ments
	should they occur.	4.0
5.	Identify the significant differences between amide and ester-type local anesthetics.	4.0

## **Therapeutic Uses**

~		
6.	Identify the common routes of administration of local anesthetics.	4.0

- 7. Identify the anesthetics that cannot be used topically or for infiltration, and why. 4.0
- 8. Explain the methods used to restrict local anesthetics to a desired site of action and indicate how these methods reduce adverse effects.
   4.0
- 9. Explain epidural and intrathecal administration of selected opioids and local anesthetics. **3.0**

## D. Opioid Analgesics, Agonist-antagonists and Antitussives

1. Discuss the following with regard to subsequent objectives:

a. Ago	onists	
i.	Morphine	4.0
ii.	Hydromorphone	4.0
iii.	Hydrocodone	4.0
iv.	Oxycodone	4.0
٧.	Methadone	4.0
vi.	Meperidine	4.0
vii.	Fentanyl	4.0
viii.	Alfentanil	4.0
ix.	Codeine	4.0
х.	Diphenoxylate	2.5
xi.	Loperamide	2.5
xii.	Heroin	2.0
b. Mix	ked Agonists/Antagonists	
i.	Buprenorphine	4.0
ii.	Butorphanol	4.0

iv.       Pentazocine       1.5         v.       Buprenorphine-naloxone       2.5         c.       Antagonists       4.0         ii.       Naloxone       4.0         iii.       Naltrexone       4.0         iii.       Nalorphine       1.0         v.       Nalorphine       1.0         v.       Nalorphine       1.0         v.       Nalmefene       3.0         ii.       Dextromethorphan       2.0         Physiology, Pathophysiology, and Therapeutic Actions         2.       Discuss the pharmacological effects and sites of action of the prototype opioid agonist morphine, and its utility in relieving different types of pain.       4.0         3.       Identify potential therapeutic actions of opioids aside from analgesia in CNS and other organ systems including cardiovascular, respiratory, and GI.       4.0         4. Explain the salient differences in pharmacology between morphine and meperidine, fentanyl, methadone, and oxycodone.       3.0         Mechanism of Action         5.       Identify the molecular mechanism of action of each drug in each drug class.       4.0         9.       Explain how the pharmacokinetic processes affecting morphine, absorption, distribution, metabolism, and excretion are relevant to its therapeutic use.       3.0         7.       Ide
v. Buprenorphine-naloxone       2.5         c. Antagonists       -         i. Naloxone       4.0         iii. Naltrexone       4.0         iii. Naloxphine       1.0         v. Nalorphine       1.0         v. Nalorphine       1.0         v. Nalmefene       3.0         ii. Dextromethorphan       2.0         Physiology, Pathophysiology, and Therapeutic Actions       -         2. Discuss the pharmacological effects and sites of action of the prototype opioid agonist morphine, and its utility in relieving different types of pain.       4.0         3. Identify potential therapeutic actions of opioids aside from analgesia in CNS and other organ systems including cardiovascular, respiratory, and GI.       4.0         4. Explain the salient differences in pharmacology between morphine and meperidine, fentanyl, methadone, and oxycodone.       3.0         Mechanism of Action       -       -         5. Identify the molecular mechanism of action of each drug in each drug class.       4.0         7. Identify the opioid agonists that are metabolized to morphine and indicate the salient differences in their pharmacology from that of morphine.       3.0         7. Identify the enjoid agonists that are metabolized to morphine and indicate the salient differences in their pharmacology from that of morphine.       3.0         9. Identify the contraindications of morphine.       4.0
c. Antagonists       4.0         i. Naloxone       4.0         ii. Naltrexone       4.0         iii. Nalbuphine       1.0         iv. Nalorphine       1.0         v. Nalmefene       1.0         i. Codeine       3.0         ii. Dextromethorphan       2.0         Physiology, Pathophysiology, and Therapeutic Actions       1.0         2. Discuss the pharmacological effects and sites of action of the prototype opioid agonist morphine, and its utility in relieving different types of pain.       4.0         3. Identify potential therapeutic actions of opioids aside from analgesia in CNS and other organ systems including cardiovascular, respiratory, and Gl.       4.0         4. Explain the salient differences in pharmacology between morphine and meperidine, fentanyl, methadone, and oxycodone.       3.0         Mechanism of Action       3.0       3.0         5. Identify the molecular mechanism of action of each drug in each drug class.       4.0         9. Identify the opioid agonists that are metabolized to morphine, absorption, distribution, metabolism, and excretion are relevant to its therapeutic use.       3.0         7. Identify adverse effects of morphine on CNS, cardiovascular, Gl-biliary, respiratory and genitourinary systems.       4.0         9. Identify adverse effects of morphine on CNS, cardiovascular, Gl-biliary, respiratory and genitourinary systems.       4.0         9. Identify t
i.       Naloxone       4.0         ii.       Naltrexone       4.0         iii.       Nalbuphine       1.0         iv.       Nalorphine       1.0         v.       Nalmefene       1.0         d.       Antitussives       3.0         ii.       Dextromethorphan       2.0         Physiology, Pathophysiology, and Therapeutic Actions       3.0         2.       Discuss the pharmacological effects and sites of action of the prototype opioid agonist morphine, and its utility in relieving different types of pain.       4.0         3.       Identify potential therapeutic actions of opioids aside from analgesia in CNS and other organ systems including cardiovascular, respiratory, and Gl.       4.0         4.       Explain the salient differences in pharmacology between morphine and meperidine, fentanyl, methadone, and oxycodone.       3.0         Mechanism of Action       5.       Identify the molecular mechanism of action of each drug in each drug class.       4.0         7.       Identify the opioid agonists that are metabolized to morphine, absorption, distribution, metabolism, and excretion are relevant to its therapeutic use.       3.0         7.       Identify the opioid agonists that are metabolized to morphine and indicate the salient differences in their pharmacology from that of morphine.       3.0         8.       Identify adverese effects of morphine on CNS, cardiovasc
ii.       Naltrexone       4.0         iii.       Nalbuphine       1.0         iv.       Nalorphine       1.0         v.       Nalorphine       1.0         v.       Nalmefene       1.0         d.       Antitussives       3.0         ii.       Dextromethorphan       2.0         Physiology, Pathophysiology, and Therapeutic Actions         2.       Discuss the pharmacological effects and sites of action of the prototype opioid agonist morphine, and its utility in relieving different types of pain.       4.0         3.       Identify potential therapeutic actions of opioids aside from analgesia in CNS and other organ systems including cardiovascular, respiratory, and Gl.       4.0         4.       Explain the salient differences in pharmacology between morphine and meperidine, fentanyl, methadone, and oxycodone.       3.0         Mechanism of Action       5.       Identify the molecular mechanism of action of each drug in each drug class.       4.0         6.       Explain how the pharmacokinetic processes affecting morphine, absorption, distribution, metabolism, and excretion are relevant to its therapeutic use.       3.0         7.       Identify the opioid agonists that are metabolized to morphine and indicate the salient differences in their pharmacology from that of morphine.       3.0         Adverse Effects, Drug Interactions and Contraindications       4.0
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12. Discuss abuse liability for opioids and how it differs among the various drugs. <b>3.0</b>
13 Identify the signs and symptoms of morphine and heroin overdose and how they are managed
4.0
14. Explain precipitated abstinence and indicate under what circumstances it might occur following
the clinical use of opioid analgesics of antagonists. <b>4.0</b>
or aspirin.
Therapeutic Uses

16. Identify the clinical indications for the opioids and opioid antagonists and the basis for their use.

4.0

17. Contrast the analgesic effects of morphine with those of the nonsteroidal anti-inflar drugs; with those of antidepressants; and with those of carbamazepine and gabaper	nmatory ntin,
particularly in relation to the treatment of neuropathic pain conditions.	4.0
18. Discuss the rationale for using mixtures of opioid analgesics and NSAIDS.	4.0
19. Explain how agonist-antagonists and partial agonists differ in their utility and advers	e effect
profile when compared to morphine.	4.0
20. Explain the salient differences between naloxone and naltrexone and how these are	reflected in
clinical use of these drugs.	3.5
21. Explain how the combination of naloxone with opiate analgesics in oral and sublingu	lal
preparations permits opiate action, yet decreases abuse liability.	3.0
22. Rationalize using methadone to treat heroin abusers, and identify aspects of methad	done's
pharmacokinetics and pharmacodynamics that make it useful for this purpose.	3.0
23. Differentiate between maintenance therapy with methadone and buprenorphine.	3.0
24. Rationalize using methodone for treatment of chronic pain.	3.0

24. Rationalize using methodone for treatment of chronic pain.

#### VIII. **Centrally Acting Muscle Relaxants and Drugs for Treatment of Motor Disorders**

## A. Drugs for Parkinson's Disease

1. Discuss the following with regard to subsequent objectives:

a.	L-Dopa/Carbidopa	4.0
b.	Selegiline (Deprenyl)	4.0
c.	Pramipexole	3.0
d.	Ropinirole	2.5
e.	Bromocriptine	2.0
f.	Benztropine	1.0
g.	Entacapone	1.0
h.	Amantidine	1.0
i.	Muscle Relaxants	3.0
j.	Baclofen	3.0
k.	Dantrolene	4.0
١.	Diazepam	4.0
m.	Lorazepam	4.0
n.	Tizanidine	3.0
0.	Cyclobenzaprine	2.0
p.	Carisoprodol	3.0

## Physiology, Pathophysiology, and Therapeutic Actions

2.	Identify the major anatomical pathways and neurotransmitter systems involved in contr	rol of
	motor function.	4.0

3. Discuss current hypotheses about the etiology and pathophysiology of Parkinson's disease.

4.0

4. Describe the pathophysiological basis of rigidity, spasticity, and muscle spasm, and identify the classes of agents that are used to promote skeletal muscle relaxation. 4.0

## **Mechanism of Action**

5. Identify the molecular mechanism of action of each primary drug. 4.0

## Adverse Effects, Drug Interactions and Contraindications

6.	Identify the adverse effect profile of levodopa and how it is altered by combination with
	carbidopa. 3.0
7.	Compare and contrast the adverse effect profile of ergot and non-ergot dopamine agonists.
	3.0
The	erapeutic Uses
8.	Rationalize the use of levodopa in Parkinson's disease, as well as its use in combination with
	carbidopa. 4.0
9.	Differentiate between the two major classes of direct DA receptor agonists used for chronic
	control of Parkinson disease, and explain how they are used therapeutically, as well as how their
	therapeutic actions compare to that of levodopa. <b>3.0</b>
10.	Explain the use of anticholinergics, MAO inhibitors, COMT inhibitors, and amantadine in treating
	Parkinson's disease. 2.0
11.	Identify drugs that can cause Parkinsonism and other movement disorders, and recommend
	treatment for these drug-induced disorders. <b>3.0</b>

 Identify drugs useful for treatment of spasticity, and compare and contrast the mechanisms of action and adverse effects of benzodiazepines, baclofen, cyclobenzaprine, and dantrolene. 4.0

## B. Drugs to Treat or Prevent Seizures

1. Discuss the following with regard to subsequent objectives:

a.	Carbamazepine	4.0
b.	Gabapentin	3.0
c.	Phenobarbital	4.0
d.	Topiramate	3.0
e.	Fosphenytoin	2.0
f.	Diazepam	4.0
g.	Lamotrigine	3.0
h.	Phenytoin	4.0
i.	Valproic acid	4.0
j.	Primidone	1.0
k.	Ethosuximide	4.0
I.	Lorazepam	4.0
m.	Tiagabine	3.0
n.	Clonazepam	3.0

## Physiology, Pathophysiology, and Therapeutic Actions

- 2. Describe the pathophysiology of seizures, and identify the types and prevalence of epilepsy. **3.0**
- Explain mirror foci, kindling, post-tetanic potentiation, and long-term potentiation with respect to possible relevance in the initiation and spread of seizure activity.
   3.0

## **Mechanism of Action**

Identify the major classes of antiseizure antiepileptic drugs, the seizure types against which they are effective, and their cellular mechanisms of action, and explain how these actions might be relevant to their roles as antiseizure agents.
 4.0
# Pharmacokinetics

	<ol> <li>Identition</li> <li>particion</li> <li>Ration</li> <li>Identition</li> </ol>	Identify the pharmacokinetic factors relevant to appropriate therapy with antiseizure drugs, iparticular why the clearance of phenytoin changes with dose.3.0Rationalize monitoring plasma concentrations of many antiepileptic drugs.4.0Identify the antiseizure medications that induce hepatic enzymes and describe the			
	conse	quences treatment of epilepsy and for interactions with drugs used for other con	ditions. <b>4.0</b>		
	Adverse	Effects, Drug Interactions and Contraindications			
	8. Identi	fy the adverse and teratogenic effects of the major antiseizure drugs.	4.0		
	Therapeu	herapeutic Uses			
	9. Explai 10. Define 11. Explai their u	n the use of antiseizure medications. e <i>status epilepticus</i> and explain pharmacologic management. n the therapeutic use of antiseizure drugs for conditions other than epilepsy, incl use as analgesics and as mood stabilizers.	4.0 4.0 uding 4.0		
С.	Drugs Us	ed in Affective Disorders			
	1. Discus a. b. c. d. e. f. g. h. i. j. k. l. m n. o. p. q. r.	<ul> <li>st the following with regard to subsequent objectives:</li> <li>Amitriptyline</li> <li>Citalopram</li> <li>Nortriptyline</li> <li>Trazodone</li> <li>Despiramine</li> <li>Mirtazapine</li> <li>Bupropion</li> <li>Escitalopram</li> <li>Paroxetine</li> <li>Venlafaxine/Desvenlafaxine</li> <li>Fluvoxamine</li> <li>Phenelzine</li> <li>Duloxetine</li> <li>Fluoxetine</li> <li>Sertraline</li> <li>Clomipramine</li> <li>Mirtamine</li> <li>St John's Wort</li> </ul>	4.0 4.0 3.0 2.0 1.0 4.0 3.0 3.0 3.5 1.0 3.5 4.0 2.5 1.0 2.0 2.0		
D.	Drugs for	Manic-Depressive (Bipolar) Disorder			
	1. Discus	s the following with regard to subsequent objectives:	• •		
	a.	Carbamazepine	2.0		
	b.		4.0		
	C.	Dianzapine	4.0		
	d.	Risperidone	4.0		
	e.	Cionazepam	1.0		

f. Lithium Carbonate 4.0 g. Aripiprazole 3.5

	h. i. j.	Quetiapine Ziprasidone Clozapine		3.5 1.0 2.5
Phy	ysiology	y, Pathophysiology, and	Therapeutic Actions	
2. 3. 4.	Explain how it List the Describ	the concept of behavioral can be altered by drugs. symptoms, signs and know e the signs and symptoms	l affect, current neurochemical theories regard wn etiology of depresson. s of bipolar disorder, including subtypes and na	ing affect and 3.0 4.0 tural history,
	includir	ng manic episodes.		4.0
Me	chanis	m of Action		
5.	Identify SNRIs, a	the major classes of anticatypical antidepressants, a	depressant drugs and their primary cellular targ	gets (TCAs, SSRIs, <b>4.0</b>
ь. 7.	antidep Explain	ressants. the major theories explai	ning the presumed mechanisms of action of dru	of <b>3.0</b> ugs useful for
	treating	g bipolar disorder (lithium,	, anticonvulsants, antipsychotics).	3.0
Pha	armaco	kinetics		
8.	Discuss importa	the pharmacokinetics of t ance of active metabolite f	the different classes of antidepressant drugs, a formation, as well as how pharmacokinetics is i	nd explain the relevant when
9.	switchi Discuss sodium of lithiu	ng from one medication to the pharmacokinetics of I , effects of exercise, use o Im overdose.	o another. lithium, and explain its relationship to alteratio f diuretics, monitoring of plasma lithium levels	<b>3.0</b> n in dietary , and treatment <b>4.0</b>
The	erapeut	ic Uses		
10.	Explain panic d	the use of antidepressant isorder, post-traumatic str	s for other indications such as obsessive comp ress disorder (PTSD), neuropathic pain, smoking	ulsive disorder, g cessation,
11	enuresi	s, and generalized anxiety	disorder.	3.0
11. 12.	Discuss	the use of antiseizure dru	igs for treatment of bipolar disorder: compare	and contrast
	their ac	lvantages and disadvantag	ges compared to lithium.	4.0

#### Adverse Effects, Drug Interactions and Contraindications

- Identify the most common adverse effects of the major classes of antidepressants, and where known, explain the mechanism for these effects; as well as significant drug and dietary interactions.
   **3.0**
- 14. Identify the signs and symptoms of overdose with each of the major classes of antidepressants and the appropriate treatment for tricyclic antidepressant toxicity, serotonin syndrome, and the tyramine effect.
   4.0
- 15. Differentiate between adverse side effects of lithium from signs and symptoms of lithium overdose, and explain why lithium is contraindicated in patients with impaired renal function or cardiovascular disease.
   4.0
- E. Antipsychotic Drugs
  - 1. Discuss the following with regard to subsequent objectives:

a.	Chlorpromazine	4.0
b.	Clozapine	4.0
c.	Haloperidol	4.0
d.	Risperidone	4.0
e.	Thiothixene	1.0
f.	Aripiprazole	3.0
g.	Fluphenazine	2.0
h.	Olanzapine	4.0
i.	Quetiapine	3.0
j.	Ziprasidone	1.5

#### Physiology, Pathophysiology, and Therapeutic Actions

2.	Discuss symptoms of schizo	phrenia and theories	regarding the und	erlying neurochemical basis.

3. Contrast the actions of phenothiazines and haloperidol with those of atypical antipsychotics, and explain the implications for theories of the mechanisms of antipsychotic actions. **4.0** 

#### **Mechanism of Action**

4. Discuss current theories regarding the therapeutic mechanism of action of antipsychotic drugs, including acute and chronic effects on major dopaminergic and serotonergic systems in the CNS.

4.0

4.0

## **Therapeutic Uses**

F.

5.	Discuss the effectiveness of classical and atypical antipsychotics in the treatment of both positive and negative signs of schizophrenia.	י <b>3.0</b>
6. 7.	Explain uses of antipsychotic drugs for indications other than schizophrenia. Explain the use of dopamine antagonists in Tourette's syndrome.	3.0 2.0
Ad	verse Effects, Drug Interactions and Contraindications	
8.	Discuss the adverse effect profile of low-potency classical antipsychotics, high-potency c antipsychotics, and atypical antipsychotics.	lassical <b>4.0</b>
9.	Identify the time course, signs, and symptoms of antipsychotic drug-induced dyskinesias (dystonia, akathesia, Parkinson-like symptoms, tardive dyskinesia), and discuss their	5
	management and treatment.	3.0
10.	Define <i>neuroleptic malignant syndrome</i> and discuss its management and treatment.	4.0
Sec	dative Hypnotics and Anxiolytic Drugs	

#### 1. Discuss the following with regard to subsequent objectives:

a.	Alprazolam	4.0
b.	Flurazepam	2.0
с.	Zolpidem	4.0
d.	Lorazepam	1.0
e.	Phenobarbital	1.0
f.	Zaleplon	2.5
g.	Eszopiclone	4.0
h.	Midazolam	4.0
i.	Chloral hydrate	1.0
j.	Oxazepam	1.0

		_	
	k.	Temazepam	2.0
	Ι.	Flumazenil (antagonist)	4.0
	m.	Ramelteon	3.0
	n.	Diphenhydramine	1.0
	о.	Pentobarbital	1.0
	р.	Triazolam	2.0
Ph	ysiolog	y, Pathophysiology, and Therapeutic Actions	
2.	Define	sedation, hypnosis, anesthesia, and coma.	2.0
3.	List and	d describe the stages of sleep.	2.0
4.	Define	anxiety, discuss its relationship to the amygdale, and differe	ntiate the major anxiety
	disorde	ers.	2.0

5. Explain the GABA<sub>A</sub> receptor channel complex, the heterogeneity of its subunits, and the 4.0 physiological and therapeutic implications.

#### Mechanism of Action

6. Discuss the effects of various sedative/hypnotic/anxiolytic drugs on GABA<sub>A</sub> function and their selectivity for different receptors with different subunit subtypes, as well as differences in their sites of action on the GABA<sub>A</sub> receptor channel complex. 4.0

#### Adverse Effects, Drug Interactions and Contraindications

7.	Identify the signs and symptoms of barbiturate and benzodiazepine overdose and its tre	eatment.
		4.0
8.	Explain the interactions of the various classes of drugs used as hypnotics, sedative, and	
	anxiolytics with other CNS depressants.	4.0
~	Explore the device device Peletty and Peletty and States and States and States and States and States and States	

Explain the dependence liability and withdrawal syndromes of the various classes of drugs used as hypnotics, sedative, and anxiolytics. 4.0

#### Therapeutic Uses

- 10. Compare and contrast the effects of barbiturates, benzodiazepines, and nonbenzodiazepine agonists at the benzodiazepine site on induction and maintenance of sleep (including effects on sleep stages). 3.0
- 11. Discuss the adverse effects of benzodiazepines, nonbenzodiazepines, and barbiturates, as well as why drugs acting at the benzodiazepine receptor have virtually totally replaced barbiturates 4.0 as hypnotics.
- 12. Identify the therapeutic uses of benzodiazepines, and prototypes for each use. 3.0
- 13. Explain how pharmacokinetics of various benzodiazepines relates to their therapeutic utility. 4.0
- Identify and describe other groups of drugs with sedative/hypnotic and anxiolytic actions, including ramelteon, buspirone, chloral hydrate, and hydroxyzine. 4.0
- 15. List drugs that are used for treating anxiety disorders other than generalized anxiety: panic disorder, obsessive-compulsive disorder, and specific phobias. 4.0

#### G. Substance Abuse

#### Physiology, Pathophysiology, and Therapeutic Actions

- 1. Discuss tolerance and physical dependence abuse of drugs. 4.0
- 2. Compare and contrast the roles of drug craving and reward versus avoidance of withdrawal in

	initiation and maintenance of substance dependence.	3.0
3.	Identify major CNS pathways involved in substance dependence.	4.0
4.	Explain how pharmacokinetics influences abuse liability and withdrawal syndromes.	4.0
5.	Describe patterns and effects of substance abuse for stimulants, opioids, sedative-hyp	notics,
	and anxiolytics.	3.5
6.	Discuss morbidity and mortality, including the dangers of unregulated withdrawal, for	substance
	dependence on various classes of abused drugs.	3.0
7.	Define <i>tolerance</i> .	4.0
8.	Compare and contrast the withdrawal syndrome for classes of drugs of abuse that pro	duce
	physical dependence.	3.0
9.	Discuss techniques for detoxifying users.	4.0

#### H. Stimulants

		<b>.</b>			
1	Discuss th	o following	with regard	to subsoa	uant abiactivas:
т.	Discuss ti	ie iuliuwilig	with regard	LU SUDSEY	ueni objectives.
					,

a.	Amphetamine	4.0
b.	Cocaine	4.0
c.	Modafinil	2.0
d.	Sibutramine	1.0
e.	Atomoxetine	2.0
f.	Methamphetamine	4.0
g.	Nicotine	4.0
h.	Varenicline	2.0
i.	Caffeine	3.5
j.	Methylphenidate	4.0
k.	Ephedrine	2.0

#### **Mechanism of Action**

2.	Discuss current theories of the mechanisms of action of the stimulant and anorexigenic	: drugs
	listed above.	4.0

#### Adverse Effects, Drug Interactions and Contraindications

3.	Describe the abuse potential for the following psychostimulants listed above.	4.0
4.	Explain the toxic effects of stimulants used therapeutically, and the adverse effects of	stimulant
	misuse and abuse.	4.0

Describe the addictive properties of nicotine and the adverse effects of nicotine and other tobacco constituents.
 4.0

#### **Therapeutic Uses**

- Explain therapeutic uses of stimulants and related drugs as appetite suppressants, in attention deficit hyperactivity disorder, in narcolepsy, and for promoting wakefulness.
   4.0
- 7. Describe therapies to treat nicotine dependence, including nicotine patches and chewing gum, nicotine receptor partial agonists (varenicline), and other agents such as bupropion. **4.0**

#### I. Ethanol and Drugs for Treatment of Alcoholism

1. Discuss the following with regard to subsequent objectives:

a.	Ethanol	4.0
b.	Methanol	2.0
с.	Acamprosate	4.0

e. Fomepizole 2.0	
f. Naltrexone 4.0	
g. Disulfiram 4.0	
h. Topiramate 2.0	
Physiology, Pathophysiology, and Therapeutic Actions	
2. Identify the acute CNS actions of ethanol and discuss their relationship to blood alcohol levels.	
4.0	
Mechanism of Action	
3. Discuss current theories about the mechanism of action of alcohol in the CNS. <b>4.0</b>	
Pharmacokinetics	
4 Describe the pharmacokinetics absorption distribution metabolism and excretion of ethanol	
4. Describe the pharmacokinetics, absorption, distribution, metabolism, and excretion of ethanol. 4 0	
Adverse Effects, Drug Interactions and Contraindications	
5. Describe the acute and chronic organ toxicities of ethanol methanol and higher alcohols (e.g,	
ethylene glycol). 4.0	
6. Identify the drugs with which ethanol shows cross-tolerance and cross-dependence. <b>4.0</b>	
7. Identify drugs, both prescription and over the counter, that would entail a patient refraining	
from the use of alcoholic beverages, and describe the potential interactions. <b>3.0</b>	
8. Explain the management of methanol toxicity. 4.0	
9. Identify the signs and symptoms of chronic alcoholism and the ethanol abstinence syndrome;	
and compare and contrast the latter with abstinence syndromes following chronic use of	
barbiturates, benzodiazepines, or opioids. 4.0	
10. Identify the mechanism for the synergism between chloral hydrate and ethanol. <b>2.0</b>	
Therapeutic uses	
11. Identify the therapeutic applications of ethanol. 2.0	
12. Discuss the treatment options for acute intoxication by ethanol or other alcohols, and for the	e
ethanol abstinence syndrome. 3.0	
13. Explain the effects and the mechanistic rationale for the use of disulfiram, naltrexone, and	d
acamprosate in the treatment of chronic alcoholism. <b>3.0</b>	
Hallucinogens and Designer Drugs	
1. Discuss the following with regard to subsequent objectives:	
a. Lysergic Acid Diethylamide (LSD) 3.0	
b. Mescaline 2.0	
c. Scopolamine 1.0	
d. MDMA (Methylene Dioxymethamphetamine) 4.0	
e. Phencyclidine (PCP) <b>3.0</b>	
f. Ketamine 4.0	
Physiology, Pathophysiology, and Therapeutic Actions	

2. Differentiate between the behavioral and hallucinogenic effects of the various drugs and

J.

compare and contrast the drug-induced states with endogenous psychoses.

3. Discuss the variability in individual responses to hallucinogens and the interaction between the social setting in which hallucinogens are taken and their behavioral effects. **2.0** 

#### **Mechanism of Action**

Identify the hallucinogens with primary actions on 5HT<sub>2A</sub> receptors, and those that are NMDA receptor antagonists, and muscarinic receptor antagonists and describe their mechanisms of action.
 2.0

#### Pharmacokinetics

5. Explain how the pharmacokinetics of different drugs may influence their duration of action and their detection by screening tests for illicit drug use. **2.0** 

#### Adverse Effects, Drug Interactions and Contraindications

- 6. Explain tolerance to, and cross-tolerance among the various hallucinogens. **3.0**
- 7. Identify the toxidromes expected for LSD, MDMA, PCP, and belladonna alkaloids. 4.0
- 8. Discuss general principles of treatment for patients with known ingestion of hallucinogens. 3.0

#### K. Marijuana

L.

1.	Discuss the following with regard to subsequent objectives: a. Marijuana/Delta-9 Tetrahydrocannabinol (THC) b. Dronabinol	1.0 1.0
Ph	ysiology, Pathophysiology, and Therapeutic Actions	
2.	Identify endogenous cannabinoids, and discuss how they differ from classical neurotransmitters/neuromodulators, their receptors, and the current hypotheses at functional roles.	oout their <b>1.0</b>
3.	Explain the psychological, physiological and pharmacologic effects of smoking maunderstand the rationale for using dronabinol.	arijuana and <b>3.0</b>
Inł	halants/organic solvents and gases	
1.	Discuss the following with regard to subsequent objectives:	
	a. Carbon tetrachloride	1.0
	b. Glue	1.0
	c. Toluene	1.0
	d. Gasoline	1.0
	e. Nitrous oxide	4.0
Ad	lverse Effects, Drug Interactions and Contraindications	

Describe the epidemiology of abuse of inhalants.
 Discuss the effects of organic solvents and their toxicities.
 2.0

#### M. Opioids, Sedative-Hypnotics and Antianxiety Agents

1. Discuss the following with regard to subsequent objectives:

4.0

a.	Benzodiazepines	4.0
b.	Morphine	4.0
С.	Oxycodone (and other prescription opioids)	4.0
d.	Buprenorphine-naloxone	2.0
e.	Heroin	4.0
f.	Methadone	4.0
g.	Barbituates	2.0
h.	Naltrexone	2.0
Adverse E	ffects, Drug Interactions and Contraindications	

2.	Discuss the substance abuse potential of opioids, sedative-hypnotics, and antia	nxiety agents.
		4.0
3.	Describe the features of addiction to, and dependence on, these agents.	4.0

#### **Therapeutic Uses**

Discuss therapies for opiate dependence including maintenance therapies (methadone), and antagonist therapies (naltrexone), and explain the use of combinations of partial agonists (buprenorphine) and antagonists (naltrexone).
 4.0

#### N. Drugs and the Law: Therapeutic uses

1.	Identify the characteristics of drugs in each of the Drug Enforcement Administration		
	classification of controlled substances into Schedules I, II, III, and IV, and give examples	s of	
	specific drugs that are included in each schedule.	4.0	
2.	Discuss the ways in which this classification affects the clinical use of drugs.	4.0	

#### O. Drugs for Treatment of Alzheimer's Disease

1. Discuss the following with regard to subsequent objectives:

a.	Donepezil	4.0
b.	Galantamine	2.0
c.	Memantine	4.0
d.	Rivastigmine	4.0

#### **Mechanism of Action**

Identify drugs used for the treatment of Alzheimer's disease, their mechanisms of action, their efficacy, and their adverse effects.
 4.0

#### IX. Endocrine Pharmacology

#### Introduction, Physiology and Pathophysiology

- Identify and describe the general functions of hormones and their target organs (location and type of receptors).
   3.0
- Explain the etiology of endocrine syndromes including those due to hormone deficiency/excess, receptor defects, hormone resistance, abnormal hormone dynamics, and hormone binding proteins.
   3.0

#### **Mechanism of Action**

Identify the mechanisms of hormone action including receptors and signal transduction pathways for hormones.
 3.0

#### **Pharmacokinetics**

4. Explain the regulation of hormone synthesis/release/disposition, the role of circadian rhythms, patterns of release, binding proteins, and modulating factors. **4.0** 

#### A. Drugs and Hormones from Hypothalamus and Anterior Pituitary

1. Discuss the following with regard to subsequent objectives:

a.	Octreotide	3.0
b.	Somatomedins (IgF-1)	3.0
с.	Pergolide	3.0
d.	Ganirelix	1.0
e.	Leuprolide	4.0
f.	Pegvisomant	3.0
g.	Somatropin	3.0
h.	Prolactin	2.0
i.	GNRH (Gonadorelin)	1.0
j.	Menotropins	1.0
k.	Sermorelin	3.0
I.	Bromocriptine	4.0
m.	Abarelix	1.0
n.	Goserelin	1.0
0.	Nafarelin	1.0
p.	Somatrem	3.0
q.	Cabergoline	4.0
r.	Follitropin	1.0
s.	Human Chorionic Gonadotropin (HCG)	1.0
t.	Urofollitropin	1.0

#### **Physiology and Pathophysiology**

2.	Explain the regulation of growth hormone (GH) biosynthesis and secretion, including the roles or growth hormone releasing hormone (GH-RH) and GH-releasing peptides; glucose levels,		
	somatotatin, and dopamine; and age and body composition.	4.0	
3.	Identify the physiological conditions that elicit growth hormone secretion, and outline ho	w	
	specific diagnostic maneuvers can elicit GH secretion.	4.0	
4.	Explain the regulation of prolactin biosynthesis secretion and release by suckling, as well	as the	
	effect of dopaminergic and serotonergic agonists and antagonists.	3.0	
5.	Identify pharmacological agents that can induce hyperprolactinemia.	3.0	
6.	Describe medical problems related to hypersecretion of prolactin in the female (galactorr		
	amenorrhea, infertility) and in the male (hypogonadism, infertility).	3.0	
7. Explain the kinetics of secretion for GnRH and the relationship to the therapeuti			
	synthetic analogs, the mode of administration, and therapeutic considerations.	4.0	
Mechanism of Action			
8.	Identify the molecular mechanism of action of each drug in each drug class.	4.0	
Act	Actions on Organ Systems		

	9. 10. 11.	Explain the biological actions of growth hormone on peripheral tissues. Explain the role(s) of IGFs (somatomedins). Describe the biological actions of prolactin on breast development and lactation and exp relationship of the hormones that are involved in breast development and lactation, incl growth hormone, estrogen, progesterone, glucocorticoids, TRH, prolactin, oxytocin, and	2.0 2.0 Dain the Juding insulin.
	12.	Explain the structure-activity relationships of gonadotropin releasing hormone (GnRH) a synthetic analogs.	2.0 nd 3.0
	Ad	verse Effects, Drug Interactions and Contraindications	
	13. 14. 15.	Identify and describe the adverse effects of GH therapy in children and adults. Identify and describe the adverse effects of GH therapy in children and adults. Identify and describe the adverse effects of GnRH and analogs as therapeutic agents wh to treat infertility, prostatic carcinoma, endometriosis, and central precocious puberty.	4.0 4.0 en used 1.0
	The	erapeutic Uses	
	16. 17.	Idenfity and describe the medical problems related to hypo- or hyper- secretion of GH a role of releasing/replacement therapy and release-inhibiting drugs in the management of states. Explain the mode of administration and therapeutic considerations for intermittent (inferversus continuous administration (endometriosis, uterine fibroids, and prostate cancer) precocious puberty.	nd the of these <b>4.0</b> ertility) and <b>4.0</b>
В.	Нy	pothalamus, Anterior and Posterior Pituitary Drugs	
	1.	<ul> <li>Discuss the following with regard to subsequent objectives:</li> <li>a. Corticotropin</li> <li>b. Demeclocycline</li> <li>c. Oxytocin</li> <li>d. Cosyntropin</li> <li>e. Desmopressin</li> <li>f. Chlorpropamide</li> <li>g. Vasopressin</li> </ul>	3.0 2.0 3.0 4.0 1.0 4.0
	Phy	ysiology and Pathophysiology	
	2. 3. 4. 5.	Describe the effects of vasopressin on receptor subtypes and signal transduction system vascular smooth muscle and the kidney. Identify the drugs that affect vasopressin release/action and explain their relationship to therapy of diabetes insipidus and SIADH. Identify the drugs that can cause diabetes insipidus (nephrogenic and neurogenic) and S Explain the actions of oxytocin and roles in parturition and lactation.	s in 4.0 5 the 4.0 IADH. 4.0
	Me	echanism of Action	
	6.	Identify the molecular mechanism of action of vasopression and related hormones.	4.0
	Act	tions on Organ Systems	
	7. Pha	Explain the actions of vasopressin and analogs, such as desmopressin on organ systems. armacokinetics	4.0

	8.	Identify the route(s) by which cosyntropin is administered.	1.0
	Ad	verse Effects, Drug Interactions and Contraindications	
	9. 10.	Identify the possible (rare) side effects of cosyntropin administration. . Outline the toxicity and contraindications for oxytocin.	1.0 1.0
	Th	erapeutic Uses	
	11. 12. 13.	<ul> <li>Discuss the rapid ACTH stimulation test in diagnosing pituitary-adrenal disorders and ide what endpoint is measured.</li> <li>Discuss preparations and routes administration of vasopressin analogs available for trear neurogenic and partial diabetes insipidus, bleeding of esophageal varices, and deficient l clotting factors in hemophilia.</li> <li>Describe the diagnostic and therapeutic uses of oxytocin.</li> </ul>	entify 1.0 ting blood 2.0 1.0
С.	Ad	Irenal Cortical Drugs and Hormones	
	1. Phr 2. 3. 4.	Discuss the following with regard to subsequent objectives: <ul> <li>a. Dexamethasone</li> <li>b. Aminogluthethimide</li> <li>c. Metyrapone</li> <li>d. Prednisone</li> <li>e. Fludrocortisone</li> <li>f. Beclomethasone</li> <li>g. Fluticasone</li> <li>h. Mifepristone</li> <li>i. Triamcinolone</li> <li>j. Spironolactone</li> <li>k. Hydrocortisone</li> <li>l. Ketoconazole</li> <li>m. Mitotane</li> <li>n. Aldosterone</li> </ul> ysiology and Pathophysiology Outline the major steps in the biosynthesis of steroids. Explain the regulation of aldosterone secretion by angiotensin (I, II, and III).	4.0 3.0 4.0 4.0 4.0 3.0 4.0 4.0 4.0 3.0 2.0 4.0 3.0 4.0 4.0 4.0
	Мс	achanism of Action	
	5	Identify the molecular mechanism of action of the corticosteroids	40
	э. •		4.0
	AC	tions on Organ Systems	
	6. 7. 8.	Explain the actions of corticosteroids on intermediary metabolism, growth and developmelectrolyte homeostasis, immune, and inflammatory responses. Describe the cellular mechanism of action of corticosteroids. Discuss the structure-activity relationship of synthetic glucocorticoids, especially those modifications that enhance pharmacodynamics activity and/or determine activity based route of administration.	nent, 4.0 4.0 0n 4.0

# Pharmacokinetics

	Explain the significance of corticosteroid disposition (protein binding, biotransformate enzyme induction) that may necessitate changes in dosage regimens.	tion, <b>4.0</b>
	dverse Effects, Drug Interactions and Contraindications	
	<ol> <li>Outline the adverse effects/contraindications related to corticosteroid use.</li> <li>Outline the adverse effects of excessive mineralocorticoid activity.</li> </ol>	4.0 4.0
	herapeutic Uses	
	<ol> <li>Explain the rationale for corticosteroid use in replacement therapy, as antiinflammat immunosuppressive agents and as diagnostic agents in hypothalmo-pituitary adreno disease/dysfunction.</li> <li>Explain the rationale for alternate day therapy and the necessity for slow withdrawa chronic therapy with glucocorticoids.</li> <li>Explain the rationale for spironolactone in treating primary hyperaldosteronism.</li> </ol>	cory and cortical <b>4.0</b> I following <b>4.0</b> <b>4.0</b>
D.	rugs for the Treatment of Thyroid Disease	
	<ul> <li>Discuss the following with regard to subsequent objectives: <ul> <li>a. lodide salts</li> <li>b. Levothyroxine</li> <li>c. Methimazole</li> <li>d. Propranolol</li> <li>e. Propylthiouracil</li> <li>f. Lithium</li> <li>g. Potassium lodide</li> <li>h. Radioactive lodine (l<sup>131</sup>)</li> <li>i. Triiodothyronine</li> </ul> </li> <li>hysiology and Pathophysiology</li> <li>Explain the regulation and the key steps in thyroid hormone synthesis and periphera conversion.</li> <li>Identify the mechanisms by which thyroid hormones regulate cellular function.</li> <li>Identify the signs and symptoms of hypothyroidism (myxedema) and the consequent disease that can alter drug therapy for other concurrent diseases.</li> </ul>	3.0 4.0 4.0 2.0 4.0 4.0 3.0 1 3.0 4.0 ces of the 4.0
	lechanism of Action	
	Identify the molecular mechanism of action of each drug in each drug class.	4.0
	ctions on Organ Systems	
	Explain the relationship between thyroid hormones and the actions of catecholamine rationale for the use of propranolol in the treatment of hyperthyroidism.	es and the <b>4.0</b>
	harmacokinetics	
	Explain the pharmacokinetic rationale for selecting the most appropriate form of thy hormone as replacement therapy. Identify the best index of adequate replacement therapy with thyroid hormone. <b>4.0</b> Explain the rationale for selecting the most appropriate antithyroid drug for treating	roid <b>4.0</b>
	hyperthyroidism (diffuse toxic goiter) in a non-pregnant versus a pregnant female.	3.0

#### Adverse Effects, Drug Interactions and Contraindications

Outline the adverse effects of antithyroid medications and identify those that are potentially life threatening.
 4.0

#### **Therapeutic Uses**

- Explain the necessary cautions when replacing thyroid hormone in a patient with a history of coronary artery disease.
   4.0
- 12. Explain the rationale and order of administration of drugs given to treat thyroid storm. **4.0**
- 13. Explain the rationale for the uses of drugs/radioiodine in treating hyperthyroidism; explain their mechanism(s) of action; consequences of radioioactive iodine use.
   4.0

#### E. Drugs for Treatment of Osteoporosis

1.	Discuss	s the following with regard to subsequent objecti	ves:
	a.	Bisphosphonates	4.0
		i. Alendronate	
		ii. Ibandronate	
		iii. Zoledronate	
		iv. Etidronate	
	b.	Calcitonin	4.0
	с.	Raloxifene	4.0
	d.	Parathyroid hormone	3.0
	e.	Calcitriol	4.0
	f.	Cinacalcet	3.0
	g.	Teriparatide acetate	4.0
	h.	Calcium gluconate	4.0
	i.	Furosemide	4.0

#### **Physiology and Pathophysiology**

2. 3.	Describe the regulation of calcium homeostasis and the physiological actions of parathy hormone (PTH), calcitonin (CT) and 1,25dihydroxyvitamin D3.0 [1,25-(OH) <sub>2</sub> D <sub>3.0</sub> ]; underst role(s) of kidney, liver, and GI tract in vitamin D homeostasis. Identify the mechanisms regulating synthesis, secretion of PTH and actions and CT their mechanism(s) of action on bone, kidney and intestine.	roid and the <b>4.0</b> <b>4.0</b>
Me	chanism of Action	
4.	Identify the molecular mechanism of action of each drug in each drug class.	4.0
Ad	verse Effects, Drug Interactions and Contraindications	
5. 6.	Explain the possible adverse effects of CT, 1,25-(OH)₂D and calcium supplements. Discuss the chronic toxicity associated with long-term use of sodium fluoride.	4.0 1.0
Th	erapeutic Uses	
7. 8.	Identify the available preparations of CT, 1,25-(OH) <sub>2</sub> D, and calcium supplements and the clinical uses; compare and contrast the treatment of hypo- and hyper-parathyroidism. Identify the available preparations of CT and 1,25-(OH) <sub>2</sub> D and calcium supplements.	eir 4.0 4.0

9. Explain the clinical value of bisphosphonates and CT in the treatment of: hypercalcemia, Paget's disease, osteoporosis (postmenopausal and glucocorticoid-induced). **4.0** 

#### F. Drugs used in Treatment of Diabetes

1. Discuss the following with regard to subsequent objectives:

a.	Acarbose	4.0
b.	Exenatide	4.0
с.	Glyburide	4.0
d.	Nateglinide	4.0
e.	Rosiglitazone	4.0
f.	Chlorpropamide	1.0
g.	Glucagon	4.0
h.	Insulins (aspart, glulisine, lispro, regular, nph, detemir, glargine)	4.0
i.	Pioglitazone	4.0
j.	Sitagliptin	4.0
k.	Diazoxide	2.0
I.	Glipizide	4.0
m.	Metformin	4.0
n.	Repaglinide	4.0
о.	Tolbutamide	3.0

#### Physiology and Pathophysiology

2.	Describe the normal daily patterns of insulin secretion and changes that occur in different	ent types
	of diabetes mellitus.	4.0

- 3. Explain the effects of insulin and glucagon on intermediary metabolism and ion transport. 4.0
- 4. Explain the effects of incretin hormones, esp. GLP-1 on insulin and glucagon secretion. 3.0 4.0
- 5. Explain the effects of amylin protein on glucagon secretion.
- 6. Describe the pathophysiology of the primary types of diabetes mellitus (bihormonal disease insulin and glucagon), and their sequelae: diabetic ketoacidosis and nonketotic hyperosmolar coma. 4.0

#### Mechanism of action

7. Identify the molecular mechanism of action of each drug used to treat osteoporosis. 4.0

#### **Pharmacokinetics**

- 8. Discuss common and possible serious side effects of bisphosphonates. 4.0
- 9. Explain the pharmacokinetic (onset and duration of action) rationale for the use of insulin preparations in 'split-mixed' or continuous SC infusion. 4.0
- 10. Discuss commonly used drugs with which sulfonylurea compounds are known to interact and the postulated mechanisms for these interactions (first versus second generation). 4.0

#### Adverse Effects, Drug Interactions and Contraindications

11. Explain the clinical manifestations and management of overdose with insulin and oral hypoglycemic agents, respectively. 4.0

#### Therapeutic Uses

- 12. Explain the mechanisms by which oral anti-diabetic agents act and describe the influence these mechanisms have on selection for therapy in individual patients (eg, obese). 4.0
- 13. Identify the relative roles of insulin and oral hypoglycemics in the treatment of type I and type II diabetes mellitus. 4.0

14. Explain the potential for metformin to cause metabolic acidosis, and identify which patients it should not be used in. **4.0** 

#### G. Gonadal Hormones and Drugs

1. Discuss the following with regard to subsequent objectives:

a.	Anastrozole	4.0
b.	Drospirinone	3.0
с.	Exemestane	4.0
d.	Medroxyprogesterone	4.0
e.	Raloxifene	4.0
f.	Finasteride	4.0
g.	Testosterone	4.0
h.	Clomiphene	3.0
i.	Estradiol 17ß	4.0
j.	Levonogestrel	3.0
k.	Norethindrone	4.0
١.	Tamoxifen	4.0
m.	Flutamide	4.0
n.	Conjugated/Esterified Estrogens	4.0
0.	Estrone	3.0
p.	Mestranol	3.0
q.	Phytoestrogens	1.0
r.	Bicalutamide	1.0
s.	Leuprolide	4.0
t.	Diethylstilbestrol	1.0
u.	Ethinyl Estradiol	4.0
٧.	Mifepristone	3.0
w.	Progesterone	4.0
х.	Danazol	4.0
у.	Oxandrolone	4.0

## **Physiology and Pathophysiology**

2.	2. Explain the gametogenic and steroidogenic functions of the ovary and their regulation by	
	gonadotropins.	4.0
3.	Identify the sources of androgens (ovary, testes, adrenal) and understand their regu	ulation of
	secretion; define the roles of LH and FSH on gonadal function.	4.0
4.	Explain the importance of androgens for sexual differentiation and puberty.	3.0
5.	Describe medical problems associated with hypo- (hypogonadism) and hyperfunction	on
	(precocious puberty, hyperandrogenism) and explain rationale for therapy.	4.0
6.	Rationalize the clinical uses of androgens in replacement therapy, anemia, and cata	bolic states.
		4.0
M	echanism of action	
_		

7. Identify the molecular mechanism of action of each drug in each drug class. **4.0** 

#### **Actions on Organ Systems**

<ol> <li>Discuss the effects of estrogen on cardiovascular function, intermediary metabolism, e and water balance, cognition, reproductive function, skin, plasma proteins, and blood l hepatic function.</li> </ol>	lectrolyte lipids <b>4.0</b>
<ol> <li>Discuss the effects of estrogens on laboratory tests, including liver function, clotting factory the thyroid hormone disposition, and adrenocortical function.</li> </ol>	ctors, <b>4.0</b>
10. Discuss the effects of androgens on growth and development (anabolic actions versus androgenic actions).	4.0
<ol> <li>Discuss the importance of dihydrotesterone formation and binding to androgen recept prostate gland and other organs.</li> </ol>	tors in the <b>4.0</b>
Pharmacokinetics	
12. Differentiate between absorption, distribution, and elimination of synthetic and natura	al
estrogens.	4.0
13. Identify the routes of administration, absorption, and relative duration of action of syn androgens and testosterone.	thetic <b>4.0</b>
Adverse Effects, Drug Interactions and Contraindications	
14. Describe major adverse effects/contraindications for estrogens and progestins alone, a combination.	and in <b>4.0</b>
15. Describe the most common drug interactions with estrogens and progestins.	4.0
16. Describe the adverse effects of androgens/anabolic steroids when used in male and fer	male. <b>4.0</b>
17. Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s	tructure. 4.0
17. Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s Therapeutic Uses	tructure. 4.0
<ul> <li>17. Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>18. Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> </ul>	<b>4.0</b> cof <b>2.0</b>
<ul> <li>17. Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>18. Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>19. Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora</li> </ul>	<b>4.0</b> cof <b>2.0</b> al
<ul> <li>17. Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>18. Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>19. Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>20. List agents used for pertoxical contraception</li> </ul>	tructure. 4.0 c of 2.0 al 3.0 2.0
<ul> <li>17. Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>18. Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>19. Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>20. List agents used for postcoital contraception.</li> <li>21. Outline the types of hormonal contraceptive agents other than combination agents, and</li> </ul>	tructure. 4.0 cof 2.0 al 3.0 3.0 didentify
<ol> <li>Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>List agents used for postcoital contraception.</li> <li>Outline the types of hormonal contraceptive agents other than combination agents, ar their routes of administration.</li> </ol>	tructure. 4.0 2.0 al 3.0 3.0 d identify 3.0
<ol> <li>Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>List agents used for postcoital contraceptive agents other than combination agents, ar their routes of administration.</li> <li>Discuss therapeutic and diagnostic uses of estrogens and progestins, other than their use or a contraceptive agents.</li> </ol>	tructure. 4.0 2.0 al 3.0 3.0 didentify 3.0 utility as
<ol> <li>Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>List agents used for postcoital contraceptive agents other than combination agents, ar their routes of administration.</li> <li>Discuss therapeutic and diagnostic uses of estrogens and progestins, other than their u oral contraceptives.</li> <li>Bationalize the use of long-acting progestins for long-term suppression of ovulation</li> </ol>	2.0 al 3.0 3.0 d identify 3.0 utility as 4.0 3.0
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<ol> <li>Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>List agents used for postcoital contraception.</li> <li>Outline the types of hormonal contraceptive agents other than combination agents, ar their routes of administration.</li> <li>Discuss therapeutic and diagnostic uses of estrogens and progestins, other than their u oral contraceptives.</li> <li>Rationalize the use of long-acting progestins for long-term suppression of ovulation.</li> <li>Rationalize the replacement use of estrogens and estrogen/progestin in postmenopaus osteoporosis, cognitive disorders, and cardiovascular disease.</li> <li>Explain the use of estrogen receptor modifier (SERM) and describe their therapeutic util</li> </ol>	1.0         tructure.         4.0         2.0         al         3.0         nd identify         3.0         utility as         4.0         3.0         sal         4.0         sal         4.0         sal         4.0         ncer.         3.0         lity.
<ol> <li>Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li><b>Therapeutic Uses</b> <ol> <li>Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>List agents used for postcoital contraception.</li> <li>Outline the types of hormonal contraceptive agents other than combination agents, ar their routes of administration.</li> <li>Discuss therapeutic and diagnostic uses of estrogens and progestins, other than their u oral contraceptives.</li> <li>Rationalize the use of long-acting progestins for long-term suppression of ovulation.</li> <li>Rationalize the replacement use of estrogens and estrogen/progestin in postmenopaus osteoporosis, cognitive disorders, and cardiovascular disease.</li> <li>Explain the use of estrogen receptor antagonists and aromatase inhibitors in breast cardiocastic uses</li> </ol> </li> </ol>	1.0         structure.         4.0         2.0         al         3.0         3.0         3.0         al         3.0         sal         4.0         3.0         sal         4.0         3.0         sal         4.0         sal         4.0
<ol> <li>Correlate the hepatoxicity of certain androgens/anabolic steroids with their chemical s</li> <li>Therapeutic Uses</li> <li>Explain the use of drugs such as clomiphene and gonadotropic drugs for the treatment infertility.</li> <li>Explain the rationale for the various dosage schedules (eg, biphasics, triphasics), for ora contraception when combination (estrogen-progestin) therapy is used.</li> <li>List agents used for postcoital contraception.</li> <li>Outline the types of hormonal contraceptive agents other than combination agents, ar their routes of administration.</li> <li>Discuss therapeutic and diagnostic uses of estrogens and progestins, other than their us oral contraceptives.</li> <li>Rationalize the use of long-acting progestins for long-term suppression of ovulation.</li> <li>Rationalize the replacement use of estrogens and estrogen/progestin in postmenopaus osteoporosis, cognitive disorders, and cardiovascular disease.</li> <li>Explain the use of estrogen receptor antagonists and aromatase inhibitors in breast cardiocement use of estrogen receptor modifier (SERM) and describe their therapeutic util 27. Identify the mechanism of action of mifepristone (RU 4.086) and other abortifacients.</li> </ol>	1.0         tructure.         4.0         2.0         al         3.0         nd identify         3.0         utility as         4.0         3.0         sal         4.0         3.0         sal         4.0         3.0         sal         4.0         3.0         sal         4.0         3.0

# H. Drugs Affecting the Female Urogenital System

- 1. Discuss the following with regard to subsequent objectives:
  - a. Dinoprost

b.	Mifepristone	2.0
c.	Indomethacin	2.0
d.	Terbutaline	2.0
e.	Dinoprostone	2.0
f.	Misoprostol	2.0
g.	Magnesium sulfate	2.0
h.	Ergonovine	2.0
i.	Oxytocin	2.0
j.	Ritodrine	2.0

## **Mechanism of Action**

2.	Identify the molecular mechanism of action of drugs used for uterine stimulation or relaxation <b>2.0</b>	•
Ac	tions on Organ Systems	
3.	Identify the receptors targeted by the oxytocics and the sensitivity of the uterus during the three trimesters of pregnancy. <b>2.0</b>	
Ph	armacokinetics	
4. 5.	Describe the usual route(s) of administration, onset and duration of action of the various oxytocic agents. <b>1.0</b> Describe the usual route(s) of administration as well as onset and duration of action of the various tocolytic agents. <b>1.0</b>	
Ad	Iverse Effects, Drug Interactions and Contraindications	
6.	Identify the potential adverse effects of the oxytocic agents in the mother (uterine, extrauterine) and in the infant. <b>1.0</b>	
Th	erapeutic Uses	
7. 8. 9.	Explain the clinical use of the individual oxytocics.2.0Compare and contrast the utilization of RU4.086 (mifepristone) versus prostaglandins and oxtocics in therapeutic abortion.2.0Describe the potential benefits and risks of administering tocolytic agents to the mother and baby.2.0	
Dr	ugs Affecting the Male Urogenital System	
1.	Discuss the following with regard to subsequent objectives:a. Alprostadil2.0b. Sildenafil2.0c. Doxazosin2.0d. Tamsulosin3.0e. Alfusosin2.0f. Saw Palmetto2.0g. Terazosin3.0	

g. Terazosin

# Physiology and Pathophysiology

Ι.

2. List the neuroendocrine factors that regulate functions of the male urogenital tract. 4.0

#### Mechanism of action

3. Identify the molecular mechanism of action of the drugs used for benign prostatic hyperplasia and erectile dysfunction. **3.0** 

#### Adverse Effects, Drug Interactions and Contraindications

4. Describe the adverse effects and contraindications of the prototype agents in the drug list. 3.0

#### Therapeutic Uses

- 5. Identify drugs that can be used to treat benign prostatic hyperplasia and erectile dysfunction.
- 6. Explain the proposed mechanism of action of the drugs listed above and relate the resulting pharmacological effects to their clinical use. **3.0**

#### X. Hemostasis and Blood Forming Organs

#### A. Drugs for Treatment of Anemia and Neutropenia

1. Discuss the following with regard to subsequent objectives:

a.	Iron Products	3.0
b.	Erythropoetin Alfa	3.0
с.	Folic Acid	4.0
d.	Filgrastim	3.0
e.	Interleukin-11	2.0
f.	Deferoxamine	1.0
g.	Darbepoetin	3.0
h.	Vitamin B12/ Cyanocobalamin	4.0
i.	Sargramostim	2.0
j.	Thrombopoietin	2.0

#### **Physiology and Pathophysiology**

2.	Describe the normal physiological control of hematopoietic growth factors and the effect kidney failure on erythropoiesis.	t of <b>4.0</b>		
3.	Relate factors leading to abnormal iron balance, including genetic hemochromatosis, to absorption and transport pathways.	the iron <b>3.0</b>		
4.	Identify the biochemical systems that are impaired in B-12 and folic acid deficiency, and of cyanocobalamin and folic acid in correcting the metabolic defect in DNA thymine and methionine synthesis.	the role <b>3.0</b>		
Me	Mechanism of action			
5.	Identify the molecular mechanism of action of each drug in each drug class.	3.0		
Actions on Organ Systems				
6.	Describe the pharmacological effects of the drugs in each class on the hematopoietic sys	stem.		
		3.0		

#### Pharmacokinetics

Explain the possible etiologies that should be considered if a delayed or diminished response to doses of recombinant erythropoietin within the recommended dose range occurs.
 2.0

4.0

	8. 9.	Analyze how the pharmacokinetics and therapeutic effects of epoetin alpha and darbepo alpha differs between normal and anemic dialysis patients. Describe the sources, transport, metabolism, storage, and excretion of vitamin B-12 and acid	oetin 2.0 folic
	10.	Identify the factors that influence the bioavailability of vitamin B-12 and folic acid.	2.0
	Ad	verse Effects, Drug Interactions and Contraindications	
	11. 12. 13.	Describe the principal adverse effects of the drugs in each class. Describe the clinically important drug interactions of the drugs in each class. Describe the principal contraindications of the drugs in each class.	3.0 3.0 2.0
	The	erapeutic Uses	
	<ol> <li>14.</li> <li>15.</li> <li>16.</li> <li>17.</li> <li>18.</li> <li>19.</li> <li>20.</li> <li>21.</li> <li>22.</li> </ol>	Describe the approved therapeutic indications and contraindications and pharmacokinet recombinant erythropoietin and the erythropoietin-like drug darbepoetin. Identify the criteria used for the diagnosis of iron deficiency anemia and criteria for oral versus parenteral iron therapy. Describe associated side effects and the predicted rates of response to oral therapy and parenteral iron therapy in iron deficiency anemia. Summarize the risks of acute iron poisoning in children and its treatment. Describe the pharmacologic management of chronic iron overload disease. Explain the appropriate management, route of administration, vitamin dosage, and exper response. Identify the possible metabolic reasons that folic acid corrects the erythropoietic lesion, the neurologic lesion in Addisonian pernicious anemia. Rationalize the use of folic acid in elevated serum levels of homocysteine and in spina bit Compare the therapeutic applications for myeloid growth factors with those for thrombog growth factors.	tics for <b>3.0</b> therapy <b>2.0</b> <b>2.0</b> <b>3.0</b> rds to cted <b>3.0</b> but not <b>2.0</b> fida. <b>3.0</b> pojeitic <b>3.0</b>
В.	Ant	ticoagulant Drugs	
	1. Phy 2. 3. Me	Discuss the following with regard to subsequent objectives: a. Heparin b. Protamine Sulfate c. Vitamin K d. Bivalirudin e. Enoxaparin f. Warfarin Sodium (Coumarin) g. Lepirudin h. Argatroban ysiology and Pathophysiology Explain the importance of clotting factors and regulation of hemostasis. Describe the pathogenesis of thrombosis.	4.0 4.0 2.0 4.0 4.0 2.0 2.0 3.0 4.0
	4.	Describe the molecular mechanism of action of each drug in each drug class.	4.0

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0.	K, and discuss its importance in determining the mechanism of action of the oral antico	agulants.
7.	Describe the mechanism of action and pharmacokinetics of the following antithrombin heparin, low molecular weight heparin (eg, enoxaparin), bivalirudin.	agents: 4.0
Ac	tions on Organ Systems	
8. 9.	Explain the effects of heparin on lipolysis and its role as a growth factor. Describe the onset of action and duration of action of warfarin effect in relationship to of clotting factors and their production in the human.	2.0 half-life 4.0
10.	Explain the role of Vitamin K for the synthesis of coagulation factors (II, VII, IX and X) an Proteins C and S.	d <b>4.0</b>
Ph	armacokinetics	
11. 12.	Identify the appropriate routes of administration of heparin and warfarin. Describe the mechanism of action and onset of action of heparin with that of the oral anticoagulants	4.0 4.0
13.	. Understand the monitoring of warfarin therapy using PT, INR and the indications for me warfarin levels.	easuring 4.0
14.	Understand how pharmacogenomics can be used to predict the dose of warfarin in indi patients.	vidual <b>3.0</b>
Ad	verse Effects, Drug Interactions and Contraindications	
15. 16. 17. 18. 19.	<ul> <li>Describe the principal adverse effects and contraindications of the drugs in each class.</li> <li>Know the complications associated with heparin therapy (eg, excessive bleeding) and he induced thrombocytopenia with associated thrombosis.</li> <li>Understand how protamine and vitamin K are used as antidotes to excessive bleeding of heparin and warfarin, respectively.</li> <li>Explain the adverse effects, contraindications and toxicities of warfarin, including embra fetal toxicities.</li> <li>Describe drug-drug, drug-food, and drug-disease interactions with warfarin.</li> </ul>	4.0 eparin- 4.0 aused by 4.0 yo and 4.0 4.0
Th	erapeutic uses	
20.	Contrast the use and monitoring of standard versus low-molecular-weight heparin prep	arations. <b>4.0</b>
21. 22. 23.	<ul> <li>Explain how antithrombin agents are used clinically for anticoagulation in patients with induced thrombocytopenia.</li> <li>Identify clinical uses and goals of warfarin therapy including its use in venous thromboe diseases, atrial fibrillation, myocardial infarction, and strokes.</li> <li>Discuss the approach to the management of the patient on short term and long term or anticoagulation.</li> </ul>	heparin- 4.0 mbolic 4.0 ral 4.0
An	tiplatelet Drugs	
1.	Discuss the following with regard to subsequent objectives: a. Aspirin (acetylsalicylic acid) b. Eptifibatide	4.0 2.0

С.

- 5. Identify the sites of action of heparin and direct thrombin inhibitors in the coagulation process.
- 4.0 6. Explain the relationship between the chemical structure of the oral anti-coagulants and vitamin

		c. Tirofiban	2.0
		d. Clopidogrel	4.0
		e. Dipyridamole	2.0
		f. Abciximab	2.0
		g. Thropfullie	2.0
	Phy	ysiology and Pathophysiology	
	2. 3.	Explain the role of platelet aggregation in the regulation of hemostasis. Describe the pathogenesis of thrombosis with respect to the platelet release reaction.	4.0 4.0
	Me	echanism of Action	
	4. 5. 6.	Identify the molecular mechanism of action of each drug in each drug class. Understand how inhibition of prostaglandin synthesis affects platelet aggregation, specific the role of COX-1 and COX-2. Compare and contrast the mechanism of action for aspirin, dipyridamole, ticlopidine, clopidogrel, and abciximab.	3.0 fically 4.0 4.0
	Act	tions on Organ Systems	
	7.	Identify the site of action of each drug in the platelet aggregation process.	3.0
	Pha	armacokinetics	
	8.	Compare the effects and time course of aspirin with standard nonsteroidal anti- inflamm agents (NSAIDs) and cyclooxygenase 2 (COX2) inhibitors on platelet function.	natory <b>4.0</b>
	Ad	verse Effects, Drug Interactions and Contraindications	
	9. 10.	Identify the principal adverse effects and contraindications of the drugs in each class. Identify and describe the drug-drug, drug-food, and drug-disease interactions of each dr	<b>4.0</b> ug. <b>4.0</b>
	11.	. Explain how concomitant use of NSAIDS can interfere with the antiplatelet actions of asp	birin. <b>4.0</b>
	The	erapeutic Uses	
	12. 13. 14.	<ul> <li>Explain management of the patient on short-term and long-term antiplatelet therapy.</li> <li>Explain the role of the platelet glycoprotein IIb/ IIIa inhibitors in the management of cord disease.</li> <li>Compare the effects of aspirin, ibuprofen, and propranolol for primary post MI prophyla</li> </ul>	2.0 onary 3.0 xis. 3.0
D.	Fib	prolytic/Antifibrolytic Drugs	
	1.	Discuss the following with regard to subsequent objectives:	
		a. Streptokinase	3.0
		b. Tissue Plasminogen Activator (Alteplase)	4.0
		c. Anistreplase	2.0
	Dh.	a. Aminocaproic acia	3.0
	F (1)		
	2.	Explain the role of plasminogen in thrombolysis.	4.0

3.	Explain the role of thrombolysis in the physiology of hemostasis.	4.0
M	echanism of action	
4.	Compare the molecular mechanism and site of action of alteplase to that of aminocapro	oic acid. <b>2.0</b>
5.	Explain the differences between streptokinase and alteplase in the activation of plasmin	nogen. 3.0
Ac	tions on Organ Systems	
6.	Explain the degradation of clotting factors from streptokinase.	3.0
Ph	armacokinetics	
7.	Compare the pharmacokinetic differences of alteplase and streptokinase.	2.0
Ad	verse Effects, Drug Interactions and Contraindications	
8. 9.	Relate the major adverse effects of thrombolytic drugs to their mechanism of action. Identify primary contraindications for thrombolytic drugs.	3.0 4.0
Th	erapeutic Uses	
10. 11.	Identify the major indications for thrombolytic drug therapy. Explain why aminocaproic acid (EACAis used routinely along with desmopressin and fac replacement in dental procedures in patients with hemophilia and von Willebrand's disc	<b>4.0</b> tor ease and
	for non-dental bleeding episodes in both diseases.	2.0

#### XI. <u>Chemotherapy</u>

#### A. Basic Principles of Antimicrobial Therapy

1.	Define antibiotics, selective toxicity, therapeutic index, bacteriostatic, and bactericidal.	4.0
2.	Identify MIC and MBC values.	3.0
3.	Differentiate between synergism and antagonism.	3.0

- 4. Explain the modes of action of antimicrobial drugs. 4.0
- 5. Explain bacterial resistance and the mechanisms involved in acquiring bacterial resistance. 4.0
- 6. Understand the basic principles of combination therapy with antimicrobial drugs. **4.0**

#### B. Cell Wall Synthesis Inhibitors

1.	Discuss	the following with regard to subsequent objectives:	
	a.	Amoxicillin	4.0
	b.	Ampicillin	4.0
	с.	Carbenicillin	3.0
	d.	Clavulinic Acid	4.0
	e.	Cloxacillin	3.0
	f.	Imipenem	4.0
	g.	Meropenem	2.0
	h.	Methicillin	3.0
	i.	Mezlocillin	2.0

j.	Nafcillin	2.0
k.	Oxacillin	2.0
I.	Penicillin G	4.0
m.	Pencillin V	4.0
n.	Piperacillin	4.0
0.	Sulbactam	3.0
p.	Tazobactam	3.0
q.	Ticarcillin	3.0
r.	Cefaclor	3.0
s.	Cefazolin	3.0
t.	Cefepime	3.0
u.	Cefotaxime	3.0
٧.	Cefoxitin	3.0
w.	Cefprozil	3.0
х.	Ceftazidime	3.0
у.	Ceftriaxone	4.0
z.	Cephalexin	4.0
aa.	Vancomycin	4.0

#### Mechanism of action

1.	Discuss the structural relationship of the penicillin molecule to antimicrobial activity.	3.0
2.	Identify the mechanism of action of $\beta$ -lactam antibiotics.	4.0
3.	Discuss the principle of combination of inhibitors of $\beta$ -lactamase with penicillins.	4.0
4.	Explain the pharmacological basis for combining imipenem with cilastatin.	3.0
5.	Describe the structural differences between penicillins and cephalosporins.	3.0
6.	Identify the mechanism of action of cephalosporins.	4.0
7.	Identify the mechanism of action of vancomycin.	4.0

## Pharmacokinetics

1. 2. 3. 4.	Describe the pharmacokinetic properties of penicillins. Discuss the repository penicillins. Discuss the penicillinase-resistant penicillins. Discuss the four generations of cephalosporins with respect to the differences in their antimicrobial spectrum and pharmacokinetic properties. Discuss the pharmacokinetic properties of vancomycin.	4.0 4.0 4.0 4.0 4.0
Ad	verse Effects and Contraindications	
1. 2. 3. 4.	Identify the principal adverse effects of penicillins. Identify the principal contraindication of penicillins. Identify the adverse effects due to cephalosporins and vancomycin. Define <i>superinfection</i> and <i>cross-hypersensitivity</i> .	4.0 4.0 4.0 4.0
Th	erapeutic Uses	
1. 2.	Identify primary therapeutic indications for penicillin G. Identify the indications for broad-spectrum penicillins.	4.0 4.0

- 3. Describe the antimicrobial activity of monobactams and carbapenems. 4.0 4.0
- 4. Identify the main therapeutic indications of cephalosporins and vacomycin.

# C. Protein Synthesis Inhibitors

D.

1.	Discuss the following with regard to subsequent objectives:	
	a. Amikacin	2.0
	b. Gentamicin	3.0
	c. Neomycin	4.0
	d. Netilmicin	1.0
	e. Streptomycin	2.0
	f. Tobramycin	2.0
	g. Clindamycin	4.0
	h. Azithromycin	4.0
	i. Clarithromycin	4.0
	j. Erythromycin	4.0
	k. Linezolid	4.0
	I. Quinupristin/dalfopristin	4.0
	m. Doxycycline	4.0
	n. Minocycline	3.0
	o. Tetracycline	4.0
	p. Chloramphenicol	4.0
Me	echanism of Action	
1.	Identify the mechanism of action of each class of protein synthesis inhibitors.	4.0
2.	Identify the mechanism of acquired drug resistance.	4.0
3.	Discuss the basis for combination therapy with an aminoglycoside and a penicillin,	
	cephalosporin, or vancomycin.	4.0
Ph	armacokinetics	
4.	Discuss the pharmacokinetic properties of each class of protein synthesis inhibitors.	4.0
5.	Discuss the importance of peak and trough levels of aminoglycosides.	4.0
6.	Discuss the need for and the method of dose adjustment for aminoglycosides in patients	with
	compromised renal function.	4.0
Ad	verse Effects and Drug Interactions	
7.	Identify the main toxicities of each class of protein synthesis inhibitors.	4.0
8.	Identify the major drug interactions of macrolides due to inhibition of cytochrome P450	
	enzymes.	4.0
Th	erapeutic Uses	
9.	Identify the primary therapeutic indications for each class of protein synthesis inhibitors.	4.0
Inh	nibitors of Nucleic Acid Metabolism and Drugs Interfering with Intermediary Metabo	olism
1	Discuss the following with regard to subsequent objectives:	
т.	a Ciproflovacin	4 0
		7.0

Levofloxacin	3.0
Metronidazole	4.0
Rifampin	4.0
Trimethoprim-sulfamethoxazole (cotrimoxazole)	4.0
	Levofloxacin Metronidazole Rifampin Trimethoprim-sulfamethoxazole (cotrimoxazole)

	Me	echanism of Action	
	2. 3. 4.	Identify the mechanism of action of the antibiotics that affect metabolism. Explain the synergistic inhibition due to sequential blockade with cotrimoxazole. Identify the adverse effects of ciprofloxacin, including contraindications in children and women.	4.0 3.0 pregnant 4.0
	Ph	armacokinetics	
	5.	Discuss the pharmacokinetic properties of each class of antibiotics.	3.0
	Ad	lverse Effects	
	6.	Identify the major toxicities of each class of drugs.	4.0
	Th	erapeutic Uses	
	7. 8. 9.	Identify the therapeutic indications of each class of antimicrobial drugs that affect meta Discuss the advantages of newer fluoroquinolones over ciprofloxacin. Identify the major therapeutic indications of sulfonamides alone, and in combination wi trimethoprim (cotrimoxazole).	bolism. <b>4.0</b> th <b>4.0</b>
Ε.	An	timycobacterial Drugs	
	1.	Discuss the following with regard to subsequent objectives: <ul> <li>a. Isoniazid</li> <li>b. Rifampin</li> <li>c. Pyrazinamide</li> <li>d. Ethambutol</li> <li>e. Streptomycin</li> <li>f. Azithromycin</li> <li>g. Clarithromycin</li> <li>h. Rifabutin</li> <li>i. Dapsone</li> <li>j. Clofazimine</li> <li>k. Thalidomide</li> </ul>	4.0 4.0 3.0 3.0 3.0 3.0 3.0 3.0 3.0 3.0 3.0 3
	Me	echanism of Action	
	2. 3. 4.	Discuss the first line antitubercular drugs and understand their mechanisms of action. Discuss the various phases of active- and slow-growing <i>Mycobacterium tuberculosis</i> and compare the relative effectiveness of various drugs. Discuss the drugs used in the treatment of Hansen's disease and their mechanism of act	<b>4.0</b> <b>3.0</b> ion. <b>3.0</b>
	Ph	armacokinetics	
	5.	Identify the pharmacokinetic profile of isoniazid and rifampin.	3.0
	Ad	lverse Effects and Drug Interactions	
	6. 7.	Identify the adverse effects of isoniazid, rifampin, ethambutol and pyrazinamide. Describe the drug interactions of rifampin with anticoagulants and other drugs, such as contraceptives.	<b>4.0</b> oral <b>4.0</b>

#### Therapeutic Uses

F.

8.	Describe the regimen recommended for preventive therapy and for conventional				
	chemotherapy.	4.0			
9.	Discuss the use of rifabutin, clarithromycin and azithromycin for treatment of Mycobacteria				
	avium complex.				
10	. Identify the drugs used for reversing the lepra reactions and the erythema nodosum leprosum				
	reaction.				
11	. Explain the WHO regimen for treatment of leprosy.	3.0			
An	ntiparasitic Drugs				
1.	Discuss the following with regard to subsequent objectives:				
	a. Albendazole	3.0			
	b. Atovaquone	3.0			
	c. Diethylcarbamazine	3.0			
	d. Diloxamidine	3.0			
	e. Iodoquinol	3.0			
	f. lvermectin	4.0			
	g. Mebendazole	4.0			
	h. Metronidazole	4.0			
	i. Nifurtimox	3.0			
	j. Paromomycin	3.0			
	k. Pentamidine	3.0			
	I. Praziquantal	4.0			
	m. Pyrantel Pamaote	4.0			
	n. Sodium Stibogluconate	3.0			
	o. Sulfadiazine	3.0			
	p. Suramin	3.0			
	q. Thiabendazole	3.0			
	r. Tinidazole	2.0			
	s. Trimetrexate	2.0			

#### **Mechanism of Action**

2. Identify the mechanism of action of mebendazole, praziquantel, pentamidine, and atovaquone.

#### **Therapeutic Uses**

3.	Identify the drugs of choice and alternate drugs available for treatment of diseas	es due to
	various helminthes.	3.0

- 4. Discuss the broad spectrum antihelminthic drugs and their spectrum of activity. **3.0**
- Discuss the opportunistic infections commonly known to occur in AIDS patients and the drugs used for their treatment.
   3.0
- 6. Identify the drugs of choice for treatment of asymptomatic, mild to moderate and severe intestinal disease and hepatic abscess due to *E. histolytica*. **3.0**
- Identify the drugs used for the treatment of protozoal diseases (giardiasis, trypanosomiasis, and leishmaniasis).
   3.0
- 8. Identify the drugs used for toxoplasmosis, an opportunistic infection in AIDS patients.

3.0

4.0

# G. Antimalarial Drugs

1.

a. Artemisinin analogs (artesunate and artemether) 4.0	1
b. Atovaquone/Proguanil 3.0	)
c. Chloroquine 4.0	)
d. Mefloquine 4.0	)
e. Primaquine 4.0	)
f. Pyrimethamine 4.0	)
g. Quinine 4.0	)
h. Sulfadoxine 2.0	)

# Mechanism of Action

	2.	Describe the various locations in the life cycle of malarial parasites where the antimala	rial drugs
		are effective.	3.0
	3.	Identify the mechanisms of action of chloroquine, primaquine and pyrimethamine.	4.0
	4.	Identify the mechanism of resistance to chloroquine.	4.0
	5.	Identify the mechanism of action of artemisinin derivatives.	4.0
	Ph	armacokinetics	
	6.	Discuss the pharmacokinetic properties of chloroquine.	3.0
	7.	Discuss the pharmacokinetic properties of artesunate and artemether.	2.0
	Ad	verse Effects	
	8.	Identify the mechanism of hemolytic anemia induced by primaquine in African-Americ	an males.
			3.0
	9.	Discuss cinchonism.	4.0
	10.	Identify the toxic effects of chloroquine.	4.0
	Th	erapeutic Uses	
	11. Identify the drugs of choice for treatment of uncomplicated illness and severe illne		
		vivax, P. ovale, P. malariae and P. falciparum.	3.0
12. Describe the regimen for prophylaxis for chloroquine-sensitive and chloroquine-resista			ant areas.
			3.0
	13.	Discuss the drug combination in Fansidar and its therapeutic use.	3.0
	14.	Identify the therapeutic indications for artemisinin derivatives.	3.0
Н.	An	tifungal Drugs	
	1.	Discuss the following with regard to subsequent objectives:	
		a. Amphotericin B	4.0
		b. Caspofungin (echinocandins)	4.0
		c. Fluconazole	4.0
		d. Griseofulvin	4.0
		e. Itraconazole	4.0
		f. Ketoconazole	4.0
		g. Nystatin	3.0
		h. Sulfamethoxazole-trimethoprim (cotrimoxazole)	3.0

# **Mechanism of Action**

Ι.

J.

<ol> <li>Identify the mechanism of action of each class of antifungal drugs.</li> <li>Discuss the advantages of liposomal preparations of amphotericin B.</li> <li>4.0</li> </ol>
Pharmacokinetics
4. Discuss the pharmacokinetic properties of the various antifungal drugs. <b>4.0</b>
Adverse Effects
<ol> <li>Identify the important adverse effects of the various antifungal drugs.</li> <li>Identify the drug interactions associated with; ketoconazole, and griseofulvin and warfarin.</li> <li>4.0</li> </ol>
Therapeutic Uses
7. Identify the major therapeutic indications of the antifungal drugs. <b>4.0</b>
Antiviral Drugs
1. Discuss the following with regard to subsequent objectives:4.0a. Acyclovir/ Valacyclovir4.0b. Amantadine2.0c. Foscarnet3.0d. Ganciclovir/ Valganciclovir4.0e. Idoxuridine2.0f. Interferon alpha4.0g. Oseltamivir4.0h. Ribavarin4.0i. Rimantadine2.0j. Trifluridine2.0k. Zanamivir3.0
Mechanism of Action
<ol> <li>Classify antiviral drugs based upon their site of inhibition in the viral replication cycle.</li> <li>Identify the mechanism of action of each antiviral drug.</li> <li>4.0</li> </ol>
Pharmacokinetics
4. Discuss the pharmacokinetic properties of acyclovir and ganciclovir. <b>2.0</b>
Adverse Effects
5. Identify adverse side effects and potential drug interactions. <b>4.0</b>
Therapeutic Uses
6. Identify the major therapeutic indications for each antiviral drug.4.0
Antiretroviral Drugs
1. Discuss the following with regard to subsequent objectives :4.0a. Abacavir4.0b. Didanosine (ddl)2.0c. Lamivudine (3-TC)4.0

	d. Stavudine (D4T)	2.0
	e. Zalcitabine (ddC)	2.0
	f. Zidovudine (AZT)	4.0
	g. Amprenavir	3.0
	h. Atazanavir	4.0
		1.0
	J. LOPINAVIR k. Nolfinavir	4.0
	k. Nellillavii L. Bitonavir	2.0 4 0
	m. Saguinavir	4.0
	n. Delaviridine	1.0
	o. Efavirenz	4.0
	p. Nevirapine	3.0
	q. Enfuvirtide	3.0
	r. Maraviroc	4.0
	Mechanism of Action	
	2. Classify anti-HIV drugs based upon their site of inhibition in the viral replication cycle.	3.0
	3. Identify the mechanism of action of the nucleoside reverse transcriptase inhibitors.	4.0
	4. Identify the mechanism of action of the protease inhibitors.	4.0
	Pharmacokinetics	
	5. Discuss the use of combinations of different class of anti-HIV drugs.	4.0
	6. Describe the pharmacokinetic properties of each class of anti-HIV drugs.	3.0
	Adverse Effects	
	7. Identify the major side effects of each class of anti-HIV drugs.	4.0
	Therapeutic Uses	
	8. Discuss the various drug combinations used for the treatment of HIV infections.	4.0
К.	Basic Principles of Cancer Chemotherapy	
	1. Explain the role of chemotherapy in the management of patients with cancer.	4.0
	2. Discuss the prospects for "cure", or long term survival in cases of advanced cancer.	4.0
	3. Discuss the limitations to effective drug treatment.	4.0
	4. Define selective toxicity, mass doubling time, and growth fraction.	4.0
	5. Explain the concept of "total cell Kill" in cancer patients.	3.U
	<ul> <li>Define cell cycle specificity and classify anticalicer utugs based on the cell cycle specific</li> <li>Discuss the principles of combination chemotherapy in the treatment of cancer</li> </ul>	11. 2. 0
	<ol> <li>Identify the mechanisms of resistance to anticancer drugs.</li> </ol>	4.0
	,	
L.	Anticancer Drugs	
	1. Discuss the following with regard to subsequent objectives:	
	a. Busulfan	2.0
	b. Cyclophosphamide	4.0

~.	eyclophicsphannac	
c.	Dacarbazine	2.0

Ifosfamide	4.0
Mechlorethamine	4.0
Melphalan	3.0
Nitrosoureas (carmustine and lomustine)	4.0
Actinomycin D (Dactinomycin)	4.0
Bleomycin	3.0
Camptothecin analogs (irinotecan, topotecan)	3.0
Daunorubicin	4.0
Doxorubicin	4.0
Docetaxel	3.0
Etoposide (VP-16)	4.0
Paclitaxel	4.0
Vinblastine	4.0
Vincristine	4.0
Cetuximab	3.0
Rituximab	3.0
Trastuzumab	4.0
Capecitabine	3.0
Cytarabine	3.0
5-Fluorouracil	4.0
Gemcitabine	4.0
6-Mercaptopurine	4.0
Methotrexate	4.0
Thioguanine	2.0
Erlotinib	3.0
Gefitinib	3.0
Imatinib	4.0
Lapatinib	3.0
Sunitinib	4.0
Tamoxifen	4.0
Flutamide	4.0
Leuprolide	3.0
Goserelin	3.0
Aromatase inhibitors (anastrozole)	4.0
Sex hormone inhibitors and antagonists	3.0
n. Glucocorticoids (prednisone)	4.0
Asparaginase	2.0
Bortezomib	4.0
Carboplatin	3.0
Cisplatin	4.0
Hydroxyurea	3.0
Interteron alpha 2b	4.0
Procarbazine	2.0
Soratenio	2.0
vorinostat	2.0
	Ifosfamide         Mechlorethamine         Melphalan         Nitrosoureas (carmustine and lomustine)         Actinomycin D (Dactinomycin)         Bleomycin         Camptothecin analogs (irinotecan, topotecan)         Daunorubicin         Doxorubicin         Doxorubicin         Docetaxel         Etoposide (VP-16)         Paclitaxel         Vinblastine         Vincristine         Cetuximab         Trastuzumab         Capecitabine         Cytarabine         S-Fluorouracil         Gemeitabine         6-Mercaptopurine         Methotrexate         Thioguanine         Erlotinib         Geffinib         Imatinib         Lapatinib         Sunitinib         Tamoxifen         Flutamide         Leuprolide         Goserelin         Aromatase inhibitors (anastrozole)         Sex hormone inhibitors and antagonists         .         Suparaginase         Bortzontio         Carboplatin         Cisplatin         Hydroxyurea         Interferon alpha 2b

# Mechanism of action

	2.	2. Identify the mechanism of action of various individual anticancer drugs under each class			
		,	4.0		
	3.	Discuss the bioactivation pathways required for the action of cyclophosphamide.	2.0		
	4.	Discuss the intracellular activation pathways of different antimetabolites.	2.0		
	5.	Describe the use of leucovorin rescue n high dose methotrexate therapy.	4.0		
	Ad	verse Effects			
	6.	Identify the common toxicities of each class of anticancer drugs. 3			
	7.	Identify the specific major toxicity of individual anticancer drugs.	3.0		
	8.	Identify the cumulative dose-dependent toxicity of anthracyclines.			
	The	nerapeutic Uses			
	9.	. Identify the major therapeutic indications of various anticancer drugs.			
	10.	). Discuss the drug combinations that have shown activity against specific types of cancer.			
	11.	Explain adjuvant chemotherapy and describe various regimens used in the treatment of	cancer		
		of unreferit organ systems.	2.0		
М.	Imr	nunosuppressive Drugs			
	1.	Discuss the following with regard to subsequent objectives:			
		a. Aldesleukin	2.0		
		b. Antithymocyte globulin	2.0		
		c. Azathioprine d. Gydenbeenberide	4.0		
		e Daclizumah	2.0		
		f. Etanercept	4.0		
		g. Infliximab	4.0		
		h. Interferons (alpha, beta, and gamma)	4.0		
		i. Methotrexate	4.0		
		j. Muromonab-CD3	2.0		
		k. Mycophenolate mofetil	4.0		
		I. Prednisone	4.0		
		n Sirolimus (ranamycin)	2.0		
		o. Tacrolimus	4.0		
		p. Cyclosporine	4.0		
		q. Thalidomide	2.0		
	Me	Mechanism of Action			
	2.	Discuss the general principles of immunosuppression and immunostimulation.	4.0		
	3.	Identify the mechanism of action of immunosuppressants and immunostimulants.	4.0		
	Adverse Effects				
	4.	Identify the toxicities of antibodies and other agents used as immunosuppressants.	4.0		
	5.	identify and describe the types of allergic reactions to drugs.	4.0		
	The	erapeutic Uses			
	6. Explain the clinical uses of immunosuppressants. 4.				

# XII. <u>Toxicology and Therapy of Intoxication: Drugs Used as Antidotes</u>

1.	Define	the following and apply to subseqent objectives :	
	a.	n-Acetylcysteine	4.0
	b.	Air pollutants	2.0
	с.	Alcohols (ethanol, methanol, ethylene glycol)	4.0
	d.	Carbon monoxide	2.0
	e.	Cyanide	1.0
	f.	Naloxone	3.0
	g.	Iron	4.0
	h.	Chelators	2.0
	i.	Mercury	1.0
	j.	Pesticides (organophosphates and carbamates)/atropine/2-pam	3.0
	k.	Activated charcoal	4.0
	Ι.	Flumazenil	4.0
	m.	Methylene blue	2.0
	n.	Sodium bicarbonate	4.0
	0.	Sodium thiosulfate	2.0

# Principles of Toxicology

XIII.

2. Explain how toxicants are influenced by pharmacokinetic and pharmacodynamic pr	ocesses such			
as absorption, distribution, biotransformation, excretion and cellular targets.	2.0			
3. Explain the principles of bioactivation of chemicals to toxic species.	1.0			
<ol><li>Identify cellular defense mechanisms.</li></ol>	1.0			
5. Explain the concept of threshold levels for toxicity.	1.0			
6. Describe measures for determining drug safety and therapeutic ratio.	2.0			
Drugs/Chemicals				
7. List the signs and symptoms of toxic exposure to common toxins and toxic drugs.	3.0			
8. Explain how exposure to the primary and secondary toxicants can occur, and identi	fy the			
mechanisms of toxicity.	2.0			
9. Compare the toxicity induced by various metals.	2.0			
10. Compare the toxicity induced by the neurotoxic pesticides.				
11. Recall the antidote and/or treatment for each toxicant, and explain how to manage	acute			
intoxication.	3.0			
Environmental Toxicology/Risk Assessment				
12. Differentiate between mutagenicity and carcinogenicity.	1.0			
Herbal Medicine				
1. Discuss the following with regard to subsequent objectives:				
a. Gingko	2.0			
b. Echinachea	3.0			
c. Glucosamine Chondroitin	3.0			
d. Saw Palmetto	3.0			
e. Black Cohosh	2.0			
f. Guarana	2.0			
g. Valerian	2.0			

		<ul> <li>h. Ephedra</li> <li>i. Ginseng</li> <li>j. Kava</li> <li>k. St. John's Wort</li> <li>l. Chamomile</li> <li>m. Milk Thistle</li> <li>n. Yohimbe</li> </ul>	3.0 1.0 3.0 4.0 1.0 1.0 2.0
	Ac	ctions on Organ Systems	
	2. 3.	Explain the mechanisms through which herbal products exert their pharmacological effect Discuss the concept that there is evidence towards effectiveness for some herbal product some have shown to have no beneficial effect.	cts. <b>3.0</b> ts, but <b>4.0</b>
	Ad	dverse Effects, Drug Interactions and Contraindications	
	4. 5. 6. 7.	Identify and discuss the serious drug interactions that occur between herbal products and prescription medicines. Identify serious side effects of herbal products. Identify herbal products that should be avoided during pregnancy. Discuss the lack of FDA regulation of herbals and what that means regarding safety and e testing.	d 4.0 4.0 4.0 fficacy 4.0
XIV.	<u>Vi</u> t	tamins	
	1.	Discuss the following with regard to subsequent objectives: a. Vitamins A, D, E, K b. Vitamin C, nicotinamide, cyanocobalamin, pyridoxine c. Nicotinic acid, folic acid	3.0 3.0 3.0
	Ph	nysiology and Pathophysiology	
	2. 3. 4.	Differentiate between vitamins and antioxidants. Identify populations that have the highest risk of vitamin deficiency. Explain vitamin deficiency related problems that are commonly found in persons that chr abuse ethanol.	<b>1.0</b> <b>3.0</b> ronically <b>3.5</b>
	M	echanism of action	
	5.	Explain the mechanism of action of the water-soluble and lipid-soluble vitamins.	3.0
	Ac	ctions on Organ Systems	
	6.	Describe effects of the deficiency of each of these water and lipid-soluble vitamin types a relate to disease processes.	is they <b>3.0</b>
	Ad	dverse Effects and Toxicities	
	7. 8. 9.	Explain the principal adverse effects and toxicities for overdose and toxic levels of both w soluble and lipid-soluble vitamins. Identify the clinically important drug interactions of the drugs in each class. Explain the principal contraindications of the drugs in each class.	vater- 3.5 3.5 3.0

# Therapeutic Uses

10.	Explain vitamin regulation.	3.5
11.	Relate recommended dietary allowances (RDA) to vitamin use.	2.0
12.	Describe the therapeutic uses of the fat-soluble vitamins, including that of isotretinoin.	2.5
13.	Describe the use of thiamine in the emergency treatment of alcoholism.	3.0

# **GERIATRICS LEARNING OBJECTIVES**

# I. <u>Geriatrics</u>

1. Discuss the present and future care and economic issues resulting from demographic		
	reflect a steadily rising geriatric population within the U.S.	4.0
2.	Apply principles of the biology of aging related to geriatric pharmacotherapy and diagno	ostic
	laboratory values.	4.0
3.	Explain the role of the podiatrist in a multi-disciplinary geriatric healthcare team.	4.0
4.	Identify and discuss clinical situations wherein life expectancy, functional status, patient	t
	preference, and/or goals of care override standard recommendations for treatment in t	he
	geriatric patient.	4.0
5.	Create a management plan for falls, balance disorders and gait disorders in the geriatric	patient.
		4.0
6.	Identify challenges associated with the evaluation and management of urinary incontine	ence.
		3.0
7.	Discuss cognitive and behavioral disorders in the geriatric patient.	4.0
8. List and differentiate between types of code status, healthcare proxies, and advanced d		
	as indicated by the clinician's state of practice/training.	4.0
9.	Discuss the unique needs of the geriatric patient in institutional settings.	4.0
10.	. Explain the spectrum of institutional healthcare settings available to the geriatric patien	t.
		4.0
11	Recognize signs of elder abuse and explain protocol for reporting abuse	4.0
12	Discuss the spectrum of end-of-life care as a positive active treatment option for a patie	ent with
	advanced disease	<b>// 0</b>
		7.0
# **MEDICINE LEARNING OBJECTIVES**

Infectious diseases Neurologic disorders Cardiovascular disorders Rheumatologic disorders Metabolic and endocrine disorders Hematologic disorders, including anemias and leukemia Immunologic disorders (allergic and sensitivity reactions, and immunosuppressive states) Respiratory disorders (including asthma, emphysema, infectious pneumonitis) Behavioral medicine (depression, abuse, anger disorders, and noncompliant patients) Emergency medicine (medical/surgical) Dermatology Gastroenterology Geriatrics Pre- and Postoperative Treatment

# I. Infectious diseases

	1.	Explain the performance of a focused history and physical to identify patients with acute	e
		infectious disease.	4.0
	2.	Identify laboratory, physiologic, or imaging data that is utilized in diagnosing and recogn	nizing
		acute and or chronic infectious disease.	4.0
	3.	Order the stages of an infectious disease.	2.2
	4.	List and describe the potential outcomes of an infection.	3.6
	5.	List common endogenous and exogenous sources of infectious agents.	3.9
	6.	Discuss common invasion sites and methods of colonization and proliferation.	3.6
	7.	Discuss factors that determine the virulence of an organism.	2.3
	8.	Discuss potential host defenses against an invading organism.	2.9
	9.	Describe common host responses to infection.	2.8
	10.	List specific types of bacterial infections to which a host is susceptible when host defense	es are
	10.	defective or inactivated	29
	11	Define fever of unknown origin (ELIO) list common causes and describe how ELIOs are	2.5
	11.	classified	20
		classified.	2.5
А.	Ва	sterial	
	1	List common bacterial infections and the most likely causative organism in skin and join	ts
	±.		39
	2	Identify clinical laboratory studies used to diagnose infection	2.9
	2.	Recommend proper aptibiotic selection and usage for a given organism	3.0 2 0
	ס. ⊿	Discuss the eticlomy procentation diagnosis and treatment of joint space infections an	<b>3.0</b>
	4.	Discuss the ethology, presentation, diagnosis, and treatment of joint space methods	u 40
	_	puncture wounds.	4.0
	5.	Classify osteomyelitis and puncture wounds.	3.5
	6.	List the symptoms of, and common antibiotics used for, the treatment of urinary tract	
	_	infections.	2.4
	7.	List the treatment modalities available for patients with sexually transmitted diseases.	1.8
	8.	Differentiate between colonization and infection in the diabetic foot ulcer.	3.8
	9.	Distinguish cellulitis from erysipelas.	3.0
	10.	Compare and contrast antibiotic prophylaxis in site versus distal infection.	2.8
	11.	Discuss the Centers for Disease Control and Prevention (CDC) guidelines for hand hygier	ne.
			3.6
	12.	Describe preventive strategies for needlestick and sharps injuries intended to reduce th	e
		transmission of blood borne pathogens (hepatitis B, hepatitis C, and HIV).	3.9
	13.	Identify the indications, efficacy, and side effects of post-exposure prophylaxis for hepa	tits B
		and HIV/AIDS.	3.0
	14.	Compare and contrast cat, dog, and human bite wounds with respect to prevalence, usu	lal
		etiologic agents, risk of infection, treatment options, and potential complications.	3.1
р	Vin		
В.	VII	u	
	1.	Identify incidence and prevalence of HIV.	2.5
	2.	Describe the pathophysiology of AIDS.	2.6
	3.	List the manifestations of AIDS on the lower extremity with respect to dermatological,	
		neurological, vascular, and musculoskeletal findings.	3.5
	4.	Explain the significance and complexity of the AIDS epidemic from a historical perspectiv	ve. <b>1.8</b>
	5.	Identify known routes of HIV transmission.	3.3

	6. 7.	Explain how the HIV-1 virus attacks CD4 T-cell lymphocytes and replicates. Distinguish between the "primary infection," "clinical latency," and "symptomatic" phase clinical course of HIV-infected individuals.	<b>1.6</b> ses in the
	8.	Explain how the Absolute CD4 (T-helper) Lymphocyte Count, CD4 Lymphocytes, and Virare used as predictors of outcome in HIV.	al Load <b>2.6</b>
	9.	Distinguish between HIV Infection and AIDS, and list current criteria for AIDS-defining illu	nesses. <b>2.5</b>
	10.	Discuss symptoms and signs of acute HIV seroconversion.	2.3
	11.	Discuss the basic principles of highly active antiretroviral therapy (HAART), including the	j
		different classes of antiviral medications and their uses, as well as common side effects	and
	10	drug-drug interactions.	1.6
	12.	Discuss basics of post-exposure prophylaxis.	3.1
	13. 14.	Discuss the significance of basic liver function studies.	2.6 3.8
	15.	Discuss the route of transmission, incubation period, duration of illness, duration of vira	al
		shedding, duration of (uncomplicated) illness, and the timing of the "flu season", lab dia and vaccination of the population.	agnosis, <b>2.3</b>
	16.	Discuss the composition, ideal time (of the year) for administration, the dose/route/presite of injection, efficacy, priorities for vaccination, contraindications, and side effects or influenza vaccine.	ferred f the <b>1.6</b>
	17.	Explain the laboratory diagnostic technique and positive findings of viral infections.	2.0
	18.	Describe the diagnosis, clinical findings, prevention, treatment, and complications of HS Varicela and Zoster, Mononucleosis and CMV	VI & 2, <b>2.6</b>
	19.	Describe the diagnosis, clinical findings, prevention, treatment, and complications of Me	easles,
	20	Numps, Pollomyelitis, and Rubella.	1.9
	20.	Colorado Tick Fever, Hemorrhagic Fevers and Yellow Fever.	<b>1.5</b>
	21.	Describe the diagnosis, clinical findings, prevention, treatment, and complications of Co Cold, Uncute Undifferentiated Respiratory Diseases, Pharyngoconjunctival, keratoconju and Cystitis.	mmon nctivitis, <b>1.5</b>
С.	Fur	ngal	
	1.	Discuss the past and current scope of tuberculosis.	2.1
	2.	Outline the natural history of TB.	1.5
	3.	Explain the role of TB skin testing in TB screening and discuss conditions which may pro false negative or false positive results.	duce <b>2.4</b>
	4.	List the populations most at risk for TB.	2.1
	5.	List the treatment and prophylaxis regimen for mycobacterium tuberculosis.	2.0
	6.	Discuss risk: benefit considerations of INH prophylaxis, based upon patient age and desirisk group.	gnated <b>2.0</b>
	7.	Discuss recommendations for tuberculin skin testing and antimicrobial prophlylaxis of	
		individuals who have had close exposure to a patient with active tuberculosis.	2.1
	8.	Explain how to perform and interpret a KOH.	3.3
	9.	Explain how and when to use a Wood's light and how to interpret the results.	3.3
	10.	Define yeast, mold hyphae, mycelium, , and fungemia.	2.3
	11.	Differentiate between macro- and microconidia; mono- and dimorphic organisms; supe and deep-seated fungal infections; and opportunistic and nonopportunistic fungal infec	rticial tions. <b>2.3</b>

12.	Classify and compare nystatin, amphotericin B, clotrimazole, miconazole, ketoconazole,	
	fluconazole, and itraconazole.	3.1
13.	Define histoplasmosis, blastomycosis, coccidioidomycosis, sporotrichosis, candidiasis,	
	aspergillosis, and cryptococcosis.	2.0

### II. <u>Neurologic disorders</u>

1.	Explain the basic pathophysiology, diagnostic methods, and treatment regimens for the	
	common podiatric complaints, such as neuromas, and metatarsalgia.	4.0
2.	List the basic instrumentation and set-up for an NCV and EMG.	2.4
3.	List the pathologies that can be diagnosed via NCV and EMG.	3.1
4.	List the concepts of amplitude, duration, and latency as it pertains to NCV.	2.5
5.	Understand the use of electromyography to evaluate peripheral neuropathies.	2.9
6.	Understand basic patterns derived from electromyography.	1.9
7.	List the methods of obtaining a normal electromyogram.	2.0
8.	Understand the peripheral nervous system, pathological changes and anatomy of periph	eral
	nervous system.	3.8
9.	Understand the clinical features of peripheral nervous system disorders and the clinical	
	syndromes.	3.8
10.	Understand the treatment of peripheral neuropathies.	3.9
11.	List the parts and anatomy of the peripheral nervous system.	3.6
12.	Discuss the pathologic disorders with neuropathies.	3.7
13.	List the clinical features of peripheral nerve disorders.	3.9
14.	List the clinical syndromes and clinical features of peripheral nerve diseases.	3.9
15.	Identify the entrapment neuropathies and their clinical features.	3.9
16.	Identify the evaluation and treatment of the peripheral neuropathies.	3.9
17.	Generate a differential diagnosis, associated clinical features, and appropriate treatment	t for a
	given patient presenting signs and symptoms consistent with peripheral nerve diseases.	3.8
18.	Identify and recommend management for a complex regional pain syndrome.	3.8
19.	Identify and describe the common locations of nerve entrapment and/or injury in the fo	ot.
		3.6
20.	Explain the various causes of these nerve entrapments and/or injuries.	3.8
21.	Describe the treatment protocols for nerve entrapments and/or injuries pathologies.	3.5
22.	Describe the etiology, pathophysiology, clinical presentation, laboratory studies, diagnos	sis,
	treatment/management, course, complications, and prognosis in sciatic nerve damage.	2.8
23.	Describe the etiology, pathophysiology, clinical presentation, laboratory studies, diagnos	sis,
	treatment/management, course, complications, and prognosis in femoral nerve damage	•
		2.9
24.	Describe the etiology, pathophysiology, clinical presentation, laboratory studies, diagnos	sis,
	treatment/management, course, complications, and prognosis in common peroneal ner	ve
<u> </u>	damage.	4.0
25.	Describe the etiology, pathophysiology, clinical presentation, laboratory studies, diagnos	SIS,
20	treatment/management, course, complications, and prognosis in tibial nerve damage.	4.0
26.	Describe the etiology, pathophysiology, onset, clinical presentation and findings, laborat	ory
	studies, diagnosis, treatment/management, course, complications, and prognosis of refl	ex
	sympathetic dystrophy (causaigia).	3.9

- 27. Describe the types, etiology, clinical presentation, clinical evaluation, electrophysiologic evaluation, differential diagnoses, treatment, and prognosis of posterior tarsal tunnel syndrome.
- Describe the types, etiology, clinical presentation, clinical evaluation, electrophysiologic evaluation, differential diagnoses, treatment and prognosis of anterior tarsal tunnel syndrome.
   3.8
- 29. Discuss the etiology, clinical presentation, clinical evaluation, electrophysiologic evaluation, differential diagnoses, treatment, and prognosis of superficial peroneal and sural nerve entrapments.
   4.0
- Classify neuropathy due to poison, deficiency states, and metablic disorder; neuropathy secondary to neoplasm; angiopathic neuropathy; inflammatory and infectious neuropathy; genetically determined neuropathy; and inherited polyneuropathies with specific metabolic disorder by etiology, including basically damaged nerve structures and predominant symptomology.
   30

31.	Describe the different forms of sensory neuropathy and their distinguishing clinical
	manifestations commonly noted in diabetic patients.

- 32. Describe the clinical presentation, diagnosis, management, and prognosis in motor neuropathy commonly noted in diabetic patients. **3.9**
- 33. Describe the clinical presentation, diagnosis, management, and prognosis in autonomic neuropathy commonly noted in diabetic patients.4.0
- 34. Understand the combined nature of sensory, motor, and autonomic neuropathies in a given diabetic patient.4.0
- 35. Describe Charcot Joint, including definition, etiology, stages, clinical findings of each stage, diagnostic studies, differential diagnoses, diagnosis, concepts of treatment, and prognosis from the neurological perspective.
   4.0

36. Describe the role of autonomic, sensory, and motor neuropathy in Charcot joint. **4.0** 

- 37. Describe the most commonly affected areas of the foot in Charcot foot.4.0
- D. Central Nervous System Disorders, Including Diseases of the Spinal Cord

1.	Differentiate between the forms of generalized epilepsy and partial epilepsy.	2.0
2.	Describe the value of electroencephalogram in diagnosis of epilepsy.	1.8
3.	List principles of management of patients with epilepsy, including the rationale for use	of the
	common pharmacologic agents.	2.3
4.	Discuss pathogenesis of bacterial meningitis and potential for prevention.	2.1
5.	Describe pathophysiology and resultant dysfunction in the CNS.	2.0
6.	Identify how to select antimicrobial therapy based on clinical/laboratory epidemiologic	
	evaluation.	2.8
7.	Demonstrate a neurologic examination with emphasis on reflex, sensory, and strength	esting.
		3.9
8.	Describe the clinical manifestations of movement disorders, with emphasis on Parkinso	n's, and
	their treatments.	3.0
9.	Describe the clinical symptoms of movement disorders in terms of anatomy, physiology	, and
	neurotransmitters of the basal ganglion, cortex, and brainstem nuclei.	2.6
10.	Describe how pallidotomy and other neurosurgical procedures decrease the symptoms	of
	Parkinson's disease and other movement disorders.	1.6
11.	Describe management of restless leg syndrome and related disorders.	3.1
12.	Classify head injuries.	1.6
13.	Describe the physical assessment of a patient with a head trauma.	2.1

3.8

14.	Select a	appropriate diagnostic studies for a patient suffering from a severe head injury.	2.1
15.	Describ	be the clinical manifestations of the following tumors of the CNS	
	a.	Gliomas: Astrocytoma; fibrillary astrocytoma, glioblastoma	1.4
	b.	Multiforme: Pilocytic astrocytoma, oligodendroglioma, ependymoma	1.3
	С.	Neuronal: Poorly differentiated neoplasm, medulloblastoma	1.3
	d.	Meningiomas	1.4
	e.	Metastatic tumors	1.8
	f.	Peripheral nerve sheath tumors	2.8
	g.	Schwannoma	2.8
	h.	Neurofibroma	2.8
16.	Describ	be features of coma.	2.0
17.	List crit	eria of irreversible coma or cerebral death.	1.3
18.	Define	dementia.	2.6
19.	Discuss	staging of dementia.	1.4
20.	Explain	the staging, diagnostic work-up, and treatment of dementia and pseudodement	ia.
	•		1.8
21.	Discuss	the physiology of pain.	3.1
22.	Discuss	clinical principles of pain management.	3.4
23	Differe	ntiate between acute and chronic pain, in terms of characteristics and approache	es in
	manag	ement	3.0
2/	Descrit	the clinical manifestations, course of illness, treatment, and prognosis of demy	lenating
۲.	disease	e with emphasis on multiple sclerosis	3.5
25	Discuss	current concents in nathogenesis of other demyelinating disease	2.6
25.	Identify	types of headaches including characteristics treatment and prevention	1.6
20.	Evolain	secondary causes of headaches	1.0
27.	List the	types of lesions that can affect the spinal cord	2.2
20.		represented to the solution of spinal conductions of spinal conductions	2.5
29.	List spe	contestions final can indicate revers and locations of spirial cord resions.	2.0
50. ⊃1	Descrit	the relationships of modules, and intra modules, losions of the spinol cord	2.0
31.	Differe	ntiate between extra-medullary and intra-medullary lesions of the spinal cord.	2.3
32.	Identify	risk factors, diagnosis, and treatment for cerebrovascular diseases.	3.0
33.	Identity	risk factors for cerebrovascular disease.	3.4
34.	List the	e diagnostic procedures and treatments used in care of patients with acute stroke	2.
			2.4
35.	Describ	be pathophysiologic considerations of hypoxia, ischemia, and infarction.	2.1
36.	Differe	ntiate between intracranial hemorrhage, intracerebral hemorrhage, subarachnoi	d
	hemor	rhage, and vascular malformations.	2.0
37.	Differe	ntiate between hypertensive cerebrovascular disease, lacunar infarcts, and hype	rtensive
	enceph	alopathy.	1.9
38.	Discuss	the clinical aspects of neurofibromatosis.	3.0
39.	Discuss	the clinical aspects of the other syndromes that involve the skin and central ner	vous
	system		1.8
40.	Identify	cerebellar diseases and describe their clinical presentation and management in	the
	lower e	extremity.	3.6
41.	Identify	and recommend management of basal ganglion diseases and their effect on	
	locomo	otion.	3.5
42.	Describ	e ALS (Combined Upper and Lower Motor Neuron Syndrome) in clinical terms.	2.3
43.	Describ	be the incidence, etiology, pathophysiology, clinical presentation, diagnosis, treat	ment,
	course	and prognosis of ALS.	3.0

- 44. Describe bilateral pyramidal syndrome; pain and paresthesia; urinary frequency, urgency, or incontinence; and mental symptoms associated with ALS. **1.6**
- 45. Describe the pathophysiology, symptoms and signs, diagnosis, treatment, course, complications, and prognosis of Syndeham's chorea. **2.6**
- 46. Describe the pathophysiology, symptoms and signs, diagnosis, treatment, course, complications, and prognosis of Huntington's disease. **2.1**
- 47. Describe the pathophysiology, symptomatology, stations and gait (equilibrium), test, and etiology of the cerebellar syndrome3.3
- 48. Describe the etiology, incidence, pathophysiology, clinical presentation, laboratory studies, diagnosis, treatment, course, and prognosis of syringomyelia. **3.0**

### III. Cardiovascular Disorders

### A. Major Cardiac

1.	Distinguish the major types of myocardial injury and relate specific principles of medica	ıl 👘
	management.	3.9
2.	Describe the physiologic basis of congestive heart failure and relate specific principles of	of medical
	management.	3.5
3.	Describe major cardiovascular diseases including endocarditis, aortic pathology, and	
	cardiomyopathies, and relate to specific principles of medical management.	3.5
4.	Distinguish the major types of pediatric cardiac disorders.	2.0
5.	Identify the lower extremity manifestations associated with cardiovascular disease.	3.8
6.	Identify factors associated with atherosclerotic vascular disease.	3.8
7.	Identify dietary components implicated in modifying the risk of vascular disease.	3.5
8.	Explain origins of hypertension and the role of lifestyle changes in restoring normotens	ion.
		3.4
9.	Explain how to perform a focused history and physical for the cardiac system.	4.0
10.	Identify the laboratory, physiologic, or imaging data that is utilized in identifying cardia	С
	pathology.	3.9
11.	Identify the cardinal symptoms and signs of cardiac pathology.	3.9
12.	Outline how to perform a cardiac examination.	3.8
13.	Outline how to perform a cardiac assessment in the context of clinical podiatric situation	ons.
		3.6
14.	Identifythe cardinal symptoms of cardiac disease state.	3.9
15.	Explain general concepts of electrocardiography.	3.6
16.	Explain how to correlate EKG findings with the patient's clinical presentation from a	
	perioperative standpoint.	3.4
17.	Identify acute coronary heart disease.	4.0
18.	Identify malignant arrhythmias.	3.5
19.	Describe the spectrum of ischemic heart disease.	3.3
20.	Explain how acute and/or chronic hypertension disease states affect and interact with o	clinical
	podiatric problems, such as perioperative assessment, as well as podiatric medicine issue	ues.
		3.8
21.	Describe the autonomic nervous system's role in cardiovascular regulation.	3.3
22.	Describe the pathogenesis, pathophysiology, and symptoms of atherosclerotic coronary	y and
	peripheral vascular disease.	3.7

(eg. cholesterol fractionation, CPK isoenzymes, troponin levels).       3.7         24. Describe the clinical manifestations of heart failure.       3.0         25. Identify common types of valvular heart disease, such as aortic stenosis, mitral regurgitation, and (rheumatic) mitral stenosis.       3.1         26. Explain the roles and pharmacology of commonly prescribed cardiac medications in the treatment of cardiac diseases (e.g. digoxin, diuretics, beta-blockers, ACE inhibitors, anti-arrhythmetics, nitrates, anti-coagulants and lipid lowering agents).       3.3         27. Define hypertension       4.0         28. Define secondary hypertension and identify the most common causes.       3.8         29. Rationalize the use of different pharmacological interventions for hypertension.       3.6         30. Discuss appropriate use of diet, exercise, and lipid lowering agents in the management of hyperlipidemia.       2.9         31. Discuss the complications associated with hyperlipidemia.       3.3         32. Discuss the condictions associated with hyperlipidemia.       3.0         33. Discuss the condications associated with hyperlipidemia.       3.0         34. Discuss the condications associated with hyperlipidemia.       3.0         35. Discuss the stabilization of critical patient under local, regional, and general anesthetics under an emergent situation.       3.6         35. Discuss the stabilization of critical patient under local, regional, and general anesthetics under an emergent situation.       3.6 <th>23.</th> <th>Describe the use of laboratory tests in the diagnosis and treatment of cardiovascular</th> <th>disease</th>	23.	Describe the use of laboratory tests in the diagnosis and treatment of cardiovascular	disease
<ol> <li>Describe the clinical manifestations of heart failure.</li> <li>Joentify common types of valvular heart disease, such as aortic stenosis, mitral regurgitation, and (rheumatic) mitral stenosis.</li> <li>Explain the roles and pharmacology of commonly prescribed cardiac medications in the treatment of cardiac diseases (eg. digoxin, diuretics, beta-blockers, ACE inhibitors, anti-arrhythmetics, nitrates, anti-coagulants and lipid lowering agents).</li> <li>Define <i>hypertension</i>.</li> <li>Rotionalize the use of different pharmacological interventions for hypertension.</li> <li>Define secondary hypertent pharmacological interventions for hypertension.</li> <li>Describe clinical manifestations and treatments of dyslipidemia.</li> <li>Discuss appropriate use of screening procedures for hyperlipidemia.</li> <li>Discuss the complications associated with hyperlipidemia.</li> <li>Discuss the complications associated with hyperlipidemia.</li> <li>Discuss the stabilization of critical patient in the field and the role of advanced life support.</li> <li>Discuss the standards for monitoring a patient under local, regional, and general anesthetics under an emergent situation.</li> <li>Discuss the valuation and management of shock, including hemorrhagic, hypovolemic, cardiogenic, and neurogenic shock.</li> <li>Discuss the valuation and management of shock, including hemorrhagic, hypovolemic, cardiogenic, and neurogenic shock.</li> <li>Describe the clinical manifestations of pericarditis.</li> <li>Discuss treatment options for a patient with pericarditis.</li> <li>Discuss treatment options for a patient with pericarditis.</li> <li>Describe the clinical manifestations of pericarditis.</li> <li>Describe the signs, symptoms, and treatment for cardia carrest and myocardial infarction.</li> <li>Explain the role of advanced cardiac life support.</li> <li>Describe the clinical manifestations of pericarditis.</li> <li>Describe the clinical man</li></ol>		(eg, cholesterol fractionation, CPK isoenzymes, troponin levels).	3.7
<ul> <li>Identify common types of valvular heart disease, such as aortic stenosis, mitral regurgitation, and (rheumatic) mitral stenosis.</li> <li>Explain the roles and pharmacology of commonly prescribed cardiac medications in the treatment of cardiac diseases (eg. digoxin, diuretics, beta-blockers, ACE inhibitors, anti-arrhythmetics, nitrates, anti-coagulants and lipid lowering agents).</li> <li>Define hypertension.</li> <li>Define hypertension and identify the most common causes.</li> <li>Rationalize the use of different pharmacological interventions for hypertension.</li> <li>Discuss appropriate use of diet, exercise, and lipid lowering agents in the management of hyperlipidemia.</li> <li>Discuss appropriate use of diet, exercise, and lipid lowering agents in the management of hyperlipidemia.</li> <li>Discuss the complications associated with hyperlipidemia.</li> <li>Discuss the complications associated with hyperlipidemia.</li> <li>Discuss the complication of critical patient in the field and the role of advanced life support.</li> <li>Discuss the standards for monitoring a patient under local, regional, and general anesthetics under an emergent situation.</li> <li>Explain the role of advanced cardiac life support.</li> <li>Discuss the evaluation and management of shock, including hemorrhagic, hypovolemic, cardiogenic, and neurogenic shock.</li> <li>Discuss treatment options for a patient with pericardial diseases</li> <li>List potential causes of acute and constrictive pericardial diseases</li> <li>Discuss treatment options for a patient with pericardial diseases</li> <li>List the heart valves most commonly affected by rheumatic fever in decreasing frequency.</li> <li>Describe the clinical manifestations of pericardial diseases</li> <li>List the heart valves most commonly affected by rheumatic fever in decreasing frequency.</li> <li>Describe the clinical manifestations of rheumatic fever in decreasing frequency.</li> <li>Describe the clinical manifestations of rheumatic fever in decreasing frequency.</li></ul>	24.	Describe the clinical manifestations of heart failure.	3.0
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<ul> <li>35. Discuss the stabilization of critical patient in the field and the role of advanced life support.</li> <li>3.0</li> <li>36. Discuss the standards for monitoring a patient under local, regional, and general anesthetics under an emergent situation.</li> <li>3.6</li> <li>37. Describe the signs, symptoms, and treatment for cardiac arrest and myocardial infarction. Explain the role of advanced cardiac life support.</li> <li>3.5</li> <li>38. Discuss the evaluation and management of shock, including hemorrhagic, hypovolemic, cardiogenic, and neurogenic shock.</li> <li>3.4</li> <li>39. Define <i>acute pericarditis</i> and <i>restrictive pericarditis</i>.</li> <li>2.4</li> <li>40. List potential causes of acute and constrictive pericardial diseases</li> <li>2.4</li> <li>42. Describe the clinical presentation and physical findings in a patient with constrictive pericarditis.</li> <li>2.4</li> <li>43. Discuss treatment options for a patient with pericarditis and treatment of potential cardiovascular complications.</li> <li>8. Rheumatic Fever and Endocarditis</li> <li>1. Describe the clinical manifestations of rheumatic fever and its clinical complications with an emphasis on valvular heart disease and endocarditis.</li> <li>3.3</li> <li>2. List the heart valves most commonly affected by rheumatic fever, using "major" and "minor" criteria.</li> <li>2.4</li> <li>4. Describe appropriate treatment and prevention for rheumatic fever.</li> <li>2.8</li> </ul>	34.	Discuss the chain of activation in the EMS system from 911 to ER admission.	3.0
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4. Describe appropriate treatment and prevention for rheumatic fever.       2.8         C. Arterial, Venous, Lymphatic       1. Identify clinical signs and symptoms of venous insufficiency.       4.0         2. Identify notantial complications and recommend appropriate treatment concents of venous		criteria	2.4
<ul> <li>C. Arterial, Venous, Lymphatic</li> <li>1. Identify clinical signs and symptoms of venous insufficiency.</li> <li>4.0</li> </ul>	4	Describe appropriate treatment and prevention for rheumatic fever.	2.8
<ul> <li>C. Arterial, Venous, Lymphatic</li> <li>1. Identify clinical signs and symptoms of venous insufficiency.</li> <li>4.0</li> <li>2. Identify notantial complications and recommend appropriate treatment concents of venous</li> </ul>			
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2. Identify entential complications and recommend appropriate treatment concents of venous	1	Identify clinical signs and symptoms of venous insufficiency	4.0
	2	Identify potential complications and recommend appropriate treatment concents of	venous
insufficiency.		insufficiency.	4.0
3. Identify clinical signs and symptoms of carotid and aortic vascular disease. <b>3.0</b>	3.	Identify clinical signs and symptoms of carotid and aortic vascular disease.	3.0

	4.	Identify potential complications and recommend appropriate treatment concepts of car aortic vascular disease.	otid and <b>3.0</b>
	5.	Discuss the proper work-up and instrumentation contained in the noninvasive vascular <b>4.0</b>	exam.
	6.	Identify the clinical findings and sequelae associated with venous disease.	3.9
	7.	Outline the physical examination of the vascular system.	3.9
	8.	Explain how to perform a focused history and physical, to identify patients with acute ar	nd or
		chronic peripheral vascular disease.	4.0
	9.	Identify laboratory, physiologic, or imaging data that is utilized in identifying acute and c	or
		chronic peripheral vascular disease.	3.9
	10.	Explain the cardinal symptoms and signs of acute and or chronic peripheral vascular dise	ease. 3.9
	11.	Outline the physical examination of the peripheral vascular system.	3.9
	12.	Describe the various tests and techniques to evaluate the patient with peripheral vascul	ar
		disease.	4.0
	13.	Interpret the findings of a complete clinical vascular lower extremities evaluation.	4.0
	14.	Define and discuss lymphedema.	4.0
	15.	Determine, classify, and compare and contrast diabetic versus non-diabetic vascular dise	ease.
			4.0
	16.	Determine, classify, and compare and contrast conservative versus surgical treatments i	n PVD.
			4.0
	17.	Describe the etiology, pathophysiology, differential diagnoses, and complications of dee	р
		venous thrombosis.	4.0
	18.	Describe the clinical findings of DVT, clinical laboratory studies, medical, and surgical tre	atment
		(including the Greenfield filter).	4.0
	19.	Describe the etiologies, differential diagnoses, laboratory studies, and principles of man	agement
		of localized edema.	3.6
	20.	Describe the etiologies, differential diagnoses, laboratory studies, and principles of treat	ment of
		primary and secondary lymphedemas.	3.4
	21.	Explain acute arterial occlusion, including intrinsic and extrinsic etiology, reperfusion, cli	nical
		findings, diagnosis, management, and morbidity/mortality rates.	3.4
	22.	Describe the etiologies, clinical manifestations, and management of blue toe syndrome.	4.0
	23.	Describe the diagnosis, prognosis, surgical workup, and complications of aneurysms.	3.1
	24.	Describe the following variants of cold injury:	
		a. Chilblains (pernio) – mild frostbite	3.6
		b. Trench foot (immersion foot)	3.6
		c. High altitude frostbite	3.5
•	<u>Rh</u>	eumatologic Disorders	
А.	Мy	opathies (Primary, Secondary)	

1.	Describe the clinical features and assessment of myopathies.	3.8
2.	Explain the impact of neurodegenerative diseases on gait function.	4.0
3.	Describe major characteristics and the natural history of specific diseases in each or	gan system.
		2.5
4.	Understand the classification, clinical manifestations, diagnosis, and treatment of	
	neuromuscular diseases.	3.5

IV.

5.	Understand the types of clinical syndromes and treatments associated with muscular dy	strophy.
6	Identify the diagnostic test used for neuromuscular diseases	3.1
7.	Discuss the definition, clinical manifestation, lab findings, and treatment of idiopathic	0.1
	inflammatory myopathies.	3.0
8.	Discuss the types of alcoholic myopathies and associated clinical features.	3.3
9.	Given a set of signs and symptoms, be able to give a differential diagnosis, clinical feature	es, and
	treatment of patients with myopathies.	3.0
10.	List the types of muscular dystrophies.	3.0
11.	List the etiologies, clinical features and treatment of muscular dystrophies.	3.0
12.	Describe the etiology, incidence, pathophysiology, clinical presentation, diagnosis, treat	ment,
12	Describe the incidence etiology nathonhysiology clinical presentation laboratory stud	jec
15.	diagnosis, treatment/management, course, and prognosis of alcohol malnutrition	163,
	polyneuropathy.	3.1
14.	Define Guillian-Barre-Strohl syndrome.	3.3
15.	Define Guillian-Barre-Strohl syndrome, and describe the incidence, clinical findings,	
10	diagnostic studies, course, treatment, and prognosis.	3.0
16.	treatment/management, and progracis of neuropathy in patients with AIDS	2.4
17	Define Charcot Marie Tooth disease (Peropeal Muscular Atrophy)	2.4
17. 18	Describe the etiology nation hysiology incidence clinical presentation laboratory stud	<b>5.0</b>
10.	diagnosis course treatment/management and prognosis of Charcot-Marie-Tooth disea	ISP.
		3.6
19.	Define muscular dystrophies.	3.8
20.	Describe the incidence, postulated etiology, pathophysiology, clinical presentation, diag	nostic
	studies, diagnosis, course treatment/management, and prognosis of Duchenne's muscu	lar
	dystrophy.	2.9
21.	Describe the incidence, postulated etiology, pathophysiology, clinical presentation, diag	nostic
	studies, diagnosis, course treatment/management, and prognosis of Becker's disease.	2.8
22.	Define and describe the etiology, clinical presentation, diagnosis, course, treatment, and	ł
	prognosis of disorders of the neuromuscular junction with emphasis on Myasthenia Gra	vis.
		3.0
23.	Explain the clinical use of seizure drugs.	1.9
24.	Identify the drugs used in the treatment of Parkinsonism.	2.1
25.	Describe mechanisms of action, pharmacodynamics of key other drugs in treatment of	2.0
26	Define arthritidas	2.0
20.	Describe the demographics and historical physical radiographic and laboratory finding	<b>4.0</b>
27.	octeoarthritis RA and seronegative snondyloarthronathies	<b>40</b>
28	Explain the management of osteoarthritis, RA, and seronegative spondyloarthonathies.	4.0
29.	Identify the typical historical, physical, radiographic, and laboratory findings seen in rhe	umatoid
-	arthritis.	3.8
30.	Define ankylosing spondylitis, Reiter's disease, psoriatic arthritis, and enteropathic arthr	opathy.
31.	Describe the demographics and historical, physical, radiographic, and laboratory finding	s of
	ankylosing spondylitis, Reiter's disease, psoriatic arthritis, and enteropathic arthropathy	. 3.4
32.	Explain the management of ankylosing spondylitis, Reiter's disease, psoriatic arthritis, and	nd
	enteropathic arthropathy.	4.0

33.	Define SLE and give examples of other select connective tissue diseases.	4.0
34.	Describe the demographics and historical, physical, radiographic and laboratory findings	of SLE
	and other select connective tissue diseases.	3.8
35.	Explain the management of SLE and other connective tissue diseases.	3.8
36.	Explain how to perform a focused history and physical so as to identify patients with acu	ute and
	or rheumatologic disease.	4.0
37.	Explain the laboratory, physiologic, or imaging data that is utilized in identifying acute a	nd or
	chronic rheumatologic disease.	4.0
38.	Define gout and CPPD.	4.0
39.	Describe the demographics and historical, physical, radiographic and laboratory findings	s of gout
	and CPPD.	4.0
40.	Explain the management of gout and CPPD.	4.0
41.	Analyze the signs and symptoms of connective tissue syndromes such as lupus, sclerode	rma,
	and dermatomyosytis/polymyositis.	3.1
42.	Define <i>fibromyalgia</i> .	3.1
43.	Describe the demographics and historical, physical, radiographic, and laboratory finding	s of
	Fibromyalgia.	3.1
44.	Explain the management of fibromyalgia.	3.1
45.	Analyze the signs and symptoms of and polymyalgia rheumatica (PMR) and Giant Cell An	rteritis.
		3.1
46.	Analyze the signs and symptoms of Bechet's disease.	3.0
47.	Identify the most common infecting organisms responsible for infectious arthritis and the	neir risk
	factors.	3.4
48.	Describe the pathology and pathogenesis of systemic lupus erythematosus (Lupus, SLE).	2.3
49.	Describe the etiology, clinical presentation, differential diagnoses, studies, diagnosis, tre	atment,
	and complications of discoid lupus erythematosus	2.3
50.	Describe the etiology, clinical presentation, differential diagnoses, studies, diagnosis, tre	eatment,
	and complications of systemic lupus erythematosus	2.4
51.	Describe the etiology clinical presentation, differential diagnoses studies, diagnosis trea	tment,
	and complications of Systemic Sclerosis	3.0
52.	Describe the etiology clinical presentation, differential diagnoses studies, diagnosis trea	tment,
	and complications of Localized Linear Scieroderma.	2.3
53.	Describe the pathology and pathogenesis of limited cutaneous systemic sclerosis.	2.4
54.	Describe the pathology and pathogenesis of progressive systemic sclerosis.	2.3
55.	Describe the etiology clinical presentation, differential diagnoses studies, diagnosis trea	tment,
- 6	and complications of Sjogren's disease.	3.0
56.	Describe the etiology clinical presentation, differential diagnoses studies, diagnosis trea	tment,
	and complications of Lyme disease.	3.1
57.	Describe the etiology, clinical presentation, differential diagnoses, studies, diagnosis, tre	eatment,
<b>F</b> 0	and complications of dermatomyositis.	2.3
58.	Describe the etiology, clinical presentation, differential diagnoses, studies, diagnosis, tre	eatment,
F.0	and complications of kneumatold Arthritis and its directly related syndromes.	<b>3.</b> 8
59.	Describe the etiology, clinical presentation, differential diagnoses, studies, diagnosis, tre	atment,
60	and complications of vasculitis.	<b>3.</b> b
60.	Describe the etiology, clinical presentation, differential diagnoses, studies, diagnosis, tre	eatment,
	and complications of Slogren's Syndrome.	2.3

## V. <u>Metabolic and Endocrine Disorders</u>

### A. Diabetes

1 2	•	Identify the types of diabetes mellitus. Describe clinical presentations of diabetes mellitus.	4.0 4.0
3		Outline the diagnostic process of interpretation of laboratory testing in diabetes mellitu	s.
-		······································	4.0
4		Discuss diabetic emergencies involving ketoacidosis, hypoglycemia, and hyperglycemia.	
			3.6
5		Explain the basis for diabetic management in the following setting: outpatient, inpatient	t, and
		perioperative clinical scenarios.	3.4
6	•	Describe indications and contraindications for oral hypoglycemic and insulin therapies.	3.5
7	•	Recommend a monitoring program for diabetes mellitus using current guidelines.	3.6
8	•	Describe manifestations of the lower extremity as it relates to a clinical correlation of the	e
		endocrine system.	4.0
9	•	Describe the metabolic response to anesthesia and surgery of the diabetic patient.	4.0
1	0.	Explain medical management of serum glucose in the perioperative period.	4.0
1	1.	Identify most effective preventative and treatment strategies for diabetes.	3.9
1	2.	Explain the laboratory, physiologic, or imaging data that is utilized in identifying diabete	S
		mellitus.	4.0
1	3.	Explain how acute and or chronic diabetes mellitus affects and interact with clinical pod	iatric
		problems such as perioperative assessment as well as podiatric medicine issues.	4.0
1	4.	Write insulin orders for the perisurgical diabetic patient.	4.0
1	5.	Discuss microvascular and macrovascular complications of diabetes mellitus.	4.0
1	6.	Define microalbuminuria and macroalbuminuria.	3.9
1	7.	Discuss the pathogenesis, treatment, and prevention of nephropathy.	3.5
1	8.	Discuss the pathogenesis and resulting effects of peripheral neuropathy.	3.9
1	9.	Discuss the clinical features and management of diabetic foot infections.	4.0
2	0.	Compare and contrast soft tissue infections in diabetics versus non-diabetics.	4.0
2	1.	Discuss clinical features of diabetic muscle infarction.	4.0
2	2.	Define Fornier's Gangrene.	3.0
2	3.	Define aneorobic cellulitis.	3.5
2	4.	Define pyomyositis.	2.9
2	5.	Discuss the clinical features of acute pyelonephritis in diabetics.	3.0
2	b.	Discuss the clinical features of renal corticomedullary abscesses in diabetics.	2.8
2	./.	Discuss the clinical features of renal carbuncle in diabetics.	2.1
2	8.	Discuss the clinical features of perinephric abscess in diabetics.	2.1
В.	Go	ut	
	1.	Describe the etiology clinical presentation, differential diagnoses studies, diagnosis trea	tment.
		and complications of gout.	4.0

- Describe the etiology clinical presentation, differential diagnoses studies, diagnosis treatment, and complications of CPPD.
   4.0
- 3. Describe radiographic signs associated with acute and chronic gout. 4.0
- 4. Describe the clinical presentation, radiographic signs, and pathophysiology associated with Calcium Pyrophosphate Deposition Disease (CPDD).
   4.0
- 5. Differentiate crystal induced monoarticular disease from osteoarthritic disease. **4.0**
- 6. Explain management of the crystal-induced synovitis over the long-term emphasizing newer pharmacological agents from the perspective of a practicing rheumatologist. **3.9**

7. Outline treatment options and preventive measures of each disease state (including screening).

### C. Adrenal and Pituitary

1	. Describe, diagnosis, clinical manifestations, and laboratory abnormalities in patients w	ith
	adrenal dysfunction.	3.0
2	. Identify perioperative issues in patients with adrenal dysfunction.	3.0
3	. Describe diagnosis, clinical manifestations, and laboratory abnormalities in patients wi	th
	pituitary disease.	3.0
4	. Describe perioperative issues in patients with pituitary disease.	3.0
5	. Describe diagnosis, clinical manifestations, and laboratory abnormalities in patients wi	th thyroid
	dysfunction.	3.0
6	. Recognize perioperative issues in patients with thyroid dysfunction.	3.0
7	. Describe diagnosis, clinical manifestations, and laboratory abnormalities in patients wi	th
	parathyroid dysfunction.	3.0
8	. Identify perioperative issues in patients with parathyroid dysfunction.	3.0
9	. List the laboratory evaluation of patients with Addison's disease and pheochromocyto	ma.
		2.3
1	0. Explain the clinical and lab findings, differential diagnosis, treatment complications and	k
	prognosis of Pheochromocytoma.	1.9
7	burgid and Darathurgid	
'		
1	. Correlate the clinical picture seen with hyposecretion and with hypersecretion of each	hormone
	to the physiological effects of each hormone.	2.8
2	. Describe the basic work-up for a patient presenting with thyroid nodules.	1.8
3	. Identify the more common types of thyroid cancer.	1.6
4	. Explain the complications and treatment options for thyroid cancer.	1.6
5	. Describe a reasonable management plan for a patient with hyper/hypothyroidism.	2.4
6	. Discuss the etiology, clinical signs and symptoms, and treatment of the common cause	of
	hypercalcemia and hypocalcemia, with emphasis on the disorders of the parathyroid g	land.
		2.8
7	. Explain the pathophysiology of the hypo- and hyperparathyroid disease.	
		2.8

- 8. Identify major clinical signs of each disease state.
  9. Identify and recommend management of specific complications of the disease.
  10. Outline treatment options and preventive measures of each disease state.
  11. Identify the diagnoses of major thyroid disorders
  12. Describe the diagnosis and treatment of hypothyroidism and hyperthyroidism.
  13. Describe the diagnosis and treatment of hyperparathyroidism and hypoparathyroidism.
  2.4
- 14. Identify the pathophysiologies of diseases affecting the adrenal cortex and the parathyroid glands. 2.4

### E. Renal

D.

1.	Identify types of tumors of the urinary bladder, prostate and kidney.	1.3
2.	Explain laboratory diagnostic parameters to make a diagnosis of the tumor of the kidney	y and
	urinary bladder.	1.3
3.	Describe classical manifestation presented by the patient with tumor of the kidney and	urinary
	bladder.	1.4

4.	Identify components of a urinalysis.	3.4
5.	Explain the implications of the results of a urinalysis.	3.5
6.	Identify clinical manifestations and etiology of nephrolithiasis.	4.0
7.	Describe lower extremity surgical outcomes in this patient population.	4.0
8.	Explain the clinical impact of end-stage renal disease on the lower extremity, including	lower
	extremity surgical outcomes.	4.0
9.	Describe complications associated with administering antibiotics to patients with renal	disease.
		4.0
10	. Identify laboratory tests and interpret the results for bacteriuria.	2.9
11	. Describe the epidemiology and organisms of complications of bacteriuria.	2.6
12	. Explain diagnosis and perioperative management of podiatric patients with renal disea	se.
		3.6
13	. Explain how to perform a focused history and physical to identify patients with acute a	nd or
	chronic renal disease.	3.4
14	. Describe the laboratory and physiologic or imaging data that is of value in identifying a	cute and
	or chronic renal disease.	3.0
15	. Explain the cardinal symptoms and signs of acute and or chronic renal disease.	3.0
16	. Identify and evaluate appropriate laboratory, physiologic, and imaging data specific for	the
	diagnosis of acute and or chronic renal disease.	2.6
17	. Explain how acute and/or chronic renal disease states affect and interact with clinical p	odiatric
	problems, such as perioperative assessment as well as podiatric medicine issues.	3.7
18	. Explain how to perform a focused history and physical to identify patients with fluid an	d
	electrolyte disorders.	3.5
19	. Describe the laboratory, physiologic, or imaging data that is of value in identifying acut	e and or
	chronic fluid and electrolyte disorders.	3.0
20	. Explain the cardinal symptoms and signs of acute and/or chronic fluid and electrolyte d	lisorders.
		3.4
21	. Identify and evaluate appropriate laboratory, physiologic or imaging data specific for the	ne
	diagnosis of acute and or chronic fluid & electrolyte disorders.	2.6
22	. Explain how acute and/or chronic fluid and electrolyte disorders affect and interact wit	h clinical
	podiatric problems such as perioperative assessment as well as podiatric medicine issu	es.
		3.3
Bo	ne	
20		
1.	Identify the basic radiographic changes that accompany benign and malignant bone tu	mors.
		4.0
2.	Describe the basic characteristics of the more common bone tumors.	4.0
3.	Describe the pathophysiology of the bone diseases.	3.9
Λ	Describe metabolic hone disease including types nathology appropriate tests and tre	atment

- Describe metabolic bone disease, including types, pathology, appropriate tests, and treatment.
   4.0
- 5. Explain the causes and mechanisms for osteoporosis and osteomalacia. **4.0**

### VI. Hematologic Disorders, Including Anemias and Leukemia

F.

- Identify the role of cyanocobalamin and folate in the maturation of the red cell, and understand the implications of a deficiency of either.
   2.3
- 2. Identify clinical implications of red blood cell, WBC and platelet abnormalities. **3.5**

3.	Identify a differential diagnosis for a case of thrombocytopenia, given a clinical scenario	o. <b>3.5</b>
4.	Discuss the factors that lead to "pathologic" thrombosis.	4.0
5.	Identify a differential diagnosis of a young person with DVT / PE.	4.0
6.	Identify the inherited causes of thrombophilia.	2.3
7.	Discuss the effects and diet recommendations relating to nutritional disorders or medi	cations
	which affect the clotting factors of blood and bone density.	2.5
8.	Discuss the risks and benefits of transfusion therapy.	3.0
9.	Discuss the consequences of an ABO mismatch.	3.1
10.	Describe the clinical presentation of solid and hematologic malignancies, and discuss m	norbidity
	and mortality of the various solid and hematologic malignancies.	2.3
11.	Explain the kinetics of iron absorption, transport and storage and understand the diffe	rence
	between transferrin, ferritin, and hemosiderin.	2.0
12.	Discuss the causes of B12 deficiency, folate deficiency, and iron deficiency.	2.5
13.	Discuss the diagnosis of "anemia of chronic disease", the role of cytokines, the inflamm	natory
	disorders in which it is seen, how the diagnosis is made, and the characteristic iron dyn	amics.
		3.0
14.	Discuss the differential diagnosis and diagnostic work up, given a clinical case of normo	ocytic,
	microcytic or macrocytic anemia.	3.1
15.	Discuss how blood plasma and blood cells are affected by alterations in the availability	of
	nutritional factors.	2.1
16.	Describe the abnormalities of these structures, which occur in patients with hereditary	,
	spherocytosis and hereditary elliptocytosis.	1.4
17.	Compare and contrast "intrinsic" and "extrinsic" causes of hemolytic anemia.	2.3
18.	Identify and contrast the effects of red cell lysis that occurs within the circulation versu	IS
	hemolysis that occurs outside of the circulation. Describe the laboratory abnormalities	that
	would occur with predominantly intravascular hemolysis and predominantly extravasc	ular
4.0	nemolysis.	1.9
19.	identify the morphologic characteristics of different types of hemolytic anemia, includi	ng
	spherocytosis, schistocytosis, nemogiobinopathies, liver disease, renai disease, and pyi	ruvate
20	Kinase deficiency.	1.5 uding that
20.	compare and contrast different types of drug-induced immune hemolytic anemia, inclu-	uuing that
	reaction associated with high doces of intravenous penicillin	20
21	Pelate the structure of hemoglohin to the physiologic effect of ovugen carrying capacit	<b>5.0</b>
21.	ability to carry carbon dioxide	2 G
22	Explain substitutions in the globin chains that produce	2.0
22.	a nolymerizing hemoglohins.	13
	h congenital methemoglohinemia	1.3
	c. congenital polycythemia: and	2.0
	d. hemolytic anemia (the unstable hemoglobins).	2.3
23.	Explain the clinical manifestations of patients with sickle cell disease.	3.0
24.	Explain the perioperative management of patients with sickle cell disease.	3.0
25.	Identify the risks and benefits of narcotic pain medication in patients with sickle cell dis	sease. <b>2.5</b>
26.	Explain the clinical manifestations of patients with sickle cell disease.	2.6
27.	Compare and contrast the biochemical abnormalities in alpha and beta thalassemia.	1.9
28.	Compare and contrast the clinical manifestations of alpha and beta thalassemia.	1.6
29.	Compare and contrast the laboratory diagnoses of alpha and beta thalassemia.	1.4

30.	Discus: patient	s the clinical manifestations of leukemia and lymphoma and implications for t	he podiatric <b>3.0</b>
31.	Discus	the consequences of staging a malignancy and how it may affect therapy.	2.2
32.	Discus	s the goals of a lymphoma classification system and the basic organization of	the WHO
	classifi	cation of lymphoid malignancies.	1.3
33.	Descrit	be the clinical features and most likely lymphocyte markers of	
	a.	classic Hodgkin's disease;	1.6
	b.	chronic lymphocytic leukemia;	1.6
	с.	follicular lymphomas;	1.5
	d.	hairy cell leukemia;	1.5
	e.	burkitt's lymphoma;	1.5
	f.	adult T-cell leukemia/lymphoma; and	1.5
	g.	diffuse large B cell lymphoma.	1.5
34.	Define he	emostasis and discuss the role of the vessel, the platelet, and the plasma prot	eins, as well
	as the na	tural anticoagulants and fibrinolytics in normal hemostasis.	3.4
35.	Describe	the clinical and laboratory significance of PT, PTT, TT, bleeding time, INR, and	l mixing
	study.		4.0

# VII. Immunologic Disorders (Allergic and Sensitivity Reactions and Immunosuppressive States)

1.	Define antigen, antiboay, and immunoglobulin.	3.9
2.	Describe the structure and function of the following for immunoglobulins:	
	a. classes of immunoglobulins	3.1
	b. four-chain basic unit	2.1
	c. fragments of immunoglobulins	2.7
	d. fc fragment	1.8
	e. fab fragment	1.8
	f. fd fragment	1.8
3.	Describe antigen-antibody Immunofluorescence reactions of:	
	a. direct technique; and	1.9
	b. indirect technique.	1.9
4.	Describe the complement system, including classical and alternate complement pa	athway, and
	explain the biological significance of the complement system.	2.0
5.	Describe the following cells involved in and their role in the immune response:	
	a. neutrophils	3.1
	b. monocytes-macrophages	3.1
	c. lymphocytes	3.1
	d. T-lymphocytes (T cell)	3.0
	e. B-lymphocyte (B cell)	3.0
	f. basophiles and mast cells	3.1
	g. eosinophiles	3.1
6.	Define <i>allergy</i> .	3.0
7.	Describe allergies in terms of classification, clinical manifestations, complications,	and
	treatment.	3.0
8.	Define hypersensitivity and identify and describe the two major types.	2.9
9.	Describe the types of allergic diseases (reactions) according to classification of Gel	l and Coombs
	and types (I, II, III, and IV).	1.6

10. Describe the following Type I allergic reactions:	
a. reaginic antibodies	2.1
b. allergen	1.8
c. tissue cells	1.8
d. basophils and mast cells	1.8
e. pharmacologically active amines in man	1.5
f. histamine	1.8
g. slow reacting substance of anaphylaxis (SRS-A)	1.8
h. eosinophil chemotactic factor of anaphylaxis (ECF-A)	1.8
i. serotonin	1.8
11. Describe the following Type IV Reactions:	
a. clinical implications of transplant biology	3.0
b. tumor antigen induced	3.0
c. response to infectious agents	2.1
12. Describe the role of the EB virus and immunosuppression in the development	of lymphoma.
	1.6
13. Explain the clinical presentation of solid and hematologic malignancies, and dis	scuss morbidity
and mortality of the various solid and hematologic malignancies.	3.0
14. Discuss the morphologic findings in the peripheral blood and marrow of a patient	ent with multiple
myeloma.	2.4
15. Discuss the complications of Waldenström's macroglobulinemia and hypervisc	osity syndrome.
	2.1
16. Discuss myeloproliferative disorders, including current classification.	1.8
17. Evaluate a patient with neutrophilia and recommend the proper work-up to di	ifferentiate a
reactive leukocytosis, chronic myelogenous leukemia and leukemoid reaction.	2.4
18. Evaluate thrombocytosis and understand the clinical significance of this finding	g. <b>2.5</b>
19. Discuss the current concept and classifications of myelodysplastic syndromes.	Z.U
20. Formulate a differential diagnosis for a patient with cytopenia and suggest app	
10 help commin a diagnosis.	2.0
21. Discuss the significance of blacts in the peripheral blood, he able to formulate	a differential
diagnosis and define an Auer rod	a unierentiai <b>2 1</b>
22 Discuss the clinical and laboratory findings in acute lymphoblastic (ALL) and ac	ute myeloblastic
	1 8
24 Discuss hone marrow transplant indications and protocol	1.0
25. Compare and contrast the nationhysiology treatment and prognosis of ALL a	nd AMI 16
23. compare and contrast the pathophysiology, treatment and prognosis of ALL a	
Respiratory Disorders (Including Asthma Emphysema Infectious Proumo	nitic)
הפקורמנטו אי שושטימבוש (ווונוממווה אשנוווומ, בוווטוואשבוומ, וווובננוטמש דוובמווט	11(15)

1.	Discuss clinical manifestations and treatment of chronic bronchitis and emphysema and Cystic	
	Fibrosis.	3.3
2.	Identify risk factors leading to COPD.	3.0
3.	Discuss the epidemiology of COPD.	2.8
4.	Compare and contrast chronic bronchitis and emphysema.	2.8
5.	Identify symptoms and physical exam findings associated with chronic bronchitis and	
	emphysema.	2.9
6.	Interpret radiographic findings associated with COPD.	2.5

VIII.

7.	Discuss the prognosis of COPD.	2.6
8.	Identify general principles used in the primary care treatment of COPD.	2.6
9.	Identify preventive strategies for reducing the morbidity and mortality of COPD.	2.6
10.	Discuss pitfalls in the diagnosis of pneumonia.	2.9
11.	Identify the populations most at risk for the following types of pneumonia:	
	a. <i>S. pneumoniae</i> (pneumococcus)	2.8
	b. mycoplasma pneumoniae	2.8
	<b>c.</b> influenza	2.8
	d. gram negative bacilli	2.8
	e. legionella pneumonia	2.5
	f. viral	2.5
12.	Discuss treatment approaches for pneumonias.	3.0
13.	Describe the pathogenesis of cystic fibrosis and the most common resulting complication	ns.
_0.		1.9
14	Identify presenting signs and symptoms of CE	1.8
15	Describe treatment of CE in general terms	1.8
16	Identify risk factors for DVT / PF	4.0
17	Discuss preventive measures to reduce the risk of DVT	4.0
12	Identify the most common area of the venous system contributing to venous thromboe	mholism
10.	identity the most common area of the venous system contributing to venous thromboe	<b>1 1 1</b>
10	Identify strengths and weaknesses of DVT diagnostic modalities	4.0
19. 20	Describe the signs and symptoms that suggest PE	4.0
20.	Identify the laboratory tests appropriate for the diagnosis of DE	4.0
21.	Define the role of Imaging used in the diagnoses of DE	4.0
22.	Identify a V/O mismatch consistent with DE	5.I 2.1
23.	Discuss the mainstaux of treatment for an equite DE	3.1 2.5
24.	Discuss the mainstays of treatment for an acute PE.	3.5
25.	Describe SIADH and other Para-neoplastic syndromes associated with Lung Cancer	3.0
20.	Describe the epidemiology of lung cancer.	2.3
27.	identify the four most common types of lung cancers and describe characteristic feature	es or
20	each.	1.6
28.	Describe ventilation in terms of PaCO2, common stimuli to breatne, and components ne	cessary
20	to maintain ventilation.	2.9
29.	Describe the signs and symptoms associated with lung cancer.	2.8
30.	Describe the staging of lung cancer.	1.0
31.	Discuss the prognosis of lung cancer.	2.0
32.	Discuss most common sites for metastases of lung cancer and possible presenting signs	and
	symptoms of each.	2.0
33.	Describe the following syndromes according to etiology, symptoms, signs, diagnosis and	1
	treatment:	
	a. common cold/influenza/URI/laryngitis/epiglottitis	2.5
	b. otitis media and externa	2.1
	c. acute and chronic sinusitis	2.1
	d. acute bronchitis	2.5
	e. pleurisy	2.1
34.	Describe the process of assessing the patency of an airway and its associated airway	
	classification systems.	3.0
35.	Discuss the common supplies and techniques in managing the airway in an emergent sit	uation.
		3.0

36.	Describe how ventilation-perfusion mismatch and altered alveolar gas diffusion rate magas exchange.	y affect <b>2.1</b>
37.	Explain the importance of hemoglobin to oxygen delivery and the relationship between oxygen content, hemoglobin saturation, and PaO2.	blood <b>3.5</b>
38.	Describe how pulse oximetry can help evaluate gas exchange and discuss the use in a ch setting.	nemical <b>3.3</b>
39.	Describe how each of the components of the arterial blood gas report relates to the pat	ient's
	status.	3.0
40.	Describe the PFT abnormalities in relation to obstructive and restrictive lung diseases.	3.0
41.	Describe the PFT abnormalities characteristic of restrictive lung disease.	2.9
42.	Compare the use of spirometry and peak flow meters for diagnosing and monitoring ast	:hma,
	including the pros and cons of each.	2.6
43.	Identify an underlying disorder as obstructive or restrictive, given a case presentation a	nd
	pulmonary function study results.	3.0
44.	Discuss the clinical implications for management of the patient with lung disease.	3.0
45.	Describe diagnosis, clinical manifestations, and laboratory abnormalities in patients with	h
	interstitial lung disease.	3.0
46.	Describe perioperative considerations for patients with interstitial lung disease, including	ng
	<ul> <li>primary lung disease, ie, sarcoidosis, idiopathic pulmonary fibrosis;</li> </ul>	2.1
	b. environmental/occupational exposures, ie, asbestosis and hypersensitivity pneu	umonitis
	(including Farmer's lung);	2.0
	c. interstitial lung disease secondary to systemic medical diseases, (eg, connective	tissue
	diseases); and	2.1
	d. drug-induced.	2.1
47.	Describe the clinical presentation of interstitial airway disease.	2.1
48.	Discuss the evaluation of a patient with suspected interstitial airway disease.	2.1
49.	Discuss monitoring and treatment strategies for patients with interstitial lung disease.	2.1
50.	Define <i>asthma</i> and discuss the change of the definition in recent years.	3.0
51.	Describe the epidemiolgy of asthma.	2.3
52.	Discuss the pathophysiology of asthma.	2.5
53.	Describe the clinical presentation of asthma, including the different asthma patterns, su	ich as
	exercise-induced, episodic, chronic, virus-induced, and allergic.	2.8
54.	Describe physical exam findings suggestive of asthma.	3.3
55.	Discuss the use of spirometry and peak flow measurements in the diagnosis and manag	ement of
<b>F</b> C	asthma.	2.3
56.	Identify the diagnostic criteria for status asthmaticus.	2.5
57. E0	Define usual respiratory junute.	2.9
50. E0	Discuss the approach to management of nations, with acute respiratory failure.	2.3
59. 60	Define APDS	2.0
00.		2.9

## IX. <u>Behavioral Medicine (Depression, Abuse, Anger Disorders, and Noncompliant Patients)</u>

1.	Identify and describe the major signs and associated symptoms of common psychiatric	
	disorders.	3.0
2.	Discuss clinical features, course, prognosis, treatment, and mental status findings in the	
	psychoses and personality disorders.	3.0

	3.	Discuss the treatment of the psychoses and personality disorders.	1.3	
A. Substance Abuse				
	1.	Describe etiologies, comorbidities, clinical features, and treatment plans for patient wit	h	
		substance dependence and abuse.	2.5	
	2.	Define codependence.	2.4	
	3.	List the etiologies of substance abuse.	2.1	
	4.	List the common comorbidities associated with substance abuse.	2.1	
	5.	Diagnose and recommend a treatment plan for a patient, given a history and clinical fea	itures of	
		substance abuse.	1.5	
	6.	List epidemiological features of alcohol related disorders.	2.3	
	7.	Discuss the effects of alcohol including absorption, metabolism, on brain, behavior, and	sleep.	
			2.3	
	8.	Compare and contrast dependence versus abuse.	2.4	
	9.	Discuss affects and side effects of withdrawal of alcohol.	2.6	
	10.	Discuss neurological effects of alcohol (alcohol-induced persisting amnestic disorder).	2.6	
	11.	Discuss features of Wernicke-Korsakoff syndrome.	2.0	
	12.	Discuss treatment of Wernicke-Korsakoff syndrome.	2.0	
	13.	Discuss alcohol-induced psychotic disorder.	2.0	
	14. Discuss alcohol-induced mood disorder and anxiety disorder.			
	15. Discuss treatment and rehabilitation of alcohol-related disorders.			
	16. Explain the basic anatomy of pain perception.			
	17. Differentiate between acute and chronic pain.			
	18.	Explain the pharmacology and physiology of pain control.	3.0	
	19.	Explain the emotional and psychologial impact of chronic pain.	2.9	
В.	Alt	ered Mental Status		
	1.	Differentiate between delirium, dementia, and depression.	2.4	
	2.	Discuss the pathophysiology, signs, and symptoms of the most common and serious cau altered mental status:	uses of	
		a. Metabolic causes	2.4	
		b. Structural lesions	2.4	
		c. Vascular	2.4	
		d. Infectious etiologies	2.4	
		e. Seizures	2.8	
		f. Hypertensive encephalopathy	2.4	
		g. Low perfusion states	2.0	
	3.	Describe the risk factors for developing altered mental status.	2.6	
	4.	Explain the diagnostic evaluation of altered mental status.	2.3	
	5.	Describe indications, contraindications, and complications of lumbar puncture.	2.0	
	6.	Explain principles of management of the common causes of altered mental status.	2.1	
	7.	Describe nonpharmacologic measures to reduce agitation and aggression.	2.1	
	8.	Identify key drugs used in the treatment of depression including knowledge of mechani	sm of	
		action and pharmacokinetics and pharmacodynamics.	3.0	
	9.	Identify key drugs used in the treatment of bipolar mania including knowledge of mecha	anism of	
		action and pharmacokinetics and pharmacodynamics.	2.1	
	10.	Identify symptoms of substance abuse and substance dependence.	3.1	

11.	11. Identify diagnostic assessment tools, rehabilitation and treatment programs for substance		
	dependence.	2.4	
12.	Describe the podiatrist's role and obligations in dependent adult, child abuse and negle	ct. <b>4.0</b>	
13.	Identify signs and symptoms of dependent adult abuse.	3.3	
14.	Identify signs and associated symptoms of child abuse and neglect.	3.3	
15.	Identify guiding principles governing physicians' actions of end-of-life care.	3.0	
16.	Explain the concept of patient autonomy and its implications in caring for dying patients	5. <b>3.0</b>	
17.	Outline the Kübler-Ross stages of dying.	3.0	
18.	List the components of the mental status exam.	3.9	
19.	Discuss the sections of the mental status exam.	2.9	
20.	Discuss classifications, signs, symptoms, differential diagnosis, and treatment of panic d	isorders.	
		1.9	
21.	Discuss signs and symptoms of anxiety.	3.0	
22.	Define OCD, Agoraphobia, and social phobia	3.0	
23.	Explain post-traumatic stress disorder (PTSD).	2.0	
24.	Discuss etiology, course, prognosis, and treatment of PTSD.	2.0	
25.	Explain the classification phobias.	1.3	
26.	Discuss etiology, diagnosis, clinical features, course, prognosis, and treatment of agorage	hobia.	
		1.0	
27.	Discuss etiology, diagnosis, clinical features, course, prognosis, and treatment of social	phobias.	
		1.0	
28.	Discuss etiology, diagnosis, clinical features, course, prognosis, and treatment of specifi	С	
	phobias.	1.0	
29.	Diagnose and recommend a treatment plan for a patient, given a history and clinical fea	itures of	
	anxiety disorders, PTSD and phobias.	1.0	
30.	List the epidemiology of disorders of mood.	1.1	
31.	Discuss the differential diagnosis of disorders of mood.	1.5	
32.	Discuss the diagnosis of disorders of mood.	1.5	
33.	Discuss clinical features and mental status findings of disorders of mood.	1.5	
34.	Discuss the treatment of disorders of mood.	1.4	
35.	List the epidemiology of schizophrenia, schizophreniform disorder, schizoaffective disor	der, and	
	delusional disorder.	1.4	
36.	List the etiologies of schizophrenia, schizophreniform disorder, schizoaffective disorder,	and	
	delusional disorder.	1.5	
37.	Compare and contrast etiologies of schizophrenia, schizophreniform disorder, schizoaff	ective	
	disorder, and delusional disorder.	1.3	
38.	Discuss the differential diagnosis of schizophrenia, schizophreniform disorder, schizoaff	ective	
	disorder, and delusional disorder.	1.3	

### X. <u>Emergency Medicine (Medical/Surgical)</u>

1.	List the components of medical history and physical examination necessary for the trea	tment of
	the emergency patient.	3.5

- Discuss symptoms and signs of chest pain due to an acute coronary syndrome such as unstable angina or acute myocardial infarction.
   4.0
- 3. Discuss symptoms and signs of chest pain that are characteristic of angina pectoris. **4.0**
- 4. Differentiate the signs and symptoms of cardiac versus noncardiac chest pain, including

	a. atypical or variant angina (coronary vasospasm, Prinzmetal angina);	2.4
	b. cocaine-induced chest pain;	2.3
	c. pericarditis;	2.4
	d. aortic dissection ;	2.6
	e. valvular heart disease (aortic stenosis, mitral valve prolapse);	2.6
	f. nonischemic cardiomyopathy; and	2.6
	g. Syndrome X.	2.5
5.	Discuss symptoms and signs of chest pain due to gastrointestinal disorders, includir	ng
	esophageal disease (GERD, esophagitis, and esophageal dysmotility), biliary diease	(cholecystitis
	and cholangitis), peptic ulcer disease, and pancreatitis.	2.9
6.	Discuss symptoms and signs of chest pain due to musculoskeletal causes, including	
	costochondritis, rib fracture, myofascial pain syndromes, muscular strain, and herp	es zoster.
_		3.0
7.	Discuss symptoms and signs of chest pain due to psychogenic causes, including son	natoform
_	disorders.	2.0
8.	Discuss symptoms and signs of chest pain due to psychogenic causes, including pan	ic disorders
_	and hyperventilation.	2.0
9.	Identify the diagnostic discrimination between common causes of abdominal pain l	based on
	history, physical exam, laboratory testing, and imaging procedures.	2.1
10.	Discuss symptoms and signs indicative of an acute/surgical abdomen.	3.3
11.	Describe the physiology of the acute febrile response, including the beneficial and o	detrimental
	effects of fever in a host, as well as the clinical manifestations of immunocompeter	it and
	immunocompromised patients.	2.4
12.	Identify risk factors and comorbidities that are important in determining the host re	esponse to
	infection.	3.0
13.	Discuss the etiology of fever in the following special populations:	
	a. neutropenia due to cancer-related myelosuppression	2.4
	b. HIV disease	2.4
	c. Intravenous drug abuse	2.4
	d. recent international travel or immigration	2.0
	e. concomitant skin rash and lymphadenopathy	2.4
14.	Discuss the clinical manifestations, lab findings, and treatment of patients with sep	SIS
	syndromes.	3.0
15.	Describe symptoms and signs of DVT and PE.	4.0
16.	Discuss the differential diagnosis of DVI.	3.8
17.	Discuss venous stasis, and the postphiebtic syndrome, lymphedema, cellulitis, supe	rficial
	thrombophlebitis, ruptured popliteal cysts, musculoskeletal injury, and arterial occ	usive
	disorders as causes of unilateral leg pain and swelling.	3.8
18.	Explain treatment modalities for DVI/PE, including, unfractionated heparin, low-m	olecular-
4.0	weight heparin, warfin, and thrombolytics.	2.6
19.	Describe the differential diagnosis of acute back pain.	2.4
20.	Recommend the diagnostic studies and treatment of the following:	
	a. ligamentous/muscle strain (nonspecific musculoskeletal back pain)	2.3
	<ul> <li>degenerative arthritis (spondylosis)</li> </ul>	2.1
	c. disc nerniation	3.0
	a. spinal stenosis	3.0
	e. vertebrai compression tracture	2.4
	t. traumatic fracture	2.9

	g.	sacroileitis	2.6
	h.	spinal metastases	2.3
	i.	spinal epidural abscess	2.4
	j.	cauda eguina syndrome	2.4
21.	Explain	the role of the following diagnostic studies in the evaluation of the back pain, inc	cluding
	indicati	ions, limitations, and cost:	-
	a.	plain radiography	2.4
	b.	CT/ MRI	2.4
	с.	myelogram	1.6
	d.	electrodiagnosis	2.9
	e.	bone densitometry	3.0
22.	Describ	e response of back pain to	
	a.	bed rest;	2.7
	b.	exercise;	2.7
	с.	analgesia;	2.7
	d.	NSAIDs;	2.7
	e.	heat/ice;	2.7
	f.	ultrasound;	2.7
	g.	spinal manipulation; and	2.3
	h.	surgical interventions.	2.4
23.	Describ	e the clinical manifestations, treatments, differential diagnosis, pathophysiology	, and
	typical	presentations of the cutaneous manifestations of syphilis, disseminated gonorrhe	ea
	infectio	on, human papilloma virus, and herpes simplex virus.	2.4
24.	Explain	the role of the emergency department the health care system.	3.6
25.	Explain	the role of advanced cardiac life support.	3.6
26.	List the	common types of cardiac arrhythmias, clinical manifestations, symptoms, and	
	treatm	ents.	3.4
27.	Explain	the methods of establishing an airway and intravenous line.	3.3
28.	List the	signs and symptoms associated with each common cardiac arrhythmias.	
			2.9
29.	Describ	be the signs and symptoms of acute asthma, pulmonary embolus, and pneumothe	orax.
			3.7
30.	List the	types of hypertensive emergencies, and describe their symptoms.	3.0
31.	Explain	the situations in which blood pressure lowering is urgent.	3.0
32.	Explain	the evaluation and management of coma patients.	1.6
33.	Explain	the emergency management of gunshot wounds, severe lacerations, and crush i	njuries.
<u>.</u> .	<b>.</b> .		3.0
34.	Discuss	the evaluation and management of severe head injuries.	2.5
35.	Explain	the evaluation and management of animal and human bites.	3.5
36.	Explain	the examination and management of comatose patients.	1.9
37.	Discuss	the etiology, signs and symptoms, and the treatment of syncope.	3.6
38.	Explain	the evaluation and management of seizure patients.	3.5
39.	List the	etiology, signs, symptoms, and treatment for anaphylaxis.	3.9
40.	Explain	the indications for tetanus immunoprophylaxis.	3.9
41.	Describ	be the pathophysiology of thermal injuries, including systemic manifestations, and	d clinical
	manag	ement.	3.0
42.	Explain	the practical management of office emergency procedures.	4.0
43.	Explain	anaphylaxis and list the main mediators.	3.3

44.	Identify the most common allergens in allergy and anaphylaxis.	3.0
45.	Identify various sources of mast cell degranulation and mediator release.	1.9
46.	Discuss history questions useful in diagnosing a patient with allergies.	3.0
47.	List the indications and contraindications for immunotherapy.	1.4

48. Discuss the diagnosis and treatment of food allergies. **1.0** 

## XI. <u>Dermatology</u>

Δ	Diaano	sis
А.	Diugnos	515

	1.	Explain the process of epidermal regeneration.	3.1
	2.	Identify the appropriate therapeutic agents for the disorders for eczema and papulosqu dermatoses and the ichthyoses	amous
	z	Describe the different mechanisms of contact dermatitis and how to perform patch test	ting
	5.	besende the unreferent meenanisms of contact dermatics and now to perform pater tes	<b>2.3</b>
	4.	Describe the morphology of atopic dermatitis and list the associated clinical features of	atophy.
			3.0
	5.	Describe the morphology of Reiter' syndrome, psoriasis, lichen planus, lichen nitidus, lic	hen
		sclerosus, erythema annulare centrifugum (EAC), allergic contact dermatitis, pityriasis r	osea,
		seborrheic dermatitis, and icthyosis.	3.4
	6.	Explain the clincial manifestations, etiological agents, diagnosis, and treatment of cutan	eous
		fungal infections.	4.0
	7.	Explain the clinical manifestations, etiological agents, diagnosis, and treatment of viral	
		infections.	4.0
	8.	Diagnose and develop an appropriate treatment plan for tinea pedis.	4.0
	9.	Discuss the present illness, clinical appearance etiology, differential diagnosis, and treat	ment of
		hyperkeratotic, intertriginous, and vesicular tinea pedis.	4.0
	10.	Explain the clinical forms of verrucae.	4.0
	11.	Explain the viriology and epidemiology of verrucae.	4.0
	12.	Identify the features of the regression process in warts.	4.0
	13.	Explain the various treatment options for pedal warts.	4.0
	14.	List and describe the various treatment options and their indications for pedal warts.	4.0
	15.	Describe the procedure for curettage of pedal warts.	3.4
В.	Dei	rmatoses	
	1.	Differentiate clinically and histologically of the Keratodermas the IPK, PPD, verruca and	КРРН
		lesions.	3.5
	2.	Discuss the characteristic concominant systemic physical findings associated with the	
		keratodermas discussed.	3.5
	3.	Identify and recommend treatment for the clinical distinguishing characteristics of helo	ma
		durcin HD), IPK - (Intractable plantar Keratoses), PPD- Porokeratosis Plantaris Discreta),	and
		describe the treatments associated with these.	4.0
С.	Loc	al and Systemic Manifestations	

1. Explain the relationship between diseases of internal organs and cutaneous manifestations.

	2.	Explain the necessity to refer patients with underlying systemic diseases to a specialist for				
		management of the primary disease.	3.7			
	3.	Describe the cutaneous manifestation of sytemic disease.	3.5			
	4.	Integration of Dermatologic Concepts per Instruction.	3.2			
	5.	Describe the following conditions:				
		a. toxic eruption	2.6			
		b. collagen-vascular disease	2.6			
		c. porphyria cutanea tarda	2.5			
		d. necrobiosis lipoidica diabeticorum	3.4			
		e. sarcoidosis	2.6			
		f. vitiligo	3.0			
		g. Bowen's Disease	2.6			
		h. neurofibromatosis	2.9			
		i. Kaposi's Sarcoma	3.0			
		j. mycosis fungoides	2.5			
		k. Hodgkin's Disease	2.5			
		I. leukemia	2.5			
		m. metastatic carcinoma - breast	2.3			
		n. vasculitis	3.0			
D.	Tui	mors				
	1.	Identify the clinical characteristics distinguishing a benign and malignant lesion.	3.6			
	2.	Outline the process of dysplastic change.	3.3			
	3.	List the types of benign, premalignant, and malignant skin tumors.	4.0			
	4.	Define <i>premalignant</i> .	3.9			
	5.	5. Describe the significant differences between a malignant and benign lesion, from a				
		perspective.	4.0			
	6.	Describe the clinical features of basal cell carcinoma, squamous cell carcinoma, and	d malignant			
		melanoma.	4.0			
_	~					
E.	Spe	ecial Disorders of Nails and Appendages of the Skin				
	1.	Discuss the diagnosis and treatment of onychocryptosis.	4.0			
	2.	Describe the nail units, growth and development.	4.0			
	3.	Explain the nail units reaction patterns.				
	4.	Explain the effects of systemic diseases on the nail unit.				
	5.	5. Label the anatomical parts of the nail unit. <b>4.0</b>				
	6.	Describe normal nail growth and regeneration of the nail plate in detail.	3.33			
	7.	Describe the patterns of disturbances that can occur in the nail matrix. nail fold and	d nail bed. <b>3.3</b>			
	8.	Differentiate tinea ungium from onychomychosis.	3.3			
	9.	Describe the changes that can occur in the nail unit in the presence of PVD, arthriti	s. and			
	5.	pulmonary disease.	3.7			
	10.	Discuss the types and causes of digital clubbing.	3.6			
	11	1. Identify and define the benign and malignant tumors of the nail.     4.0				
	12	Identify and describe the nail dystrophies	3.8			
	13	3. Discuss the possible recreational and occupational factors causing skin changes within the lower				
	10.	extremity.	3.3			
	14.	Discuss lesions associated with acquired immune deficiency syndrome.	3.3			
	15.	Recommend a management plan for pedal hydration problems.	3.4			

<ol> <li>Diagnose and manage dyshidrosis and juvenile plantar dermatitis.</li> <li>Identify the special sports related pedal skin problems.</li> </ol>	3.6 3.6
Treatment	

1. Describe the treatment and control of infections, eczematous dermatoses and ichthyoses.

		3.4
2.	Describe the treatment and control of nail disorders.	3.6

3. Describe the treatment and control of tumors of the skin. **3.6** 

### XII. <u>Gastroenterology</u>

F.

1.	Identify and evaluate the significance of abnormal liver functions tests.	3.5
2.	Identify the clinical manifestations and significance of GI bleeding/ peptic ulcer disease.	3.1
3.	Identify the clinical manifestations and significance of Inflammatory Bowel Disease.	3.0
4.	Identify the clinical manifestations and significance of pancreatitis.	3.0
5.	Identify the clinical manifestations and significance of colon cancer.	3.0

## XIII. <u>Geriatrics</u>

1.	Identify and evaluate urinary incontinence and retention in the perioperative period.	3.0
2.	Identify dementia, delirium, and depression in the perioperative period, with special en	nphasis
	on delirium postoperatively.	3.0
3.	Discuss nutritional issues in the geriatric population.	3.0
4.	Explain the evaluation of podiatric problems in the nursing home patient, as well as the	
	recognition of age-associated medical and psycho-social issues.	3.9
5.	Explain the recognition, prevention, and treatment of deep tissue injury (Decubitis) in the	ne
	geriatric, as well as classifications of Pressure Ulcers.	3.9
6.	Explain the significance of advanced directives and the POLST (Physician Order for Life	
	Sustaining Treatment) form for the geriatric patient.	3.0

### XIV. <u>Pre- and Postoperative Assessment</u>

Explain the evaluation of preoperative laboratory, physiologic, and imaging data.				
Explain evaluation of specific organ systems in the preoperative geriatric and pediatric patier				
Discuss	the assessment of the following postoperative problems			
a.	Fever	4.0		
b.	Altered mental status	3.9		
с.	Fluid & electrolyte disturbances	3.6		
d.	Acute Kidney Injury	3.3		
e.	Chest pain and shortness of breath	4.0		
f.	Postoperative hypotension and hypertension	3.6		
	Explain Explain Discuss a. b. c. d. e. f.	<ul> <li>Explain the evaluation of preoperative laboratory, physiologic, and imaging data.</li> <li>Explain evaluation of specific organ systems in the preoperative geriatric and pedia</li> <li>Discuss the assessment of the following postoperative problems <ul> <li>a. Fever</li> <li>b. Altered mental status</li> <li>c. Fluid &amp; electrolyte disturbances</li> <li>d. Acute Kidney Injury</li> <li>e. Chest pain and shortness of breath</li> <li>f. Postoperative hypotension and hypertension</li> </ul> </li> </ul>		

# **RADIOLOGY LEARNING OBJECTIVES**

1.	. Describe the components of a lower extremity x-ray unit, including tubehead, beam limitation			
	devices, and control panel.	2.3		
2.	<ol><li>Locate and use basic x-ray tubehead components, including</li></ol>			
	a. cathode with filament(s), focusing cup, anode with embedded target, anode angle,			
	window, filtration, tube housing, and collimator;	2.3		
	<li>b. rotating versus stationary anodes; and</li>	2.3		
	c. line-focus principle and central ray.	2.3		
3.	Outline the steps in x-ray production-within the x-ray tube, and detail basic cathode and	anode		
	interactions.	2.0		
4.	Describe x-ray production in terms of			
	a. cathode interactions: thermionic emission and space charge formation;	2.0		
	b. functional cathode design considerations: focusing cup;	2.0		
	c. functional anode design considerations: stationary versus rotating, line-focus pr	inciple;		
		2.0		
	d. anode angle, the line focus principle, and the effect on image sharpness versus h	neel		
	effect;	2.0		
	e. anode interactions: Bremsstralung and characteristic x-ray production;	2.0		
	f. significance of milliamperage and kilovoltage; and	2.0		
	g. graphic polyenergenic x-ray beam with characteristic spikes.	2.0		
5.	Define x-ray beam intensity in terms of photonic quantity and quality and unit of measu	re.		
	, , , , , , , , ,	2.0		
6.	Illustrate how the following basic factors affect beam intensity			
	a. intensity = quantity x quality of photons in beam	2.0		
	b. units of exposure (Roentgens)	2.0		
	c. heel effect ( nonuniform intensity)	2.0		
7.	Discuss applications of Bremsstralung curves.	2.0		
8.	Identify and describe four basic factors that affect x-ray beam intensity via photon quan	titv.		
	, , , , , , , , , , , , , , , , , , , ,	2.0		
9.	Identify and describe two main factors that affect x-ray beam quality.	2.0		
10.	Summarize the major interactions of diagnostic x-rays within matter, centering on the co	oncepts		
	of coherent/elastic scattering, photoelectric interactions, and Compton scattering.	1.7		
11.	Contrast the differences between photoelectric and Compton scatter interactions in ma	tter.		
		1.7		
12.	Relate the significance of photoelectric interactions and Compton scattering in terms of	safetv		
	and imagine and quality.	1.7		
13.	Define the following terms used to quantify radiation absorption in matter and biologics	systems		
	a. Rad	1.4		
	b. Grav	1.4		
	c. Rem	1.4		
	d. Sievert	1.4		
14.	Define the exposure, absorbed dose, dose equivalent, and effective equivalent dose.	2.1		
15.	Identify the image receptors used in plain-film radiographic and fluoroscopic imaging.	2.3		
16.	Discuss film, in terms of structure of film and its emulsion: types of film and direct versus	S		
	indirect (screen): and relationship of film type with speed/latitude/detail.	2.3		
17.	Discuss film intensifying screens in terms of how they work: reduction in dosage: calcium	1		
	tungstate versus rare earth types; relationship of screen speed to detail/resolution: and			
	absorption versus conversion efficiency.	2.3		

- 18. Discuss computed radiography (CR) and direct digital image receptors, with reference to computed radiography (CR) barium fluorohalide screen with plate construction and direct digital radiography (DR). 2.3
- 19. Discuss fluoroscopic image intensifiers, with special attention to image intensifier construction, including input phosphor, photocathode, output phosphor, and focusing lens; and television image monitoring. 2.3
- 20. Describe how radiographic images are formed for film, CR/digital, and fluoroscopic image 2.0 receptors.
- 21. Discuss film and latent image formation, including photon interactions with silver halide crystals that result in latent image formation; reduction of ionic silver; and sensitivity speck and latent image center formation. 2.0
- 22. Discuss digital (non-film) CR and image formation in terms of photostimulable phosphor (PSP) luminescence and the barium fluorohalide phosphor storage screen and CR imaging plate; interactions with x-ray photons with screen during exposure, some energy trapped as latent image, some released as light; trapped energy released later through latent image processing via laser scanning of plate; light release (blue green) proportional x-ray photon energy stored; erasure; and distinction between CR and Direct digital. 2.0
- 23. Discuss fluoroscopic image intensifiers, in terms of input phosphor and photoemission Incident x-rays on input phosphor converted/recruited to light photons; electron output by photocathode; acceleration to output phosphor; and flux gain. 2.0
- 24. Discuss the process of converting the latent to manifest image with both film and CR image receptors. 1.6
- 25. Explain film development in terms of film processing, the basic sequence of processing, and the distinction between clearing and fixing time. 1.6

26. Explain digital CR latent image processing, in terms of laser scanning the barium fluorohalide plate; erasure of image; interference by x-ray exposure; exponential image fade with time and post-processing. 1.6

- 27. Discuss the use of film imaging systems. 2.4 28. Explain the proper handling and storage of film. 2.4 29. Identify factors and/or determinants of fog and scatter. 2.4
- 30. Compare and contrast manual processing versus automatic film processing, including equipment used. 2.4
- 31. Explain darkrooms in terms of safelight/spectral matching. 2.4 32. Discuss the usage of film identification. 2.4 33. Define *radiographic density*. 2.4 34. Discuss the factors that influence radiographic density, and how they affect it, including a. film density; 3.0 b. milliamperage, mAs; 3.0 c. distance; 3.0 d. kilovoltage, kVp; 3.0 e. 15% rule; 5% rule; and 3.0 f. application to digital image receptors (ie, linear response to radiation). 3.0 35. Define radiographic contrast. 3.0 36. Delineate between film and subject contrast. 3.0
- 37. Correlate basic subject factors with their influence on final image contrast, including a. film contrast as defined by thickness differences;
  - 2.9 2.9 b. density differences; 2.9
  - c. atomic number (Z) difference;

	d. effects of kilovoltage; and	2.9
	e. applications to digital radiography.	2.9
38.	List the factors that typically result in films being too light or too dark.	2.9
39.	Explain image detail and identify the factors that influence appearance.	3.3
40.	Identify the basic causes for a blurred image, and alteration of an object's shape or positi	on.
		3.3
41.	Define distortion and identify factors that influence its appearance.	3.3
42.	Discuss the biological effects of ionizing radiation, and how radiation may affect the hum	an
	body.	2.7
43.	Recount the basic molecular and macromolecular effects of ionizing radiation within the	cell,
	both direct and indirect.	2.7
44.	Distinguish between threshold and non-threshold dose/response curves.	2.7
45.	Contrast the relative/differential radiosensitivity of somatic cells.	2.7
46.	Compare and contrast deterministic and stochastic effects of radiation.	2.7
47.	Compare and contrast acute and long-term effects of ionizing radiation.	2.7
48.	Discuss the major early (acute) effects of ionizing radiation on the human body.	2.9
49.	Discuss the late (long term) effects of ionizing radiation.	2.7
50.	Compare and contrast the effects of acute and long-term effects of ionizing radiation on	
	biological systems.	2.7
51.	Explain principles and basic techniques available to reduce exposure to patients and oper	rators.
		3.4
52.	Explain how time, distance, and shielding from a radiation source generally influence the	
	amount of exposure.	3.4
53.	Explain the "ALARA" principle.	3.4
54.	Outline the adverse effects of improper collimation.	3.4
55.	Discuss the use of intensitying screens and added x-ray filtration to reduce radiation expo	osure.
ГC	Define effective dese	3.4
50.	Define effective dose.	<b>5.4</b>
57.	radiation desimption badges	али Эл
58	Describe current methods and techniques consistent with safe operating procedures for	<b>3.4</b> hoth
50.	nation and the operator	34
59	Differentiate the relative dose equivalent of plain-film radiographic pedal/extremity stud	jes
55.	versus typical nonnedal studies.	3.4
60.	Discuss basic scatter radiation "maps" and explain where to stand relative to orientation	of tube
	head and image intensifier.	3.4
61.	Outline the current annual effective dose limits of thyroid, skin, hands, and feet; lens of t	he eve;
	cumulative lifetime; and whole body dose limits for radiation workers, the general public	, and
	the fetus.	3.4
62.	Define position, projection, and view.	3.1
63.	Explain the significance of positioning the foot and ankle in the angle and base of gait.	3.0
64.	Explain the proper technique for obtaining the following weight-bearing, nonweight-be	aring,
	or partially weight-bearing views:	
	a. foot: anteroposterior, lateral, lateral oblique, medial oblique, axial calcaneal (Ha	rris-
	Beath), and axial sesamoid	3.0
	b. ankle: anteroposterior, mortise, medial oblique, lateral oblique, lateral, lateral si	tress,
	push-pull stress, and inversion stress	3.0

65.	. Demonstrate the working ability to safely and properly position the following foot and a views:	inkle
	a. foot: anteroposterior, lateral, lateral oblique, medial oblique, axial calcaneal (H Beath), and axial sesamoid	arris- <b>3.0</b>
	b. ankle: anteroposterior, mortise, medial oblique, lateral oblique, lateral, lateral stress, push-pull stress, and inversion stress	(sagittal) <b>3.0</b>
66.	Describe the basic indications for each of the following x-ray views:	0.0
	a. foot: anteroposterior, lateral, lateral oblique, medial oblique, axial calcaneal (H	arris-
	Beath), and axial sesamoid	3.9
	b. ankle: anteroposterior, mortise, medial oblique, lateral oblique, lateral, lateral	stress,
	push-pull stress, and inversion stress	3.9
67.	Identify the normal radiographic anatomy for the following foot and ankle views:	
	a. foot: anteroposterior, lateral, lateral oblique, medial oblique, axial calcaneal (H	arris-
	Beath), and axial sesamoid	3.9
	<ul> <li>ankle: anteroposterior, mortise, medial oblique, lateral oblique, lateral, lateral nush-null stress, and inversion stress</li> </ul>	stress, <b>39</b>
68.	Identify the angular, spatial, and positional x-ray relationships that are used in biomech	anical
	radiography.	3.9
69.	. Relate typical changes associated with flatfoot, cavus foot, bunion deformity, metatarsu	IS
	adductus, and clubfoot.	3.7
70.	Identify the accessory ossicles of the foot and ankle.	3.7
71.	List the foot and ankle bones present at birth.	3.7
72.	. Describe the time of appearance, variance, and completion of ossification of the primar	y and
	secondary ossification centers of the foot and ankle for both male and females.	3.7
73.	. Discuss the major differentials associated with both acceleration and delay in osseous	
	maturation.	3.7
74.	. Explain the basic techniques of administration, optimal scan times, and general indication	ons and
	usages in current podiatric practice of the following nuclear medicine studies: Tc-99 MD	P, Tc-99
	HMPAO, Indium-111, gallium-67, sequential marrow/WBC scanning, and PET scanning.	3.7
75.	. Compare and contrast IC-99 MDP, IC-99 HMPAO, Indium-111, gailium-67, sequential	
	marrow/ wBC scanning, and PET scanning in terms of	logu
	a. Sensitivity versus specificity issues as it relates to common foot and ankie patho	10gy,
	h hasic nedal positioning for scanning, and	4.0
	c hone scanning - spot images and rectilinear scanning: "hot" spots and cold (nho	topenic)
	spots: RSD, stress fractures. Paget's disease. HPOA.	4.0
76.	Explain the basic interpretation of the following nuclear medicine studies as applied to	
	complicated diabetic foot infections, including Charcot neuroarthropathy	
	a. triphasic Tc-99 MDP	4.0
	b. Tc-99 HMPAO	4.0
	c. sequential Tc-Indium-111	4.0
	d. sequential marrow/WBC scanning	4.0
77.	Discuss the basic principles and application of ultrasound as applied to foot and ankle	
	musculoskeletal imaging.	4.0
78.	. Explain sonographic basics, including physical principles and physics of ultrasound funda	amental
	to imaging musculoskeletal tissue.	4.0
79.	. Identify the main components of the ultrasound unit.	4.0

80.	Relate how gain, tissue gain compensation, electronic focusing, spatial compounding, tis	sue			
	harmonics, read zoom, write zoom, and frequency affect image optimization.	4.0			
81.	Identify and describe anisotropy; edge shadowing, posterior acoustic enhancement; pos	terior			
	acoustic shadowing; partial volume artifact; and reverberation. 4.				
82.	Define hyperechoic, anechoic, hypoechoic, fibrillar, and isoechoic.4				
83.	Describe the main indications and limitations of musculoskeletal diagnostic ultrasound.	4.0			
84.	Recognize the normal appearance on short axis and longitudinal axis of plantar fascia, te	ndons			
	(Achilles tendon, posterior tibial tendon), ligaments (anterior talofibular), and joints (First	st MPJ).			
		4.0			
85.	Define <i>anisotropy</i> and explain how it applies to tendons.	4.0			
86.	Discuss general sonographic findings/acoustic appearance of ligamentous, tendinous, ar	nd fascial			
~-	pedal pathologies, as well as arthropathic pathology (eg, effusion).	4.0			
87.	Discuss the principles of sectional x-ray imaging that forms the basis for CT scanning.	4.0			
88.	Identify sectional anatomy and imaging planes as seen on CT sections.	4.0			
89.	List basic pedal indications for CT scanning.	<b>4.0</b>			
90.	Discuss MRI of the foot and ankie in terms of technology and equipment; physics and es	sential			
	steps of image formatting; relaxation time events; basic pulse sequence in musculoskele				
	imaging, as well as uses, advantages, and disadvantages; spatial resolution; common per				
01	attracts, WK 11 contrast agent, and general chinical applications.	4.0			
91.	weighted images: pulse sequences and imaging planes; normal pulse sequence depende	nt signal			
	intensities of the basic musculoskeletal tissues: I-coupling and ESE T2 imaging; and norm	al MRI			
	anatomy of the foot and ankle including.				
	a. Tumor/tumor-like lesions	4.0			
	i. lipoma – ST				
	ii. Morton's neuroma				
	iii. plantar fibroma				
	iv. ganglionic cyst				
	b. Tendonopathy	4.0			
	i. achilles tendon				
	ii. posterior tibial tendon				
	iii. peroneal (Fibularis)				
	c. Trauma	4.0			
	i. fractures (stress, etc.)				
	ii. OCD				
	iii. hematoma				
	iv. AVN				
	v. Lisfranc injuries				
	vi. ankle ligament sprain				
	d. Infections	4.0			
	I. SOTT LISSUE				
	II. addscess				
	in. cenunus iv. hono				
	iv. Done				
	v. acute osteomyentis e Miscellaneous	4.0			
	i nlantar fasciitis	ч. <b>U</b>			
	ii Charcot disease				

92.	Recognize open, closed, comminuted, greenstick, compression, distraction, avulsion, strepathological, displaced, nondisplaced, angulated, rotated, complete, incomplete, bayone	ess, et, and
	compound fractures.	4.0
93.	Explain what is meant by apposition and alignment of fractures in terms of angulation, re	otation,
	displacement, and distraction.	4.0
94.	Describe congruity, dislocation, subluxation, diastasis, and effusion as realted to the	
	radiographic appearance of joints.	3.9
5.	Identify and describe transverse, oblique, spiral, impacted, and intra-articular fracture pa	atterns.
		3.9
5.	Identify and describe delayed union, nonunion (hypertrophic, oligotrophic, atrophic), ma	alunion,
	and pseudoarthrosis, in relation to improper fracture healing.	4.0
7.	Describe and identify on x-ray the Berndt and Harty classification of talar dome fractures	S.
		3.9
3.	Describe and identify on CT the Sanders classification of calcaneal joint depression fractu	ures.
		4.0
Э.	Describe and identify on x-ray the Salter-Harris classification of epiphyseal plate fracture	es.
		4.0

iii. tarsal coalition iv. foreign body

95.

96.

97.

98.

99.

- 100. Describe and identify the radiographic changes/stages of avascular necrosis (osteonecrosis) in both adult and pediatric bone. 4.0 101. Define *Hawkins' sign* and the *crescent sign*. 3.9
- 102. Identify the location and explain the physiology of Legg-Calve-Perthes, Osgood-Schlatter, Blount, Sever, Kohler, Iselin, Bushke, Freiberg, Treves, Renandier, and Mouchet-Diaz diseases. 4.0

103. Discuss the three stages of the Eichenholtz radiographic classification of neuropathic bone disease (Charcot), along with the clinic-radiographic correlation with each stage. 4.0

- 104. Describe the radiographic stages of osteomyelitis in terms of acute, subacute, or chronic; and hematogenous and direct extension/direct inoculation. 4.0 105. Identiy and describe the radiographic changes of pyogenic septic arthritis and tuberculous septic arthritis (Phemister's triad). 4.0
- 106. Identify and describe the radiographic changes of soft tissue infections. 4.0 107. Discuss the appropriate use of radiographic modalities for diagnosis of osteomyelitis and its
- differentiation from neuropathic bone disease and diabetic osteolysis. 108. Identify on plain film radiographs the features of the following pedal arthropathies:
  - a. adult onset rheumatoid arthritis 3.9 3.9 b. seronegative spondyloarthropathies c. gout/tophaceous gout 3.9 d. CPPD/Pseudogout 3.9 e. diffuse Idiopathic Skeletal Hyperostosis (DISH) 3.9 f. osteoarthritis, in terms of: 3.9 i. Oloff-Jacobs classification
    - ii. Kellgren-Lawrence radiologic grading scale

109.	)9. Identify on plain film radiographs the features of the following pedal metabolic bone di			
	a.	osteoporosis;	3.9	
	b.	radiographic manifestations of osteoporosis;	3.9	

- c. generalized versus regional osteopenia; and 3.9 3.9
- d. complex regional pain syndrome (CRPS).
- 110. Discuss the WHO criteria for fracture threshhold and DEXA scanning (T and Z scoring). 3.9

111.	Differe	ntiate betwe	een rickets and osteomalacia and distinguish the radiographic feature	res of
112.	Identify and describe the radiographic features of primary and secondary hyperparathyroid			
				3.3
113.	Identify	and describ	be the radiographic features of renal osteodystrophy.	3.4
114.	Identify	and describ	be the radiographic features of Paget's disease.	3.6
115.	Identify	and describ	be the radiographic features of pedal acromegaly.	3.0
116.	Discuss	the following	ng basic differentials for generalized periostitis:	
	a.	hypertroph	ic pulmonary osteoarthropathy (prototypic disorder)	3.6
	b.	chronic ver	nous edema	3.6
	с.	thyroid acr	opachy	3.6
	d.	secondary	hyperparathyroidism	3.6
	e.	hypervitam	ninosis A	3.6
	f.	acromegaly	/	3.6
	g.	tuberous so	clerosis	3.6
	h.	seronegativ	ve arthropathy	3.6
	i.	normal rap	id growth (pediatric)	3.6
	j.	congenital	syphilis (pediatric)	3.6
117.	Identify	and describ	be the radiographic features of enostosis, lead intoxicationn, vitamin	n A- and
	vitamir	D-osteopet	rosis, melorheostosis, osteopoikilosis, osteopathia striata, and scler	osing
	osteon	yelitis.		3.1
118.	Discuss	the radiogr	aphic features of myositis ossificans, including localized/heterotopic	c new
	bone fo	rmation and	d myositis ossificans progressive.	3.6
119.	Describ	e and radio	graphically delineate Monckeberg medial calcific sclerosis,	
	ASO/at	nerosclerosi	is, and phleboliths.	3.6
120.	Recogr	ize the basio	c disorders associated with calcinosis, including metastatic, dystropl	nic,
	calcino	sis interstitia	alis universalis, and tumoral calcinosis.	3.6
121.	RAD 57	Describe,	identify, and differentiate between the general radiographic feature	es of
	benign	and maligna	ant bone tumors in relation to sclerotic margin, appearance of bone	matrix,
	and pe	iosteal reac	tion.	4.0
122.	Identify	and describ	be the radiographic characteristics of the following bone tumors and	d/or
	tumor-	ike lesions:		
	a.	Cartilagino	us	3.9
		i. Ost	teochondroma	
		ii. end	chondroma	
		iii. cho	ondroblastoma	
		IV. Cho	ondromyxoid fibroma	• •
	D.	FIDrous		3.9
		I. NOI	nossifying fibroma	
		II. TIDI	rous cortical defect	
			rous dyspiasia	2.0
	С.	Usseous	racid octooma	3.9
		i. OST	eulu usteullid	
		II. UST		
		in. DOI	ne infarction	
	Ь	IOU .VI		20
	u.		ind's sarcoma	5.9
		1. EW	ing 5 sai cullia	

- ii. chondrosarcoma
- iii. conventional osteogenic sarcoma
- iv. metastases
- v. solitary (unicameral) bone cyst
- vi. aneurysmal bone cyst
- vii. giant cell tumor
- viii. osteomyelitis (including Brodie's abscess)
- 123. Identify the plain film radiographic characteristics of tarsal coalitions, including Talar beaking, "halo sign" of the subtalar joint, "rounded" lateral process of talus, joint space narrowing, asymmetry of the posterior and middle facets on an axial calcaneal image, and "anteater" or "comma" sign for a calcaneonavicular bar.
- 124. Describe the method of image production and indications for an ultrasound of the foot and ankle, including venous duplex Doppler imaging for venous thromboembolic disease. 3.9
- 125. Identify anatomical structures as seen on conventional ultrasound imaging. **3.4**
- 126. Describe the indications, normal anatomy, and duplex Doppler imaging for venous<br/>thromboembolism.3.7

# **ORTHOPEDICS LEARNING OBJECTIVES**

Biomechanics Pathomechanics Sports Medicine General Orthopedics Pediatric Orthopedics
### I. <u>Biomechanics</u>

В.

## A. Basic Terminology

1. Identify and describe motion	ons, positions, and fixed positions that occur in each of the o	cardinal
planes as they pertain to th	le lower extremity with emphasis on the foot and ankle.	4.0 4.0
		4.0
Basic Mechanics		
1. Define center of mass and a	center of gravity.	4.0
2. Define torques, couples, an	d moments.	3.5
3. Differentiate between ener	gy, kinetic energy, and potential energy.	3.5
4. Define <i>centripetal force</i> , <i>ce</i>	ntripetal acceleration, and angular acceleration.	3.0
5. Identify the equations of ro	tational motion.	3.0
6. Define <i>linear motion</i> and id	entify the equations of linear motion.	3.0
7. Define <i>power</i> .		3.5
8. Define <i>work</i> .	And the second distance of the state of the second s	3.0
9. Discuss Newton's Laws of N	Notion and their application to the process of human galt.	4.0
10. Explain the principle of con	servation of angular momentum.	3.0
11. Differentiate between rota	tional and linear motion.	4.0
12. Explain the philippe of con	servation of inteal motion.	5.U 2 E
14 Describe the relationship b	etween kinetic and power.	3.5
15 Discuss the basic concents	of inertia momentum and motion as they relate to the low	9.9 /er
extremity.	or mercla, momentani, and motion as they relate to the low	4.0
16. Discuss the concepts of stre	ess and strain physics as applied to Wolf's Law.	4.0
17. Discuss the concept of frict	ion as a force, and explain the laws of friction and coefficier	nts of
friction.	·	4.0
18. Differentiate between scala	ar and vector quantities.	3.0
19. Describe the concept of a le	ever and the types of levers with reference to the lower ext	remity.
		3.5
20. Describe a stress/strain dia	gram.	4.0
21. Identify and describe the di	ifferent loading modes.	3.5
22. Describe basic elements of	bone and tendon physics.	4.0
23. Differentiate between the l	behaviors of adult bones under different loading modes.	4.0
24. Explain combined loading c	of bone.	4.0
25. Explain functional adaption	of bone.	4.0
26. Explain the effect of muscle	e contraction on bone during the gait cycle.	4.0
Soft Tissue Physiology Mech	anics	
1. Explain the relationship bet	tween the sarcomere and the development of muscle tension	on.
		3.5
2. Describe the biomechanica	l properties of cartilage.	3.5
3. Discuss the characteristics	of ligaments and tendons.	4.0
4. Describe the length-tensior	n relationship of muscles.	4.0
<ol><li>Compare and contrast sing</li></ol>	le and multiple joint muscles.	4.0

6. Describe the effects of various injuries/pathologies on the mechanical properties of the different biological tissues.
4.0

	7. 8. 9.	Describe the different functions of muscles during gait and give examples. Describe factors that affect mechanical efficiency. Define <i>elastic response</i> and give examples of elastic response during the gait cycle.	4.0 4.0 4.0
С.	Sta	atistics and Compensation	
С.	Sta 1. 2. 3. 4. 5. 6. 7. 8. 9.	Define <i>compensation</i> Define <i>compensation</i> and distinguish normal and abnormal compensation. Discuss the effect of deviation of the trunk or leg on the foot. Discuss the effect of deviation in one part of the foot on the other. Discuss the effect of deviation of the terrain on the foot. Explain the theorems of compensation. Describe the distribution of body weight during static stance, as well as the role that con of the gastrocnemius has on maintaining it. Explain osseous restraining mechanisms, and provide examples of that. Contrast and compare the contributions of bone, muscles, and ligaments in stability dur static stance. Explain what happens when rotatory moments induced by ground reactive forces canned	4.0 4.0 4.0 4.0 4.0 0 0 0 0 0 0 0 0 0 0
		compensated.	4.0
_	10.	Explain why subtalar and midtarsal joints are primarily involved in compensation.	4.0
D.	Foi	rces and Functional Anatomy	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> <li>11.</li> <li>12.</li> </ol>	Describe the production of different forces during stance, including when they peak. Explain forefoot pathology as caused by abnormal shear forces during propulsion. Explain the production of abnormal shear forces during propulsion. Explain why the swing limb is thought to cause forward movement of the body. Compare and contrast the structure and function of the medial and lateral columns of t Describe the effect that distortion of anatomy has on function. Explain the function of the plantar fascia. Explain why the midtarsal joint is maximally pronated during midstance. Describe the locking function of the midtarsal joint and relate the midtarsal motion and to subtalar joint (STJ) position. Explain oblique toe break. Describe the ontogenic etiology of foot dysfunction.	4.0 4.0 4.0 4.0 he foot. 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.
	13.	Explain beam and truss and relate to contact and propulsion.	3.0 3.0
Е.	Mr	anual Muscle Testina	
	1. 2.	Describe the techniques used to test muscle strength for the major muscle groups cross ankle, as well as the intrinsic foot muscles. Discuss the standard five point grading scale used to evaluate muscle strength.	ing the 4.0 4.0
	Ph	asic Muscle Activity	
	1. 2. 3.	Identify the factors that influence a muscle's ability to produce power. Differentiate between concentric, eccentric, and isometric muscle contractions and und the roles that they play in ambulation. Determine the type of muscle contraction that lower extremity muscles are undergoing each phase of the gait cycle.	4.0 erstand 4.0 during 4.0

	<ol> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> <li>11.</li> <li>12.</li> <li>13.</li> </ol>	Identify the normal phasic muscle activity of the anterior thigh; medial thigh; posterior to anterior leg; lateral leg; posterior leg; and intrinsic foot muscle groupsduring gait. Differentiate between monophasic and biphasic muscle activity. Describe the segment of gait cycle, the function, the percentage of gait cycle and the po pathology for the soleus; gastrocnemius; anterior tibial; extensor digitorum longus; exter hallucis longus; peroneal brevis; peroneal longus; posterior tibial; flexor hallucis longus; digitorum longus. Describe the timing of muscle activity and its relation to function during actual phase of cycle. Describe the consequences of anterior muscle dysfunction relative to gait. Describe the consequences of lateral muscle dysfunction relative to gait. Describe the consequences of intrinsic foot muscle dysfunction relative to gait. Discuss the consequences of intrinsic foot muscle dysfunction relative to gait. Discuss the muscle function above the knee relative to the gait. Identify tendons in relation to the joint axis.	high; 4.0 4.0 ssible nsor flexor 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0
F.	Pri	nciples of Shock Absorption	
	1. 2. 3. 4. 5. 6.	Define shock absorption and identify the gait parameters that influence it. Describe the roles that the subtalar, ankle, knee, and hip joints play in shock absorption. Explain the influence that timing sequence of the shock absorbing mechanism has on the ability to absorb shock. Describe the pathology and parameters that may lead to poor shock absorption. Describe the role of the plantar fat pad in shock absorption. Describe the role that shoes play in shock absorption.	4.0 4.0 e body's 4.0 4.0 4.0 4.0
G.	Pri	nciples of Stability	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> </ol>	Define <i>stability</i> and <i>instability</i> . Describe the attributes of joint stability. Describe the relationship the subtalar joint and midtarsal joint have with respect to stab Describe the role that the arch of the foot plays with respect to stability. Define <i>joint hypermobility</i> and <i>joint congruity</i> . Describe the role that the soft tissues have on stability. Contrast and compare positive and negative mechanical advantages. Explain the theory of proximal stability and apply it to the joints of the lower extremity.	4.0 4.0 ility. 4.0 4.0 4.0 4.0 4.0 4.0
Н.	Kin	etics and Kinematics	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> </ol>	Define <i>kinetics</i> and <i>kinematics</i> . Define <i>inverse kinematics</i> and <i>forward kinematics</i> . Interpret a kinematic graph. Define <i>momentum</i> and explain its relation to any given point in the gait cycle. Define <i>acceleration</i> and how it relates to the gait cycle. Identify and explain the factors that influence a muscle's ability to produce power. Explain a joint moment and determine whether it is an internal or external moment. Discuss the moment of any given joint at any particular point in the gait cycle. Explain ground reactive force and determine the position and orientation of the force w respect to the joints of the lower extremity during each phase of the gait cycle.	3.5 3.0 3.5 3.5 4.0 4.0 4.0 ith 4.0
	10.	Define <i>pronation</i> and its role in motion.	4.0

	11. Describe motion in terms of linear and angular relationships.	2	4.0
Ι.	. Functional Axes and Planes of Motion		
	1. Describe the cardinal planes.	4	1.0
	2. Describe axis of motion.	4	1.0
	3. Differentiate between uniaxial, biaxial, and triaxial joints.	2	4.0
	4. Differentiate between uniplanar, biplanar, and triplanar joints.	2	4.0
	5. Discuss the concept of planar dominance as it relates to a joint.	2	4.0
	6. Describe the subtalar joint in terms of axis, location, and range of mo	tion. 4	4.0
	7. Describe the midtarsal joint in terms of axis, location, and range of m	otion. 4	1.0
	8. Describe the first ray range of motion.	4	1.0
	9. Describe the fifth ray range of motion.	2	1.0
	10. Describe the first metatarsophalangeal joint range of motion.	4	1.0
	11. Describe the role of the lesser metatarsophalangeal joint range of mo	otion. 4	1.0
	12. Describe the common motions and positions of the foot using body p	lanes. 4	1.0
	13. Describe and demonstrate freedom of motion.	4	4.0
	14. Describe and give examples of pathology that develops relative to joi	nt axes of motion. 4	1.0
	15. Describe the motions involved in closed kinetic chain supination.	4	4.0
	16. Describe the motions involved in closed kinetic chain pronation.	2	4.0
	17. Describe the motions involved in open kinetic chain supination.	2	4.0
	18. Describe the motions involved in open kinetic chain pronation.	2	4.0
J.	. Spine		
	1. Identify and describe the axes of motion and biomechanics of the spi	ne.	3.4
	2. Discuss the etiologies, locations, and types of scoliosis.	3	3.0
	3. Discuss the signs and symptoms associated with scoliosis.	3	3.0
	4. Describe and perform a screening exam for scoliosis.	3	3.5
	5. Discuss radiographic techniques to diagnose scoliosis.	3	3.0
	6. Describe common gait changes associated with scoliosis.	2	4.0
	7. Discuss the etiology and locations of lordosis and kyphosis.	3	3.5
	8. Describe the dynamics of lordosis and kyphosis in static stance and ga	ait. 4	4.0
К.	K. Limb Length Discrepancy		
	1. Differentiate normal and abnormal variances in limb length.	4	4.0
	2. Discuss etiologies of LLD.	4	1.0
	3. Differentiate between structural and functional LLD.	4	1.0
	4. Differentiate between the techniques used to measure true limb lenge	th versus functional	limb
	length.	4	1.0
	5. Discuss radiographic techniques to diagnose limb length discrepancy.	2	1.0
	6. List other points of evaluation to determine the presence of a limb le	ngth discrepancy. 4	1.0
	7. Describe signs, symptoms, and gait changes associated with asymmetry	trical limb length. 4	1.0
	8. Identify and describe nonsurgical methods of relieving symptoms ass	ociated with LLD.	1.0
	9. Describe the effects on the body associated with eliminating compen	satory changes in the	e feet
	for patients with limb length discrepancy and scoliosis.	2	4.0
L.	Hip Joint		
	1. Identify the axis of motion and biomechanics of the hip joint.	2	4.0

	<ol> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> <li>8.</li> <li>9.</li> <li>10.</li> <li>11.</li> <li>12.</li> </ol>	Explain the technique used to measure the sagittal plane hip range of motion. Explain the technique used to measure transverse plane range of motion of the hip. Explain the technique used to measure frontal plane motion of the hip. List the normal sagittal plane ranges of motion for the hip. List the normal frontal plane ranges of motion for the hip. List the normal transverse plane ranges of motion for the hip. Discuss the various planal abnormalities about the hip. Describe signs, symptoms, and gait changes associated with abnormal hip range of motion. Describe the limiting factors in hip flexion with the knee flexed and with the knee exten	4.0 4.0 4.0 4.0 4.0 4.0 4.0 500. 4.0 ded. 4.0
	13. 14. 15. 16.	Describe the limiting factors in transverse plane hip range of motion with hip flexed and extended. Discuss the position of the hip during the various periods of the gait cycle. Discuss neutral position versus closed-packed position of the hip. Calculate the transverse plane neutral position of the hip.	l while 4.0 4.0 4.0 4.0
М.	Kne	ee Joint	
	1. 2. 3. 4. 5. 6. 7. 8. 9. 10. 11. 12. 13.	Identify and describe the knee joint axes and the motion of the knee joint. Discuss the position of the knee joint during the phases of gait. Discuss the relationship of the knee joint function on the hip, leg, and foot. Discuss the muscles governing the knee joint function and describe their role during gait Discuss normal patella-femoral joint function. Discuss the provisions for stability and flexibility at the knee. Discuss the establishment of knee joint stability, both functionally and anatomically. <b>4.0</b> Explain the technique used to measure knee range of motion. Explain the techniques used to evaluate the frontal and sagittal plane position of the kn Differentiate between true tibial torsion and malleolar position. List normal values for malleolar position. Describe etiologies, signs, symptoms, and gait changes associated with abnormal malleo position.	4.0 4.0 4.0 4.0 4.0 4.0 6 4.0 4.0 4.0 4.0 4.0 0 1ar 4.0
	Fur	nctional Deviations of the Knee	
	1. 2. 3. 4.	Discuss the planal abnormalities of the knee, inlcuding genu varum, tibial varum, genu v and tibial valgum, and genu recurvatum. Describe signs, symptoms, and gait changes associated with abnormal knee position. Discuss the effects of ankle equinus on the knee. Discuss the effect of pronation on the knee.	algum 4.0 4.0 4.0 4.0
N.	An	kle Joint	
	1. 2. 3. 4.	Identify the axis of motion and biomechanics of the ankle joint. Explain the technique used to measure ankle joint dorsiflexion. Describe the common mistakes made when measuring ankle joint dorsiflexion. List normal ranges of motion for the ankle joint.	4.0 4.0 4.0 4.0

- Discuss ankle joint function during the phases of gait.
   Describe the bones involved in the ankle joint.
   4.0
- 7. Describe neutral position of the ankle joint.4.0

## **Functional Deviations of the Ankle**

	1. 2	Explain equinus deformity of the ankle.	4.0 4.0
	2. 3.	Differentiate between bony block, gastrocnemius, gastro-soleus, and pseudoequinus equinus equi	uinus.
			4.0
	4.	Discuss the general clinical features associated with ankle equinus.	4.0
	5.	Discuss compensation mechanisms in the lower extremity in the presence of equinus.	4.0
	6. -	Discuss gait patterns associated with equinus.	4.0
	7.	Discuss the general treatment principles for equinus.	4.0
	8.	Discuss the various functional adaptations of the ankle joint as a result of surgery or tra-	uma.
	_		4.0
	9.	Describe how the foot compensates for congenital and neuromuscular ankle joint equir	ius.
			4.0
	10.	Describe the clinical and radiographic manifestations of ankle joint equinus.	4.0
	11.	Discuss the prognosis of ankle joint equinus regarding conservative care.	4.0
	12.	Diagnose and recommend treatment for ankle equinus, given a clinical scenario.	4.0
	13.	Describe the adverse effects of loss of neuromuscular activity on ankle function.	4.0
О.	Suk	otalar Joint	
	1.	Identify the axes of motion and biomechanics of the subtalar joint.	4.0
	2.	Differentiate between open and closed kinetic chain subtalar joint function.	4.0
	3.	Describe the position of the subtalar joint in each of the phases of the gait cycle.	4.0
	4.	Describe the technique used to measure subtalar joint range of motion and neutral posi-	ition.
			4.0
	5.	Describe normal subtalar joint neutral position and ranges of motion.	4.0
	6.	Discuss the factors limiting subtalar joint range of motion.	4.0
	7.	Describe the bones involved in the STL	4.0
	8.	Describe and demonstrate rotational equilibrium and apply it to STJ.	4.0
	Fur	nctional Deviations of the Subtalar Joint	
	1.	Describe the sagittal pane deviations of the subtalar joint axis and discuss the possible	
		outcomes.	4.0
	2.	Describe the transverse plane deviations of the subtalar joint axis and discuss the possib	ble
		outcomes.	4.0
	3.	Describe the effects of subtalar joint range of motion by variations in the position of its	axis.
			4.0
	4.	Describe etiologies, signs, symptoms, and gait changes associated with abnormal subtal	ar joint
		ranges of motion and/or neutral position.	4.0
	5.	Identify laterally and medially displaced calcaneus and discuss the possible outcomes of	each.
			4.0
Ρ.	Red	arfoot Function	
	1.	Differentiate rearfoot varus and rearfoot valgus.	4.0
	2.	Identify and discuss the etiologies of rearfoot varus and rearfoot valgus.	4.0
	3.	Distinguish rearfoot varus from subtalar joint varus, identify a subtalar joint varus, and o	discuss
		its possible outcomes.	4.0

	4.	Distinguish rearfoot valgus from subtalar joint valgus, identify a subtalar joint valgus, and	d discuss
	_	its possible outcomes.	4.0
	5. c	Define tibial varum and tibial valgus.	4.0
	6.	Identify tibial varum and discuss the possible outcomes.	4.0
	/.	Identify tibial valgus and discuss the possible outcomes.	4.0
	ð.	Define and discuss resting calcaneal stance and neutral calcaneal stance position.	4.0
	9.	Describe the measurement of resting calcaneal stance position and neutral calcaneal sta	ince
	10	position.	4.0
	10.	Describe the measurement of tibial influence	4.0
	11.	Describe the measurement of tibial influence.	4.0
	12.	compensation.	4.0
	13.	Discuss factors affecting rearfoot position.	4.0
	14.	Discuss the effects of rearfoot pathology on the gait cycle.	4.0
	15.	Identify and discuss possible scenarios that lead to an inverted resting calcaneal stance i	position.
			4.0
	16.	Identify and discuss possible scenarios that lead to an everted resting calcaneal stance p	osition.
			4.0
	17.	Identify and discuss possible scenarios that lead to a perpendicular resting calcaneal sta	nce
		position.	4.0
	18.	Explain why neutral calcaneal stance position represents total rearfoot deformity.	4.0
	19.	Explain why resting calcaneal stance position represents compensation that has occurre	d at the
		subtalar joint.	4.0
	20.	Describe deformities that may cause abnormal findings in the stance positions.	4.0
	21.	Identify the possible compensations for a rearfoot varus and rearfoot valgus and discuss	the
		possible outcomes.	4.0
	22.	Describe the clinical and radiographic manifestations of rearfoot varus and differentiate from nartially compensated	fully <b>4.0</b>
	23	Discuss conservative treatment of rearfoot varus and differentiate fully from partially	4.0
	20.	compensated.	4.0
	24.	Discuss prognosis of rearfoot varus when treated with functional orthoses.	4.0
	25.	Define <i>calcaneal valaus</i> .	4.0
_			
Q.	Mi	dtarsal Joint	
	1.	Identify the axis, location, and range of normal motion of the midtarsal joint.	4.0
	2.	Describe the bones involved in the midtarsal joint.	4.0
	3.	Describe the relationship between subtalar joint position and midtarsal joint motion.	4.0
	4.	Describe the function of the normal midtarsal joint during gait with respect to ground re	active
		forces and muscular activity.	4.0
	5.	Discuss the factors that limit or increase midtarsal joint range of motion.	4.0
	6.	Discuss the locking mechanism of the midtarsal joint and the significance of the locking	
		mechanism in normal gait.	4.0
	7.	Describe the technique used to measure midtarsal joint locked or maximally pronated p	osition.
			4.0
	8.	Discuss the common errors in measuring midtarsal joint position.	4.0
	9.	List the values for the normal midtarsal joint position.	4.0

## Function Deviations of the Midtarsal Joint

1.	Identify the medial deviation anomalies of the oblique midtarsal joint axis and discuss the possible outcomes.	he <b>4.0</b>
2.	Identify the superior deviation anomalies of the oblique midtarsal joint axis and discuss possible outcomes.	the 4.0
3.	Discuss the treatment implications of a foot with a superiorly deviated midtarsal joint as	xis.
4.	Describe etiologies, signs, symptoms, and gait changes associated with abnormal midta maximally pronated position.	rsal joint 4.0
Inv	verted Forefoot Deformities	
1.	Define <i>forefoot varus</i> .	4.0
2.	Define forefoot supinatus.	4.0
3.	Define <i>metatarsus primus elevatus</i> .	4.0
4.	Identify and discuss the etiologies of forefoot varus.	4.0
5.	Identify and discuss the etiologies for forefoot supinatus.	4.0
6.	Differentiate between a forefoot varus and a forefoot supinatus.	4.0
7.	Identify the signs, symptoms, and compensation patterns of the specific inverted forefo	ot
	deformities.	4.0
8.	Distinguish between congenital and acquired metatarsus primus elevatus.	4.0
9.	Discuss the potential outcomes of metatarsus primus elevatus.	4.0
10.	Describe the compensation of the foot for forefoot varus.	4.0
11.	Describe the clinical and radiological manifestations of forefoot varus.	4.0
12.	Discuss conservative treatment for forefoot varus.	4.0
13.	Accurately describe the prognosis of forefoot varus.	4.0
14.	Diagnose and recommend an acceptable treatment plan for forefoot varus.	4.0
Eve	erted Forefoot Deformities	
1.	Define <i>forefoot valgus</i> .	4.0
2.	Identify the possible compensations for forefoot valgus and discuss the outcomes.	4.0
3.	Identify and discuss the etiologies of forefoot valgus.	4.0
4.	Define plantarflexed first ray.	4.0
5.	Distinguish between congenital and acquired plantarflexed first ray.	4.0
6.	Discuss the potential outcomes of a plantar flexed first ray.	4.0
7.	Describe the etiologies of plantarflexed first ray deformities.	4.0
8.	List the clinical signs and symptoms associated with plantarflexed first deformities.	4.0
9.	Describe the compensation mechanism for the different types of plantarflexed first ray	
	deformities.	4.0
10.	Discuss etiology of rigid plantarflexed first ray deformity.	4.0
11.	Describe the compensation of the body on rigid plantarflexed first ray deformity.	4.0
12.	Discuss clinical signs and symptoms of rigid plantarflexed firstfirst ray deformity.	4.0
13.	Explain how rigid plantarflexed first ray is treated.	4.0
14.	Discuss the prognosis of rigid plantarflexed first ray.	4.0
15.	Compare and contrast rigid and nonrigid forefoot valgus.	4.0
16.	Diagnose and recommend an appropriate treatment plan, when given a clinical present	ation
	with rigid plantarflexed first ray.	4.0
17.	Identify and discuss a dorsiflexed cuboid.	4.0
18.	Discuss the possible outcomes of a dorsiflexed cuboid.	4.0

18. Discuss the possible outcomes of a dorsiflexed cuboid.

	<ol> <li>Discuss etiology, compensation, clinical findings, prognosis, and treatment of ri forefoot valgus.</li> </ol>	gid and nonrigid <b>4.0</b>
R.	First Ray Function	
	<ol> <li>Identify the axis, location, and range of normal motion of the firstfirst ray.</li> <li>Describe the bones involved in the first ray.</li> <li>Describe the technique used to evaluate first ray range of motion.</li> <li>Discuss the common errors in measuring first ray range of motion.</li> <li>List the normal values for first ray range of motion.</li> <li>Calculate the first ray neutral position.</li> <li>Differentiate between congenital and acquired first ray deformity.</li> <li>Differentiate between flexible and rigid first ray deformity.</li> <li>Define hypermobile first ray.</li> <li>Describe normal motion of the first ray during gait.</li> <li>Describe etiologies, signs, symptoms, and gait changes associated with abnorm of motion and/or neutral position.</li> <li>Discuss the relationship of STJ position and first ray motion.</li> <li>Discuss mechanical treatments for first ray deformities.</li> <li>List the biomechanical deformities associated with hypermobile first ray.</li> </ol>	4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0
S.	First Metatarsophalangeal Joint	
	<ol> <li>Identify the axis, location, and range of normal motion of the first MPJ.</li> <li>Describe the techniques used to measure first metatarsophalangeal joint range</li> <li>Discuss the common errors in measuring first metatarsophalangeal joint range</li> <li>Describe the normal values for first metatarsophalangeal joint range of motion</li> <li>Discuss the effect of the first ray position on the first metatarsal phalangeal joint motion.</li> <li>List the components of first MPJ dorsiflexion during gait and the normal amount available from each component.</li> <li>Describe etiologies, signs, symptoms, and gait changes associated with abnorm metatarsophalangeal joint range of motion.</li> </ol>	4.0 of motion. 4.0 of motion. 4.0 at range of 4.0 at of motion 4.0 al first 4.0
Т.	Fifth Ray Function	
	<ol> <li>Identify and describe the axis of motion and biomechanics of the fifth ray.</li> <li>Discuss the fifth ray range of motion.</li> <li>List the clinical signs and symptoms associated with a plantarflexed fifth ray.</li> <li>List the clinical signs and symptoms associated with a dorsiflexed fifth ray.</li> </ol>	4.0 4.0 4.0 4.0
U.	Central Ray Function	
	<ol> <li>Identify the axis, location, and range of motion of the lesser metatarsophalange</li> <li>Describe normal and abnormal metatarsal parabola, including radiographic assessments</li> </ol>	eal and digits. 4.0 essment of these
	<ol> <li>Describe the clinical signs and symptoms associated with abnormal metatarsal</li> <li>Describe etiologies, signs, symptoms, and gait changes associated with abnorm</li> </ol>	<b>4.0</b> parabola. <b>4.0</b> al lesser
	metatarsophalangeal joint range of motion.	4.0

	5.	Describe etiologies, signs, symptoms, and gait changes associated with abnormal positi and/or range of motion of digits.	on <b>4.0</b>
	6.	Define and describe the axes of motion and biomechanics of the phalanges.	4.0
	7.	Describe the clinical findings and gait changes associated with a plantarflexed lesser me	etatarsal
		deformity.	4.0
	8.	Describe the clinical signs and symptoms associated with a dorsiflexed lesser metatarsa	l. <b>4.0</b>
V.	Ga	it	
	1.	Explain the subdivision of gait into phases and periods.	4.0
	2.	Describe the periods of the stance phase of the gait cycle.	4.0
	3.	Describe swing phase of the gait cycle.	4.0
	4.	Define cadence, step length, and stride length.	4.0
	5.	Describe the relationship between limb length and cadence.	4.0
	6.	Describe the foot's distribution of forces across its structure during gait.	4.0
	7.	Describe the basic motions of the foot and leg during the gait cycle.	4.0
	8.	Describe the role the leg and body play in participation and support of gait.	4.0
	9.	Identify the position and the motion of each joint in the lower extremity for any given p	oint of
		the gait cycle.	4.0
	10.	Discuss the function of each muscle or muscle group during gait.	4.0
	11.	Describe the position of the hip, ankle subtalar, and midtarsal joints in each of the phas	es of the
		gait cycle and the moments that are being applied to them during these phases.	4.0
	12.	Describe the position of the first ray in each of the phases of the gait cycle and the mon	nents
		that are being applied to it during these phases.	4.0
	13.	Describe the position of the forefoot in each of the phases of the gait cycle.	4.0
	14.	Define velocity and explain its relationship to gait.	4.0
	15.	Identify and describe each body segment as it moves in the three body planes.	4.0
	16.	Describe gait analysis findings of asymmetry.	4.0
	17.	Describe function of upper extremity to lower extremity during galt.	3.5
	18.	Apply the concept of ground reactive force to abnormal positions of the foot in the gait	cycle.
	10	Discuss wath along that may accuracy as a result of almormalities within the crit such	3.0
	19.	Discuss pathology that may occur as a result of abnormalities within the gait cycle.	4.0
	Fui	nctional Deviations of Gait	
	1.	Describe and discuss circumduction, hip hiking, vaulting, abnormal hip rotation, excessi	ve knee
		flexion or extension, inadequate dorsiflexion, abductory twist, early heel lift, foot drop,	and
		wide base gait.	4.0
	2.	Differentiate the abnormal gait findings associated with foot pathology.	4.0
	3.	Describe Steppage gait and discuss the possible causes.	4.0
	4.	Describe Trendelenburg gait and discuss the possible causes.	4.0
	5.	Describe Parkinsonian gait and discuss its clinical features.	4.0
	6.	Differentiate between spastic diplegia and hemiplegia.	4.0
	7.	Describe calcaneus gait and discuss its possible causes.	4.0
W.	Bio	mechanical Radiographic Interpretation	
	1.	Identify normal radiographic angles.	4.0
	2.	Describe the standard position for taking radiographs for biomechanical evaluation.	4.0
	3.	Identify radiographic signs in the normal foot, pronated foot, and supinated foot.	4.0
	4.	Identify the cyma line on a radiograph.	4.0

5. 6.	Identify normal sagittal, transverse, and frontal plane relationships in the foot. Identify abnormal sagittal, transverse, and frontal plane relationships in the foot.	4.0 4.0
. OI	thoses	
1. 2. 3.	Define orthoses, prosthetics, and pedorthics. Describe the role of the orthotist, prosthetist, and pedorthist in treating foot disorders. Describe the general purpose of orthoses.	4.0 4.0 4.0
4.	Differentiate between custom orthoses, prefabricated orthoses, and prefabricated arch	
5	supports. Discuss the usage as well as the pros and cons of prefabricated orthoses and prefabricated o	4.0 ated arch
5.	supports.	4.0
6.	Identify and describe types of materials used for prefabricated orthoses and prefabricat supports.	ed arch <b>4.0</b>
Fu	nctional Foot Orthoses	
1. 2.	Explain the purpose of foot orthoses. Identify the component parts of a functional orthoses.	4.0 4.0
3. 4.	Discuss the role of an orthoses in managing forefoot deformities. Discuss the role of an orthoses in resisting abnormal forces in the rearfoot (both pronat	4.0 ory and
5.	Describe how to incorporate motion into the rearfoot.	4.0 4.0
6.	Describe the limitation of orthoses on subtalar joint motion.	4.0
7.	Describe types of materials used for component parts of the orthoses.	4.0
8.	Discuss the goals of therapy of a functional orthoses.	4.0
9.	Discuss the function of orthoses in gait.	4.0
10	. Describe the foot pathologies that would benefit from functional orthoses managemen	t. <b>4.0</b>
11	. Identify relative contraindications for functional orthoses.	4.0
12	Discuss indications for rigid, semingid, and flexible materials.	4.0
10	Identify the materials commonly used for propated orthoses	4.0
15	Discuss the treatment goals of a custom foot orthoses	4.0
16	Describe the appearance of a dress shoe orthoses.	4.0
17	. Discuss the limitations of dress shoe orthoses.	4.0
18	. Identify the materials commonly used in dress shoe orthoses.	4.0
19	. Describe the appearance of a typical sport orthoses.	4.0
20	. Discuss sport-specific modifications for sports orthoses.	4.0
21	. Discus limitations of a sports orthoses.	4.0
22	. Identify some of the materials commonly used in sports orthoses.	4.0
23	. Discuss the effect of the heel contour, heel seat depth, and heel counter of the shoe on function of the orthoses.	the <b>4.0</b>
24	. Discuss the effects of the shank, midsole, lasts, and uppers of the shoe on the function orthoses.	of the <b>4.0</b>
Ac	commodative Foot Orthoses	
1.	Identify the component parts of an accommodative orthoses.	4.0

- 1. Identify the component parts of an accommodative orthoses.4.02. Discuss the goals of therapy of an accommodative orthoses.4.0
- 3. Identify and differentiate types of materials used for an accommodative orthoses. **4.0**

	4.	Describe the foot pathologies that would benefit from accommodative orthoses manage	ement.
	-		4.0
	5.	identify relative contraindications for an accommodative orthoses.	4.0
Υ.	Cas	sting Techniques	
	1.	Explain the steps for obtaining both neutral and prone suspension casts.	4.0
	2.	Explain a systematic approach to evaluating a negative cast.	4.0
	3.	Explain the effect of errors in technique on the negative cast.	4.0
	4.	Discuss the advantages and disadvantages of various neutral position casting techniques	5.
			4.0
	5.	Describe the type of pathology that is captured in a negative plaster cast.	4.0
	6.	Identify the biomechanical deformities based on the negative cast.	4.0
	7.	Describe the types of casting techniques used to fabricate foot orthoses.	4.0
	8.	Recommend an appropriate casting technique, given a particular orthoses type.	4.0
	9.	Describe the rationale for obtaining an impression of the foot.	4.0
	10.	Discuss the pros and cons of using plaster versus fiberglass (STS).	4.0
	11.	Discuss the pros and cons of computerized imaging techniques.	4.0
Ζ.	Ort	thoses Fabrication	
	1	Discuss the steps involved in fabricating an orthoses and identify manufacturing errors i	n each
	1.	of these steps.	3.5
	2.	Discuss the types of pouring techniques and when each technique is utilized.	3.5
	3.	Describe the proper technique for forming a positive cast.	3.0
	4.	Compare and contrast positive and negative casts.	4.0
	5.	Discuss the different posting techniques and corrections incorporated into an orthoses	device.
			4.0
	6.	Discuss the theories of soft tissue accommodations on a positive cast.	4.0
	7.	Select the appropriate positive cast modifications, given a clinical scenario.	4.0
	8.	Describe the different materials used in fabrication of orthoses devices and their advant	ages
		and disadvantages.	4.0
	9.	Discuss the difference between a milled and a vacuum pressed orthoses.	3.0
	10.	List indications and contraindications to the use of forefoot and rearfoot posts.	4.0
	11.	Select appropriate plate additions, given a clinical scenario.	4.0
	12.	Discuss the indications for a heel lift.	4.0
	13.	Discuss the indications for a metatarsal raise.	4.0
	14.	Differentiate between top cover materials.	4.0
	15.	Select the appropriate forefoot extensions, given a clinical scenario.	4.0
	16.	List the required components of an orthoses prescription.	4.0
	17.	Describe the effect that changing the thickness and width of an orthoses device has on	foot
		function.	4.0
	18.	Explain the way to determine the number of degrees the rearfoot should be posted, as	well as
		how much motion should be allowed in the rearfoot post.	4.0
	19.	Describe the effect of medial and lateral heel modifications on foot function.	4.0
	20.	Describe forefoot balancing techniques including intrinsic and extrinsic posting, and exp	lain
		when to balance in positions other than zero.	4.0
	Or	thoses Evaluation	

1. Describe and demonstrate the technique used to fit an orthoses into the shoe. **4.0** 

2. 3.	Discuss the proper procedure in dispensing an orthoses to a patient. Discuss the evaluation process for a patient who has been wearing an orthoses.	4.0 4.0
0	rthoses Troubleshooting	
1. 2.	List the possible casting errors that would lead to orthoses problems. Discuss the implications of a supinated longitudinal midtarsal joint; a dorsiflexed 4th and metatarsophalangeal joint; a supinated oblique midtarsal joint axis; and a pronated sub- ioint axis.	<b>4.0</b> d 5th talar <b>4.0</b>
3.	Discuss the implications of choosing the wrong forefoot and rearfoot posts for an orthog	ses. <b>4.0</b>
4. 5.	Discuss the implications of choosing the wrong heel cup height, motion, and/or arch fill. Explain the possible ramifications of choosing the wrong material for an orthoses.	4.0 4.0
AA. SI	noe Therapy	
1. 2.	Describe the anatomy of a shoe. Describe the types of special shoes used in the scope of the podiatric practice, including extra depth and custom molded and the indications of each.	4.0 the 4.0
3. 4.	Describe the types of materials employed in shoe construction. Describe the types of modifications that can be made to shoe gear to assist with treatmedifferent foot and ankle disorders.	<b>3.0</b> ent of <b>4.0</b>
5. 6. 7	Discuss the various last shapes available for specific pathologies. Describe the determinants of proper shoe fit.	3.5 4.0
7. 8. 9.	Discuss the placement of the rocker relative to the pathology being treated. Differentiate between a rocker and a bar.	4.0 4.0 4.0
1( 1: 1:	<ul> <li>Explain a SACH heel (Solid Ankle Cushion Heel).</li> <li>Discuss the functions of a SACH heel.</li> <li>Discuss the indications and contraindications for a SACH heel</li> </ul>	3.5 4.0 3.5
13 14	<ol> <li>Biscuss the indications and contraindications for a SACH field.</li> <li>Recommend a shoe prescription for common podiatric pathologies.</li> <li>Discuss the role of the insole in regard to shoe function.</li> </ol>	4.0 4.0
1: 1(	<ol> <li>Discuss the importance of a removable insole.</li> <li>Evaluate the tread patterns for various types of function.</li> <li>Identify the types of past on share and discuss the advertance, discdurate and india</li> </ol>	4.0 4.0
1	of each.	<b>3.5</b>
10	indications for each.	es, and <b>4.0</b>
1	<ol> <li>Identity various types of healing sandals and discuss the advantages, disadvantages, and indications for each.</li> </ol>	4.0
20	<ol> <li>List several different types of metatarsal bars and discuss the differences and indication each.</li> </ol>	s for <b>4.0</b>
2: 2:	<ol> <li>L. List the steps used in the application of a metatarsal bar to a shoe.</li> <li>Distinguish between a flange, a flare, and a wedge.</li> </ol>	3.0 3.0
23 24	<ol> <li>B. Differentiate between a flange, a flare and a wedge.</li> <li>Discuss indications and contraindications for a flange, a flare and a wedge.</li> </ol>	4.0 4.0
2! 2(	<ol> <li>5. List the height limitations for in-shoe and external shoe lifts.</li> <li>5. Discuss the sole modifications that are required to use a full-length external lift.</li> <li>7. Identify indications and contrain disctions for full bouth lift.</li> </ol>	4.0 3.0
2.	<ul> <li>identity indications and contraindications for full-length lift versus a heel lift modificatio</li> </ul>	n. <b>4.0</b>

	28.	Discuss the indications for a tongue pad.	3.0
	29.	Discuss the indications for a metatarsal pad.	3.5
	30.	Discuss the indications for unilateral and for bilateral heel lifts.	4.0
	31.	Identify a Mayo pad and discuss its indications.	3.5
	32.	Discuss the indications and contraindications for a shoe excavation.	3.0
	33.	Discuss the steps in performing a shoe excavation.	2.0
	34.	Discuss methods of widening the sole of the shoe and list specific indications for doing s	ole
		modifications.	3.0
	35.	Discuss various methods for shoe stretching and list specific indications for each method	ł.
			3.0
	36.	Discuss the use of elastic laces for specific pathologies.	3.0
	37.	Discuss the use of specific lacing techniques based on specific types of pathology and ac	tivities.
			3.0
	38.	Discuss the benefits of bilaminar and trilaminar materials.	3.0
	39.	Identify indications for toe filler modifications to an insole.	4.0
	40.	Discuss insole wedging and cobra pad type modifications.	3.5
	Cus	stom Molded Shoes	
	1.	Identify indications for custom molded shoes.	4.0
	2.	Identify the materials required to make a bivalve cast.	3.0
	3.	Describe the technique used for bivalve casting.	3.0
	4.	Discuss the benefits and limitations of this casting technique.	3.0
	5.	Identify the materials required to make a univalve cast.	3.0
	6.	Describe the technique used for univalve casting.	3.0
	7.	Discuss the benefits and limitations of the univalve casting technique.	3.0
	8.	Identify the materials required to make a cast using an STS sock.	3.0
	9.	Describe the technique used for casting with an STS sock.	3.5
	10.	Discuss the consequences of applying the STS sock inappropriately.	3.5
	11.	Discuss the benefits and limitations of the STS sock.	3.5
	12.	Discuss the use of positive last modifications in the manufacture of a custom molded she	oe.
			3.0
	13.	Discuss the positive last modifications that are marked and made on the positive cast.	3.0
	14.	Discuss the upper modifications that are available and identify the indications for each.	3.0
	15.	Discuss the sole modifications available and identify the indications for each.	3.0
BB.	Bra	ices and Prosthetics	
	1.	Define an ankle-foot-orthoses (AFO) type device.	4.0
	2.	Discuss the functions of an AFO type device.	4.0
	3.	Discuss the indications and contraindications of an AFO type device.	4.0
	4.	Identify the therapeutic goals of an AFO.	4.0
	5.	Describe materials from which an AFO is made.	4.0
	6.	Define a knee-ankle-foot-orthoses (KAFO) type device.	4.0
	7.	Discuss the functions of a KAFO type device.	4.0
	8.	Discuss the indications and contraindications of a KAFO type device.	4.0
	9.	Identify and describe the custom stirrup orthotic.	4.0
	10	Discuss the casting technique used for manufacture of a custom stirrup orthotic (eg. Rite	chie
		brace).	4.0
	11.	List indications and contraindications for a custom stirrup orthotic (eg. Ritchie brace).	4.0
	-		

17	Describe the nexterior culiet type of culde fact outboard	4.0
12.	Describe the posterior splint type of ankie-toot-orthoses.	4.0
13.	Discuss the ordering and/or casting for a posterior splint type of AFO.	4.0
14.	Discuss the indications for a posterior splint AFO.	4.0
15.	Describe the functionality of a patellar-tendon bearing brace.	4.0
16.	Discuss the indications and limitations of a patellar tendon bearing brace.	4.0
17.	Identify a patellar tendon bearing brace.	4.0
18.	Describe the functionality of a gauntlet brace functions.	4.0
19.	Discuss the indications and limitations of a gauntlet brace.	4.0
20.	Identify a Charcot Restraint Orthotic Walker (CROW).	4.0
21.	Describe the functionality of a CROW.	4.0
22.	Discuss the indications and limitations of a CROW.	4.0
23.	Describe the function of a removable cast in the treatment of Charcot neuroarthropath	у.
		4.0
24.	Discuss the indications and limitations of a removable cast.	4.0
25.	Discuss the indications for a short leg versus long leg walking boot.	4.0
26.	Describe the function of a double upright brace.	4.0
27.	Discuss the indications and limitations of a double upright brace.	4.0
28.	Identify a double upright brace AFO.	4.0
29.	Identify a hinged brace type AFO.	4.0
30.	Describe the function of a hinged brace type AFO.	4.0
31.	Discuss the indications and limitations of a hinged brace type AFO.	4.0
32.	Describe the function of a dorsiflexion assist AFO.	4.0
33.	Discuss the indications and limitations of a dorsiflexion assist AFO.	4.0
34.	Discuss the concept of a Tone Reducing Ankle-Foot Orthoses (TRAFO).	4.0
35.	List the indications and contraindications for the TRAFO.	3.5

## II. <u>Pathomechanics</u>

## A. Digital Deformities

1.	Describe in detail the origin, course, and insertions, and functions of all tendons insertin	g into
	the lesser digits.	4.0
2.	Explain the effect of the extensor hood ligament on extensor tendon function.	4.0
3.	Describe the dynamic balance of tendons necessary for maintaining normal digital positi	ioning
	during the normal gait cycle.	4.0
4.	Describe the etiology and definition of Hammer Digit Syndrome.	4.0
5.	Describe 3 major theories of the etiology of hammertoes: Extensor Substitution, Flexor	
	Substitution and Flexor Stabilization.	4.0
6.	Explain other factors that could affect the etiology of hammer toes.	4.0
7.	Describe the associated foot deformities that may occur due to hammer digit syndrome	. 4.0
8.	Describe the various conservative treatments of hammer digit syndrome.	
		4.0
9.	Identify the anatomical structures that govern the function of the digits with emphasis of	on the
	role of the extensor expansion.	4.0
10	. Recognize how the extensor expansion and associated structures can create a "rigid bea	m
	effect."	4.0
11	Describe the pathomechanics of hammer toe syndrome.	4.0
12	Identify the clinical signs and symptoms associated with hammertoe deformity.	4.0

<ol> <li>Recognize maller</li> <li>Describe the pat</li> <li>Describe digit ab</li> <li>Describe digit qui</li> <li>Describe curly to</li> <li>Describe hallux i</li> <li>Discuss the etion</li> <li>Digital assessme positional digital</li> <li>Discuss associate joint dislocation.</li> </ol>	t toe and claw toe deformities. homechanics of mallet and claw toe deformities. oductus and adductus deformities and be able to explain their etiologies inti varus deformities and subtypes. be deformity. nterphalangeus. ogy and pathomechanics of functional digital imbalance. nt approach to the identification, classification, and treatment of struct deformity. ed digital conditions with predislocation syndrome and metatarsophalan	4.0 4.0 4.0 4.0 4.0 4.0 4.0 4.0 ural or 4.0 ngeal 4.0
Hallux Abducto Val	lgus and Bunion Deformities	
<ol> <li>Define hallux about the analysis of the analysis</li></ol>	ducto valgus. Atomical structures that govern the function of the first metatarsal phala as of hallux abducto valgus. cessive pronation results in the development of HAV. Aur stages of HAV development with their associated findings radiograp	4.0 angeal 4.0 4.0 4.0 nically.
6. Relate how othe	r lower extremity deformities contribute to the development of an HAV	4.0 7.
<ol> <li>Beginning with v information of se</li> <li>Describe the ada Abducto Valgus.</li> </ol>	veakness of pull of the Peroneus Longus, list the biomechanical steps th evere Hallux Abducto Valgus. aptive soft tissue and osseous changes that could result from severe Hal	4.0 at result 4.0 lux 4.0
<ol> <li>Discuss the pred</li> <li>Discuss the othe</li> </ol>	isposing factors to Hallux Abducto Valgus Formation. r predisposing factors to bunion formation besides Hallux Abducto Valg	<b>4.0</b> us. <b>4.0</b>
<ol> <li>Identify the max and Bunion Defo</li> <li>Discuss the indic</li> <li>Discuss the cons including indicat</li> <li>Define and comp</li> <li>Demonstrate exa</li> <li>Describe crepitu</li> <li>Demonstrate exa</li> <li>Discuss first ray</li> </ol>	imum and minimum range of the goals of treatment for Hallux Abducto ormities. actions for treatment of Hallux Abducto Valgus and Bunion Deformities. ervative treatment options for Hallux Abducto Valgus and Bunion Defor ions and complications. pare <i>tracking</i> and <i>trackbound</i> of the first metatarsophalangeal joint. am technique to identify if the First MPJ is trackbound. s of the first metatarsophalangeal joint. am technique to identify crepitus. hypermobility as an etiology of HAV and hallux limitus/rigidus.	Valgus 4.0 4.0 mities 4.0 4.0 4.0 4.0 4.0 4.0 4.0
Hallux Limitus, Hall	lux Rigidus & Metatarsus Primus Elevatus	
<ol> <li>Define hallux lim</li> <li>Define functiona</li> <li>List the etiologie</li> <li>Discuss the stage</li> <li>List the clinical si</li> <li>Describe the con</li> </ol>	nitus and hallux rigidus. In hallux limitus. In hallux limitus/rigidus, both biomechanical and non-biomechanical as of hallux limitus/rigidus. In hallux limitus/rigidus. In pensation and mechanisms for hallux limitus and rigidus.	4.0 4.0 4.0 4.0 4.0 4.0
	<ol> <li>Recognize maller</li> <li>Describe digit ab</li> <li>Describe digit qu</li> <li>Describe curly to</li> <li>Describe hallux i</li> <li>Discuss the etiol</li> <li>Digital assessme positional digital</li> <li>Discuss the etiol</li> <li>Digital assessme positional digital</li> <li>Discuss associate joint dislocation.</li> <li>Hallux Abducto Val</li> <li>Define hallux ab</li> <li>Describe the ana joint.</li> <li>List the etiologie</li> <li>Describe how ex</li> <li>Recognize the for</li> <li>Relate how other</li> <li>Beginning with v information of so</li> <li>Describe the ada Abducto Valgus.</li> <li>Discuss the pred</li> <li>Discuss the other</li> <li>Identify the max and Bunion Defo</li> <li>Discuss the cons including indicat</li> <li>Describe crepitu</li> <li>Describe crepitu</li> <li>Describe crepitu</li> <li>Define hallux limital</li> <li>Define hallux limital<td><ol> <li>Recognize mallet toe and claw toe deformities.</li> <li>Describe the pathomechanics of mallet and claw toe deformities.</li> <li>Describe digit adjutus and adductus deformities and be able to explain their etiologies of Describe acity toe deformity.</li> <li>Describe curly toe deformity.</li> <li>Describe to git adjuttive and adjutus deformities and be able to explain their etiologies of Describe acity toe deformity.</li> <li>Describe acity toe deformity.</li> <li>Discuss the etiology and pathomechanics of functional digital imbalance.</li> <li>Digital assessment approach to the identification, classification, and treatment of struct positional digital deformity.</li> <li>Discuss associated digital conditions with predislocation syndrome and metatarsophalar joint dislocation.</li> <li>Hallux Abducto Valgus and Bunion Deformities</li> <li>Define hallux abducto valgus.</li> <li>Describe the anatomical structures that govern the function of the first metatarsal phale joint.</li> <li>List the etiologies of hallux abducto valgus.</li> <li>Describe how excessive pronation results in the development of HAV.</li> <li>Recognize the four stages of HAV development with their associated findings radiograph</li> <li>Relate how other lower extremity deformities contribute to the development of an HAV</li> <li>Beginning with weakness of pull of the Peroneus Longus, list the biomechanical steps th information of severe Hallux Abducto Valgus.</li> <li>Discuss the predisposing factors to Hallux Abducto Valgus Formation.</li> <li>Discuss the other predisposing factors to bunion formation besides Hallux Abducto Valgus.</li> <li>Discuss the oncharities.</li> <li>Discuss the oncharities.</li> <li>Discuss the oncharities and and minimum range of the goals of treatment for Hallux Abducto and Bunion Deformities.</li> <li>Describe repetives of the first metatarsophalangeal joint.</li> <li>Describe the adaptive soft treatment of Hallu</li></ol></td></li></ol>	<ol> <li>Recognize mallet toe and claw toe deformities.</li> <li>Describe the pathomechanics of mallet and claw toe deformities.</li> <li>Describe digit adjutus and adductus deformities and be able to explain their etiologies of Describe acity toe deformity.</li> <li>Describe curly toe deformity.</li> <li>Describe to git adjuttive and adjutus deformities and be able to explain their etiologies of Describe acity toe deformity.</li> <li>Describe acity toe deformity.</li> <li>Discuss the etiology and pathomechanics of functional digital imbalance.</li> <li>Digital assessment approach to the identification, classification, and treatment of struct positional digital deformity.</li> <li>Discuss associated digital conditions with predislocation syndrome and metatarsophalar joint dislocation.</li> <li>Hallux Abducto Valgus and Bunion Deformities</li> <li>Define hallux abducto valgus.</li> <li>Describe the anatomical structures that govern the function of the first metatarsal phale joint.</li> <li>List the etiologies of hallux abducto valgus.</li> <li>Describe how excessive pronation results in the development of HAV.</li> <li>Recognize the four stages of HAV development with their associated findings radiograph</li> <li>Relate how other lower extremity deformities contribute to the development of an HAV</li> <li>Beginning with weakness of pull of the Peroneus Longus, list the biomechanical steps th information of severe Hallux Abducto Valgus.</li> <li>Discuss the predisposing factors to Hallux Abducto Valgus Formation.</li> <li>Discuss the other predisposing factors to bunion formation besides Hallux Abducto Valgus.</li> <li>Discuss the oncharities.</li> <li>Discuss the oncharities.</li> <li>Discuss the oncharities and and minimum range of the goals of treatment for Hallux Abducto and Bunion Deformities.</li> <li>Describe repetives of the first metatarsophalangeal joint.</li> <li>Describe the adaptive soft treatment of Hallu</li></ol>

	8. 9. 10. 11. 12.	Describe the etiologies of Metatarsus Primus Elevatus. Describe the clinical signs and symptoms associated with Metatarsus Primus Elevatus. Describe the compensations for Metatarsus Primus Elevatus. Describe the principles of orthoses prescription writing for patients with hallux limitus, limitus, limitus and Metatarsus Primus Elevatus. Identify the conservative interventions for alleviation of symptoms associated with hallu limitus/rigidus.	4.0 4.0 1.0 1.0 1.0 1.0 4.0 4.0
D.	На	llux Varus	
	1. 2. 3. 4. 5.	Define <i>hallux varus</i> . List the etiologies of hallux varus. Compare the juvenile and adult forms of hallux varus. List the clinical signs and symptoms associated with hallux varus. Identify the conservative interventions for alleviation of symptoms associated with hallu <b>4.0</b>	4.0 4.0 4.0 4.0 x varus.
Ε.	Les	ser Rays	
	1.	List the anatomical structures that govern the function of the fifth metatarsal phalangea	al joint. <b>4.0</b>
	2. 2	Describe the pathomechanics of the fifth ray.	4.0 4.0
	3. 4.	Discuss the five biomechanical causes of Tailor's bunions.	4.0
	5.	List the clinical signs and symptoms associated with a plantarflexed fifth ray.	4.0
	6.	List the clinical signs and symptoms associated with a dorsiflexed fifth ray.	4.0
	7.	Describe the concept of splayfoot and the associated clinical features.	4.0
	8.	Describe the normal and abnormal metatarsal parabola, including radiographic assessmentiation this parabola.	ent of <b>4.0</b>
	9.	Describe the clinical signs and symptoms associated with abnormal metatarsal parabola	. 4.0
	10.	Describe plantarflexed and dorsiflexed lesser metatarsal deformities.	4.0
	11.	Describe the clinical findings and gait changes associated with a plantarflexed lesser me	tatarsal. <b>4.0</b>
	12.	Describe the clinical signs and symptoms associated with a dorsiflexed lesser metatarsa	
	10	deformity.	4.0
	13.	List the causes of abnormal lesser metatarsal nead shape.	3.5 4 0
	14. 15.	Recognize the pathomechanics seen with predislocation syndrome/plantar plate dysfun	ction.
	16.	Identify the principles of orthoses prescription writing for a patient with metatarsalgia.	4.0 3.0
F.	Pes	s Cavus	
	1.	Describe the different etiologies of pes cavus.	4.0
	2.	Recognize the incidence of neuromuscular disease involved.	4.0
	3.	Describe the pathomechanics of pes cavus.	4.0
	4.	List and describe the common clinical findings of pes cavus.	4.0
	5.	Describe a diagnostic work-up of a patient with pes cavus.	4.0
	ט. ד	Discuss conservative treatment options for pes cavus.	4.U 4.0
	7. 8.	Discuss different theories of pathogenesis.	4.0 4.0

	9. 10. 11.	Discuss how foot and leg compensate. Describe treatment considerations and recognize surgery may be required. Diagnose and treat neuromuscular pes cavus, when given a clinical presentation.	4.0 4.0 4.0
G.	Fla	tfoot Deformities	
	1. 2. 3. 4.	Describe different etiologies, including abnormal ontogeny, of flatfoot deformity. Describe pathomechanics resulting from joint instability. Describe signs and symptoms of flatfoot deformities. Describe common treatment plans for flatfoot deformities.	4.0 4.0 4.0 4.0
Н.	Не	el Pain	
	1. 2. 3. 4. 5.	Define <i>heel pain syndrome</i> . Describe the various etiologies and pathomechanics of heel pain. Identify the clinical signs and symptoms of heel pain. Explain conservative treatments for heel pain. Discuss the subjective and objective heel pain assessment methods to differentiate heel systemic origin versus pathomechanical involvement with appropriate treatment option	4.0 4.0 4.0 pain of as. 4.0
Ι.	Sin	us Tarsi Syndrome	
	1. 2. 3.	Describe the pathomechanics of sinus tarsi syndrome. Identify the clinical signs and symptoms of sinus tarsi syndrome. List the conservative treatments for sinus tarsi syndrome.	4.0 4.0 4.0
J.	Evo	aluation and Management of the "At Risk" Foot	
	1. 2.	Describe the biomechanical management of the "at-risk foot" due to diabetes, peripher vascular disease, neurological, or other metabolic disorders. Discuss conservative treatment options for the "at-risk foot."	al 4.0 4.0
III.	<u>Sp</u>	orts Medicine	
А.	Spo	orts Medicine Practice	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> </ol>	Describe the psychological, social and physical characteristics unique to the sports medi patient. Differentiate between a general medical history and physical exam versus a sports medi history and physical. Discuss the psychological aspects of the competing, elite and special needs athlete. List the benefits, challenges and factors unique to a sports medicine practice. Discuss the benefits of being part of a sports medicine team. Compare the evaluation and management of the child athlete with that of an adult athlete compare and contrast the approach and surgical management of the athlete patient ve nonathlete patient	cine 3.0 cine 3.5 3.0 3.0 3.0 ete. 4.0 rsus the 4.0
	8. 9. <b>Th</b>	Identify the effects of gender on training, conditioning, endurance, and injury. Describe the assessment the injured athlete on the field.	4.0 4.0

## 1. Describe the psychological, sociological, and cultural challenges facing a female athlete. **3.0**

2.	Describe the problems associated with amenorrhea and osteoporosis in the female athl	ete.
3. 4. 5.	Describe the effect of diet and eating disorders in the female athlete and their effects of menstrual cycle. Identify the effects of oral contraceptives on the female athlete. Discuss the current trends in exercise during pregnancy in the female athlete.	3.0 3.0 3.0 3.0
The	e Aging Athlete	
1. 2. 3. 4.	Describe the psychological, sociological, and cultural challenges facing an aging athlete. Identify the effects of age on training, conditioning, endurance, and injury. Identify the effects of chronic conditions and medications on the aging athlete. Describe the nutrition needs to the unique to the aging athlete.	3.0 3.0 3.0 3.0
The	e Child Athlete	
<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> </ol>	Discuss the physical, anatomical, and biomechanical differences between the child/imm athlete and the adult athlete. Describe the general growth process and its effect on athletic participation. List the areas of structural weakness in the child athlete. Discuss the various injuries and conditions specific for the child athlete. Recommend prescription or treatment modalities and protocols for specific injuries con the child athlete. List the effects and limitations of training and conditioning in the child athlete. Discuss the unique challenges of understanding and treating the adolescent athlete.	ature 3.5 4.0 4.0 4.0 mon in 4.0 3.0 3.0
The	e Special Needs Athlete	
1. 2. 3.	Describe the psychological, sociological, and cultural challenges facing a special needs at Identify the effects of special needs on training, conditioning, endurance, and injury. Identify the effects of medical conditions and medications on the special needs athlete.	thlete. 3.0 3.0 3.0
Spo	orts Nutrition	
1. 2. 3.	Describe the nutritional needs of the athlete and how they differ from the general population of the common nutritional supplements and fluid replacement products used by athletes. List a variety of "doping" or banned substances used by athletes.	ilation. 3.0 3.0 3.0
Тес	chniques of Training	
1. 2. 3. 4. 5.	Discuss the basic training techniques and nomenclature used by athletes, such as long si distance, intervals, tempo runs, circuit training, sets, and plyometrics. Discuss the basic techniques and benefits of unique training forms such as dance, yoga, and martial arts. Explain the principles of conditioning, stretching, and strength training. List the factors affecting endurance and performance. Discuss the rationale for sports-specific training.	low 3.0 Pilates 3.0 3.0 3.0 3.0
Bio	mechanics of Running	

1. Differentiate between the gait cycles of running versus walking.4.0

	2. 3. 4. 5.	Identify the effects of speed on the running gait cycle. Describe how the loads through the foot differ between walking and running. Compare the differences in phasic muscle activity in walking as compared to running. Describe the abnormal running biomechanics and its relationship to athletic performance the development of injury. Compare and contrast the gait variances of shod versus unshod running.	4.0 3.5 3.0 :e and 3.5 3.0
	Fo	ot Orthoses in the Athlete	
	1.	Describe the unique considerations when prescribing orthoses for the sports medicine p	atient. 3.5
	2. 3.	Outline the indications for various orthotic modifications used for treatment of specific injuries. Differentiate between the specialized orthoses used in specific sports, such as skiing, ma	sports 3.5 arathon,
		track, cycling, dance, skating, and basketball.	3.0
	Atl	hletic Footwear	
	1. 2.	Describe the anatomy, construction, and function of a various athletic shoes, such as run walking, court, turf, dance, and cycling. Discuss the current techniques and modifications used in the fabrication of athletic foot	nning, <b>4.0</b> wear
	3.	Identify and describe common running shoe wear patterns and the biomechanical, clinic therapeutic significance, including the shoe prescription.	3.0 cal, and 4.0
	Spo	orts Equipment & Training Aids	
	1.	Explain basic bike fit techniques and describe the common lower extremity injuries seer improper fit.	with <b>3.5</b>
	2.	Identify and describe the high tech training aids used by athletes to assess fitness, such rate monitors and power meters.	as heart 4.0
р	5. Cn/		4.0
ь.	spo	orts injuries	
	Asy	ymmetry in the Athletic Patient	
	1.	Explain the pathological basis of asymmetrical function and the presence or potential fo	r injury. <b>4.0</b>
	2.	Review the appropriate treatment plans for the athlete with various limb length discrep	ancies.

4.0

#### Stress Fractures of the Lower Extremity

- 1. Discuss the pathomechanics of loads and their relationship to injury to bony tissues. **4.0**
- Describe how training errors, nutritional status, gender, age, and other special considerations contribute to development of stress fracture.
   4.0
- 3. Discuss the clinical presentation and management of tibial stress fracture. **4.0**

#### Capsular/Joint Impingement Syndromes

- 1. Explain the pathomechanics producing impingement syndromes.4.0
- 2. Differentiate between soft tissue impingement syndromes and bony impingement syndromes, such as Hallux IPJ, First MPJ, lesser MPJ's, calcaneocuboid joint (subluxed cuboid

syndrome), subtalar joint (sinus tarsitis), and ankle joint (anterior, posterior, medial and lateral impingement syndrome). 4.0

#### Lower Extremity Tendonopathy

- 1. Discuss the causes for acute or chronic injuries to specific tendons. 4.0
- 2. Differentiate between the different types of tendonopathy, such as tendonosis, tenosynovitis, and enthesopathy. 4.0
- 3. Explain the relationship between abnormal biomechanical function and development of injury to specific tendons.
- 4.0 4. List the signs and symptoms associated with tendonopathy.
- 5. Describe the various methods of treatment for tendonopathy.
- 6. Discuss diagnositic imaging techniques used in the evaluation of tendonopathy. 4.0
- 7. Describe how training errors, nutritional status, gender, age, and other special considerations 4.0 (eg, antibiotics and steroid) contribute to development of tendonopathy.

#### Hip and Thigh

- 1. Describe clinical presentation, evaluation, and management of athletic injuries of the hip and thigh, such as trochanteric bursitis, iliotibial band friction syndrome, piriformis syndrome," snapping hip," hamstring strain, and quadriceps strain. 3.5
- 2. Discuss the pathomechanical factors contributing to sport specific hip and thigh injuries. **3.5**

#### Knee

- 1. Describe clinical presentation, evaluation, and management of athletic injuries of the knee, such as chondromalacia patella, patellofemoral joint syndrome, popliteal tendonitis, iliotibial band friction syndrome, meniscal tears, ACL and PCL tears, collateral ligament sprain, plica, impingement syndrome, patellar tendonitis ("jumper's knee"), and pes anserine bursitis.
- 2. Discuss the pathomechanical factors contributing to sport-specific knee injuries.

4.0

4.0

4.0

- 3. Describe and demonstrate the evaluation techniques used to determine the integrity of the patellar tendon, collateral ligaments, cruciate ligaments and menisci of the knee joint. 4.0 4.0
- 4. Discuss how the "Q" angle relates to patellar tracking.
- 5. Describe the effect of foot dysfunction on knee pathomechanics. 4.0

#### Leg

- 1. Describe clinical presentation, evaluation, and management of athletic injuries of the leg, such as acute and chronic compartment syndromes, "shin splints" [ie, anterior compartment myositis, deep posterior compartment myositis/Medial Tibial Stress Syndrome (MTSS)], peroneal tendonitis, and tibial stress fracture. 4.0 4.0 2. Discuss the pathomechanical factors contributing to sport-specific leg injuries.
- 3. Describe clinical presentation, evaluation, and management of tennis leg. 4.0

#### Ankle

- 1. Describe and demonstrate how to identify and assess the integrity of the lateral collateral ligaments of the ankle. 4.0
- 2. List the pathomechanical factors that may predispose patient to lateral ankle sprains. 4.0

- Describe and demonstrate the clinical evaluation of lateral ankle sprains, including the need for assessment of non-ankle structures such as the base of the fifth metatarsal, anterior process of the calcaneus, Achilles tendon insertion, peroneal groove, and the proximal fibula.
   4.0
- 4. Differentiate between types of ankle sprains, such as lateral, medial, and high ankle sprains.
- 5. Discuss of clinical presentation, imaging, and treatment of talar dome injury.
- Describe the specialized radiographic techniques utilized to assess lateral ankle injuries and grade their severity.
   4.0
- Describe the treatment and return-to-activity protocols for lateral ankle injuries based on grade/severity of injury.
   4.0
- Describe the biomechanical etiology, clinical presentation, specialized radiographic findings, and the management of fibular (peroneal) tendon subluxation.
   4.0
- 9. Describe the clinical presentation and management of injuries to the os trigonum. **4.0**
- Describe the biomechanical etiology, clinical presentation, specialized radiographic findings, and the management of sport specific anterior and posterior impingement syndrome.
   4.0

#### Rearfoot

- Describe clinical presentation, evaluation, and management of athletic injuries of the rearfoot, such as Achilles tendinitis, Achilles tendon rupture, paratenonitis, adhesive tendinopathy, calcaneal stress fractures, calcaneal apophysitis, cuboid impingement syndrome, and plantar fasciitis.
   4.0
- 2. Discuss the pathomechanical factors contributing to sport-specific rearfoot injuries. **4.0**

#### Midfoot

- Describe clinical presentation, evaluation, and management of athletic injuries of the midfoot, such as navicular stress fracture, and midfoot sprains.
   4.0
- 2. Discuss the pathomechanical factors contributing to sport-specific midfoot injuries. **4.0**

#### Forefoot

- Describe clinical presentation, evaluation, and management of athletic injuries of the lesser metatarsal, such as stress fractures, avulsion fracture, Jones fracture, capsulitis, and plantar plate rupture.
- Describe clinical presentation, evaluation, and management of athletic injuries of the first metatarsophalangeal joint, including turf toe, soccer toe, sesamoiditis, plantar plate injuries, and impingement syndrome.
   4.0
- 3. Discuss the pathomechanical factors contributing to sport-specific forefoot injuries. **4.0**

#### Dermatology

- Describe the etiology, clinical presentation, management, and prevention of sports-related dermatological conditions, such as subungual hematoma ("Tennis Toe"), blisters, fungal and bacterial infections, taping/bracing skin reactions, and dermal abrasions.
- 2. Discuss the implications of MRSA infections in the athlete. **4.0**
- 3. Discuss the importance of the appropriate sport-related sock. **4.0**
- C. Physical Medicine and Rehabilitation

#### Patient Assessment

4.0

4.0

ROM, strength, power, endurance, and improving balance and proprioception. 4.0 7. Describe the methods for the determination of the patient's readiness to return to activities. 4.0 **Physical Therapy Modalities** 1. Describe the various types of active and passive range of motion exercises and their indications and contraindications. 4.0 2. List the indications and contraindications for the use of therapeutic cold and heat in the lower extremities. 4.0 3. List the indications and contraindications of phonophoresis, iontophoresis, electrical stimulation, and ultrasound. 3.5 4. List the indications and contraindications for massage, soft tissue mobilization, traction, and manipulation techniques of the lower extremities. 3.5 5. Outline the indication for hydrotherapy as it pertains to the treatment of lower extremity 3.5 pathology. 6. Discuss the indications and contraindications of strength training and methods for specific muscle groups. 3.5 7. Discuss specific strengthening techniques such as isometric, isotonic, isokinetic, concentric/eccentric, and open and closed kinetic chain in the rehabilitation. 3.5 Describe the concepts of proprioceptive retraining of the lower extremities and its importance in injury management and prevention of further injury. 3.5 9. Write a therapeutic exercise prescription. 3.0 10. Discuss the indications and contraindications of intermittent compression as a modality. 3.0 11. List the compression garments and explain when to prescribe them as a treatment. 3.0 **Rehabilitative Equipment** 4.0 1. Describe indications for canes and crutches and other ambulatory assistive devices. 2. Demonstrate the proper fit, use, and patient instructions for a variety of ambulatory assistive devices, including cane, crutches, walker, and wheelchair. 4.0 3. Describe the indications, contraindications, and adverse effects for the use of immobilizing devices such as casts, ambulatory boots, and AFOs. 4.0 4. Demonstrate the proper donning and doffing of commonly used immobilization devices. 4.0 5. Discuss the indications and contraindications for the use of a variety of available knee and ankle braces and supports. 4.0

1. Discuss the role of the physical therapy and physical medicine in the treatment of lower

2. Distinguish which patients are appropriate for referral to physical therapy and physical

3. Discuss the range of motion of the joints of the lower extremities discriminating between active

reducing inflammation, pain spasm, edema, and scar tissue and adhesions, as well as increasing

5. Identify the indications for additional objective testing of strength and power (eg, isokinetic

6. Describe the common goals of physical therapy for lower extremity conditions, including

extremity pathology.

and passive motion.

4. Assess muscular strength and power manually.

instrumentation and computerized gait analysis).

medicine.

#### IV. General Orthopedics and Disorders of Bone

4.0

4.0

4.0 4.0

4.0

#### A. Soft Tissue Neoplasms

- Describe the general histopathological classification, etiology, and pathophysiology of soft tissue neoplasms.
   3.0
- 2. Describe the diagnostic modalities utilized in assessing soft tissue neoplasms.
- Discuss the clinical presentation and management of benign fibrous tumors, as well as malignant fibrosarcoma.
   4.0
- Discuss the clinical presentation and management of lipomatous tumors such as the lipoma and the malignant liposarcoma.
   4.0
- 5. Discuss the clinical presentation and management of benign tumors of smooth muscle including the leiomyoma, as well as the malignant leiomyosarcoma. **4.0**
- 6. Discuss the clinical presentation and management of benign tumors of skeletal muscle including the rhabdomyoma, as well as the malignant rhabdomyosarcoma. **4.0**
- 7. Discuss the clinical presentation and management of benign tumors of the vasculature including hemangioma, pyogenic granuloma, glomus tumor, as well as the malignant angiosarcoma.
  - 4.0

4.0

4.0

- Discuss the clinical presentation and management of benign tumors of tendon and synovial tissue, including synovial cyst (ganglion), tenosynovial giant cell tumor (pigmented villonodular synovitis), as well as the malignant synovial sarcoma and clear cell sarcoma.
   4.0
- Describe the clinical presentation and management of benign tumors of nerve tissue, including nerve sheath ganglion, neurilemmoma, and neurofibroma.
   4.0
- Describe the clinical presentation and management of quasi-tumors of the foot, including foreign body inclusion cyst.
   4.0

#### B. Osseous Neoplasms

- 1. Describe the clinical approach to the radiographic finding of an osseous neoplasm. **4.0**
- 2. List the characteristics utilized to categorize osseous tumors.
- Describe the most common benign osseous tumors, including osteoma, osteoid osteoma, chondroblastoma, enchondroma, chondromyxoid fibroma, osteochondroma, unicameral bone cyst, aneurysmal bone ccyst, fibrous dysplasia, nonossifying fibroma, and intraosseous ganglion and lipoma, as well astheir individual radiographic presentations.
- Describe the most common malignant osseous tumors, including osteogenic sarcoma, chondrosarcoma, Ewing's sarcoma, fibrosarcoma, lymphoma, and myeloma, as well as their individual radiographic presentations.
   4.0
- 5. Discuss the most common quasi-malignant osseous tumors, including giant cell tumor, as well astheir individual radiographic presentations. **4.0**
- Describe the clinical and radiographic characteristics that allow the clinician to differentiate benign from malignant tumors.
   4.0

#### C. Rheumatology

#### **Systemic Sclerosis**

- Discuss scleroderma with regards to epidemiology, clinical presentation, diagnosis, treatment, and prognosis.
   4.0
- Describe Raynaud's phenomenon and differentiate Raynaud's phenomenon from Raynaud's disease.
   4.0

#### Lupus Erythematosus

 Discuss systemic lupus erythematosus (SLE) with regards to epidemiology, clinical presentation, diagnosis, treatment, and prognosis.
 4.0

#### Polymyalgia Rheumatica and Giant Cell Arteritis

- Discuss polymyalgia rheumatica with regard to epidemiology, clinical presentation, diagnosis, and treatment.
   Discuss giant cell arteritis with regards to epidemiology, clinical presentation, complications
- Discuss giant cell arteritis with regards to epidemiology, clinical presentation, complications, diagnosis, and treatment.
   4.0
- Compare and contrast polymyalgia rheumatica and giant cell arteritis with each other and with other rheumatologic diseases.
   3.0

#### Fibromyalgia & Chronic Myofascial Pain

1.	Discuss the epidemiology of fibromyalgia.	4.0
2.	Discuss the diagnostic criteria for fibromyalgia.	4.0
3.	Discuss disorders that are associated with fibromyalgia.	4.0
4.	Describe the clinical presentation of fibromyalgia.	4.0
5.	Define <i>trigger point</i> and discuss the clinical relevance of trigger points.	4.0
6.	Outline treatment strategies for fibromyalgia.	4.0
7.	Discuss other possible etiologies of myofascial pain.	4.0

#### D. Mechanical/Structural Conditions of the Spine

Ε.

1.	Describe the normal anatomy of the spine.	4.0
2.	Describe the normal ontogeny of the spine.	3.0
3.	Discuss the various etiologies and types of scoliosis including the possible locations for t	he
	deformity.	3.0
4.	Discuss the signs and symptoms associated with scoliosis.	3.0
5.	Describe and perform a screening exam for scoliosis.	4.0
6.	Discuss radiographic techniques to diagnose scoliosis.	3.0
7.	Describe common gait changes associated with scoliosis.	4.0
8.	Describe the effects on the rest of the body of eliminating compensatory changes in the	feet for
	patients with scoliosis.	4.0
9.	Discuss the clinical findings, associated function and/or gait disturbances, and treatmen adult spinal disorders, including spinal osteoarthritis, spinal stenosis, kyphosis, herniated intervertebral disk and lumbosacral strain, cervical strain, cervical spondylosis, whiplash injury, fracture of spinal process, flexion fracture of the neck, partial dislocation from hyperextension injury, atlas fracture, and odontoid process fracture.	t of d cervical <b>3.0</b>
Me	chanical and Structural Conditions of the Hip	
1.	Measure the ranges of motion for the hip.	4.0
2.	Evaluate the strength of the muscles crossing the hip joint.	4.0
3.	Evaluate the effect of the hamstrings on the amount of hip flexion available.	4.0

- 4. Evaluate the effect of the quadriceps on the amount of hip extension available. **4.0**
- 5. Evaluate a patient for the presence of coxa varum or coxa valgum.
- 6. Discuss the effect of coxa varum and coxa valgum on the gait cycle.4.0
- Evaluate a patient for iliotibial band syndrome and discuss biomechanical etiologies or factors associated with this diagnosis.
   4.0

4.0

8. Describe the clinical findings, associated function and/or gait disturbances, and treatment of adult hip disorders including osteoarthritis, trochanteric bursitis, acute fracture and/or dislocation, and hip replacement. 4.0

#### F. Mechanical and Structural Conditions of the Knee

	1.	Evaluate the knee to determine the integrity of the collateral ligaments (varus and valge	us stress
		test).	4.0
	2.	Evaluate the knee to determine the integrity of the cruciate ligaments (anterior and pos	terior
		drawer test).	4.0
	3.	Evaluate the knee to determine the integrity of the menisci of the knee.	4.0
	4.	Evaluate a patient for the presence of genu varum or genu valgum.	4.0
	5.	Determine the Q angle on a patient.	4.0
	6.	Evaluate a patient for "tracking" of the patella.	4.0
	7.	Identify and describe the signs and symptoms of chondromalacia patella.	4.0
	8.	Evaluate a patient for quadriceps tone and the presence of chondromalacia patella.	4.0
	9.	Evaluate a patient for pes anserine bursitis and discuss possible biomechanical etiologie	S
		associated with this diagnosis.	4.0
	10.	Differentiate between patello-femoral syndrome and chondromalacia patella.	4.0
	11.	Evaluate the muscles crossing the knee joint.	4.0
	12.	Describe the clinical findings, associated function and/or gait disturbances, and treatme	nt of
		adult knee disorders including Baker's cyst, prepatellar bursitis and infrapatellar bursitis	,
		sprain/rupture of the collateral ligaments, sprain/rupture of the cruciate ligaments, tea	r/rupture
		of the menisci, osteoarthritis with or without loose bodies, and knee joint replacement.	4.0
	13.	Describe synovial joint examination, technique, and analysis.	4.0
G.	Воі	ne Healing and Facture Management	
	1.	Discuss the development and pathomechanic implications of stress reaction and stress	fracture.
		······································	4.0
	2.	Discuss tissue healing principles and bone healing/remodeling.	4.0
	3.	Discuss the common fracture types and management.	4.0
	4.	Discuss the common diagnostic tests used in the diagnosis of orthopedic pathology.	4.0
	5.	Discuss the general conservative and operative management of orthopedic disorders.	4.0
	6.	Discuss the regional interdependence and its implications in treating orthopedic pathole	ogv.
			<b>4.0</b>
	7.	Discuss pathophysiology of bone healing and fracture management.	4.0
	_		
Н.	Ort	hopedic Strapping	
	1.	Discuss the indications, contraindications, and alternatives for orthopedic strapping.	4.0
	2.	Identify the materials and basic techniques for orthopedic strapping.	4.0
Ι.	Ort	hopedic Padding	
	1.	Discuss the indications, contraindications, and applications for paddings.	4.0
	2.	Discuss the alternatives, if any, for L & M pad, metatarsal pad, metatarsal raise,	
		dancer's/sesamoid pad, heel lift, longitudinal arch pad, mayo pad. cuboid pad. varus/va	lgus pad.
		morton's extension, reverse morton's extension, digital/ buttress/crest pad. and horses	hoe pad.
			4.0

3. Identify the materials available for orthopedic padding. 4.0 Apply a L & M pad, metatarsal pad, metatarsal raise, dancer's/sesamoid pad, heel lift, longitudinal arch pad, mayo pad, cuboid pad, varus/valgus pad, morton's extension, reverse morton's extension, digital/ buttress/crest pad, and horseshoe pad.
 4.0

#### V. <u>Pediatric Orthopedics</u>

#### A. Prenatal Development, Birth, and Perinatal Development

	1.	Describe normal prenatal development.	3.0
	2.	Describe embryology, ontogeny, and developmental changes in the lower extremities.	3.0
	3. ⊿	List the important milestenes of each trimester	3.0
	4. 5	Describe both normal and abnormal labor and delivery	3.0
	5. 6	list important differential factors implications and variations in the normal and abnorm	al hirth
	0.	process	3.0
	7.	Review significant factors that affect neurological maturation.	3.0
	8.	Describe normal neonatal development.	3.0
	9.	Discuss perinatal development as a function of neurological maturation.	3.0
	10.	Given a description of a newborn, determine the APGAR score and discuss the significar	ice of
		the score.	2.0
	11.	Describe maternal health as related to age, weight, smoking, fetal alcohol syndrome, dia	abetes,
		hypertension, and substance abuse.	3.0
В.	Рес	diatric History	
	1.	Discuss the chronology of the complaint.	3.0
	2.	Discuss the developmental landmarks and provide normal ages for each of the landmark	ks to be
		achieved.	3.0
	3.	Obtain a family history including number, age, and significant medical history of siblings	and
		adult history information.	3.0
	4.	Discuss the systems review in the pediatric patient.	3.0
	5.	Discuss the relevance of the medication allergy and immunization histories.	3.0
	6.	Discuss comorbidities found in children that make their treatment unique.	3.0
	7.	Discuss diseases unique to infancy and childhood, such asmeasles, mumps, rubella, chic	ken pox,
	_	fifths disease, rheumatic fever, and polio.	3.0
	8.	Discuss problems associated with the patient not being the historian when executing a	medical
		history.	3.0
С.	Рес	diatric General Physical Examination	
	1.	Recognize the differences in general physical examination results for an infant, toddler,	and
		child compared to an adult.	3.0
	2.	Describe techniques used to obtain vital signs in the infant, toddler, and older child. <b>3.0</b>	
	3.	Provide age-related normal values for vital signs.	3.0
	4. -	Provide possible etiologies, given an abnormal vital sign.	3.0
	5.	Discuss the significance of including and evaluating height and weight as part of the vita <b>3.0</b>	i signs.
	6.	Discuss the evaluation of the skin including color, temperature, texture, and adnexa.	3.0
	7.	Discuss the skin as a marker for disease.	3.0

## D. Osseous Growth Centers

	1. 2.	Identify osseous growth centers and chronological presentation. Identify and describe bones that are present at birth.	4.0 4.0
	3.	age five.	n and 3.0
	4.	List and identify the appearance of sesamoids, epiphyseal plates, and apophysis in the p	ediatric
	F	toot. Identify and describe normal variants that may be confused as pathology	4.0
	5. 6	Discuss the histology and physiology of the growth plate	4.0
E	0.	tooshondrosos	-110
E.	US		
	1.	Define osteochondroses.	4.0
	2. ว	Compare the mechanisms that may cause osteochondroses.	4.0
	э. Д	Indicate clinical significance of common osteochondroses	4.0
	 5.	List treatment options in the osteochondroses.	4.0
r	Co	mmon Accossory Bonos	
г.	CO	minori Accessory Borres	
	1.	List the common accessory bones of the pediatric foot.	4.0
	2.	List and describe the appearance and chronological presentation of the accessory bones	5. <b>4.</b> 0
	3. ⊿	Recognize radiographic appearance of common accessory bones.	4.0
c	4. Ca	nuicate the chinical significance of accessory bolles.	4.0
G.	Ge	neral Disease/Metabolic Disease/ Genetic Disease/Congenital Problems	
	1.	Describe anemia, lead poisoning, bone dysplasia, bone tumors, fracture management, ri and osteogenesis imperfecta as conditions associated with delayed bone maturation.	ckets,
	_		5.0
Н.	Pe	diatric Arthritides and Infections	
	1.	Discuss pain in the child and provide an algorithmic approach to pain.	3.0
	2.	Discuss "growing pains."	3.0
	3.	Define <i>juvenile myalgia</i> .	3.0
	4.	Discuss the signs, symptoms, diagnostic techniques, and treatment for the systemic forn	n of 20
	5	Juverine meumaticita artificits.	3.0
	5. 6.	Define invenile rheumatoid arthritis	4.0
	7.	Compare and contrastiuvenile rheumatoid arthritis to other inflammatory processes.	4.0
	8.	Summarize the value of lab tests used to diagnose juvenile rheumatoid arthritis.	4.0
	9.	State the clinical presentation of juvenile rheumatoid arthritis.	4.0
	10.	List the common treatment regimens in juvenile rheumatoid arthritis.	4.0
	11.	Discuss the Polyarticular variants of Juvenile Rheumatoid Arthritis.	3.0
	12.	Discuss the Pauciarticular variants of Juvenile Rheumatoid Arthritis.	3.0
	13.	Discuss the less common pediatric collagen vascular syndromes.	3.0
	14.	Define Septic Arthritis. Differentiate Septic Arthritis from Iuvenile Phaymotoid Arthritis or esteemuslitis	3.0
	15. 16	State lab tests needed to diagnose pediatric sentic arthritis	4.U 1 0
	17	Explain the clinical significance of sentic arthritis	4.0
	±7.		

	18.	Define pediatric hematogenous osteomyelitis.	4.0
	19.	State lab tests needed to diagnose pediatric hematogenous osteomyelitis.	4.0
	20.	Differentiate pediatric hematogenous osteomyelitis from juvenile rheumatoid arthritis.	4.0
	21.	Summarize the clinical significance of hematogenous osteomyelitis.	4.0
	22.	Discuss the etiology and pathology involved with hematogenous osteomyelitis.	4.0
	23.	Discuss the signs, symptoms, pathology, diagnostic techniques, treatment, and prognosi	s of
		early acute osteomyelitis.	4.0
	24.	Discuss the signs, symptoms, pathology, diagnostic techniques, treatment, and prognosi	s of late
		acute osteomyelitis.	4.0
	25	Discuss the signs, symptoms pathology, diagnostic techniques, treatment, and prognosis	of
	20.	subacute (chronic attenuated) osteonyelitis	4.0
	26	Outline clinical work-up for suspected osteomyelitis	4.0
	20.	Outline laboratory work-up for suspected osteomyelitis	4.0
	27.	Outline "bedside" work-up for suspected osteomyelitis	4.0
	20.	Outline imaging work-up for suspected osteomyelitis	4.0
	29.	Outline a treatment plan for esteemyelitis including antibiosis and surgical intervention.	4.0
	30. 21	Describe HIV and treatment available	4.0
	51.		4.0
Ι.	Net	uromuscular Diseases	
	1.	Define <i>cerebral palsy</i> .	3.0
	2.	Discuss the etiologies of cerebral palsy.	3.0
	3.	Discuss motor and sensory changes associated with neurological/neuromuscular disease	es.
		,	4.0
	4.	Discuss the orthopedic sequel of cerebral palsy.	4.0
	5.	Discuss the basic treatment for cerebral palsy.	4.0
	6.	Discuss the types of hereditary sensorimotor neuropathies.	4.0
	7.	Discuss the clinical picture associated with hereditary sensorimotor neuropathies.	4.0
	8.	Discuss the basic treatment of hereditary sensorimotor neuropathies.	4.0
	9.	Discuss the types of muscular dystrophies.	4.0
	10.	Discuss the clinical picture associated with muscular dystrophy.	4.0
	11.	Discuss principles of management of muscular dystrophy.	3.0
	12.	List common congenital medical problems, such as Down syndrome, cerebral palsy, mus	cular
		dystrophy, Ehler-Danlos syndrome, hypotonia, and neuromuscular disease.	4.0
	13.	Describe and discuss the causes/mechanisms for spasticity, athetosis, paresis, ataxia, pa	ralysis,
		atonia, ballismus, and rigidity.	3.0
	14.	Describe gait changes associated with neurological/neuromuscular diseases, including co	erebral
		palsy, Guillain-Barre, muscular dystrophy, Charcot-Marie-Tooth, polio/post-polio syndro	me,
		multiple sclerosis, post-cerebral vascular, Tabes dorsalis, accident, and Parkinsons.	3.0
	15.	Describe circumducted gait, cerebellar gait, foot slap, Trendelenberg gait, drop foot, pill	rolling,
		and scissors and explain why each occurs.	4.0
	16.	Discuss in general terms the treatment options available for gait problems associated wi	th
		neurological/neuromuscular diseases.	4.0
	17.	Recognize, identify, and describe the lower extremity manifestations and the signs and	
		symptoms and be able to suspect the neuromuscular, upper motor neuron and lower m	otor
		neuron disorders found in children including, cerebral palsy, spina bifida and disastamet	amyelia,
		muscular dystrophies, myopathies, peripheral neuropathies, hypotonia and poliomvelitis	s,
		Downs, Prader-Willi, Adams-Oliver, achondroplasia, Apert, nail-patella, Morquio. Sturge	-Weber,
		Mafucci, Goltz, Fetal Alcohol, Marfan's osteogenesis imperfect, and Ehlers-Danlos.	4.0

18.	Discuss the need for referral of the patient with congenital medical problems.	4.0
19.	Discuss the techniques used to determine muscle tone.	4.0
20.	Discuss the techniques used to determine muscle strength.	4.0
21.	Discuss the technique and location used for evaluation of deep tendon reflexes.	4.0
22.	Discuss techniques and locations for superficial reflexes.	4.0
23.	Discuss gait evaluation as a component of the neuromuscular examination.	4.0
Me	tatarsus Adductus	
1.	Define metatarsus adductus.	4.0
2.	Explain the etiological factors seen in metatarsus adductus.	4.0
3.	Describe the clinical appearance of metatarsus adductus.	4.0
4.	Describe the radiographic appearance of metatarsus adductus.	4.0
5.	Differentiate metatarsus adductus from other forefoot pathologies such as forefoot add	uctus
	andc-foot.	4.0
6.	Identify the patients that benefit from conservative treatment for metatarsus adductus.	4.0
7.	Discuss conservative treatment of metatarsus adductus.	4.0
8.	List the complications from cast therapy for metatarsus adductus.	4.0
9.	Identify which patient may need surgical correction for metatarsus adductus.	4.0
10.	Discuss significant familial factors associated with the chance of occurrence for metatars	sus
-	adductus.	4.0
11.	Differentiate between metatarsus adductus and the variants of talipes equinovarus.	4.0
12.	Discuss the physical exam findings associated with metatarsus adductus.	4.0
13.	Discuss comorbidities associated with metatarsus adductus.	4.0
14.	Provide a step-wise treatment plan for metatarsus adductus.	4.0
15.	Discuss the evaluation of the patient's response to treatment to determine resolution of	fthe
	metatarsus adductus.	4.0
16.	Discuss conservative measures, including manipulation and casting, shoegear and bracing	ig. for
-	the treatment of metatarsus adductus.	4.0
17.	Provide a step-wise treatment plan for metatarsus adductus.	4.0
18.	Discuss the evaluation of the patient's response to treatment to determine resolution of	fthe
-	metatarsus adductus.	4.0
19.	Discuss surgical options available based on the patient's age and the severity of the met	atarsus
	adductus.	4.0
20.	Discuss possible long-term sequelae of metatarsus adductus.	4.0
Tal		
Idi	ipes Equillovarus	
1.	Define talipes equinovarus.	4.0
2.	List the etiological factors of talipes equinovarus.	4.0
3.	Review the pathological anatomy of talipes equinovarus.	4.0
4.	Describe the clinical presentation of talipes equinovarus.	4.0
5.	List the three component deformities of talipes equinovarus.	4.0
6.	Discuss the familial factors for talipes equinovarus.	4.0
7.	List the four different types of talipes equinovarus and discuss the comorbidities, respor	ise to
	therapy, and other factors associated with type.	4.0
8.	Discuss the radiographic findings associated with talipes equinovarus.	4.0
9.	Discuss the techniques used in the radiographic evaluation of talipes equinovarus.	3.0
10.	List and describe the conservative treatments for talipes equinovarus.	4.0
11.	Discuss complications of treatment for talipes equinovarus.	4.0

	12.	Outline the order of approach to the deformities involved in talipes equinovarus when with casting	treated
	12	With Casting.	4.0
	13. 14	Discuss the possible complications of treatments for talpes equinovarias.	4.0
	15.	List and describe surgical approaches and procedures, for complicated and uncomplicated	ted TFV.
	10.		4.0
	Со	ngenital Dislocated Hip	
	1.	Define congenital dislocated hip.	4.0
	2.	Identify the incidence and etiology of congenital dislocated hip.	3.0
	3.	Summarize the clinical findings seen in congenital dislocated hip.	4.0
	4.	Describe, and explain the significance of, limitation of abduction, asymmetrical gluteal	folds,
		trendelenberg, sign, anchor sign, and perineal angle.	4.0
	5.	Describe the Ortolani test and discuss its clinical significance.	4.0
	6.	Describe the Barlow test and discuss its clinical significance.	4.0
	7.	Describe the Galleazzi/Allis test and discuss the clinical significance.	4.0
	8.	Discuss the radiographic views required, as well as evaluation and interpretation, for C	DH,
		includingShenton's line, Perkin's line, and acetabular index.	3.0
	9.	Describe the Thomas test and discuss its clinical significance.	3.0
	10.	Describe the Ely test and discuss its clinical significance.	3.0
	11.	Describe the Ober test and discuss its clinical significance.	3.0
	12.	Discuss imaging techniques used to evaluate for a congenital dislocated hip.	<b>3.</b> 0
	13.	List and describe treatments available for congenital dislocated hip, including success r	
	14.	Discuss the incidence of any comorbidities associated with congenital hip dislocations.	4.0 4.0
J.	Sag	gittal, Frontal, and Tranverse Plane Deformities of the Hip, Knee, and Foot	
	1.	Recognize the normal position of the newborn hip, knee, and foot.	4.0
	2.	State the normal position of the pre-walker's hip, knee, and foot.	4.0
	3.	Describe the normal position of the beginning walker's hip, knee, and foot.	4.0
	4.	Describe the normal position of the toddler's hip, knee, and foot.	4.0
	Hip	Joint	
	1.	Discuss normal transverse and frontal plane development of the hip and femur.	4.0
	2.	Describe the gait pattern associated with femoral antetorsion.	4.0
	3.	Describe the gait pattern associated with femoral retrotorsion.	4.0
	4.	Describe the gait pattern associated with femoral anteversion.	4.0
	5.	Describe the gait pattern associated with femoral retroversion.	4.0
	6.	Discuss the physical exam techniques used to distinguish between versional and torsion	nal
		problems.	4.0
	7.	Discuss the possible treatments for versional and torsional problems.	4.0
	8.	Discuss the normal frontal plane development of the femur.	4.0
	9.	Provide normal values for the angle of inclination related to age.	4.0
	10.	Define <i>coxa varum</i> and discuss associated deformities and gait abnormalities.	4.0
	11.	Define <i>coxa valgum</i> and discuss associated deformities and gait abnormalities.	4.0
	12.	Discuss treatments available for coxa varum and coxa valgum.	4.0
	13.	compare the internal and external transverse plane hip pathology.	4.0

14.	Discuss transverse plane hip range of motion, including logic and techniques for measur the hip flexed and the hip extended, age-related normal values, and clinical significance abnormal findings	ing with of
15.	Discuss frontal plane hip range of motion including method of measurement, age-relate	d
	normal values, and discuss clinical significance of abnormal findings.	4.0
16.	Discuss sagittal plane hip range of motion including method of measurement, age-relate	ed
	normal values, and clinical significance of abnormal findings.	4.0
Kn	ee Joint	
1.	Discuss the normal frontal plane development of the knee/tibial segment.	4.0
2.	Discuss the physical exam findings associated with tibial varum.	4.0
3.	Discuss possible etiologies of pathological tibial varum.	4.0
4.	Describe the pathological process involved in Blount's disease.	4.0
5.	Describe the resultant gait and possible long-term sequelae of pathological tibial varum	4.0
6.	Differentiate between genu varum and tibial varum.	4.0
7.	Discuss the physical exam findings associated with tibial valgum.	4.0
8.	Discuss possible etiologies for pathologies tibial valgum and genu valgum.	4.0
9.	Discuss the resultant gait and possible long-term sequelae of pathological tibial valgum	or genu
4.0	valgum.	4.0
10.	Describe the method used to evaluate tibial torsion and provide normal values for the c	linical
11	measurements.	4.0
11.	Differentiate between tibilal torsion and malleolar position.	4.0
12.	Define retrotorsion and discuss the gait nattern associated with retrotorsion of the tibia.	2.0
13. 1/	Define antetorsion and discuss the gait pattern associate with antetorsion of the tibia	3.0
14.	Discuss the treatments available for tibial retrotorsion and tibial antetorsion	3.0
16	Discuss the recurrence available for riskin recrotorsion and riskin arrectorsion.	l clinical
10.	significance of abnormal findings.	3.0
17.	Discuss knee motion including method of measurement, age-related normal values, and	l clinical
	significance of abnormal findings.	3.0
18.	Discuss the normal frontal plane development of the knee/tibial segment.	3.0
Peo	diatric Gait	
1	Describe normal and abnormal gait as a function of age	4.0
1. 2	Identify and discuss abnormal gait for nediatric age	4.0
3.	Recognize, identify, describe, and evaluate deviations from normal gait, including their	-110
5.	management.	4.0
4.	Recognize, identify, describe, and evaluate causes of toe-walking and their management	t in
	children.	4.0
5.	Summarize the use of external devices for assistance in pediatric gait.	4.0
In-t	toe Gait	
1	Differentiate between physiological in-toe gait and pathological in-toe gait	40
2	Discuss early childhood gait as a function of anatomical position and neuromuscular	-7.0
	development.	4.0
3.	Describe the pediatric entity of in-toe gait.	4.0
4.	List the etiology and incidence of in-toe gait.	4.0
5.	Discuss transverse plane changes related to in-toe gait.	4.0

6.	List and describe the non-ambulatory devices used in the treatment of in-toe gait.	4.0
7.	List and describe the orthotic devices used in the treatment of in-toe gait.	4.0
8.	Compare treatment versus benign neglect for iln-toe gait.	4.0
9.	Dscribe the complications of the treatment of in-toe gait.	4.0
10.	Identify and describe appropriate footwear for children and the types, and indications for	or,
	prescription footwear in the management of pedal pathology.	4.0
Fla	tfoot Deformities	
1.	Differentiate between flexible and rigid flatfoot deformities.	4.0
2.	Define talipes calcaneovalgus.	.0
3.	Discuss possible etiologies for talipes calcaneovalgus.	4.0
4.	Discuss the physical findings associated with talipes calcaneovalgus.	4.0
5.	Discuss the radiographic findings for talipes calcaneovalgus.	4.0
6.	Provide a treatment plan for a patient with talipes calcaneovalgus.	4.0
7.	Differentiate between talipes calcaneovalgus and congenital convex pes valgus.	4.0
8.	Describe the soft tissue and bony pathology involved in talipes calcaneovalgus, as well a	s any
	associated deformities.	4.0
9.	Describe any long-term sequelae associated with talipes calcaneovalgus.	4.0
10.	List the physical examination tests used to determine the presence of ligamentous lax.	4.0
11.	Discuss the signs and symptoms associated with ligamentous laxity.	4.0
12.	List any associated systemic pathologies associated with ligamentous laxity such as triso	my 21,
	biochemical disorders, and neuromuscular diseases.	4.0
13.	List common biomechanical deformities associated with flexible flatfoot.	4.0
14.	Describe treatments available for flexible flatfoot.	4.0
15.	Define congenital convex pes valgus and list the synonyms for this deformity.	4.0
16.	Discuss possible etiologies for congenital convex pes valgus.	4.0
17.	Discuss the physical exam findings associated with congenital convex pes valgus.	4.0
18.	Describe the soft tissue and bony pathology involved in congenital convex pes valgus, as	s well as
	any associated pathology.	4.0
19.	Describe the radiographic findings associated with congenital convex pes valgus.	4.0
20.	List and describe choices of treatments for congenital convex pes valgus, and describe a	ny long-
	term sequelae associated with the deformity.	4.0
21.	Describe the surgical procedures used in the treatment of congenital convex pes valgus.	4.0
22.	Define oblique talus and differentiate from vertical talus.	3.0
23.	Describe the etiology, diagnosis, examination, and treatment of oblique talus.	3.0
24.	Define tarsal coalition.	4.0
25.	Describe the signs and symptoms associated with tarsal coalitions.	4.0
26.	List the different types of coalitions in order of their frequency of occurrence.	4.0
27.	Discuss pertinent radiographic projections and expected findings for each of the tarsal	
	coalitions.	4.0
28.	Discuss more advanced imaging techniques that may be used for the evaluation of tarsa	d
	coalitions.	4.0
29.	List and discuss conservative and surgical options for each of the tarsal coalitions.	4.0
30.	Describe the long-term sequelae for tarsal coalitions.	4.0
31.	Discuss peroneal spastic flatfoot as a symptom of tarsal coalitions.	4.0
32.	Discuss the diagnosis, treatment, and other etiologies of peroneal spastic flatfoot.	4.0
33.	Define <i>flexible pes planus</i> .	4.0
34.	Describe flexible pes planus in the pediatric patient.	4.0

35.	List the etiologies for flexible pes planus.	4.0
36.	Describe the clinical and radiographic findings in flexible juvenile pes planus.	4.0
37.	List the common conservative treatment plans for juvenile pes planus.	4.0
38.	Differentiate treatment plans for flexible and rigid pes planus.	4.0
39.	Describe the orthotic control and devices used for juvenile pes planus.	4.0
40.	Describe the treatment of the asymptomatic severe juvenile pes planus. <b>4.0</b>	
41.	Review the non-treatment of the mild flexible pes planus.	4.0
42.	Differentiate pes planus from normal childhood ontogeny.	4.0
43.	Define <i>rigid pes planus</i> .	4.0
44.	List the possible etiologies for rigid pes planus.	4.0
45.	Describe the clinical findings in pes planus.	4.0
46.	Describe the radiographic evaluation of rigid pes planus.	4.0
47.	State the natural history of rigid pes planus.	4.0
48.	Describe orthotic control devices and other conservative treatment (eg, shoes, shoe	
	modification, bracing) prescribed in the treatment of rigid pes planus.	4.0
Ca	vus Deformities	
1.	Recognize pes cavus (congenital cavus, calcaneocavus, and cavovarus).	3.0
2.	Describe the appearance of the cavovarus foot type.	4.0
3.	Describe the soft tissue and bony involvement in the cavovarus deformity.	4.0
4.	Describe any other pathology associated with cavovarus deformity.	4.0
5.	Describe the treatment options for cavovarus.	4.0
6.	Describe the appearance of the cavoadductus foot type.	4.0
7.	Describe the soft tissue and bony involvement in the cavoadductus deformity.	4.0
8.	Describe any other pathology associated with cavoadductus deformity.	4.0
9.	Discuss treatment options for cavoadductus.	4.0
10.	Describe the appearance of the calcaneocavus foot type.	4.0
11.	Describe the soft tissue and bony involvement in the calcaneocavus deformit.	4.0
12.	Describe pathology associated with calcaneocavus.	4.0
13.	Discuss treatment option for calcaneocavus.	4.0
14.	Discuss the likelihood of a concurrent neurological disease with the presence of a cavu deformity.	s foot <b>4.0</b>
15.	Outline the neurological and/or neuromuscular diseases associated with cavus foot de	formity.
	4.0	•
Juv	enile Hallux Valgus and Digital Deformities	
1.	Identify common congenital digital deformities.	4.0

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2.	Recognize the etiological factors in congenital digital deformities.	4.0
3.	Describe the conservative management of digital deformities.	4.0
4.	Outline common surgical approaches for juvenile digital deformity.	4.0
5.	Define and discuss congenital hallux valgus.	4.0
6.	Define and discuss infantile hallux valgus.	4.0
7.	Define and discuss juvenile hallux valgus.	4.0
8.	Define and discuss adolescent hallux valgus.	4.0
9.	Discuss the clinical recognition of hallux valgus.	4.0
10.	Outline the radiographic interpretation of hallux valgus.	4.0
11.	Discuss clinical and surgical decision making for the treatment of hallux valgus.	4.0
12.	Describe and discuss the sagittal plane deformities of the second and fourth toes.	4.0

К.

	13. Describe and discuss the varus rotation deformities of the third and fourth toes.	
		4.0
	14. Discuss surgical decision making for the treatment of second, third, and fourth toe digi	tal
	deformities.	4.0
	15. Define digiti quinti varus.	4.0
	16. Define hallux abducto valgu.	4.0
	17. Define hallux varus.	4.0
	18. Define curly toe.	4.0
19. Describe and discuss the etiology, clinical appearance, radiographic assessme		eatment
	of polydactyly, brachymetatarsia, and syndactaly.	4.0
L.	Pediatric Trauma and Child Abuse	
	1. Discuss the physician's role and legal responsibilities in suspected child abuse.	4.0

- 2. Discuss the different types of child abuse and the signs and symptoms of each type. **3.0**
- 3. Discuss the common fracture types associated with child abuse. **4.0**

# **SURGERY and ANESTHESIOLOGY**

## **LEARNING OBJECTIVES**

Anesthesiology Hospital Protocol Tumor Surgery **Operating Room Technique** Postoperative Complications First Metatarsal Surgery Lesser Digital Surgery Flat Foot Surgery **Cavus Foot Surgery** Equinus Conditions and Surgery Traumatology Nerve Surgery Heel Surgery Soft Tissue Surgery Specific Conditions Involving Surgery Pediatric Surgery General Surgical Conditions Tarsal Coalitions Arthroscopy and Endoscopy of the Foot and Ankle
#### I. Anesthesiology

#### A. Perioperative Management\_of the surgical patient

		inoperative management <u>o</u> f the sargiear patient	
	1. 2.	Describe the components of pre-anesthetic evaluation, including importance and applic the ASA Physical Classification System. Describe anesthetic implications for the common disease states affecting the cardiovase pulmonary, neurologic, metabolic and endocrine, hepatic and renal, hemopoietic and	ation to <b>4.0</b> cular,
	3.	musculoskeletal systems. Discuss the impact of perioperative medications on outpatients and inpatients with co-edisease.	<b>3.0</b> existing <b>3.0</b>
	4.	Discuss allergic reaction prophylaxis and infection prophylaxis with respect to the anest patient.	hetic <b>3.0</b>
В.	Int	ra-operative Management of the Surgical Patient	
	1. 2.	<ul> <li>Describe the indications for and goals of monitoring for patients undergoing procedure local, regional, and general anesthesia</li> <li>Describe indications for the following types of monitors in anesthesia: <ul> <li>a. blood pressure</li> <li>b. pulse oximetry</li> <li>c. EKG</li> <li>d. temperature (aural and esophageal)</li> <li>e. capnography</li> <li>f. neuromuscular injury that may result from poor positioning</li> </ul> </li> </ul>	s under 3.0 3.0 3.0 3.0 3.0 3.0 3.0 3.0 3.0
С.	Air	way Management for Patients Undergoing Anesthesia	
	1. 2.	Discuss assessment methods for airway patency and classify common airway systems. Describe conditions that predispose a patient to airway impairment.	4.0 3.0
D.	Loc	cal Anesthesia	
	1. 2.	Classify nerve fiber in relation to local anesthetic actions. Make pharmacologic recommendations for the use of amide and ester local anesthetic and non-plain solutions in podiatric medicine, including mechanism of action,	<b>4.0</b> for plain
		pharmacodynamics, and pharmacokinetics.	4.0
	3.	Identify known toxic doses for local anesthetics used in podiatric medicine, and recogniz symptoms, and management of toxic reaction to local anesthesia.	ze signs, <b>3.0</b>

 Differentiate between toxic and allergic reaction to local anesthesia, including clinical findings, and management of anaphylactic shock.
 3.0

#### E. Intravenous Anesthesia

- Explain the concept of "ideal" anesthetic, and describe advantages and disadvantages of IV anesthetics.
   3.0
- 2. Distinguish between opioid and non-opioid IV anesthetics. **3.0**
- Recall the pharmacology, including mechanism of action, pharmacodynamics, pharmacokinetics, clinical uses, contraindications, and adverse effects of benzodiazepines, barbiturates, etomidate, and Ketamine.
   4.0

	4.	Recall the pharmacology, including the mechanism of action, pharmacodynamics, pharmacokinetics, clinical uses, contraindications, and uses Fentanyl meperidine and methods and methods are set to a set the set of the set o	orphine.
			4.0
	5.	Give examples of opioid antagonists and mixed agonist antagonist opioids.	4.0
	6.	Describe indications and goals of Total IV Anesthesia (TIVA).	3.0
F.	Ge	neral Anesthesia	
	1.	Define general anesthesia, and describe its advantages and disadvantages.	3.0
	2.	Describe the general mechanism of action, stages, and planes of general anesthetics. Ar	nesthesia
			4.0
	3.	Recall the pharmacology, including the mechanism of action, pharmacodynamics,	
		pharmacokinetics, and toxicity of N2O and volatile anesthetics.	4.0
	4.	Describe risks and benefits of inhaled anesthetics, including risk for developing malignal	nt
		hyperthermia, manifestations, and treatment.	3.0
	5.	Recall the pharmacology, including mechanism of action, pharmacokinetics, pharmacod	ynamics,
		clinical uses, and contra-indications of the commonly used muscle relaxants.	4.0
	6.	Describe the use and limitations for monitoring neuromuscular blockade, and identify d	rugs
		used to reverse neuromuscular blockade.	3.0
G.	Re	gional Anesthesia	
	1	Recall the anatomy of the spinal column and peripheral pervous system in relation to	
	т.	administration	3.0
	2	Describe the advantages and disadvantages of administering regional anesthesia includ	ing
	۷.	associated safety issues	40
	z	Describe principles of neuravial anesthesia, including the indications and contra-indicati	005
	Э.	not not a not a set of the set of	s utilized
		for sninal anesthesia	<b>4 0</b>
	4	Describe indications contra-indications physiologic effects mechanism of action	-1.0
		complications, and drugs utilized for endural anesthesia	4.0
	5	Indicate general principles of peripheral perve blockade iniciduding indications contra-	-1.0
	5.	indications, and complications.	3.0
	6	Describe the common local anesthetic agents used in and the techniques used for the c	ommon
	0.	regional blocks of the lower extremity, including sciatic, femoral, popliteal, common per	oneal.
		nosterior tibial sural sanhenous, and Bier block	3.0
н.	<u>Ho</u>	spital Protocol	
А.	Ch	arting and Orders	
	1	Explain assential components of admission history and physical potes	3.0
	1. 2	Explain essential components of a pre-operative note post-operative note, and operative	<b>J.U</b>
	۷.	report.	<b>3.0</b>

3. Explain essential components of admission orders, peri-operative orders, pre-operative orders, and post-operative orders. **3.0** 

# B. Informed Consent

1. Explain informed consent, including medico-legal implications. **3.0** 

2. Identify the party that may give informed consent, and in what circumstances it is required. 3.0

#### C. Admitting and Consulting Protocol

1. Describe JCAHO regulations pertaining to podiatric physicians performing histories and physicals for the purposes of hospital admission. **3.0** 

## D. Organization of Hospital Staff

Differe	ntiate between hospital medical staff and other staff, such as allied health.	3.0	
Explain principles of granting hospital privileges to clinical staff.			
Describe clinical privileges granted to hospital staff, including			
a.	active;	3.0	
b.	admitting;	3.0	
с.	consulting;	3.0	
d.	courtesy; and	3.0	
e.	surgical.	3.0	
	Differe Explain Describ a. b. c. d. e.	Differentiate between hospital medical staff and other staff, such as allied health. Explain principles of granting hospital privileges to clinical staff. Describe clinical privileges granted to hospital staff, including a. active; b. admitting; c. consulting; d. courtesy; and e. surgical.	

#### III. <u>Tumor Surgery</u>

#### A. Biopsy Techniques

	1.	Describe general indications for performing biopsies.	4.0
	2.	Differentiate between excisional, incisional, punch, shave, fine needle, and needle c	ore biopsies.
			4.0
	3.	Summarize indications and contra-indications for excisional, incisional, punch, shave	e, fine
		needle, and needle core biopsies.	4.0
В.	Sof	ft Tissue Tumors	
	1.	Describe the salient clinical features and surgical treatment of the following types of lesions of fat, muscle, and nerve origin of	f malignant
		a. liposarcoma;	3.0
		b. rhabdomyosarcoma; and	3.0
		c. neurofibrosarcoma.	3.0
	2.	Explain the significance of skin metastases in terms of primary disease state, and ide	entify the
		most common primary lesions in males and females that give rise to metastases to t	he skin. <b>3.0</b>
С.	Во	ne Tumors	
	1.	Describe the salient clinical features and surgical treatment of the following types of bone tumors:	fbenign
		a chondroma	3.0
		h chondrohlastoma	3.0
		c enchondroma	3.0
		d. ossifving and non-ossifving fibroma	3.0
		e. anneurysmal and unicameral bone cysts	3.0
		f. osteoid osteoma	3.0

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j.	giant cell tumor	3.0
k.	intraosseous ganglion	3.0
I.	intraosseous lipoma	3.0

#### D. General principles of Cancer Staging

1.	Describe the staging of cancer via the TNM System.	3.0
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2. Describe the role of the American Cancer Society in staging various cancers that affect the skin and musculoskeletal systems. 3.0

#### IV. **Operating Room Technique**

#### A. Asepsis

	1.	Explain and apply Universal Precautions and their application to the sterile technique and the OP and income and the Precautions and their application to the sterile technique and the Precautions and the Precautions and the Precautions and the Precaution and t	nd within
	2	the OR environment.	3.0
	2.	Describe and apply essential components of sterile technique.	3.0
	3.	Describe and apply the concept of "Surgical Conscience" and explain potential conseque	ences of
		breeches in sterile technique, with respect to self and operating field.	3.0
	4.	Explain routine and biohazard waste handling procedures, as well as general cleaning st	andards
		for the OR.	3.0
	5.	Discuss principles of asepsis, sterilization, and autoclaving.	3.0
В.	Ins	trumentation	
	1.	Classify, including uses of, non-power instrumentation commonly found in a basic foot/	ankle
		surgery tray.	3.0
С.	AO	Technique/External Fixation Principles	
	1.	Explain principles of A-O fixation.	3.0
	2.	Discuss the application of A-O technique to foot and ankle surgery and its role in bone h	nealing.
			3.0
	3.	Describe the mechanical basis of stable and rigid internal fixation.	3.0
	4.	Explain "lag screw" and the techniques utilized in insertion.	3.0
	5.	Describe the concepts and techniques utilized in static and dynamic interfragmental	
		compression.	3.0
	6.	Explain screw plates and screws.	3.0
	7.	Identify and describe instrumentation found in the Mini, Small, and Large, and Cannulat	ted
		Fragment A-O Sets.	3.0
	8.	Explain the principles and techniques that dictate the use of K wires and cerclage wires.	3.0
	9.	Explain the principles and types of external fixation device used in foot and ankle surger	ry.
			3.0
Л	Sur	turos/Tochnique	

### D. Sutures/Technique

1. Explain general principles of usage of the following in foot and ankle surgery sutures: stainless steel wire, nylon, polyester, polyethylene, polypropylene, polyglycolic acid, and polydioxanone.

- 2. Classify and describe commonly used suture material utilized in foot and ankle surgery. 3.0
- 3. Describe biological and mechanical properties of absorbable and non-absorbable sutures.

4.	Discuss surgical needles commonly used in foot and ankle surgery, including material use construction, and classify them according to needle type, size, curvature, and cross-section	ed for on, with
	reference to the needle coding system.	4.0
5.	Describe the commonly used suture techniques in foot and ankle surgery, including the u	use and
	performance of the following techniques: simple, mattress (vertical and horizontal), rete	ntion
	(superficial and deep), subcuticular, and running.	3.0
6	Describe indications for Kessler, Ruppell, and Krakow suture technique in feet and ankle	curgory

- Describe indications for Kessler, Bunnell, and Krakow suture technique in foot and ankle surgery, as well as other types of technique used in tendon repair.
   3.0
- 7. Explain general principles and instrumentation and techniques which may be used for repairing:

a.	tendon:tendon	3.0
b.	tendon:bone	3.0
c.	soft tissue anchor: bone	3.0

#### E. Other Biomaterials

- Describe physical and mechanical properties of materials used for implants in foot and ankle surgery.
   3.0
- Describe physical and mechanical properties of non-metallic materials used in foot and ankle surgery.
   3.0
- Describe physical and mechanical properties of bone morphogenic proteins used in foot and ankle surgery.
   3.0
- 4. Describe the use of topical hemostatic agents used in foot and ankle surgery. **3.0**
- 5. Describe the indications for, and types of bone stimulator used in, foot and ankle surgery. 3.0
- Explain basic principles and functions of surgical dressings, including description of dressing materials and the anatomy of a surgical dressing.
   3.0
- 7. Describe the types of surgical dressing employed in the practice of foot and ankle surgery.
- 3.08. Describe the role of immobilization in foot and ankle surgery.3.0

### V. <u>Postoperative Complications</u>

#### A. Systemic Medical (Inpatient Only)

	1.	Identify the causes of and recognize altered mental status in the postoperative period i	n a
		patient.	4.0
	2.	Identify the causes and recognize the signs, symptoms, and sources of postoperative	
		dehydration.	4.0
	3.	Identify potential causes and recognize the signs and symptoms of chest pain (Atelectat	sis versus
		MI versus PE versus Other) in the postoperative period.	4.0
	4.	Recognize the signs, symptoms and diagnostic indicators of postoperative urinary tract	
		infection.	4.0
	5.	Identify the causes and recognize the signs, symptoms and diagnostic indicators of	
		postoperative blood glucose anomalies (diabetic ketoacidosis, hypoglycemia).	4.0
В.	Ou	tpatient and Inpatient	

Identify the causes of and risk factors for postoperative gastrointestinal pathology to include constipation, fecal impaction, nausea, vomiting, and diarrhea (include pseudomembranous colitis secondary to antibiotic).
 4.0

	2.	Recognize the signs and symptoms of postoperative gastrointestinal pathology.	4.0
	3.	Identify the management strategies for postoperative gastrointestinal pathology.	4.0
	4.	Recognize the signs and symptoms of postoperative superficial phlebitis.	4.0
	5.	Understand and recommend appropriate workup, management strategies, and prophyl	axis for
		superficial phlebitis.	4.0
	6.	Identify the causes and risk factors for postoperative deep venous thrombosis.	4.0
	7.	Recognize the signs and symptoms of deep venous thrombosis.	4.0
	8.	Recommend appropriate workup, management strategies, and prophylaxis for postope	rative
	_	deep venous thrombosis.	4.0
	9.	Identify the causes and risk factors for postoperative pulmonary embolism.	4.0
	10.	Recognize the signs and symptoms of pulmonary embolism.	4.0
	11.	Recommend appropriate workup, management strategies, and prophylaxis for postope	rative
		pulmonary embolism.	4.0
	12.	Identify the potential causes of postoperative fever, including atelectasis and pneumon	ia 
		(aspiration), DVT, infection at the surgical site, other infection (UTI/catheter), and medi-	cation
	40	related (anticholinergic).	4.0
	13.	Recognize the signs and symptoms of postoperative fever.	4.0
	14.	Understand and recommend appropriate workup, diagnostic indicators, and manageme	ent
	1 -	strategies for postoperative fever.	4.0
	15.	identify the causes and recognize the signs and symptoms of normal postoperative bloc	
	10	Versus excessive blood loss secondary to bleeding disorders and coagulopathies.	4.0 ding
	10.	including prophylavic	
	17	Dictinguish between normal nectonorative nain and intractable alledunia	4.0
	17.	Identify causes of intractable postoperative pain to include CPDS, post-tourniquet com	<b>4.0</b>
	10.	neuralgia, handage/cast related pain	<b>1 0</b>
	19	Recommend diagnostic modalities and management strategies for abnormal nostonera	tive nain
	19.	Recommend diagnostic modalities and management strategies for abnormal postopera	<b>4 0</b>
	20	Identify risk factors and causes of nostonerative CRPS	4.0
	20.	Recognize the signs and symptoms of CRPS	4.0
	22.	Recommend appropriate workup, management strategies, and prophylaxis for postope	rative
		CRPS.	3.0
-	_		
С.	Foc	ot and Ankle Specific	
	1.	Identify the risk factors and causes for postoperative ischemia, including digital and tota	al limb
		ischemia.	4.0
2		Recognize the signs and symptoms of postoperative ischemia.	4.0
3	•	Recommend appropriate workup and diagnostic measures, management strategies, and	b
		prophylaxis.	4.0
4	•	Identify the risk factors and causes for postoperative wound/surgical site infection.	4.0
5	•	Recognize the signs and symptoms of postoperative wound/surgical site infection, inclu	ding the
		cardinal signs of infection erythema, edema, calor, dolor, malodor, and loss of function.	4.0
6	•	Recommend appropriate workup/diagnostic measures, management strategies, and pro-	ophylaxis
		for postoperative wound/surgical site infections.	4.0
7	•	Identify risk factors and causes of postoperative wound/skin complications to include ex	kcessive
		edema, hematoma, seroma, suture abscess, wound dehiscence, and hypertrophic/keloi	d scar.
			4.0
8	•	Recognize the signs and symptoms of postoperative wound/skin complications.	4.0

9.	Recommend appropriate workup/diagnostic measures and management strategies for	
	postoperative wound/skin complications.	4.0
10.	Identify risk factors and causes of complications associated with bone healing, including	
	nonunion (septic and aseptic), delayed union, and malunion.	4.0
11.	Recognize signs and symptoms of complications associated with bone healing.	4.0
12.	Recommend appropriate workup /diagnostic measures and management strategies for	
	complications associated with bone healing.	4.0
13.	Identify risk factors and causes of hardware complications, inlcuding pin site complication	ons and
	internal/external hardware failure.	4.0
14.	Recognize signs and symptoms of hardware complications in the postoperative patient.	4.0
15.	Recommend appropriate management strategies for hardware complications in the	
	postoperative patient.	4.0
16.	Recognize clinical signs and symptoms, as well as radiographic markers of avascular nec	osis.
		4.0
17.	Recommend workup /diagnostic measures, as well as management strategies for postop	perative
	avascular necrosis.	4.0
18.	Identify the causes and recognize the signs and symptoms of specific foot and ankle surg	gery
	related complications, including transfer lesions, alignment complications (under/over	

correction) capsulitis, joint stiffness, and bandage/cast attributed wounds. **4.0** 

#### VI. First Metatarsal Surgery

#### A. Etiology and Radiographic Assessment of Hallux Abducto Valgus Deformity

- Explain the etiology of hallux abducto valgus deformity, including the biomechanics, heredity, inflammatory rheumatogic diseases, neurological disorders, environment factors, trauma and surgical complications.
   4.0
- Explain the importance in performing a clinical and physical evaluation of a patient with hallux abducto valgus deformity.
   **3.0**
- Describe normal and abnormal angles used in the radiographic evaluation of a hallux abducto valgus deformity in transverse, sagittal, and frontal planes, including metatarsus adductus angle, IM angle, hallux abductus angle, PASA, DASA, hallux abductus interphalangus angle, metatarsal protrusion distance, and sesamoid position.
- B. Soft Tissue Procedures for Correction of Hallux Valgus Deformity
  - Describe the surgical anatomy of the first metatarsal and sesamoid complex, as well as the ligamentous attachments of the First MTPJ.
     4.0
  - Describe soft tissue procedures utilized in correction of hallux valgus deformity, including muscle tendon balancing procedures and the concepts of the lateral release including the ligamentous attachments of the First MTPJ.
     3.0
- C. Phalangeal Procedures for the Correction of Hallus Valgus Deformity
  - 1. Summarize the procedures, indications, and contraindications of hallux osteotomies. **4.0**
  - Identify potential complications that may arise from performing hallux osteotomies to correct hallux valgus deformity.
     4.0
  - Summarize the procedures, indications, and contraindications of hallux IPJ fusion as part of hallux valgus deformity.
     4.0

	4.	Identify complications that may arise from performing hallux IPJ fusion to correct hallux deformity.	valgus <b>4.0</b>
D.	Dis	tal Osteotomies of the First Metatarsal for the Correction of Hallux Valgus Deform	ity
	1.	Summarize the procedures, indications, and contraindications of distal osteotomies as procedures used in correction of hallux valgus deformity.	<b>4.0</b>
	Ζ.	hallux valgus deformities.	<b>4.0</b>
Ε.	Sh	aft Osteotomies of the First Metatarsal for the Correction of Hallux Valgus Deform	ity
	1.	Summarize the procedures, indications, and contraindications of the shaft osteotomies of First metatarsal as procedures used in correction of hallux valgus deformity.	of the <b>4.0</b>
	2.	metatarsal used to correct hallux valgus deformities.	4.0
F.	На	llux Varus	
	1.	Explain the etiology of the pathomechanics, including iatrogenic versus non-iatrogenic h varus deformity.	allux <b>4.0</b>
	2.	Describe the treatment plan to correct hallux varus deformity including surgical technique both soft tissue and osseous.	ues, <b>4.0</b>
G.	На	llux Limitus/Rigidus	
	1.	Discuss the pathomechanics, etiology, and clinical presentation of hallux limitus and hall rigidus.	ux <b>4.0</b>
	2.	Describe joint preserving surgical procedures used to correct hallux limitus/rigidus inclue chielectomy, and osteotomy.	ding <b>4.0</b>
	3.	Describe procedures used for joint resection including arthroplasty, interposition arthropland replacement arthroplasty for hallux limitus/rigidus.	plasty, <b>4.0</b>
	4.	Identify the biomaterials used in joint replacement procedures, including design and fun surgical techniques, and complications due to material failure, design function, and host	ction,
	5.	Identify postoperative complications that may result from surgery for hallux limitus/rigio	<b>4.0</b> lus. <b>4.0</b>
Н.	Ba.	se Procedures of the First Metatarsal for the Correction of Hallux Valgus	
	1.	Explain procedures, indications, and contraindications for performing base osteotomies First metatarsal to correct hallux valgus deformity, including the concepts of osteotomy	of the design
	2.	Explain the hinge axis concept including the components of the hinge, the placement of hinge the axis, the motion about the hinge, and the orientation of the axis.	<b>4.0</b> the <b>4.0</b>

- Identify potential complications that arise from performing base osteotomies to correct hallux valgus deformity.
   4.0
- I. Juvenile Hallux Valgus
  - 1. Explain etiologies for juvenile hallux valgus deformity and the mechanism of action. **4.0**
  - Describe indications and contraindications for performing juvenile hallux valgus surgery, including muscle tendon balance procedures, base osteotomies, head osteotomies, epiphysiodeses, and ancillary procedures.
     4.0

4.0

2.	Evaluate the pathophysiology or pathomechanics of digital deformity, including effects of equinus, nes cavus and extensor substitution: flatfoot and flexor stabilization; muscle weak	ness
	and flever substitution; and first ray instability and load transfer on digital deformity	1033
С	Discuss the normal and abnormal accords of the history and physical evamination, including	
5.	becass the normal and abnormal aspects of the history and physical examination, including	any
	fallowing.	
	Ionowing:	
	a. sont tissue digital procedures: <b>4.</b>	)
	i. capsulotomy	
	II. tenotomy	
	III. tenectomy	
	iv. tendon Lengthening	
	a) "Z" type	
	b) extensor recession	
	b. MIPJ sequential release: 4.0	)
	I. Kelikian push-up test between step evaluation	
	II. sequential steps: dorsal capsule, extensor brevis, collateral ligaments, flexo	r
	plate (plantar capsule release), extensor longus	
	c. rendon transfers: 4.0	)
	i. flexor tendon transfer FDB, FDL, combined	
	ii. extensor tendon transfer, Hibbs	
	d. syndactylism 4.0	)
	e. osseous digital procedures: 4.0	)
	i. ostectomy/exostectomy/condylectomy	
	ii. phalangectomy: partial/complete	
	iii. arthroplasty (IPJ)	
	iv. PIPJ implant arthroplasty	
	v. diaphysectomy	
	vi. phalangeal osteotomy	
	vii. arthrodesis fusion (IPJ)	
	viii. amputation	
	a) partial:terminal Symes	
	b) complete	
	1) transphalangeal	
	2) MTPJ	
4.	Discuss the indications, contraindications, advantages, and disadvantages of each digital	

VII. <u>Lesser Digital Surgery</u>

procedure.

Lapidus type procedure.

- 1. Identify, classify, and evaluate lesser (2–5) digital deformities and conditions. **4.0**
- Identify potential complications that arise from performing first metatarsal surgeries for the Lapidus type procedure.
   4.0

# 3. Identify postoperative complications following juvenile hallux valgus surgery.

J. First Metatarsal Cuneiform Arthrodesis for the Correction of Hallux Abducto Deformity

1. Describe indications and contraindications for performing first metatarsal surgery for the

	5.	Discus	the risks and benefits of performing or not performing digital procedures.	3.0
	6.	Discus	s regional anatomy of the lesser digits.	4.0
	7.	Explair	appropriate incisional approach(es) and outline their respective procedural step	s.
				4.0
	8.	Explair	the instrumentation and material needs for performance of digital procedures.	4.0
	9.	Explair	fixation materials and techniques, including physical characteristics,	
		advant	ages/disadvantages, indications/ contraindications, and applications.	4.0
	10.	Explair	the graft materials, including physical characteristics, advantages and disadvanta	ages,
		indicat	ions and contraindications, and application ofgrafting techniques.	4.0
	11.	Discus	the immediate perioperative care requirements and postoperative managemen	t of each
		digital	procedure.	4.0
	12.	Explair	the potential complications of each digital procedure and its management.	3.0
A.	Cei	ntral M	etatarsal Surgery (Surgery distal to the tarsometatarsal joints of rays 2, 3,	and 4)
	1.	Evalua	te the central (2–4) metatarsal deformities and conditions	
		a.	shortened metatarsal;	4.0
		b.	elongated metatarsal (transverse plane digital deviation with Kelikian push-up t	est);
				4.0
		с.	plantarflexed metatarsal;	4.0
		d.	prominent plantar condyle;	4.0
		e.	MTPJ stress syndrome;	4.0
			i. predislocation phase	
			ii. dislocation phase	
		f.	dislocated MTPJ;	4.0
		g.	arthritic MTPJ; and	4.0
		h.	rupture of flexor plate.	4.0
	2	Evolair	nathonhysiology or nathomochanics of the metatarcal deformity, including the	offect of

- Explain pathophysiology or pathomechanics of the metatarsal deformity, including the effect of equinus, pes cavus, and extensor substitution; flatfoot and flexor stabilization; muscle weakness and flexor substitution; and first ray instability and load transfer on digital deformity.
   4.0
- 3. Discuss normal and abnormal aspects of the history and physical examination, including laboratory studies, diagnostic tests, or imaging studies that indicate or contraindicate the following central (2–4) metatarsal procedures:
  - a. central metatarsal procedures:

- i. metatarsal shortening procedures
  - a) oblique shortening osteotomy (Weil)
  - b) step down osteotomy
  - c) chevron shortening osteotomy
  - d) cylindrical shortening osteotomy
- ii. metatarsal lengthening procedures
  - a) sagittal "Z" lengthening osteotomy
  - b) cylindrical lengthening osteotomy with bone graft
  - c) callous distraction (refer to section on congenital deformity)
- iii. metatarsal elevating procedures
  - a) vertical "V" osteotomy
    - b) dorsal wedge basal osteotomy
    - c) sagittal "Z" osteotomy
- iv. metatarsal lowering procedures, including sagittal "Z" plantarflexing osteotomy

		b. metatarsal abducting procedures, including multiple osteotomy management o	f
		metatarasus adductus	4.0
		c. metatarsal resection (eliminating) procedures:	4.0
		i. partial metatarsal head resection (MTPJ arthroplasty)	
		a) distal metatarsal head (hemi (4 mm) joint resection)	
		b) plantar condylectomy, inlcluding MTPJ impant arthroplasty	
		ii. metatarsal head resection	
		a) single	
		b) multiple: pan metatarsal head resection	
		III. amputation	
		a) isolated lesser ray amputation,	
		b) transmetatarsal amputation (TMA)	
		c) chopart amputation	
		iv. partial ostectomy, including metalarsal cureiform exostectomy	
		d MTDI flevor plate repair	10
	Λ	u. Mirrinexul place repair Discuss the indications, contraindications, advantages, and disadvantages of each meta	tarcal
	4.	nrocedure	
	5	Discuss the risks and henefits of performing or not performing metatarsal procedures	4.0
	6.	Discuss the regional anatomy of the lesser metatarsals	4.0
	7.	Discuss the appropriate incisional approach(es) and outline procedural steps related to	each
		metatarsal procedure.	4.0
	8.	Explain the instrumentation and material needs for performance of metatarsal procedu	res.
			4.0
	9.	Explain the fixation materials and techniques, including physical characteristics,	
		advantages/disadvantages, indications/ contraindications.	4.0
	10.	Explain the graft materials, including physical characteristics, advantages and disadvant	ages,
		indications and contraindications.	4.0
	11.	Discuss the immediate perioperative care requirements and postoperative managemer	t of each
		metatarsal procedure.	4.0
	12.	Explain potential complications of each metatarsal procedure and its management.	4.0
В.	Fift	h Metatarsal Surgery (Surgery Distal to the Tarsometatarsal Joint of Ray 5)	
	1.	Identify, classify, and evaluate level(s) of the following fifth metatarsal deform conditions:	ities and
		a. Tailor's Bunionette deformity	4.0
		i. soft tissue defrormity: bursitis, neuritis lateral to fifth met head	
		ii. enlarged lateral condyle	
		iii. lateral bowing of distal metatarsal shaft (lateral deviation angle increas	ed)
		iv. lateral splaying of fifth metatarsal at metatarsal base (intermetatarsal a	ngle
		increased)	
		b. Arthritis Fifth MTPJ	4.0
	2.	Discuss the pathophysiology or pathomechanics of the Tailor's bunionette deformity in	cluding
	~	the effect on forefoot abduction when foot in neutral calcaneal stance position (NCSP).	4.0
	3.	Discuss normal and abnormal aspects of the history and physical examination including	. h
		laboratory, diagnostic, or imaging studies or tests that would indicate or contraindicate	the
		ronowing procedures:	

a. fifth ray procedures: Tailor's bunionette

b. fifth metatarsal	4.0
c. bunionectomy of the fifth metatarsal without osteotomy	4.0
d. bunionectomy of the fifth metatarsal with osteotomy	4.0
i. distal shalt/field osteotomy	
e. metatarsal head resection	4.0
4. Discuss the indications, contraindications, advantages, and disadvantages of digital pro	ocedures.
	4.0
5. Discuss the risks and benefits of performing or not performing metatarsal procedures.	3.0
6. Discuss regional anatomy.	4.0
<ol> <li>Explain incisional approach(s) and outline procedural steps related to each.</li> <li>Discuss the instrumentation and material needs for performance of fifth metatarsal procedural steps are steps.</li> </ol>	4.0 ocedures
b. Discuss the instrumentation and matchar needs for performance of intrimetatarsarpr	<b>4.0</b>
9. Explain fixation materials and techniques to fifth metatarsal surgery, including physica	
characteristics, advantages/disadvantages, indications/ contraindications.	4.0
10. Explain graft materials, including physical characteristics, advantages/disadvantages,	
indications/ contraindications.	4.0
11. Discuss immediate perioperative care requirements and postoperative management of	t each
12. Explain potential complications of each fifth metatarsal procedure and its managemer	4.0 t. 4.0
Flat Foot Surgery	
1. Recognize that there is not universal terminology when referring to flatfoot deformity	3.0
2. Recognize characteristic clinical findings associated with flatfoot of, including everted	neel,
abduction of the forefoot on the rearfoot, collapse of the medial column flexibility, an	d rigidity.
	4.0
<ol> <li>Recognize evaluate and diagnose ankle equinus as either a primary force or secondary adaptation with flat fact.</li> </ol>	4.0
4 Identify etiological factors that require compensation and result in flatfoot deformity	4.0 4.0
<ol> <li>Explain planal dominance and determine the primary plane of compensation.</li> </ol>	3.0
6. Perform a biomechanical evaluation for flat foot and correlate radiographic findings ar	nd
determine planal dominance.	4.0
7. Recognize and evaluate a flat foot (pes valgus deformity) that is rigid and determine the	ie
etiology.	<b>4.0</b>
a. Identify the pathologic collapsing pes valgus foot that requires surgical treatment (den instability, nain, progression)	<b>4 0</b>
<ol> <li>Explain the pathology of ankle equinus and its surgical management.</li> </ol>	3.0
10. Describe indications for medial column soft tissue procedures utilized for flat foot (pes	valgus
deformity).	3.0
11. Describe indications for medial column arthrodesis procedures utilized for flat foot (pe	s valgus
deformity).	3.0
<ol> <li>Describe indications, techniques, and implants utilized for subtalar arthroefelsis.</li> <li>Explain extraarticular calcaneal osteotomies with an arthroefelsis effect on the subtala</li> </ol>	<b>3.U</b> ar ioint
15. Explain extraor ficular calcanear osteotomies with an artificereisis effect on the subtale	<b>3.0</b>
14. Describe indications and technique of Evans calcaneal osteotomy for transverse flat fo	ot
deformity (pes valgus deformity).	3.0

VIII.

- Describe indications and techniques of posterior calcaneal osteotomies for frontal plane flat foot deformity (pes valgus deformity).
   3.0
- 16. Recognize severe hind foot degenerative joint disease and recommend hindfoot arthrodesis.

4.0

#### IX. <u>Cavus Foot Surgery</u>

Χ.

# A. Perioperative Management of the Surgical Patient

1. Define, describe, and identify a cavus foot as primarily being a sagittal plane deformity	of	
plantarflexion of the forefoot on the rearfoot, with secondary multiplane forefoot and	rearfoot	
deformities.	3.0	
2. Classify cavus foot as flexible or rigid and evaluate its possible association with neuro-r	nuscular	
disorders.	4.0	
3. Identify neurologic conditions associated with cavus foot as progressive or static.	3.0	
4. Classify pes cavus as congenital or acquired lesser tarsus cavus and forefoot cavus.	4.0	
5. Recognize transverse, frontal and sagittal plane (fore and hind foot) deformity associat	ed with	
pes cavus.	3.0	
6. Recognize and understand pseudo equinus associated with pes cavus.	3.0	
7. Diagnose progressive neurologic pes cavus and recommend joint stabilization or arthro	odesis	
procedure of the fore and hindfoot.	3.0	
8. Describe and interpret the Coleman block test for evaluation of pes cavus.	4.0	
9. Recommend radiographic views for pes cavus, draw and interpret angular measureme	nts for	
surgical decision making.	4.0	
10. Delineate the flexible and rigid components of pes cavus for surgical decision making.	4.0	
11. Describe indications for and recommend plantar soft tissue release as a component of	pes cavus	
surgery.	3.0	
12. Describe indications for and recommend specific tendon transfer procedures for musc	le	
imbalance associated with pes cavus.	4.0	
13. Describe indications and role of metatarsal osteotomies in the surgical management of	fanterior	
pes cavus.	4.0	
14. Recognize and recommend midtarsal osteotomies for pes cavus with a mid-foot apex.	4.0	
15. Recognize and recommend calcaneal osteotomy for rigid frontal and sagittal hindfoot	deformity.	
	4.0	
16. Recognize and recommend tarsal fusion procedures for rigid and or arthritic pes cavus	4.0	
17. Evaluate digital deformity associated with pes cavus and recommend surgical treatment	nt options	
based on etiology and muscular imbalance.	4.0	
18. Recognize and evaluate lateral ankle instability associated with pes cavus deformity.	4.0	
Equinus Conditions and Surgery		

1.	Describe the anatomy and function of the triceps surae and Achilles tendon.	4.0
2.	Define equinus and differentiate muscular from osseous equinus or combined muscular	-osseous
	equines.	4.0
3.	Perform and interpret the Silverskiold test.	3.0
4.	Identify proximal and distal compensations for equinus deformity.	4.0

	<ol> <li>Recommend conservative treatment modalities, when appropriate, for muscular equinus deformities.</li> <li>Discuss spastic muscular equinus and surgical treatment of proximal recession.</li> <li>Identify nonspastigastrocnemius equinus and recommend distal gastrocnemius recession procedures.</li> <li>Identify and diagnose spastic and nonspastic gastrosoleus equines.</li> <li>Describe and recommend anterior advancement Achilles tendon procedures for spastic gastrosoleus equines.</li> <li>Describe and recommend Achilles tendon tenotomies and lengthening procedures for nonspastic gastrosoleus equines.</li> <li>Recommend talotibial exostosis or other osseous block resection for osseous equines.</li> </ol>	s 3.0 3.0 4.0 4.0 4.0 4.0 4.0
XI.	Traumatology	
А.	General Principles of Management of the Traumatized Patient	
	1. Describe the basic concepts of initial patient evaluation and emergency triage.	3.0
В.	Nail Trauma	
	<ol> <li>Discuss common mechanisms of injury associated with acute and chronic nail trauma.</li> <li>Describe appropriate management of nail trauma, including subungual hematoma, nail b laceration with and without fracture.</li> </ol>	<b>3.0</b> bed <b>3.0</b>
С.	General Principles of Fracture management	
	<ol> <li>Evaluate radiographs, CT, MRI, as well as other special imaging modalities to identify fore midfoot, and rearfoot trauma.</li> <li>Describe the concepts of closed reduction, percutaneous fixation, and external fixation.</li> <li>Discuss the determination for a closed reduction versus an open reduction.</li> <li>Explain the concepts of open reduction and internal fixation.</li> </ol>	efoot, 4.0 3.0 4.0 3.0
D.	Open Fracture Management, Including Gunshot Wounds	
	<ol> <li>Discuss basic management of soft tissue trauma, including imaging, wound care, tetanus appropriate antibiotic prophylaxis.</li> <li>Describe the Gustillo and Anderson classification and its significance in the treatment and management of soft tissue injuries involving bone.</li> <li>Recognize the basic characteristics of particular soft tissue wounds.</li> <li>Describe and select appropriate wound treatment and the types of closure techniques.</li> </ol>	and 3.0 d 3.0 3.0 3.0 3.0
Ε.	Digital Trauma	
	<ol> <li>Discuss common mechanisms and configurations of digital fractures.</li> <li>Describe the concepts of closed reduction and open reduction of digital fractures.</li> <li>Describe the long-term complications of digital fractures.</li> </ol>	3.0 3.0 3.0
F.	First Metatarsal Fractures	
	1. Discuss the basic principles of closed reduction utilized in the treatment.	3.0

- Recognize and evaluate the basic clinical and imaging characteristics to enable appropriate treatment in reference to closed versus open reduction.
   4.0
- Describe the advantages and disadvantages of closed versus open reduction in first metatarsal fractures.
   4.0
- 4. Describe the external and internal fixation principals in reference to the first metatarsal. 3.0
- 5. Describe common metatarsal anatomical fracture types, including neck, midshaft, and base fractures; as well as joint dislocations, intra-articular fractures and avulsion fractures. **3.0**
- 6. Describe common metatarsal fracture subtypes and discuss appropriate treatment and common long-term complications associated with such trauma.
   4.0
- G. Central Metatarsal Fractures (2, 3, 4)
  - 1. Discuss the basic principles of closed reduction utilized in the treatment of metatarsals. **3.0**
  - Recognize and evaluate the basic clinical and imaging characteristics to enable appropriate treatment in reference to closed versus open reduction.
     4.0
  - 3. Describe the advantages and disadvantages of closed versus open reduction in central metatarsal fractures.
  - 4. Describe the external and internal fixation principals in reference to metatarsals. **3.0**
  - 5. Describe common metatarsal anatomical fracture types, including neck, midshaft, and base fractures; as well as joint dislocations, intra-articular fractures and avulsion fractures. **3.0**
  - 6. Describe common metatarsal fracture subtypes and discuss appropriate treatment and common long-term complications associated with such trauma.
     4.0

#### H. Fifth Metatarsal Fractures

- Differentiate between head, midshaft, proximal shaft, base, and avulsion fifth metatarsal fractures.
   3.0
- Recognize and evaluate the basic clinical and imaging characteristics to enable appropriate treatment in reference to closed versus open reduction.
   4.0
- Describe the advantages and disadvantages of closed versus open reduction in fifth metatarsal fractures.
   4.0
- 4. Describe the external and internal fixation principals in reference to the fifth metatarsal. **3.0**
- 5. Describe the complications and concerns with avascular nonunion of a Jones fracture. **3.0**

#### I. Lis Franc's Fracture

- Discuss the basic principles of closed reduction of Lis Franc's fractures.
   Recognize and evaluate the basic clinical and imaging characteristics to enable appropriate treatment in reference to closed versus open reduction.
   Describe the advantages and disadvantages of closed versus open reduction in Lis Franc's fractures.
   Describe external fixation and internal fixation principals in reference to metatarsals.
   Describe common Lis Franc fracture subtypes and discuss appropriate treatment and common
- long-term complications associated with such trauma.
- J. Midfoot Fractures (Navicular, Cuneiforms, Cuboid)
  - Discuss the basic classifications and mechanisms of midfoot fractures.
     Recognize and evaluate the basic clinical and imaging characteristics to enable appropriate
  - Recognize and evaluate the basic clinical and imaging characteristics to enable appropriate treatment in reference to closed versus open reduction.
     4.0
  - 3. Describe the advantages and disadvantages of closed and open reduction in midfoot fractures.

4.0

3.0

	4.	Describe external fixation and internal fixation principals in reference to the midfoot.	3.0
К.	Cal	caneal Fracture	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> </ol>	Discuss common mechanisms of injury associated with calcaneal fractures and describer most common classification schemes and incidence of associated injuries. Describe the most useful imaging modalities to ensure appropriate management. Evaluate common radiographic angles, such as Gissane's and Bohler's angle, and explain implications of the normal and abnormal values of each. Describe and select appropriate conservative and surgical treatment options of intra- ar articular calcaneal fractures. Describe the common classifications of Rowe, Essex-Lopresti, and Saunders. Discuss contra-indications to surgical intervention, including advantages and disadvanta internal and external fixation, in reference to the timing of surgical intervention. Discuss common long term pathology associated with calcaneal trauma.	the 3.0 3.0 the 3.0 d extra- 4.0 4.0 ges of 4.0 3.0
L.	Tal	ar Fractures	
М.	1. 2. 3. 4. 5. 6.	Describe normal talar anatomy, including vascular supply. Describe the pathophysiology of talar aseptic necrosis and explain clinical and imaging characteristics to aide in the diagnosis and treatment. Evaluate controllable and uncontrollable factors that can influence the normal bone hea process. Describe the Hawkin's talar fracture classification and the sequella of these injuries. Describe and select appropriate surgical and conservative treatment options of various fractures. Discuss the Berndt-Hardy classification with mechanism and long-term sequellae of osteochondral lesions and talar fractures.	3.0 3.0 aling 4.0 3.0 talar 3.0 4.0
	1. 2. 3. 4. 5.	Explain the Lauge-Hansen and Denis Weber ankle fracture classification schemes. Describe advantages and possible disadvantages of conservative and surgical treatment for ankle fracture types. Recognize and categorize different ankle fracture types, using imaging modalities. Describe basic principles of appropriate internal and external fixation. Describe the common short-term and long-term complications associated with trauma a fracture of the ankle.	3.0 options 4.0 4.0 3.0 and 4.0
N.	Pilo	on Fractures	
	1. 2. 3. 4. 5.	Explain Lauge-Hansen and Denis Weber ankle fracture classification schemes. Describe advantages and possible disadvantages of conservative and surgical treatment for ankle fracture types. Recognize and categorize different ankle fracture types, using imaging modalities. Describe basic principles of appropriate internal and external fixation. Describe the common short-term and long-term complications associated with trauma a fracture of the ankle.	3.0 options 4.0 3.0 and 4.0

# O. Physeal Plate injuries

1. Discuss basic anatomical characteristics of pediatric anatomy associated with physeal injuries.

	2.	Describe the Salter-Harris classification schemes used to describe physeal injuries and en	valuate
	3.	Imaging modalities used to classify such injuries. Describe and select appropriate conservative and surgical treatment options for physea	<b>3.0</b> l iniuries.
	-		3.0
	4.	Discuss common pathological sequellae associated with physeal injures.	4.0
Ρ.	Со	mpartment Syndrome	
	1.	Describe the mechanism of compartment syndromes (acute, traumatic or chronic, excer	rtional).
			3.0
	2.	Discuss physical evaluation and pressure testing of compartment syndromes.	3.0
	3.	Describe the treatment options of compartment syndromes.	3.0
	4.	Describe the challenges of nerve damage and muscle tissue loss defects.	4.0
Q.	Ac	ute and Chronic Tendon Trauma	
	1.	Discuss basic tendon anatomy and physiology, including tendo-Achilles, tibialis posterior peroneals.	r, and <b>3.0</b>
	2.	Describe the normal phases of tendon healing and explain how local and systemic factor	rs may
	3.	Recognize the basic subjective and objective characteristics consistent with tendon trau the lower extremity including tendo-Achilles runtures, tibialis posterior dysfunction, and	ma of
		subluxing peroneals.	3.0
	4.	Discuss the most appropriate imaging tools to aide in the evaluation and treatment of te	endon
		trauma of the lower extremity.	3.0
	5.	Describe and select appropriate conservative and surgical treatment options in reference	e to
		tendon trauma in the lower extremity.	3.0
R.	An	kle Sprains and Talar Dome Injuries, Lateral Ankle Instability	
	1.	Describe normal ankle and subtalar joint anatomy.	3.0
	2.	Describe the biomechanics of ankle and subtalar joint dislocations.	3.0
	3.	Describe clinical and imaging characteristics to aide in the diagnosis and treatment.	3.0
	4.	Describe the common talar fracture classification schemes.	3.0
	5.	Describe and select appropriate surgical and conservative treatment options of talar fra	ctures,
	c	Including osteochondral lesions.	3.0
	б. 7.	Discussiong-term sequalae of osteochondral lesions. Describe ankle stabilization procedures.	4.0 3.0
S.	Th	ermal Injuries	
	1	Describe the types and classifications of burns, thermal necrosis and frostbite	3.0
	2.	Discuss the importance of host response, circulation, wound healing, risk factors, and in	fections.
	3	Evaluate controllable and uncontrollable factors that can influence the normal wound h	ealing
	5.	process.	4.0
	4.	Describe the options and materials available for skin substitutes and grafting.	3.0
	5.	Describe the challenges of tissue loss defects.	4.0
Т.	Pu	ncture Wounds	

1. Describe the complications of foreign body and marine puncture wounds and infections. **3.0** 

2.	Discuss the importance of host response, risk factors in reference to the development	nt, and
	management of postoperative infections.	4.0
3.	Evaluate controllable and uncontrollable factors that can influence the normal wour	nd healing
	process.	4.0
4.	Recognize the basic characteristics of edema, hematoma, and infections and formula	ate
	appropriate evaluation and treatment options for each.	3.0
5.	Describe the normal anatomical compartments of the lower extremity, including the	e foot.
		3.0
6.	Discuss common etiologies of compartment syndrome, as well as diagnostic and treat	atment
	options.	3.0
7.	Discuss the pathophysiology of fracture blisters, as well as treatment options.	3.0
8.	Describe the treatment of infected wounds and human, animal, and insect bites.	3.0

#### XII. <u>Nerve Surgery</u>

#### A. Nerves of the Lower Leg, Ankle, and Foot

- 1. Identify, classify, and evaluate nerve entrapments that affect the foot and ankle. **4.0**
- 2. Discuss gross and microscopic lower extremity regional neuroanatomy.
- Discuss the pathophysiology of mechanically and metabolically induced neuropathy and classification of nerve injury, specifically Seddon and Sunderland Classification.
   4.0
- Discuss the normal and abnormal aspects of history and physical examination, including laboratory studies, and diagnostic tests (electrodiagnostic testing and imaging studies) based upon the chief complaint.
   4.0
- Discuss neurological surgical procedures, including neurolysis, neurectomy, and neurectomy with implantation.
   4.0
- Discuss the indications, contraindications, advantages, and disadvantages, of neurolysis, neurectomy, and neurectomy with implantation.
   4.0
- Discuss the immediate perioperative care requirements and postoperative management of neurolysis, neurectomy, and neurectomy with implantation.
   3.0
- Discuss the potential complications of nerve surgery, such as amputation neuroma and complex regional pain syndrome, and its management.
   4.0

### XIII. Heel Surgery

1.	Explain the etiology and pathogenesis of common heel deformities, including heel spurs a	ind
	heel pain syndrome and plantar fasciitis.	1.0
2.	Explain the etiology and classification of heel pain, including anatomical consideration,	
	biomechanical and systemic causes.	3.0
3.	Explain the incidence of heel pain syndrome and its clinical and radiographic evaluation. 4	I.0
4.	Explain the surgical treatment of heel spur surgery, including indications, contraindication	s,
	procedures, and complications.	3.0
5.	Explain the surgical approaches to the plantar fasciotomy, heel spur surgery, and the	
	complications that can occur in both.	3.0
6.	Discuss new forms of treatment, including low/high wave electromagnetic shock therapy	as
	well as autologous platelet concentration injections.	3.0

# B. Haglunds Deformity

	1.	Explain the etiology of Haglund's deformity, including biomechanical and systematic cau	ises, as
		well as anatomical considerations.	4.0
	2.	Explain the evaluation of a patient with Haglund's deformity, both clinically and radiogra	aphically,
		in a differential diagnosis.	4.0
	3.	Explain the surgical treatment including indications, contraindications, procedures, and	
		complications of Haglund's deformity.	3.0
С.	Ret	trocalcaneal Extotosis and Tendo Achilles Calcifications	
	1.	Explain the etiology and pathogenesis of the retrocalcaneal exostosis and the tendo ach	illes
		calcifications, including biomechanical and systematic causes.	4.0
	2.	Explain the clinical and radiographic evaluations of retrocalcaneal and tendo achilles	
		calcifications.	4.0
	3.	Explain surgical treatment including indications, contraindications, procedures, and	
		complications of the retrocalcaneal exostosis.	3.0
•	Sof	ft Tissue Surgery	

# A. Principles

XIV.

	1.	Discuss basic principles of soft tissue surgery, incision placement, healing, and basic postoperative management strategies.	4.0	
В.	Na	il Surgery: Chemical and Non-chemical procedure		
	1.	Identify and describe normal nail unit anatomy.	4.0	
	2.	Explain indications for nail surgery, including identification of various types of nail patho	ology	
	_	that may require surgical intervention.	4.0	
	3.	Discuss basic contraindications, as well as risks associated with nail surgery.	4.0	
	4.	Correlate appropriate nail procedure to underlying nail pathology.	4.0	
	5.	Explain the difference between elective and nonelective nail procedures.	4.0	
	6.	Identify various local anesthetic techniques, including type of anesthetic agent used for	nail	
		procedures.	4.0	
	7.	Describe skin plasties used to address nail pathology.	4.0	
	8.	Explain the terminology differences and between matrixectomy, I&D, and avulsion (part	tial and	
		total).	4.0	
	9.	Describe the surgical techniques for both partial and total nail avulsion.	4.0	
	10.	0. Differentiate between chemical and nonchemical matrixectomy, and explain advantages and		
		disadvantages of the various surgical matrixectomy techniques.	4.0	
	11.	Identify the chemicals used for chemical matrixectomy.	4.0	
	12.	Describe surgical technique and necessary instrumentation for both partial and total ch	emical	
		matrixectomy.	4.0	
	13.	List and describe the nonchemical matrixectomy procedures.	4.0	
	14.	Explain indications for, as well as risks and benefits of, nonchemical matrixectomy proce	edures.	
			4.0	
	15.	Explain and describe the clinical features of nail unit lesions that require biopsy.	4.0	
	16.	Describe nail unit biopsy techniques.	4.0	
	17.	Describe appropriate postoperative care following various nail procedures.	4.0	
	-		-	

	18.	<ul> <li>Explain complications that may occur following nail matrixectomy including recurrence, bleeding, extended healing times, scar formation, swelling, pain, infection, residual dyst excessive granulation tissue, deformity of the nail bed.</li> </ul>	rophy, <b>4.0</b>
С.	Su	bungual Exostosis	
	1.	Explain the pathoanatomy of subungual exostosis and the corresponding nail pathology	,
		associated with it.	4.0
	2.	Explain the origin of subungual exostosis.	4.0
	3.	Describe incisional approaches to subungual exostosis.	4.0
	4.	Explain surgical technique and instrumentation used to resect/remove the subungual explain surgical technique and instrumentation used to resect/remove the subungual explain surgical technique and instrumentation used to resect the subungual explain surgical technique and instrumentation used to resect technique and technique and instrumentation used to resect technique and tec	xostosis.
			4.0
	5.	Explain the role of pathology and microbiology with respect to surgical resection of sub-	ungual
	_	exostosis.	4.0
	6. 7.	Describe postoperative care for surgery related to subungual exostosis resection. Discuss complications associated with resection of subungual exostosis.	4.0
			4.0
D.	Ve	rruca Surgery	
	1.	Explain the etiology of pedal verruca, including specific viral origins.	4.0
	2.	List the differential diagnosis for both benign and malignant pedal verruca.	4.0
	3.	List of clinical characteristics of verruca, including divergent skin lines, pin-point bleedin	g with
		debridement, and pain with lateral pressure.	4.0
	4.	Explain the treatment options available and commonly used for pedal verruca.	4.0
	5.	Rationalize that no treatment works every time, but all treatments work some of the tir	ne.
			4.0
	6.	Explain the role of curettage in the treatment of pedal verruca.	4.0
	7.	Explain the technique of curettage of verrucous lesions, including necessary instrument	s and the
	_	concept of avoidance of penetration of the basement membrane.	4.0
	8.	Explain why any excised verrucous tissue should be sent to pathology for examination.	4.0
	9.	Explain that other modalities can be used to augment surgical curettage of pedal vertue	a,
		ablation	<b>10</b>
	10	Explain the use of various forms of laser in the treatment of pedal vertucal including car	- <b>H</b> on
	10.	dioxide laser and nulse dve laser	4.0
	11.	Explain the use of other forms of verrucous destruction, including electrocautery.	
		electrodessication and cryoablation.	4.0
	12.	. List postoperative management strategies for methods of surgical management of verru	uca.
			4.0
	13.	. List complications associated with surgical management of verruca to include scarring,	
		recurrence, delayed healing, infection, pain, and swelling.	4.0
Ε.	Os.	sicle/Sesamoid Surgery	
	1.	Identify pathology requiring excision of a pedal ossicle.	4.0
	2.	Differentiate normal variants from pathologic ossicles or sesamoids and explain the cau	se of
		such pathology.	4.0
	3.	Explain the surgical approach, technique, postoperative management, and complication	าร
		following ossicle excision.	4.0

#### XV. Specific Conditions Involving Surgery

#### A. Surgical Considerations and Surgery for the Rheumatory Arthritic patient

	1. 2. 3.	Discuss the surgical considerations of medications and systemic disease. Recognize the advantages and disadvantages of implants versus fusions. Describe the procedure of pan metatarsal head resection.	3.0 4.0 3.0
В.	Sui Rei	rgical Considerations and Surgery for the Diabetic Patient (Including Charcot construction)	
	1. 2. 3.	Describe the basic indications and risks for diabetic patients. Describe surgical options of muscular imbalance, including tenotomy and tendon transf Discuss the advantages and disadvantages of internal and external fixation.	3.0 ers. 4.0 4.0
С.	Sur	rgical Infections (Soft tissue/Bone) and Amputations	
	1. 2. 3. 4. 5.	Discuss diabetes and lower extremity healing. Describe tests for wound healing including arterial, venous, and oxygenation. Describe the surgical reconstruction of vessels. Choose the appropriate surgical procedure for various foot or leg ulcers. Discuss the diagnosis and treatment of osteomyelitis, including bone scan, MRI, biopsy, and plastic reconstruction.	3.0 3.0 4.0 3.0 excision, 3.0
D.	Ne	urologic Conditions Amenable to Surgery	
	1	Discuss the clinical presentation and examination of perve degeneration including gait	40

- Discuss the clinical presentation and examination of nerve degeneration, including gait. 4.0
   Describe muscle tendon imbalance and joint abnormalities. 3.0
- Describe muscle tendon imbalance and joint abnormalities.
   Recognize the advantages and disadvantages of tendon transfers and joint arthrodesis.
   4.0
- 4. Propose acceptable postoperative protocol and expectations for various procedures. **3.0**

### XVI. <u>Pediatric Surgery</u>

### A. General

- Execute a thorough birth and developmental milestone history interview and perform a physical exam on a pediatric patient.
   4.0
- 2. Discuss the perioperative management of a pediatric patient including pain control. **3.0**

# B. Juvenile Hallux Abducto Valgus

- 1. Be able to describe the etiology of juvenile hallux abducto valgus. **4.0**
- 2. Be able to describe the physical radiology exam findings for juvenile hallux abducto valgus.

- Be able to list and describe the surgical procedures for the correction of juvenile hallux abducto valgus. Also discuss the long term outcomes and potential complications.
   4.0
- C. Digital Deformities

	one step bone grafting and their potential complications.	4.0
Me	tatarsus Adductus	
1. 2. 3.	Discuss the etiology of metatarsus adductus. Discuss the gait, physical, and radiographic findings of metatarsus adductus. Identify and describe procedures in the surgical correction of metatarsus adductus inclu- tissue procedures (tendon releases/transfers, capsulotomies- Thompson, Heyman Herno Strong), osteotomies, including etatarsal and cuboid and cuneiform osteotomies.	<b>4.0</b> <b>4.0</b> ding soft don <b>4.0</b>
Cor	ngenital Pes Planus	
1.	Discuss the etiology of pes planus is rigid or flexible including rigid etiologies (tarsal coali	itions). <b>4.0</b>
2.	Detail the differences in gait and the physical exam findings, including planal dominance determining the rigidity o flexibility of the pes planus, including rigid etiologies (tarsal co and radiographic findings.	, in alitions) <b>4.0</b>
3.	Discuss the surgical options for the treatment of rigid or flexible pes planus including the arthroeresis, soft tissue correction, and osseous correction.	e role of <b>4.0</b>
4.	correction, and osseous correction.	4.0
Ver	rtical Talus	
1. 2. 3.	Discuss the etiology of vertical talus. Identify vertical talus upon physical examination and radiographically. Discuss soft tissue releases and osseous surgical correction of vertical talus and their lon outcomes and potential complications.	<b>4.0</b> <b>4.0</b> g term <b>4.0</b>
Clu	bfoot	
1. 2.	Discuss the etiology of clubfoot deformity. Identify clubfoot deformity upon gait examination, physical examination and radiograph	<b>4.0</b> iically. <b>4.0</b>
3.	Discuss soft tissue release, including capsulotomies and Achilles tenotomies for the surg correction of clubfoot deformities and their long term outcomes and potential complica-	ical tions. <b>4.0</b>

## D. Brachymetatarsia

1. Perform a history, physical examination, and radiographic evaluation as related to brachymetatarsia.

1. Be able to identify upon physical examination and radiographically: curly toe, congenital

possible surgical interventions. Also discuss the long term out comes and potential

interventions upon physical and radiographic examination.

minimus digitus varus, congenital hallux varus, and macrodactyly deformities along with their

2. Identify ectrodactyly, syndactyly, and polydactyly deformities along with their possible surgical

3. Discuss the long term outcomes and potential complications of ectrodactyly, syndactyly, and

2. Identify surgical options in the correction of brachymetatarsia including callous distraction or

#### Ε.

complications.

polydactyly deformities.

#### *F*.

4.0

4.0

4.0

4.0

### Н.

G.

#### XVII. **General Surgical Principles**

#### A. Instruments and Materials

1.	List and describe methods of obtaining hemostasis including tourniquets, bovies, and	
	hemostatic agents, and discuss the safety concerns of each.	4.0
2.	List the types of surgical drains utilized in surgery.	4.0

2. List the types of surgical drains utilized in surgery.

#### B. Perioperative Management

- 1. List the elements of a preoperative history and physical and the implications if the patients has comorbidities, including diabetes, hypertension, renal disease, and heart disease. 4.0
- 2. Discuss the proper use and selection of fluids and electrolyte management in the perioperative patient. 4.0
- 3. Discuss blood typing, the various blood products, proper administration including adjunctive medications administration, and transfusion reactions. 4.0
- 4. Identify and discuss commonly prescribed medications, including narcotics and antibiotics in the management of the perioperative patient along with their indications, contraindications, and alternatives. 4.0

#### XVIII. **Tarsal Coalitions**

- 4.0 1. Differentiate between a fibrous, cartilaginous, and bony coalition.
- 2. Describe the signs, symptoms, gait, and physical examination findings of talo-navicular, calcaneo-cuboid, calcaneo-navicular, and talo-calcaneal coalitions. 4.0
- 3. Describe the radiographic, CT or MRI findings as related to each specific tarsal coalition. 3.0
- 4. Describe the surgical approaches to the correction of tarsal coalitions. 4.0

#### XIX. Arthroscopy and Endoscopy of the Foot and Ankle

#### A. Historical

1. Explain the historical developments associated with arthroscopic and endoscopic foot and ankle surgery. 3.0

#### **B.** Principles

- 1. Explain the basic principles of arthroscopy and endoscopy. 3.0
- 2. Explain arthroscope visualization concepts including field of view, inclination of view, and clarity.
- 3. Explain why field of view is determined by lens angles. 3.0
- 4. Explain basic concepts of arthroscopic movement including positioning, sweeping, angulation (obliquity), triangulation, and rotation. 3.0

#### C. Preoperative Evaluation

1. Explain general indications for arthroscopic surgery, including (diagnostic) inability to make a specific diagnosis and (therapeutic) treatment of a known condition. 4.0

	2. 3. 4. 5.	<ul> <li>Explain the absolute contraindications to arthroscopic surgery to include localized soft to infection, as well as other relative contraindications.</li> <li>Explain why intra-articular infection is not a contraindication to arthroscopy (I&amp;D).</li> <li>Explain the importance of patient history and physical examination of the ankle and foor preoperative evaluation for arthroscopic/endoscopic procedures.</li> <li>Explain the basic concepts of a focused examination of the ankle and foot, including RO ligament testing, and correlative anatomical structure location with respect to foot and arthroscopic surgery.</li> </ul>	issue <b>4.0</b> <b>4.0</b> It in the <b>4.0</b> M, ankle <b>4.0</b>
D.	Im	aging	
	1. 2.	Explain the importance of weight bearing radiographic imaging of the foot and ankle wirespect to preoperative evaluation for arthroscopic foot and ankle surgery. Explain the role of other ancillary forms of imaging of the foot and ankle such as stress radiographs, arthrography, nuclear medicine, ultrasound, CT, and MRI with respect to preoperative evaluation for arthroscopic/endoscopic foot and ankle surgery.	th 4.0 4.0
Ε.	Ins	trumentation	
	<ol> <li>1.</li> <li>2.</li> <li>3.</li> <li>4.</li> <li>5.</li> <li>6.</li> <li>7.</li> </ol>	<ul> <li>Identify and explain the different types of irrigation used in arthroscopic surgery.</li> <li>Differentiate between gravity driven inflow and pump assisted inflow.</li> <li>Identify various sizes of arthroscopes used in foot and ankle surgery.</li> <li>Explain the uses of and interactions between the obturator, trochar, cannula, and the arthroscope.</li> <li>Identify and explain the role of accessory instruments, including spinal needles, scissors dissectors, graspers, biopsy forceps, knives, curettes, osteotomes, rasps, retrieving instructions and suture delivery systems.</li> <li>Identify and explain the role of power instruments, including joint shaver systems, abrae awls, debriders (mechanical, laser, radiofrequency), power reamers, and drills.</li> <li>Explain the difference between noninvasive and invasive forms of distraction with respective.</li> </ul>	3.0 3.0 3.0 , probes, tuments, 3.0 ders, 3.0 ect of
E	On	arthroscopy and the importance of distraction to the procedure.	3.0
	1. 2. 3.	Explain anesthesia and hemostasis concepts with respect to foot and ankle arthroscopic/endoscopic procedures. Describe positioning and preparation of a patient, including all equipment necessary to the operative leg. Describe the layout of the OR with respect to equipment position and duties of all OR p	<b>3.0</b> secure <b>3.0</b> ersonnel. <b>3.0</b>
G.	Со	rrelative Surgical Anatomy (Ankle Arthroscopy)	
	1. 2. 3. 4. 5.	<ul> <li>Explain be knowledgeable of the cross sectional anatomy of the ankle.</li> <li>Identify osseous landmarks.</li> <li>Identify tendon landmarks.</li> <li>Identify the location of the DP and PT artery with respect to other landmark structures.</li> <li>Describe the anatomic location of structures in the subcutaneous layer, including the superoneal nerve, sural nerve, and saphenous nerve, as well as the venous network.</li> <li>Describe the structures in the deep fascial layer, including the flexor and extensor tendor</li> </ul>	4.0 4.0 4.0 4.0 uperficial 4.0 ons of

the foot and ankle, the two deep neurovascular bundles. **4.0** 

Describe the ligamentous structures, including the tibiofibular syndesmosis, anterior inferior tibiofibular ligament, posterior inferior tibiofibular ligament, transverse tibiofibular ligament, the interosseous membrane, and ankle joint capsule (deltoid ligament, ATF ligament, CF ligament and PTF ligament).

#### H. Diagnostic Arthroscopic Examination (Ankle Arthroscopy)

- Identify the anatomic location and underlying correlative anatomy of the anterior portals, including, anteromedial, anterocentral, medial midline, anterolateral, and accessory anterior portals; transtalar portals (medial and lateral); posterior portals (including posteromedial, modified posteromedial, posterolateral, TransAchilles, coaxial, accessory posterior portals and endoscopic portals); and transmalleolar portals (medial and lateral).
- Identify and order the steps of the surgical technique protocol for introduction of the arthroscopic equipment into the appropriate portals.
   3.0
- 3. Explain the 21-point arthroscopic ankle examination. **3.0**
- 4. Explain postoperative management strategies after various types of arthroscopic surgeries.

I. Soft Tissue Lesions

Explain the pathogenesis, identify the arthroscopic appearance, and describe the arthroscopic management techniques for forms of soft tissue ankle pathology, including congenital plicae, adhesions (fibroarthrosis), capsulitis, local synovitis, generalized synovitis (posttraumatic), infectious synovitis (pyarthrosis), various soft tissue impingements, rheumatoid synovitis, PVNS, synovial chondramatosis, hemophilia, and other inflammatory arthritides.

#### J. Osteochondral Pathology

1. Explain the pathogenesis, identify the arthroscopic appearance, and describe the arthroscopic management techniques for forms of osteochondral pathology, including surface defects, osteochondritis dissicans, loose bodies, osteophytes, talardome cysts/lesions, arthritis. **3.0** 

#### K. Other Pathology

- Explain the indications, rationale, and methods for arthroscopic treatment of acute ankle fractures and post-fracture defects.
   3.0
- Explain the indications, rationale, and methods for arthroscopic treatment of lateral ankle instability.
   **3.0**
- 3. Explain the indications, rationale, and methods for arthroscopic ankle arthrodesis. **3.0**
- 4. Explain the indication for arthroscopic foreign body removal.

#### L. Other Joint Arthroscopy

- 1. Explain the indications, rationale and methods for arthroscopic subtalar joint surgery including subtalar arthroscopic arthrodesis. **3.0**
- Explain the indications, rationale and methods for arthroscopic calcaneal cuboid joint surgery, and First metatarsophalangeal joint arthroscopy.
   **3.0**

#### M. Rehabilitation after Foot and Ankle Arthroscopy

 Describe appropriate rehabilitation modalities following foot and ankle arthroscopic surgery and Explainexplain the timing for implementation of each phase of rehabilitation.
 4.0

3.0

 Explain the goals of the rehabilitation modalities and their effects on ROM/flexibility, strength, and coordination.
 4.0

#### N. Complications in Ankle and Foot Arthroscopy

- Identify and discuss possible complications following foot and ankle arthroscopy, including neurovascular injury associated with portals, tendon injuries, ligament injuries, articular cartilage injury, accelerated DJD (related to direct cartilage injury), instrument breakage, fluid management complications, compartment ischemia, wound complications, infection, postoperative swelling, thrombophlebitis and PE, CRPS, and postoperative stress fractures.
  - 4.0
- ExplainDiscuss techniques or measures to minimize or avoid surgical and postsurgical complications with respect to arthroscopic foot and ankle surgery.
   4.0

#### **O.** Endoscopic Procedures

- 1. Identify nonarticular soft tissue pathologies treatable with endoscopic surgical methods. 3.0
- ExplainDiscuss endoscopic procedures to treat nonarticular soft tissue pathology, inlcuding plantar fasciotomy, gastrocnemius recession, tarsal tunnel release, excision of a retrocalcaneal bursa, resection of Haglund's deformity, and external neurolysis.
   **3.0**

# **COMMUNITY HEALTH LEARNING OBJECTIVES**

Public Health Biostatistics Jurisprudence in Public and Community Health Epidemiology

# I. Public Health

1.	Define:	
	a. illness	4.0
	b. <i>disease</i>	4.0
	c. quality assessment	3.0
	d. quality assurance	2.0
	e. social marketing	3.0
2.	Differentiate between health care, medical care, and public health.	4.0
3.	Define wellness and prevention according to NIH standards.	1.0
4.	Distinguish between epidemic, endemic, and pandemic.	4.0
5.	Outline the historical evolution of health care in the United States, including the char	iges in
	organization structure, social structure, and technology.	2.0
6.	Describe changes of the following disease patterns as health care had evolved in the States:	United
	a. epidemics of acute infectious diseases affecting population groups	3.0
	<li>b. acute infectious and traumatic events affecting individuals</li>	3.0
	c. chronic diseases	3.0
	d. special chronic diseases (related to genetic make-up, environmental hazards	and
	individual lifestyle)	3.0
7.	Discuss events that have led to changes in disease patterns as health care has evolved	d in the US.
		4.0
8.	List and describe the major subsystems of the US health care system.	3.0
9.	Differentiate between the pathophysiologic and epidemiologic processes involved in	disease
	production.	3.0
10.	Describe indicators and predictors of health services utilization.	3.0
11.	Discuss factors contributing to an increase in health care spending.	3.0
12.	Perform a cost-benefit analysis with an outcomes-based approach.	2.0
13.	Discuss the origin and development of health insurance in the US.	2.0
14.	Describe the historic models of health care in England, Canada, Germany, and Cuba.	2.0
15.	List and describe the categories of health insurance in the United States.	3.0
16.	List and describe regulatory interventions used to regulate and monitor the health ca	re system.
17	List and describe the three major criteria areas upon which quality according to be	<b>2.0</b>
17.	List and describe the three major chteria areas upon which quality assessment is basi	20.
18	Discuss the mission of the $\Lambda C \Lambda$	1.0
10.	a Define meaninaful use	1.0
	h Explain the National Healthcare Quality Improvement Strategy	1.0
	c Outline the core quality measures	1.0
19	Explain the role of a Professional Review Organization (PRO)	3.0
20	Explain risk management	3.0
21	Describe the mission and basic layout of the Department of Health and Human Servic	es (DHHS)
22.	Discuss the various agencies in the US that provide or use public health services (eg. 1	nstitute of
	Medicine [IOM] part of the National Academy. The Department of Labor to include the	ne Hospital
	Safety and Health Administration [OSHA], National Institutes of Health [NIH]. Centers	for
	Medicare and Medicaid Services [CMS], Center for Disease Control and Prevention [C	DC], The
	Food and Drug Administration [FDA], United States Preventive Services Task Force).	2.0
23.	Explain the function of the Surgeon General.	3.0

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24. List and describe the ten essential public health services.	4.0
25. Relate the ten essential public health services to the clinical practice of podiatric m	edicine.
26. Explain the goals and focus objectives of "Healthy People 2020."	4.0
27. Describe how "Healthy People 2.002.00" relates to, and affects the clinical practice	of podiatric
medicine.	4.0
28. Explain the function of the state and local health departments.	3.0
29. Discuss advocacy as it relates to the profession of podiatric medicine and the patie	nt. <b>2.0</b>
30. Describe stakeholdership and its relation to everyday practice and program develo	pment.
31. Discuss the Health Belief Model (HBM) and the Transtheoretical Model in relation 1	0
interpersonal health and human behavior.	3.0
32. List the steps of HBM and discuss its use in patient education.	3.0
33. Discuss the relationship of HBM and the Transtheoretical Model to wellness and to	prevention.
34. Discuss the use of the Transtheoretical Model in treating addictive behavior (eg, sn	oking, sex,
and alcoholism).	2.0
35. Outline Everett Roger's Diffusion of Innovation Model of Behavior (population heal	th and
human behavior model), including its five stages and use in podiatric medicine.	2.0
36. Discuss the ecological model of community health development (Glanz and Rimer).	1.0
37. List and explain the 4 Ps of social marketing.	2.0
38. Discuss the use of social marketing in program development for the purposes of pr	oviding
podiatric medical services.	2.0

# II. <u>Biostatistics</u>

1.	Define:	
	a. inferential statistics	3.0
	b. confidence interval	3.0
	c. Bayes' theorem	1.0
2.	Define and calculate the measures of central tendency.	4.0
3.	Define and calculate the measures of dispersion.	4.0
4.	Differentiate continuous, discrete, ordinal (ranked), nominal (categorical), and dichot	omous
	data types.	4.0
5.	Compare normal, binomial, and skewed distribution.	3.0
6.	Differentiate the central limit theorem from central tendency.	3.0
7.	Differentiate between independent and dependent variables.	4.0
8.	Describe the role of hypothesis testing in research.	4.0
9.	Differentiate clinical significance from statistical significance.	4.0
10.	Compare reliability and validity.	4.0
11.	Define the <i>p</i> value and describe its role in supporting or rejecting the null hypothesis.	4.0
12.	Explain null and alternative hypotheses.	4.0
13.	Distinguish between type one (alpha) and type two (beta) errors.	3.0
14.	Describe the standard error of the mean and how this plays a role in the confidence in	nterval.
		3.0
15.	Differentiate parametric versus non-parametric testing and recognize the indications	and
	contraindications of each test.	3.0
16.	Describe the interrelationships among test efficiencies (function of the assumptions n	nade by
	the test and data types employed), sample size and magnitude of effect, and statistical	al
	significance.	3.0

17.	Select the appropriate test to measure trends, differences and interactions.	3.0
18.	Recognize and explain usage of box and whisker plots.	1.0

## III. Jurisprudence in Public and Community Health

1.	Define:	
	a. anti-kick back	
	b. Stark Law	4.0
	c. power of attorney	3.0
	d. res ipsa loquitur	3.0
	e. quid pro quo	3.0
	f. respondeat superior	3.0
	g. joint liability	3.0
	h. several liability	3.0
	i. conflict of interest	3.0
	j. transparency	3.0
2.	Differentiate between constitutional laws, statutes, administrative laws, and commo	n laws.
		3.0
3.	Describe the importance of scope of practice in the practice of podiatry.	4.0
4.	Describe legal ramification of the False Claims Act and explain Qui Tam Enforcement.	3.0
5.	Discuss standard of care and statute of limitations.	4.0
6.	Compare and contrast implied consent and informed consent.	4.0
7.	Define and recognize negligence.	4.0
8.	Explain and recognize HIPAA violation.	4.0
9.	Provide examples of things that might result in disciplinary action by a professional li	censing
	board.	4.0
10.	Describe the contract arrangement between doctor and patient.	4.0
11.	Explain what is meant by the term "unprofessional conduct."	4.0
12.	Explain investigative procedure, deposition, and discovery as they relate to medical r	nalpractice.
		3.0
13.	Recognize the legal ramifications and requirements associated with mandatory report	rting of
	child abuse and neglect.	4.0
14.	Explain what is meant by "breach of contract."	4.0
15.	Differentiate between a mission statement and a vision statement.	3.0
16.	Explain the Health Care Quality Improvement Act.	2.0
17.	Explain inurement laws and the impact on physician practice.	3.0
18.	Describe the purpose and reporting requirements of the National Practitioner Data B	ank.
		3.0
19.	Explain the ethical requirement of confidentiality of patient information.	3.0
20.	Describe the legal requirements for the prescription of a controlled substance.	4.0
21.	Explain the importance of timely and accurate charting with respect to medical malp	ractice.
		4.0
22.	Explain the Public Health Service Act.	1.0
23.	Explain the Patient Protection and the Affordable Care Act.	1.0

# IV. <u>Epidemiology</u>

1. Define:

	a.	epiden	niology	4.0		
		i.	descriptive epidemiology	4.0		
		ii.	analytical epidemiology	4.0		
	b.	relativ	e risk	3.0		
	с.	odds ra	ntio	3.0		
	d.	hazara	ratio	3.0		
2.	Differentiate between incidence and prevalence. 4.0					
3. Differentiate between sensitivity and specificity and discuss the relationship to false						
	and fal	se nega	tives.	4.0		
4.	Differe	ntiate b	etween positive and negative predictive values of a diagnostic test.	4.0		
5.	Explain crude rates. 3.0					
6.	Construct a 2x2 contingency table and demonstrate its use in calculating sensitivity, specificity					
	relative	e risk, ar	nd odds ratios.	3.0		
7.	Describe receiver operating characteristic (ROC) curves.					
8.	Define and interpret the likelihood ratio. <b>3.0</b>					
9.	Differentiate between internal and external validity.					
10.	). Recognize threats to internal validity. 4					
11.	Differentiate between observational and experimental studies. 4.0					
12.	2. Identify sources of and means to control bias, including randomization, blinding, matching,					
	inclusio	on criter	ia, exclusion criteria.	4.0		
13.	Discuss	s the hie	rarchical levels of evidence of a study based on study design.	4.0		
14. Discuss the relative values of summary investigations including Systematic revie				Meta-		
	analyse	es (eg, C	ochrane Collaboration), Clinical Practice Guidelines (CPG), Decision a	nalyses and		
	Econor	nic eval	uative studies.	4.0		
15.	15. Calculate and interpret the numbers needed to treat (NNT), numbers needed to preve			event (NNP)		
	and nu	mbers r	eeded to harm (NNH) with respect to a specific medical condition.	4.0		
16.	Describ	be the ro	ble of the Internal Review Board (IRB).	3.0		
17.	Differentiate between practice informed consent and research informed consent. <b>4.0</b>			4.0		
18.	Interpret the ethical issues in clinical research. <b>3.0</b>			3.0		
19.	Explain	ICD-10	and its role in surveillance.	1.0		

# **APPENDIX I: Bloom's Taxonomy**

**Bloom's Taxonomy (1954) and the 6 levels of the cognitive domain** - According to Kretchmar the intention of the taxonomy was to classify the change in a person created by an educational experience (2008). In this case we are focusing only on changes within the cognitive domain, which are in 6 different hierarchical levels. Although many researchers have agreed upon the hierarchical nature of the first four levels there continues to be debate around the last two levels synthesis and evaluation and whether they are in fact hierarchical or perhaps they are equal but different types of complex thinking. Many researchers have compared synthesis with creative thinking and evaluation with critical thinking. The revised taxonomy has placed these categories in the reverse order. Although it was noted by the original authors of the taxonomy that perhaps evaluation was not in fact hierarchal it is the last level in the original taxonomy, as seen below:

Level 1—Knowledge	Knowledge Verbs include:
The first level of Bloom's Taxonomy within the	Arrange
cognitive domain. At this level, instruction should	Define
focus in on enabling learners to remember, or	Describe
recognize concepts, processes, procedures,	Duplicate, Repeat
theories, or facts. This level includes both factual	Identify
knowledge and more abstract knowledge or	Label
knowledge of universals (eg, theories) or ways and	List
means of dealing with specifics (eg, recognizing	Match
how our educational system has	Name
evolved) (Kretchmar, 2008).	Order
	Recall
	Recognize
	Record
	Relate
	Remember
	re-order
	Reproduce
	Select
	State
Level 2—Comprehension	Comprehension Verbs
This is the second level of Bloom's Taxonomy	Classify
within the cognitive domain. At this level,	Convert
instruction should focus in on enabling learners to	Defend
translate facts into their own words,	Describe
understanding the interrelations enough to form	Discuss
opinions, make predictions, and make judgments	Distinguish
because the information has been integrated into	Examples
their "own frame of reference" and they can apply	Explain
the knowledge as they have been shown (or	Generalize
similarly to how they have been shown) to	Infer
apply it (Reeves, p. 610).	Paraphrase
	Predict

	Provide
	Review
	Rewrite
	Summarize
	Translate
Level 3—Application	Application Verbs
This is the third level of Bloom's Taxonomy within	Application verbs
the cognitive domain. At this level instruction	Change
should focus in on enabling learners to apply their	Compute
new knowledge within situations beyond what	Create
they have seen in the classroom setting. This is the	Demonstrate
heginning of critical thinking through hasic	Employ
nrohlem solving and the demonstration of transfer	Illustrate
of learning	Interpret
of rearring.	Maninulate
	Modify
	Practice
	Propare
	Produce
	Polato
	show
	Show
	Solvo
	036
Loval 4 - Analysis	Analysis Vorbs
Level 4—Analysis	Analysis Verbs
<b>Level 4—Analysis</b> This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level	Analysis Verbs Analyze Appraise
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to	Analysis Verbs Analyze Appraise Breakdown
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts	Analysis Verbs Analyze Appraise Breakdown
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the	Analysis Verbs Analyze Appraise Breakdown Calculate
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate Distinguish
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate Distinguish Examine
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate Distinguish Examine Experiment
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate Distinguish Examine Experiment Illustrate Model
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate Distinguish Examine Experiment Illustrate Model
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Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis VerbsAnalyzeAppraiseBreakdownCalculateCategorizeCompareContrastDiagramDifferentiateDistinguishExamineExperimentIllustrateModelQuestionRelateSeparate
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate Distinguish Examine Experiment Illustrate Model Question Relate Separate
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes.	Analysis Verbs Analyze Appraise Breakdown Calculate Categorize Compare Contrast Diagram Differentiate Distinguish Examine Experiment Illustrate Model Question Relate Separate Subdivide
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes. Level 5—Synthesis (Creative Thinking) This is the fifth level of Bloom's Taxonomy within	Analysis VerbsAnalyzeAppraiseBreakdownCalculateCategorizeCompareContrastDiagramDifferentiateDistinguishExamineExperimentIllustrateModelQuestionRelateSeparateSubdivideSynthesis Verbs
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes. Level 5—Synthesis (Creative Thinking) This is the fifth level of Bloom's Taxonomy within the cognitive domain. Many records have	Analysis VerbsAnalyzeAppraiseBreakdownCalculateCategorizeCompareContrastDiagramDifferentiateDistinguishExamineExperimentIllustrateModelQuestionRelateSeparateSubdivideArrangeArsamble
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes. Level 5—Synthesis (Creative Thinking) This is the fifth level of Bloom's Taxonomy within the cognitive domain. Many researchers have compared this level of the cognitive domain to	Analysis VerbsAnalyzeAppraiseBreakdownCalculateCategorizeCompareContrastDiagramDifferentiateDistinguishExamineExperimentIllustrateModelQuestionRelateSeparateSubdivideArrangeAssembleCombino
Level 4—Analysis This is the fourth level of Bloom's Taxonomy within the cognitive domain. At this level, instruction should focus in on enabling learners to breakdown their acquired knowledge into parts through the consideration of elements, the relationships of those elements, and the organizing principles. Key to this level within the cognitive domain is the act of deductive and inductive thought processes. Level 5—Synthesis (Creative Thinking) This is the fifth level of Bloom's Taxonomy within the cognitive domain. Many researchers have compared this level of the cognitive domain to arreative thinking. Therefore, at this level	Analysis VerbsAnalyzeAppraiseBreakdownCalculateCategorizeCompareContrastDiagramDifferentiateDistinguishExamineExperimentIllustrateModelQuestionRelateSeparateSubdivideArrangeAssembleCombineCombineCombineCombine

instruction should focus in on enabling learners to	Construct
be able to take the breakdown of parts from the	Create
analysis phase and form new relations, a new	Design
whole resulting in a creative solution to a	Develop
proposed problem, which was not covered within	Formulate
the classroom setting.	Generate
	Rearrange
	Reconstruct
	Relate
	Reorganize
	Revise
	Re-write
	Solve
	synthesize
Level 6—Evaluation (Critical Thinking)	Evaluation Verbs
This is the six level of Bloom's Taxonomy within	Appraise
the cognitive domain. Many researchers have	Argue
compared this level of the cognitive domain to	Assess
critical thinking. Therefore, at this level, instruction	Compare
should focus in on enabling learners to be able to	Conclude
take the breakdown of parts from the analysis	Contrast
phase and form new relations through the process	Defend
of evaluation by using a set of content specific	Evaluate
criteria.	Judge
	Justify
	Interpret
	Support

Sources:

Kretchmar, J. (2008). Taxonomy of Educational Objectives - The Cognitive Domain. In , *Taxonomy of Educational Objectives-Cognitive Domain -- Research Starters Education* (p. 1). Great Neck Publishing. Retrieved from EBSCO*host*.

Reeves; M, F. (n.d). An Application of Bloom's Taxonomy to the Teaching of Business Ethics. *Journal of Business Ethics*, 9(7), 609. Retrieved from EBSCOhost.

\*\*\*Table provided by Sarah S. Wormwood, ©2012.

# **APPENDIX II: Ranking Key**

The following key was used by the content area groups when developing their sections:

- 4 = Absolutely essential for preparation for podiatric medical residency
- 3 = Requires significant emphasis for preparation for residency

moderate emphasis for preparation for residency

2 = Requires 1 = Requires

marginal emphasis for preparation for residency

0 = Does not require emphasis for preparation for podiatric medical residency

# **APPENDIX III: AACPM Council of Faculties**

#### Arizona School of Podiatric Medicine at Midwestern University (AZPod)

Glendale, Arizona

Denise B. Freeman, DPM, MSE Associate Program Director

Chair

Pamela E. Potter, PhD Professor and Chair, Department of Pharmacology

#### **Barry University School of Podiatric Medicine (BUSPM)**

Miami Shores, Florida

Sanjay Sesodia, PhD Chair of Basic Medical Sciences Professor of Anatomy/Neurophysiology

Christopher Peterson, DPM Director of Clinical Education

# California School of Podiatric Medicine at Samuel Merritt University (CSPM)

Oakland, California

Bruce A. Richardson, PhD Professor and Associate Dean, Preclinical Affairs

Eric D. Stamps, DPM Associate Dean for Clinical Affairs

#### College of Podiatric Medicine and Surgery at Des Moines University (CPMS)

Des Moines, Iowa

James A. Mahoney, DPM Associate Dean for Academic Affairs

Donald G. Matz, PhD Chair and Professor, Department of Anatomy

# Kent State University College of Podiatric Medicine (KSUCPM) (formerly Ohio College of Podiatric Medicine)

Independence, Ohio
Marie M. Blazer, DPM Assistant Professor, Department of Podiatric Medicine

Ronald Wright, PhD Professor of Microbiology/Immunology and Assistant Dean of Preclinical Affairs

## New York College of Podiatric Medicine (NYCPM)

New York, New York

Eileen D. Chusid, PhD Dean of Pre-Clinical Sciences

Robert Eckles, DPM, MPH Dean of Graduate Medical and Clinical Education

## Dr. William M. Scholl College of Podiatric Medicine at Rosalind Franklin University of Medicine and Science (SCPM)

North Chicago, Illinois

John Becker, PhD Chair Elect Professor of Basic Biomedical Sciences

Karona Mason, DPM Assistant Dean of Clinical Sciences

## **Temple University School of Podiatric Medicine (TUSPM)**

Philadelphia, Pennsylvania

Helen E. Pearson, PhD Associate Professor, Department of Anatomy and Cell Biology

Marc Karpo, DPM Assistant Professor, Podiatric Medicine and Orthopedics

## Western University of the Health Sciences College of Podiatric Medicine (WUCPM)

Pomona, California

Jonathan Labovitz, DPM Associate Professor and Chair, Department of Medicine, Surgery and Biomechanics

Mathew Wedel, PhD Assistant Dean for Pre-Clinical Curriculum Assistant Professor of Anatomy Assistant Professor of Anatomy