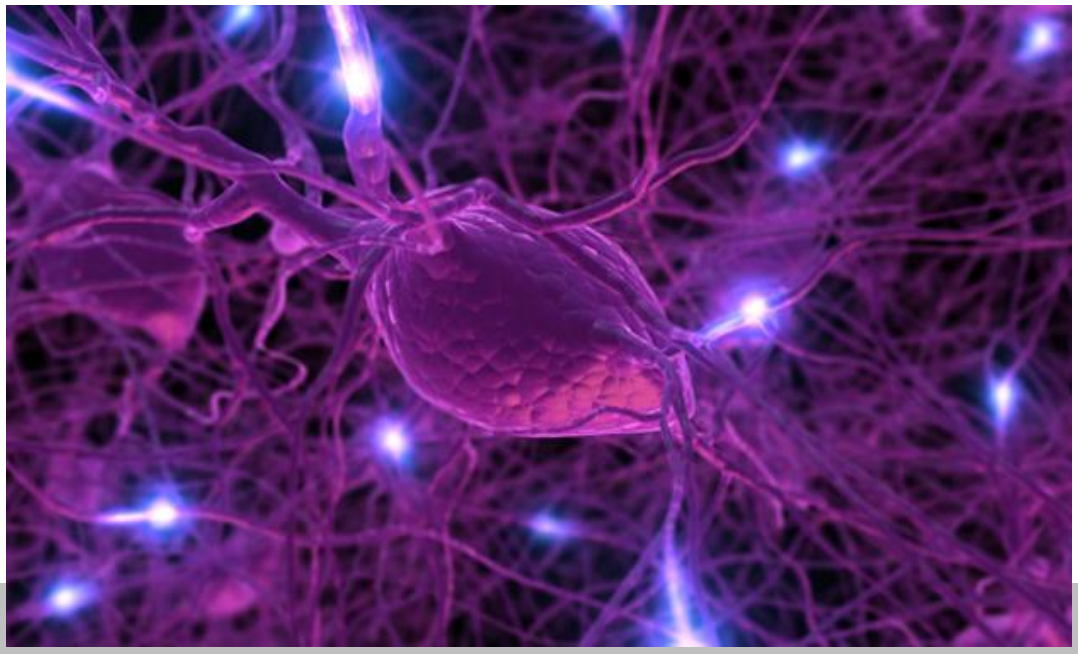
COLLEGE OF MEDICINE
Department of Medical Education
Curriculum Development & Research Unit



STUDENT'S BOOK

NEUROPSYCHIATRY BLOCK (NEUR 222)

YEAR 2 (Female Group)

2017-2018
(1438-1439)

COLLEGE OF MEDICINE
Department of Medical Education
Curriculum Development & Research Unit

THE NEUROPSYCHIATRY BLOCK

Year Two

BLOCK BOOK AND STUDENT GUIDE

(17 September 2017 to 23 November 2017)

(2017-2018) 143-1439

A message from the Dean

We are pleased with your progress in the medical program and your achievements. Being a first year medical students is a great opportunity for you to consolidate what you have learnt in the preparatory year and prepares you for the clinical skills and competencies needed in the clinical years. The Department of Medical Education through its different units is working hard to create an integrated and innovative curriculum that builds on the changes introduced in the preclinical years and enforces best teaching/learning approaches in the design of the new medical curriculum. As you are aware, the College of Medicine at King Saud University is one of the best colleges not just in the Kingdom of Saudi Arabia but proved to be one of the best in the gulf region, and the Middle East. It also has its international influence among the best colleges of medicine worldwide. This makes us proud of our achievements and provides you with an insight about the quality of teaching and research that we have reached and our continuous work to maintain our standards.

Therefore, the medical curriculum aims at preparing you and equipping you with the best training and clinical skills to become a medical graduate that fulfils the highest international standards. Therefore, the focus of the curriculum is to enhance a number of skills such as case-based learning, critical thinking, self-directed learning, deep understanding of concepts, application of knowledge learnt, and how to make decisions on the basis of evidence. The curriculum also aims at enhancing your skills in areas such as professionalism, e-learning, task-based learning, and preparing you for life-long learning. The design of the curriculum encourages small group learning, use of cases for discussion, lectures, student-led seminars, bed-side teaching, task-based learning, use of multimedia and e–learning as modes for teaching and learning. The use of wide range of teaching and learning modes and small group discussion will help you to become active learners, and work with other students in your group as a team.

I wish you all the best during your academic year and would encourage all of you to get the best out of the teaching and learning opportunities provided to you during this year. Our teaching staff and clinicians would be very happy to help you on any issue that you need help with.

Professor Khalid Ali Fouda Neel

Dean, College of Medicine and the Supervisor of University Hospitals

A Message from the Vice Dean for Academic Affairs

It is my pleasure to welcome you all to the second year of Medicine. I would like to take this opportunity to congratulate you all on your success and achievements. There is no doubt that you have worked hard during the first year to adapt to the university system and our new integrated curriculum. In the mean time, we would like you to remember that success is not a destination, success is a journey and there will be many challenges during your journey of success. A successful person would turn these challenges into opportunities for success.

As you might be aware, our faculty under the leadership of our Dean is moving into an integrated curriculum that encourages small group learning and student-centered approaches for learning. To achieve these goals we have established the Department of Medical Education under the leadership of Dr Mona Soliman and his teams to develop the new integrated curriculum. The design of the new curriculum is focused on the students not the teachers. Our aim is to equip each of you with the current teaching and learning strategies that are used in the best universities worldwide and ensure that you will be an excellent medical doctor who will be committed to the profession and willing to serve patients in our country, our region, and wherever our government and our professional bodies would ask you for help.

On these bases, our aim is not just to graduate more doctors; our aim is to ensure that doctors graduating from our university are equipped with knowledge, skills, behavior, and competencies needed for best practice of medicine anywhere in the world. This goal makes a lot of responsibility from your end and we would like you to take this opportunity and work effectively to achieve your goals. Our academic and clinical staff are expert in their areas and very eager to help and support you to achieve your dreams. I would encourage you to ask for help when needed and our support team would work with you on any challenges you might face during the course. I wish you all the best.

Dr. Saleh Adhehri

Vice Dean for Academic Affairs

College of Medicine

A Message from the Neuropsychiatry Block Chair

I would like to take this opportunity to thank all of the faculty members of the basic and clinical medical sciences and the members of Department of Medical Education for their support and cooperation to make the Nervous System Block more revealing for the students. This eight week block is designed to integrate the basic concept of neurosciences between basic and clinical sciences. It will be based on small group discussion tutorials, lectures, essential practical introduction to clinical medicine of communication skills, professionalism and self learning sessions. This course includes knowledge and skills of all basic and clinical sciences related to the nervous system and also psychological disorders. The contents of this block will also help the students to come to the differential diagnosis of the neurological diseases and reach at the final conclusion. This course is designed to facilitate the students to enhance their knowledge, develop independent thinking, communication skills and leadership qualities. I believe that, this block will bring new optimism and an improved platform for the students to learn and enhance their knowledge and skills in better understanding of nervous system and associated diseases and their management. We welcome the feedback and constructive comments from the students as well as faculty members.

Professor Sultan Ayoub Meo

Neuropsychiatry Block Chair

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General Information

Block Title	Neuropsychiatry Block
Block Code & Number	NEUR 222
Credit Hour	12
Block Duration	10 Weeks
Block Dates	17th September to 23th November 2017
Block Chairman	Dr. Sultan Ayoub Meo

Teaching Staff
Year 2 – Female Group

Department	Name	Mobile No. / Ext.	E-mail
Anatomy	Dr. Sanaa Al-Shaarawi	0507375313	salsharawi@ksu.edu.sa
	Dr. Raeesa Abdulatawab	0509019047	rmohammad@ksu.edu.sa
	Dr. Jamilah El-Medany	0508955020	galmadani@ksu.edu.sa
Histology	Dr. Raeesa Abdulatawab	0509019047	rmohammad@ksu.edu.sa
Physiology	Dr. Faten Zakaria	0559268557	fatenz3699@hotmail.com
	Dr. Fawzia Al Roug	0504113717	falrouq@ksu.edu.sa
	Dr. Hayam Gad	0559621986	Hayam_gad@hotmail.com
	Prof. Laila Al Ayadhi	Ext.52936	lyayadhi@ksu.edu.sa
	Dr. Aida Korish	0506282704	iaidakorish@yahoo.com
	Dr. Nervana Bayoumy	0550555217	nbayoumy@ksu.edu.sa
	Dr. Ola Mawlana	0541991876	olamawlana@gmail.com
Pharmacology	Prof. Hanan Hagar	0507554152	hananhhagar@yahoo.com
	Prof. Yieldez Bassioni	0565794577	Yieldez@yahoo.com
	Dr. Alia Alshawanani	0505175545	aalshanawani@ksu.edu.sa
Pathology	Dr. Hala Kfoury	0504127781	halakfoury@hotmail.com
	Dr. Shaesta Zaidi	0542028371	Snz24@yahoo.com/ snz24@hotmail.com
Microbiology	Dr. Maha Al Muhaizea	0506460990	maha_mm990@hotmail.com
	Dr. Fawzia Al-Otaibi	0553223309	ofawzia04@ksu.edu.sa / ofawzia@ksu.edu.sa
	Dr. Malak El-Hazmi	0505403663	malak_elhazmi@hotmail.com
	Prof. Hanan Habib	0504138199	hahabib@ksu.com.sa
Biochemistry	Dr. Sumbul Fatma	0598245851	sumbulfatma@gmail.com
	Dr. Rana Hasanato	0504479022	ranamomen@yahoo.com / rhasanato@ksu.edu.sa
Radiology	Dr. Faten Al-Mohaideb	0555456852	fatenalmohideb@gmail.com
Psychiatry	Dr. Abdulrahman Al-Wahibi	0551584549	abalwahibi@ksu.edu.sa
	Dr.Noor Al Modihesh	0559114399	nalmodaihesh@gmail.com
Professionalism	Prof. Hanan Habib	0504138199	hahabib@ksu.edu.sa
	Prof. Lulu Alnuaim	0505401021	lalnuaim@ksu.edu.sa
	Dr. Hala Kfoury	0504127781	halakfoury@hotmail.com

List of the Problem-Based Learning Cases

The table below summarizes the PBL cases to be discussed in the Neuropsychiatry Block.

Week	Case Number	Case Title
W1	**NO CASE**	
W2 (Monday & Thursday)	Case 1	"…I have difficulty in swallowing!"
W3 (Monday & Thursday)	Case 2	"I feel unsteady"
W 4	**NO CASE**	
W5 (Monday & Thursday)	Case 3	" Unable to talk"
W6 (Monday & Thursday)	Case 4	"I have tremor"
W7 (Monday & Thursday)	Case 5	"Absent from school"

Instructions:

The cases listed above will be discussed by students in their small groups. Each group is about 8 to 12 students. Each case will be discussed in two tutorials, on Sunday and Wednesday. Each tutorial is two hours long.

Attendance of Small Group Learning tutorials:

Students must attend all small group learning tutorials. If a student is not well she needs to provide a medical certificate from their family doctor. If a student misses out to attend four tutorials without acceptable reason, he/she might not be allowed to attend the final examination.

Students Roles in Small Group Learning Tutorials:

The design of the curriculum encourages small group discussion and student-centered learning. To achieve these goals there is a need for establishing good group dynamics, interpersonal skills, and effective communication. These elements will ensure that learning is an enjoyable process and rewarding to each member in the group. Therefore, students play a vital role in making a difference in their groups. To achieve these changes and improve your learning outcomes, we recommend that you use the paper by Professor Samy Azer, titled "Becoming a Student in a PBL Tutorial", a copy is enclosed in the Appendix. Your continuous reflection on these tips and working on identifying your role in your group will help you in reaching these goals and building up your group.

Objectives of the Block

By the end of the course, the students should be able to:

- Understand the relationship between the anatomical structures of the different parts of the nervous system and their functions.
- Understand the pathology, microbiology, pathogenesis, and factors contributing to the development of common diseases affecting the nervous system.
- Use basic sciences to explain patient's signs and symptoms, interpret investigation results, and provide justifications for the different views.
- Develop effective communication skills and explore biopsychosocial, and ethical issues in their assessment of their care.
- Use clinical cases to apply knowledge learnt, generate hypotheses, build an enquiry plan, and use evidence to refine their hypotheses, and justify their views.
- Design a management plan, and understand the pharmacological basis of drugs used in the management of common diseases affecting the nervous system.
- Master skills about professional development and professionalism in relation to the nervous system block.
- Develop basic clinical skills related to the nervous system.

1.0 Knowledge

1.1 Describe the gross anatomy and the ultra-structure of the spinal cord, brainstem, cerebral hemisphere, and the cerebellum, and correlate the anatomical structures and connections to their physiological functions.
1.2 Describe the anatomy and the ultra-structure of the eye, ear, tongue, and nose as well as the anatomy of cranial nerves, autonomic nervous system, and correlate anatomical structures to their functions.
1.3 Discuss the physiology of neurotransmitters, cerebral circulation, basal ganglia connections, descending and ascending tracts, and cerebrospinal fluid circulation and their functions.
1.4 Correlate anatomical structures of the nervous system to their radiological findings and discuss differences between different radiological modalities and their uses in patients with common neurological disorders.
1.5 Discuss the pathology and the pathogenesis of common diseases affecting the nervous system including cerebral infarction, brain/brainstem tumours, intracerebral haemorrhage, increased intracerebral pressure, and degenerative diseases. Explain possible risk factors for each of these disorders.
1.6 Discuss the pharmacology of drugs affecting the central nervous system particularly drugs used in treating Parkinsonism, epilepsy, depression, anxiety, schizophrenia, and management of conditions such as stroke.
1.7 Discuss the pharmacology of ototoxic drugs, sedatives, and drugs working on the eye.

1.8 Discuss the epidemiology, causes, clinical picture and management of drug abuse.
1.9 Discuss the microbiology, pathology, pathogenesis, clinical picture, diagnosis and brief management of infectious diseases affecting the nervous system including meningitis, encephalitis, cerebral malaria, cerebral tuberculosis, middle ear infection, conjunctivitis, and leishmaniasis.
1.10 Discuss the principles of medical professionalism and patient safety and demonstrate the ability to apply these principles to clinical case scenarios and challenging/emergency situations.
1.11 Discuss and apply the principles of self-directed learning.
1.12 Discuss the role of culture, behaviour, and social factors in disease development.
1.13 Discuss, briefly, health promotion and disease prevention.
1.14 Discuss the impact of disease on patient and family members.
1.15 Describe the gross anatomy and the ultra-structure of the spinal cord, brainstem, cerebral hemisphere, and the cerebellum, and correlate the anatomical structures and connections to their physiological functions.
1.16 Describe the anatomy and the ultra-structure of the eye, ear, tongue, and nose as well as the anatomy of cranial nerves, autonomic nervous system, and correlate anatomical structures to their functions.
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1.40 Discuss the role of culture, behaviour, and social factors in disease development.
1.41 Discuss, briefly, health promotion and disease prevention.
1.42 Discuss the impact of disease on patient and family members.

2.0 Cognitive Skills

2.1.1 Identify problems, generate hypotheses, make an enquiry plan, weigh evidence for and against a hypothesis, and make decisions on the basis of available evidence.
2.1.2 Apply knowledge learnt from dermatomes and myotomes distribution to clinical case examinations and making decisions regarding level of the neurological lesion.
2.1.3 Use available information to differentiate between normal and abnormal changes.
2.1.4 Identify their learning needs, search for new information and use new information to solve problems.
2.1.5 Integrate knowledge learnt from different disciplines such as anatomy, physiology, biochemistry, pathology, microbiology, and pharmacology to discuss a problem, make priorities, and define their action plan, and learning needs.
2.1.6 Work out how to handle uncertainty and decide on approaches to handle such situations.

3.0 Interpersonal Skills & Responsibility

1.1 Communicate effectively and demonstrate the ability to build rapport, work as a member of a small group and contribute to the learning of others.
1.2 Demonstrate the ability to apply well-being principles including how to handle stress.
1.3 Demonstrate accountability, professional behaviour, and the ability to contribute to active learning in their small groups.
1.4 Demonstrate the ability to monitor their progress, apply time management rules, and use feedback in improving their performance.

1.5 Take medical history from patients with common neurological conditions and demonstrate the ability to present their findings, and communicate with patients using simple language without technical jargon.

4.0 Communication, Information Technology, Numerical

4.1 Use computer programs in searching new information, sharing information, and analyzing data.

5.0 Psychomotor skills

5.1 Conduct clinical examination of the cranial nerves on a simulated patients and demonstrate the ability to show correct techniques, correct sequence of examination and the ability to interpret findings.
5.2 Conduct a clinical examination of the sensory and motor systems on a simulated patient and demonstrate the ability to show correct techniques, correct sequence of examination and the ability to interpret findings.
5.3 Conduct examination of the eyes and ears and demonstrate the ability to use ophthalmoscope in examining the fundus of the eye.

Teaching and Learning Modes:

In an integrated curriculum like our curriculum, we use a wide range of teaching and learning strategies to ensure that learning meets the different needs of the students. These strategies include:

- Small group discussion
- Lectures
- Student-led seminars
- Practical classes.
- Clinical skills
- Independent learning
- Writing an essay or mini thesis.

Objectives of the Lectures

Title of the lecture: Organization of nervous system	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neurpsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- List the divisions of the nervous system.
- Define the terms: receptors, effectors, grey matter, white matter, nucleus, ganglion, tract, nerve.
- Describe the development and derivatives of the neural tube.
- List the structures protecting the brain

Background:

The nervous system functions to detect changes in the internal and external environment and to bring about appropriate responses in muscles glands and organs. It is basically composed of specialized cells whose functions is to receive sensory stimuli and transmit them to the effector organs (muscles and glands)

Main concepts in the lecture:

- Central nervous system: brain, spinal cord
- Peripheral nervous system: receptors, sensory and motor neurons and nerves, ganglia
- Somatic and autonomic divisions
- Sympathetic and parasympathetic parts of autonomic nervous system
- Sensory (afferent) & motor (efferent) components
- Neurons & neuroglia
- Meninges, subarachnoid space and cerebrospinal fluid

Take home messages:

- Anatomical & functional division of the nervous system
- Structural organization of the nervous system
- Function of nervous system.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Anatomy of the spinal	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Describe the external features of the spinal cord regarding position, beginning, termination, segments and enlargements.
- Define the terms "cauda equina" and " filum terminal".
- Describe the sites of exit of spinal nerves from vertebral column.
- Describe in brief the blood supply of the spinal cord.
- List the important nuclei in the grey matter of the spinal cord and identify their location.
- List the important tracts in the white matter of the spinal cord and identify their location and types of fibres.

Background:

The spinal cord is an important structure. From a functional connectional perspective it is very interesting. It receives afferent fibres from sensory receptors of the trunk and limbs, it controls movements of the trunk and limbs, and provides autonomic innervation for most of the viscera. The spinal cord provides connection between the cerebrum, the cerebellum, and brainstem.

Main concepts in the lecture:

During this lecture, students understand about the spinal cord, position, segments and enlargements, and its blood supply, the grey matter and the white matter of the spinal cord, and important nuclei and tracts, type of fibres, and the exit of spinal nerves from vertebral column.

Take home messages:

- The spinal cord; external features and its blood supply.
- The important nuclei in the grey matter, and important tracts in the white matter of the spinal cord
- The sites of exit of spinal nerves from vertebral column.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Sensory tracts (ascending)	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Define the meaning of a nerve tract.
- Distinguish between the different types of tracts.
- Locate the position of each tract.
- Describe the sensory pathway.
- Identify the different sensory spinal tracts and their functions.
- Identify the course of each of these tracts.

Background:

All sensations arising from skin, connective tissues, voluntary muscles, periosteum, teeth, and so forth belong to the general somatic sensory system, more commonly referred to as the somatosensory system.
The general senses include light touch or tactile discrimination and sensation of pressure or deep touch, vibration, proprioception, pain and temperature.

Main concepts in the lecture:

During this lecture, students should understand that the somatosensory pathway consist of three neurones: the first order neuron is in the sensory ganglia, the second is in the spinal cord or brain stem or both and the third is in the thalamus.

Take home messages:

- Dorsal column tracts: (Gracile & Cuneate)
    - Function: Transmit
    - Proprioceptive (deep) sensations (sense of position, sense of movement, vibration sense).
    - Fine touch sensations (tactile localization, tactile discrimination, graphesthesia & stereognosis). *These senses reach a conscious level (cerebral cortex).*
- Spinothalmic tracts: Function: Transmit impulses concerned with specific sensory modalities: pain, temperature and touch, that reac a conscious level (cerebral cortex).
- Spinocerebellar tracts:
    - Function: Transmit impulses from tactile and stretch receptors (subcutaneous) to subconscious centers (cerebellum)-Muscle tone and coordination.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Embryological spinal cord and vertebral column	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Describe the layers of the neural tube forming the spinal cord (ependymal , mantle, and marginal).
- List the derivatives of basal and alar plates.
- Describe the development of the spinal ganglia, and derivatives of the neural crest.
- Describe the notochord and its significance.
- Describe the end stages of development of the vertebral column.
- List the main congenital anomalies of vertebral column (types of spina bifida with and without nervous tissue involvement).

Background:

The spinal cord is an important structure, its developmental perspective is very interesting. It starts development as a neural tube, that differentiates into three layers; ependymal , mantle, and marginal. The spinal cord is later differentiates into basal and alar plates; which give rise to the gray and white matters.
Differentiation of the neural crest, into important derivatives. The vertebral column, is mesodermal in origin; as part of the developing somites. The main congenital anomalies concerned the vertebral column is spina bifida and it connection with anomalies of the spinal cord.

Main concepts in the lecture:

During this lecture, students understand about the development of the neural tube and its derivatives; the spinal cord development and the neural crest derivatives. Development of the vertebral column from the developing somites. The main congenital anomalies of the vertebral column and it connection with anomalies of the spinal cord.

Take home messages:

- The neural tube development.
- The spinal cord development, and the important nuclei in the grey matter, and important tracts in the white matter.
- The development vertebral column, and the main congenital anomalies

Further reading:

Moore Persaud, the developing human , clinically oriented embryology 7th edition.

Title of the lecture: Brachial plexus and lumbosacral plexus	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Describe the formation of brachial plexus (site, roots & stages).
- List the main branches of brachial plexus.
- Describe the formation of lumbosacral plexus (site & root value).
- List the main branches of lumbosacral plexus.

Background:

The brachial plexus is the network of nerves, formed by the union of ventral rami of C5 through T1 spinal nerves. The lumbosacral plexus is formed by the ventral rami of L4,5, S1,2,3,4. It lies in the pelvis and gives branches supplying pelvis and lower limb.

Main concepts in the lecture:

During this lecture, students will understand the formation of brachial plexus, trunks, divisions, cords, nerves and their root values, various lesions of brachial plexus. Students will also understand the formation and distribution of the lumbosacral plexus.

Take home messages:

- *Brachial Plexus*: Ventral & dorsal roots, dorsal root ganglion, spinal nerve, ventral & dorsal rami, trunks, cords, divisions & nerves
- Erb's Palsy
- *Lumbosacral plexus:* root values, branches & distribution.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Normal cells of the nervous system	
Lecturer's name	Dr. Raeesa Abdel Tawab
Department	Anatomy
Block	Neuropsychiatry Block
Email address	drraeesama@gmail.com

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Describe the microscopic structures of neurons in correlation with their functions.
- Classify neurons according to their shapes and branches.
- Describe the microscopic structure of neuroglia in correlation with their functions.

Background:

The nervous tissue contains neurons as well as their supporting cells; neuroglia. Neurons are considered the structural and functional units of the nervous system. However, the count of their supporting elements; neuroglia, is six to ten times more than the count of neurons.

Main concepts in the lecture:

Neurons: structure, classification, function
Neuroglia: structure, classification, function

Take home messages:

- Neurons: structure, classification, function
- Neuroglia: structure, classification, function

Further reading:

- Color Textbook of Histology. L.P. Gartner & J.L. Hiatt, 3rd edition, Chapter 9: Nervous Tissue, W.B. Saunders, London, New York.

Title of the lecture: Anatomy of the brainstem	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- List the components & functions of brain stem.
- Describe the site of brain stem.
- Describe the relations between these components & their connections to cerebellum.
- Describe the external features of both ventral & dorsal surfaces of brain stem.
- List cranial nerves emerging from brain stem & the site of emergence of each nerve.

Background:

The brain stem is an important structure. It is the pathway for ascending and descending tracts between the spinal cord and the higher centers in the forebrain. It contains *Cranial Nerve Nuclei (111-x11),* It is the Site of emergence of cranial nerves (3- 12), It contains *G*roups of nuclei & related fibers known as reticular formation.

Main concepts in the lecture:

During this lecture, students understand about brain stem, its components, external features, the attached cranial nerves & the connections to the cerebellum.

Take home messages:

- Brain stem (medulla oblongata, pons & mid brain):
- Levels
- External features (for dorsal & ventral surfaces)
- Attached cranial nerves
- Contained cranial nuclei
- Connections to cerebellum

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd edition.

Title of the lecture: Anatomy of the cranial nerves IX and X	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Describe the component fibers of 9^{th} & 10^{th} cranial nerves.
- List the nuclei of the 9^{th} & 10^{th} nerves in the brain stem.
- Describe the course and relations of the 9^{th} & 10^{th} cranial nerves in the head & neck.
- List the branches of each of these 2 nerves.
- Describe how to test the integrity of these 2 nerves.
- Describe the effect of lesion of any of these 2 nerves.

Background:

The glossopharyngeal and vagus nerves arise from the medulla oblongata and carry special visceral motor, preganglionic parasympathetic and sensory fibers. All fibers enter and leave the medulla in a series of rootlets arranged in a longitudinal row posterior to the olive. They leave the cranial cavity through the jugular foramen

Main concepts in the lecture:

During this lecture, the students should understand the, deep origin, component fibers, distribution, function, effect of lesion of each of the glossopharyngeal and vagus nerves.
Also, the student will understand the reflexes and how to test the lesions of these two nerves.

Take home messages:

- Nuclei of the glossopharyngeal and vagus nerve.
- Course and branches of the glossopharyngeal and vagus nerves.
- Functions of these two nerves.
- Reflexes of these two nerves.
- 5. Testes for integrity of these two nerves.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7^{th} ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3^{rd} edition.

Title of the lecture: Internal structures of the brainstem	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Identify the structures present at each of the following level of brain stem
- The Closed Medulla
- The Mid Medulla
- The Open (rostral) Medulla
- The Pontomedullary junction
- The Mid-pontine
- The Caudal midbrain
- The Rostral midbrain

Background:

Inside the brain stem the anatomy can be studied only by looking at sections. Usually these are in the transverse plane and are stained by Weigert's technique, which colors myelinated fibers black. Areas occupied by grey matter (nuclei of the brain stem) are pale.

Main concepts in the lecture:

During this lecture, students identify the structures those appearing/disappearing/changing their shapes at different levels.
Closed medulla: Pyramidal decussation, Internal arcuate fibers
Open medulla: Inferior olivary nucleus
Pontomedullary junction: Abducens nucleus, Fibres of facial nerve
Mid-pontine level: Trigeminal nerve Pontine nuclei, Medial lemniscus
Caudal midbrain: Decussation of superior cerebellar peduncles
Rostral midbrain: Red nucleus, Oculomotor nerve

Take home messages:

- Medial leminiscus, Inferior olivary nucleus, Inferior cerebellar peduncle, Cochlear nuclei, Hypoglossal nuclei, Dorsal motor nucleus of the vagus, Vestibular nuclei, Nnucleus ambiguous, Medial longitudinal bundle, Spinal leminiscus, Solitary nucleus, & Reticular formation.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Anatomy of the cranial nerves XI and XII	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry
Email address	Jamila_elmedany@hotmail.com.

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- List the nuclei related to the 11th & 12th cranial nerves.
- Describe the type and location of each nucleus.
- Describe the emergence, intracranial course and foramina of exit for 11th & 12th cranial nerves.
- Describe the important relations of both nerves in the neck.
- Describe the branches and distribution of both nerves
- Describe the effects of lesion of accessory and hypoglossal nerves

Background:

There are12 pairs of cranial nerves that carry afferent and efferent fibres between the brain and the peripheral structures, principally of the head and neck. The cranial nerves are commonly damaged by trauma or disease, and testing for their integrity forms part of every neurological examination.

Main concepts in the lecture:

- Accessory nerve:
  - Type of nerve (functional components)
  - The cranial & spinal parts: their origin & course
  - Foramen of exit from skull.
  - Course and important relations in neck
  - Distribution
  - Effects of lesion
- Hypoglossal nerve:
  - Type of nerve (functional components)
  - Nucleus, origin & course
  - Foramen of exit from skull.
  - Course and important relations in neck, relation to C1 fibers
  - Distribution
  - Effects of lesion

Take home messages:

- The origin, course, relations and distribution of the 11th & 12th cranial nerves
- Functional components of both nerves
- Effect of lesion and how to test the integrity of each nerve

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Anatomy of the ear	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- List the parts of the ear: External, Middle (tympanic cavity) and Internal (labyrinth).
- Describe the parts of the external ear: the auricle and the external auditory meatus.
- Identify the walls of the middle ear : roof, floor and four walls (anterior, posterior, medial and lateral).
- Define the contents of the tympanic cavity:
- Ear ossicles,: (malleus, incus and stapes)
- Muscles, (tensor tympani and stapedius).
- Nerves (branches of facial and glossopharyngeal).
- List the parts of the inner ear, bony part filled with perilymph (Cochlea, vestibule and semicircular canals), in which is suspended the membranous part that filled with endolymph).
- List the organs of hearing and equilibrium.

Background:

The ear is an important structure.It is divided into the middle ear (tympanic cavity) which contains the ear ossicles, muscles and nerves and the inner ear (labyrinth) which contains the organs of hearing & equilibrium.

Main concepts in the lecture:

During this lecture, students understand about the parts of the ear (External, Middle (tympanic cavity) and Internal (labyrinth), the boundaries and contents of the tympanic cavity, the parts of the inner ear(*bony part filled* with perilymph & membranous part that filled with endolymph). The students also understand about organs of hearing and equilibrium.

Take home messages:

- External ear: auricle and external auditory meatus.
- Tympanic cavity: boundaries, connections & contents (ear ossicles, muscles & nerves).
- Bony Labyrinth: ((Cochlea, vestibule and semicircular canals).
- Membranous labyrinth.
- Organs of hearing & equilibrium.

Further reading:

- Snell clinical Anatomy by Systems, latest edition.
- Gray's Anatomy for Students, latest edition.

Title of the lecture: Cranial nerve VIII	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	alsharawi@gmail.com

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- List the nuclei related to vestibular and cochlear nerves in the brain stem.
- Describe the type and site of each nucleus.
- Describe the vestibular pathways and its main connections.
- Describe the auditory pathway.

Background:

The vestibulocochlear nerve is the 8th cranial nerve. Its nuclei are situated in the pontomedullary region. It conducts hearing and equilibrium.

Main concepts in the lecture:

Vestibular nerve: origin, course.
Cochlear nerve: origin, course.

Take home messages:

- Vestibular pathway for equilibrium and important connections.
- Cochlear pathway for hearing.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Nerve supply of the face (cranial nerves V and VII)	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawwi@ksu.edu.sa

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- List the nuclei related to trigeminal and facial nerves in the brain stem.
- Describe the type and site of each nucleus.
- Describe the site of emergence and course of trigeminal and facial nerves.
- Describe the sensory distribution of trigeminal nerve in the face.
- Describe the motor distribution of facial nerve in the face.
- Describe the main motor & sensory effects in case of lesion of trigeminal and facial nerves.

Background:

The face is supplied by both trigeminal and facial nerves. The trigeminal (5^{th} cranial) nerve carries general sensations from the face. The facial (7^{th} cranial) nerve supplies muscles of facial expression.

Main concepts in the lecture:

Trigeminal nerve: origin, course and supply.
Facial nerve: origin, course and supply.

Take home messages:

- Areas of supply of trigeminal nerve in face.
- Muscles of facial expression supplied by facial nerve.
- Effect of trigeminal nerve lesion.
- Effect of facial nerve lesion.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7^{th} ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3^{rd} ed.

Title of the lecture: Anatomy of the eye globe and cranial nerve II	
Lecturer's name	Dr. Raeesa Abdel Tawab
Department	Anatomy
Block	Neuropsychiatry Block
Email address	drraeesama@gmail.com

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Identify the microscopic structure of the outer coat of the eye
- Identify the microscopic structure of the middle coat of the eye
- Identify the microscopic structure of the inner coat of the eye
- Describe the microscopic structure of the cornea in correlation with function.
- Identify the microscopic structure of the retina in correlation with function.

Background:

The eye globe is formed of eye wall and eye contents
The eye wall consists of 3 coats
The eye contents include aqueous humor, lens, and vitreous humor
The optic nerve is the second cranial nerve. It carries the visual sensation.

Main concepts in the lecture:

Coats of the eye globe.
Layers of the retina.
Visual pathway.
Lesions of the different parts of the visual pathway.

Take home messages:

- Coats of the eye globe.
- Layers of the retina.
- Visual pathway.

Further reading:

- Color Textbook of Histology. L.P. Gartner & J.L. Hiatt, 3rd edition, Chapter 22: Special Senses; Eye, W.B. Saunders, London, New York.

Title of the lecture: Anatomy of the nose and olfactory nerve	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Describe the structures forming the walls of the nasal cavity.
- List the main structures draining into the lateral wall of the nasal cavity.
- Differentiate between the respiratory and olfactory region of the nasal cavity.
- List the main sensory and blood supply of the nose.
- Describe the olfactory pathway.

Background:

The nose is an important structure. The nasal cavity has roof, floor, lateral and medial walls. The lateral wall is marked by 3 projections or conchae. The space below each concha is called meatus. The paranasal sinuses are cavities inside the skull bones . They are Lined with mucoperiosteum; filled with air; and it communicates with the nasal cavity.Their Function to decrease he skull weight and amplify the sound as we speak. Olfactory mucous is present in the roof, superior nasal concha, and upper part of nasal cavity. It is delicate and contains olfactory nerve cells for smell sensation.

Main concepts in the lecture:

During this lecture, the students understand about the nasal cavity, boundaries, openings of the paranasal sinuses, respiratory and olfactory mucosa and their functions and the pathway of smell sensation through the olfactory nerve.

Take home messages:

Nasal cavity: lateral wall, medial wall (nasal septum).
Paranasal sinuses: their openings and functions.
Pathway of olfactory nerve.

Further reading:

Clinical Neuroanatomy by Richard S. Snell, 7th edition..
Neuroanatomy by A.R. Crossman & D. Neary 3rd edition.

Title of the lecture: Cranial nerves III, IV and VI	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- List the nuclei related to occulomotor trochlear, and abducent nerves in the brain stem.
- Describe the type and site of each nucleus.
- Describe the site of emergence and course of occulomotor trochlear, and abducent nerves.
- Describe the important relations of optic, occulomotor trochlear, and abducent nerves in the orbit.
- List the orbital muscles supplied by each of occulomotor trochlear, and abducent nerves.
- Describe the main motor effect in case of lesion of each of occulomotor trochlear, and abducent nerves.
- Describe the visual pathway and main lesions associated with it.

Background:

The cranial nerves concerned with vision(Cr II) and movement of the eye (Cr III, IV, and VI) are important from the anatomical point of view concerning the deep nuclei in the brain stem, the site of emergence and course of each nerve, and there relation in the orbit. The distribution of each nerve (Cr III, IV, and VI) for the ocular muscles, and the main motor effect in case of lesion of each. The visual pathway and main lesions associated with it.

Main concepts in the lecture:

During this lecture, students understand about the optic nerve and the visual pathway. The occulomotor, trochlear, and abducent nerves, the muscles concerned with eye movements.

Take home messages:

- The optic nerve, the visual pathway, and main lesions associated with it.
- The important nuclei related to (Cr III, IV, and VI) in the brain stem.
- The important relations of optic, occulomotor, trochlear, and abducent nerves in the orbit
- The orbital muscles supplied by each of (Cr III, IV, and VI) nerves, and the main motor effect in case of lesion of each.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Anatomy of the cerebellum and the relevant connections	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Describe the external features of the cerebellum (lobes, fissures).
- Describe briefly the internal structure of the cerebellum.
- List the cerebellar nuclei.
- Relate the anatomical to the functional subdivisions of the cerebellum.
- Describe the important connections of each subdivision.
- Describe briefly the main effects in case of lesion of the cerebellum.

Background:

The cerebellum is a part of hindbrain. It is connected to brain stem nuclei, thalamus and motor cortex. It has a role in equilibrium and in coordination of voluntary movements.

Main concepts in the lecture:

Subdivisions of cerebellum.
Important connections of each part of the cerebellum.

Take home messages:

- Anatomical and functional subdivisions of cerebellum.
- Correlation between both subdivisions in term of connections.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Anatomy of the cerebral hemispheres	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Describe the cerebral hemispheres: shape, surfaces and subdivision into lobes
- Identify the important sulci and gyri of each lobe
- Describe the internal structure of each hemisphere: cortex (grey matter), medulla (white matter), basal ganglia, lateral ventricle
- Describe the functional areas of the cerebral cortex.
- Describe different types of fibers in the hemisphere and their functions

Background:

The cerebrum is the largest and most highly developed part of the human brain. It is involved in several functions of the body including determining intelligence, thinking, perceiving, planning and organization, producing and understanding language, interpretation of sensory impulses & motor function.

Main concepts in the lecture:

Location of cerebrum and its relation to other parts of the brain
Functional importance of cerebrum
Lobes of cerebral hemisphere, and important gyri and sulci in each lobe
Internal structure of each hemisphere
Important functional areas of cerebral cortex
Different types of fibers in the medulla of each hemisphere and functions of each type.
Effect of lesions of cortex and white matter

Take home messages:

- Two hemispheres connected to each other by a band of fibers called the corpus callosum.
- Surfaces (superolateral, medial inferior) and lobes (frontal, parietal, temporal, occipital). Each lobe performs specific functions
- Internal structure of each hemisphere and the lateral ventricle.
- Important sulci and gyri, Brodmann's mapping and functional cortical areas.
- Association, commissural and projection fibers

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Embryology of the cerebral hemispheres and cerebellum	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Describe the formation of the neural tube.
- Describe the three and five vesicle stages of the neural tube development.
- List the derivatives of each of the brain vesicles.
- List the brain flexures (midbrain, cervical and pontine)
- Describe the development of the cerebrum and cerebellum.
- List the most common anomalies of brain development.

Background:

Formation of the neural tube is completed by about the middle of the fourth week of embryonic development. Its upper end dilates & shows 3 vesicle: Prosencephalon, Mesencephalon & Rhombencephalon.

Main concepts in the lecture:

During this lecture, the students should understand that:
By end of the 2nd week of development, three germ cell layers become established, ectoderm, mesoderm and endoderm. Each germ layer will give rise to particular tissues and organs in the adult. Formation of the neural plate, fold, groove and tube from the ectoderm. Formation of the brain vesicles and its derivatives.

Take home messages:

- Neural tube formation, brain vesicles and flexures.
- Transformation of the neural tube into the adult CNS.
- Derivatives of each brain vesicles.

Further reading:

- The Developing Human By Moore & Persaud (latest edition).

Title of the lecture: Cerebral blood circulation	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:

At the end of the lecture, the students should be able to:

- Describe the course and branches of vertebra-basilar artery
- Describe the course and branches of internal carotid artery
- Describe the arterial supply of cerebrum
- List the components of Circle of Willis (circulus arteriosus)
- List the main veins draining the cerebrum

Background:

The entire blood supply of the brain and spinal cord depends on two sets of branches. The vertebral arteries arise from the subclavian arteries, and the internal carotid arteries are branches of the common carotid arteries. Conjoining the two major sources of cerebral vascular supply via the circle of Willis presumably improves the chances of any region of the brain continuing to receive blood if one of the major arteries becomes occluded The physiological demands served by the blood supply of the brain are particularly significant because neurons are more sensitive to oxygen deprivation than other kinds of cells with lower rates of metabolism Sustained loss of blood supply leads much more directly to death and degeneration of the deprived cells.

Main concepts in the lecture:

Origin, course, relations and branches of vertebral artery
Origin, course, relations and branches of basilar artery
Origin, course, relations and branches of internal carotid artery
Formation of Circle of Willis, its branches and distribution
Arteries supplying cerebrum, with a focus on arterial supply of cortical functional areas
Deep and superficial cerebral veins, and dural venous sinuses

Take home messages:

- Main sources of the arterial supply of cerebrum
- Arterial supply of cortical functional areas
- Significance of Circle of Willis
- Veins draining the cerebrum & dural venous sinuses

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed

Title of the lecture: Anatomy of the basal ganglia and connections	
Lecturer's name	Dr. Jamilah El-Medany
Department	Anatomy
Block	Neuropsychiatry Block
Email address	Jamila_elmedany@hotmail.com

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Describe the subdivisions of basal ganglia and state the function of each of them.
- Understand the important relations of corpus striatum (caudate & lentiform nuclei).
- Describe the important connections of corpus striatum.
- Describe briefly the main effects in cases of lesion of basal ganglia.

Background:

The term "basal ganglia'' refers to interconnected nuclear masses of grey matter in the forebrain, deeply situated in the cerebral hemispheres, diencephalon, and midbrain. Their function are "extrapyramidal motor system" , they control posture and regulate voluntary movements. Abnormalities of basal ganglia: result in movement disorders such as Parkinsonism and Huntington diseases.

Main concepts in the lecture:

During the lecture, the students understand about basal ganglia, divisions, connections, normal functions and abnormalities.

Take home messages:

- The basal ganglia: Corpus striatum (in cerebral hemisphere), Subthalamic nucleus (in diencephalon), Substantia nigra (in midbrain).
- Normal functions and diseases.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Anatomy of the limbic system and thalamus	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa.

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Describe the important relations of the thalamus.
- Describe the subdivisions of the thalamus.
- List the function and important connections of each of the thalamic nuclei.
- Describe briefly the main effects in case of lesion of thalamus.
- List the main nuclei and tracts of limbic system.
- List the important connections between parts of limbic system.

Background:

The thalamus is formed of 2 oval masses of grey matter, below hypo- & subthalamus
It is divided into 3 main groups of nuclei: *anterior, medial & lateral.*
The anterior & medial groups are parts of limbic system.
The specific nuclei of the lateral group are related to sensory & motor areas of cerebral cortex.
The limbic system is formed of parts of CNS interconnected with fibers. It is concerned with *memory, emotions & behavior*.

Main concepts in the lecture:

During this lecture, students understand about the important relations of the thalamus, the subdivisions of the thalamus, the subdivisions of the thalamus, the function and important connections of each of the thalamic nuclei and the main effects in case of lesion of thalamus.
The students also understand about the main nuclei and tracts of limbic system and the important connections between parts of limbic system.

Take home messages:

- The thalamus: important relations, subdivisions, functions and connections of the main thalamic nuclei.
- Limbic system: the main nuclei and tracts and their functions, the important connections between parts of limbic system.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture: Anatomy of the meninges, CNS cavities and CSF circulation	
Lecturer's name	Dr. Sanaa Al Shaarawi
Department	Anatomy
Block	Neuropsychiatry Block
Email address	salsharawi@ksu.edu.sa

Objectives of the lecture:
At the end of the lecture, the students should be able to:

- Describe the cerebral meninges & list the main dural folds.
- Describe the spinal meninges & locate the level of the termination of each of them.
- Describe the importance of the subarachnoid space.
- List the cavities of the CNS and locate the site of each of them.
- Describe the formation, circulation, drainage, and functions of the CSF.

Background:

The meninges are very important structures. It covers the cerebrum and the spinal cord to protect them. The subarachnoid space contains CSF, which is produced by the choroid plexus within the cerebral ventricles. The flow of CSF can be obstructed within the subarachnoid space by adhesions following head injury or meningitis. The obstruction of the flow of CSF leads to a rise in fluid pressure causing swelling of the ventricles (hydrocephalus).

Main concepts in the lecture:

During the lecture, students understand about the layers of meninges, function, cavities in the brain, circulation of CSF, its function and abnormalities.

Take home messages:

- Meninges: Dura mater, Arachnoid mater and Pia mater.
- Function of meninges.
- CSF formation, circulation and Ventricular cavities in the brain.
- CSF obstructions.

Further reading:

- Clinical Neuroanatomy by Richard S. Snell, 7th ed.
- Neuroanatomy by A.R. Crossman & D. Neary 3rd ed.

Title of the lecture:	Brachial plexus & Lumbosacral plexus (Practical sessions)
Lecturer's name	Dr. Jamilah El Medany
Department	Anatomy
Block / week	Neuropsychiatry block
Email address	Jamila_elmedany@hotmail.com

Objectives:

At the end of the practical section, the students should be able to:

- Demonstrate the roots of brachial plexus.
- Differentiate between the branches of brachial plexus.
- Follow the courses of median, ulnar & radial nerves.
- Identify the main branches of median, ulnar & radial nerves.
- Demonstrate the roots of lumbar & sacral plexuses.
- Follow the courses of femoral & sciatic nerves in the lower limb.
- Identify the main branches of femoral nerve (muscular, saphenous).
- Identify & follow the course of the two terminal branches of sciatic nerve (tibial, common peroneal).

Materials:

- Fixed upper and lower limbs specimens.
- Plastinated upper and lower limbs specimens.

Title of the lecture:	Brain Stems (Practical sessions)
Lecturer's name	Dr. Jamilah El Medany
Department	Anatomy
Block / week	Neuropsychiatry block
Email address	Jamila_elmedany@hotmail.com

Objectives:

At the end of the practical section, the students should be able to:

- Demonstrate the site of brain stem in the cranial cavity.
- Demonstrate in a brain stem specimen or model the main external features on both surfaces:
    1. **Medulla:** pyramid, olive, gracile & cuneate tubercles.
    2. **Pons:** groove for basilar artery, transverse pontine fibers.
    3. **Midbrain:** crus cerebri, superior & inferior colliculus.
- Demonstrate in a sagittal section the different components of brain stem (medulla, pons, midbrain), cerebellum, cerebellar peduncles, 4th ventricle & cerebral acqueduct.
- Demonstrate in a brain stem specimen the site of emergence of each cranial nerve.
- Differentiate between the different levels of medulla, pons & midbrain as regard the characteristic features present in each level.
- Demonstrate the course of lower 4 cranial nerves in the neck.
- Identify the motor distribution of facial nerve in the face.
- Identify the optic, occulomotor, trochlear and abducent nerves in cranial cavity and orbit.
- Identify the frontal, lacrimal and nasociliary branches of trigeminal nerve in the orbit.
- Identify the lingual and inferior alveolar branches of trigeminal in the infratemporal fossa.
- Trace the course of maxillary nerve through the foramina of the skull.

Materials:

- Fixed brain specimens (whole brain, brain sections).
- Brain models.

Title of the lecture: Cerebral hemisphere (Practical sessions)	
Lecturer's name	Dr. Jamilah El Medany
Department	Anatomy
Block / week	Neuropsychiatry block
Email address	Jamila_elmedany@hotmail.com

Objectives:

At the end of the practical section, the students should be able to:

- Differentiate between the different lobes of the cerebral hemisphere.
- Identify the main sulci: central, precentral, postcentral, lateral, superior & inferior frontal, superior & inferior temporal, cingulate, calcarine, callosal, collateral, olfactory.
- Identify the main gyri: superior, middle & inferior frontal, precentral & postcentral, superior & inferior parietal, superior, middle & inferior temporal, paracentral, cingulate, medial frontal, cuneus, precuneus, lingual, parahippocampal, uncus, gyrus rectus.
- Identify in brain sections (sagittal, coronal, horizontal) the following: corpus callosum, internal capsule, caudate nucleus, lentiform nucleus.
- Identify and/or trace the course of the vertebro-basilar, the anterior, middle and posterior cerebral arteries.
- Identify the cerebellum in brain specimens, brain models and brain sections.
- Identify the following: vermis of cerebellum, cerebellar peduncles, primary fissure, posterolateral fissure, horizontal fissure, flocculonodular lobe, anterior and posterior (middle) lobe.

Materials:

- Fixed brain specimens (whole brain, brain sections).
- Brain models.
- Plastinated brain sections.

Title of the lecture:	Skull (Practical sessions)
Lecturer's name	Dr. Jamilah El Medany
Department	Anatomy
Block / week	Neuropsychiatry block
Email address	Jamila_elmedany@hotmail.com

Objectives:

At the end of the practical section, the students should be able to:

- Identify the different "Normas or views" of the skull.
- Identify the different bones of the skull.
- Identify the important foramina of the skull and determine the structures passing through them.
- Determine the place of the important structures in relation to the skull.

Materials:

- Skulls

Title of the lecture:	Spinal Cord (Practical sessions)
Lecturer's name	Dr. Jamilah El Medany
Department	Anatomy
Block	Neuropsychiatry block
Email address	Jamila_elmedany@hotmail.com

Objectives:

At the end of the practical section, the students should be able to:

- Differentiate between the different levels of the segments of the spinal cord (cervical, thoracic, lumbar).
- Identify the different parts of grey (dorsal, ventral and lateral horns, central canal) and white matter (dorsal, ventral and lateral white columns) of the spinal cord.
- Identify the location of the main tracts in the white matter of the spinal cord (gracile and cuneate, spinothalamic and corticospinal tracts).
- Identify the different shapes of neurons:
    1. Rounded (unipolar): e.g. dorsal root ganglion of the peripheral nerve.
    2. Spindle-shaped (bipolar): e.g. olfactory epithelium of the nose.
    3. Multipolar:
        a) Stellate: e.g. anterior horn cells of the spinal cord.
        b) Pyramidal: e.g. pyramidal cells of the cerebral cortex.
        c) Pyriform: e.g. purkinje cells of the cerebellum.

Materials:

- Histological slides.

Title of the lecture: Ageing and changes in the brain	
Lecturer's name	Dr. Aida Korish
Department	Physiology
Block	Neuropsychiatry Block
Email address	iaidakorish@yahoo.com

Objectives of the lecture:
At the end of this lecture the students should:

- Define Aging
- Enumerate theories of aging
- Describe body and brain changes in aging
- Describe memory changes in aging
- Explain carotid hypersensitivity

Background:

Aging is the progressive, universal decline first in functional reserve and then in function that occurs in organisms over time. Brain changes with are very important for clinicians to appreciate the differences between diseases states and physiological changes that occur with age.

Key Principles to be discussed:

Changes in appearance (gradual reduction in height and weight loss due to loss of muscle & bone mass)

- A lower metabolic rate
- Longer reaction times
- Declines in certain memory functions
- Declines in sexual activity and in women menopause
- A functional decline in audition, olfaction, and vision
- Dementia and delirium
- Carotid hypersensitivity

Take home messages:

- Aging is not a disease; however, the risk of developing disease is increased, often dramatically, as a function of age.

Key Words:

- Aging, Dementia, delirium, Carotid hypersensitivity, memory

Further Reading:

- Guyton & Hall Textbook of Medical Physiology 11th Ed.
-

Title of the lecture: Autoregulation of cerebral blood flow	
Lecturer's name	Prof. Faten Zakaria
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Describe cerebral circulation & circle of Willis
- Explain main arteries that supply blood to brain
- Normal Rate of Cerebral Blood Flow
- Explain auto-regulation of cerebral blood flow
- Explain the factors effecting the cerebral blood flow
- Effects of impaired cerebral blood circulation

Background:

Cerebral blood flow (CBF) is the blood supply to the brain in a given time. The normal blood flow through the brain of an adult subject is about 50-65 ml/ 100 grams of brain tissue per minute. For the entire brain, this amount is 750-900 ml/min, or 15 per cent of the resting cardiac output. CBF is regulated to meet the brain's metabolic demands. CBF is highly related to metabolism of the tissue. Three metabolic factors have potent effects in controlling the CBF, these includes carbon dioxide concentration, hydrogen ion concentration and oxygen concentration. Ischemia results if blood flow to the brain is below 18 to 20 ml per 100 g per minute and tissue death occurs if flow dips below 8 to 10 ml per 100 g per minute.

Key principles to be discussed:

- During this lecture, students understand about the Circle of Willis, normal rate of cerebral blood flow, auto-regulation and factors effecting the regulation of cerebral blood flow.

Take home messages:

- Cerebral blood flow
- Circle of Willis
- Auto-regulation cerebral blood flow
- Factors regulating the cerebral blood flow

Key words:

- Cerebral blood flow, Circle of Willis, Auto-regulation, Factors regulating.

Further reading:

- Guyton and Hall Textbook of Medical Physiology, 12th Edition; Ganong's review of medical physiology, 23rd edition.

Title of the lecture: Functions of cerebral hemisphere	
Lecturer's name	Dr. Fawzia Al Rouq
Department	Physiology
Block	Neuropsychiatry Block
Email address	falrouq@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- Cerebral hemisphere is divided into four lobes by central sulcus and lateral sulcus. E,g frontal lobe, parietal lobe, temporal lobe and occipital lobe, students are required to know the terms categorical hemisphere and representational hemisphere and should be able to summarize the difference between these hemispheres.

Background:

They should are requested to know the function of each lobe. The frontal lobe lies in front of central sulcus and is mostly motor in functions, parietal lobe in most sensory, the temporal lobe is for auditory perception, language, memory small, the occipital cortex is required for visual processing.

Key principle to be discussed:

During their lecture the students should understand about primary motor cortex, premotor area and supplementary motor cortex, they are required to know about parental lobe and its somatossensory functions.
Take home message; area of body representation in motor and sensory cortex, higher intellectual functions of the pre frontal lobe. Functions of Broca's area and comprehensive interpretative functions of the posterior superior temporal lobe to be understood.

Key words:

- motor area, somatosensory area I & II, Broca's area, wernicke's area, calcarine fissure.

Further reading:

- Gayton and Hall textbook of Medical Physiology 12th edition.
- Gamong's review of Medical Physiology, 23rd edition.

Title of the lecture: Pain modulation	
Lecturer's name	Dr. Hayam Gad
Department	Physiology
Block	Neuropsychiatry Block
Email address	Hayam_gad@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Intensity of the pain can be altered by various extrinsic and intrinsic mechanisms, extrinsic mechanism such as rubbing or shaking of an injured area. Or applying ice pack, or stimulation with an electric vibrator at the site of pain all gives some relief from pain, pain can be modulated by giving analgesic drugs e.g.morphine.

Background:

Pain modulation by medicines is a big developing field to control and reduce pain perception.

Key topics to be discussed:

Students should know gate-control hypothesis and role of body's own morphines, the opaid peptides. To know about opioid receptors and are formed in the mid brain, brainstem and spinal cord.

Take home Messages:

- Students are required to know about descending pain ratifying pathway from brain. This inhibits in coming pain signals at the spinal cord level. It is also required to the brain's opiate system e.g. endorphin and emkepalines.

Key words:

- Descending analgesic system, gate-control hypothesis, morphine & enkephalins.

Further reading:

- Gamong's review of Medical Physiology, 23rd edition.

Title of the lecture: Pathophysiology and epilepsy	
Lecturer's name	Prof. Laila Al Ayadhi
Department	Physiology
Block	Neuropsychiatry Block
Email address	Iyayadhi@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- Define Epilepsy
- Etio-pathology of Epilepsy
- Types of Epilepsy
- Role of Genetic in Epilepsy
- Clinical Features
- Role of Electro Physiological tests in the diagnosis of Epilepsy

Take home messages:

- Epilepsy, types, pathophysiology, involvement of genetic in Epilepsy, clinical features and role of Electro physiological tests in the diagnosis of Epilepsy

Key Words:

- Epilepsy, types, pathophysiology

Further Reading:

- Guyton & Hall Textbook Of Medical Physiology 11th Ed
- Text of Medicine by Parveen and Kumar

Title of the lecture: Photo transduction in light and dark	
Lecturer's name	Prof. Faten Zakareia
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Differentiate between rodes & cones concerning distribution and convergence on ganglion cells
- Contrast the phototransduction process for rods and cones in light and dark and the ionic basis of these responses
- List and compare functional properties of scotopic and photopic vision
- To know the visual cycle and rhodopsine regeneration

Background:

The potential changes that initiates action potentials in the retina generated by the action of light on photosensitive compounds in rods and cones. When light is absorbed by these substances, their structure changes, and this trigger a sequence of events that initiates neural activity.

Take home Message:

- Na+ channel in out segment of the rods and cones are open in the dark, so current flow from inner to outer segment. When light strikes the outer segment some of Na+ channels are closed and the cell is hyperpolarized.
- The retinal pigment epithelium provides crucially important support roles of photoreceptors including removal of cellular debris, and recycling of molecular substrates in the visual transduction cascades.
- The absorption of light by photopigment in rods and cones, triggers a phototransduction cascade that determines the rate at which glutamate is released from photoreceptor terminals.

Title of the lecture: Physiology of consciousness	
Lecturer's name	Prof. Laila Ayadhi
Department	Physiology
Block	Neuropsychiatry Block
Email address	Iyayadhi@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- Define consciousness and explain the different states of consciousness .
- Explain what is meant by the " Reticular Activating System "(RAS)
- Define the location and function of the Bulboreticular Facilitatory Area .
- Describe how the interaction between the Bulboreticular Facilitatory Area , Thalamus and Cerebral Cortex subserves & sustains consciousness
- Explain how a medical person can differentiate between a conscious and unconscious person by means of outward behavior as and physical signs .
- Describe the role of EEG and evoked potentials in differentiating between a conscious person , a sleeping person , a comatose patient and brain dead patient

Further reading:

- Guyton and Hall Textbook of Medical Physiology, 12th Edition
- Ganong's review of medical physiology, 23rd edition

Title of the lecture: Physiology of the hearing	
Lecturer's name	Professor Laila Al Ayadi
Department	Physiology
Block	Neuropsychiatry Block
Email address	Iyayadhi@ksu.edu.sa

Objectives of the lecture:

At the end of this lecture the students should:

- Appreciate the functions of outer, middle and inner ear
- Describe nature of sound & its characteristics
- Function of semicircular canals& utricle& saccule.
- To understand the role of middle ear in sound transmission, magnification and tympanic reflex effect
- Recognize the function of hair cells of inner ear
- Differentiate between conductive and perceptive deafness

Title of the lecture: Physiology of sleep	
Lecturer's name	Dr. Nervana Bayoumi
Department	Physiology
Block	Neuropsychiatry Block
Email address	nbayoumy@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- Explain the difference between sleep and coma.
- Define what is meant by NREM (non-rapid eye movement, SWS) and REM (rapid eye movement) sleep.
- Describe how NREM and REM sleep are distributed during a normal night sleep in the average adult human
- Describe the behavioral and autonomic features associated with NREM and REM sleep.
- Describe how the EEG, as a physiological tool, is being used to delineate in which stage of sleep (or wakefulness) a person is.
- Appreciate how the total sleep duration and different sleep stages vary with different ages in normal humans.
- Describe the current theories about the neural basis of sleep.

Title of the lecture: Physiology of basal ganglia & regulatory mechanisms	
Lecturer's name	Dr. Fawzia Al Rouq
Department	Physiology
Block	Neuropsychiatry Block
Email address	falrouq@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- Enumerate different nuclei of basal ganglia
- Know different neurotransmitters that have a role in basal ganglia functions
- Appreciate general functions of basal ganglia
- Physiological basis of basal ganglia disorders

Background :

Basal ganglia are important parts of extrapyramidal system. They are important in motor control of movements and cognition. An important disease related to these nuclei is Parkinson's disease.

Key Principles to be discussed:

- Motor loop (putamen circuit) concerned with learned movment.
- Cognitive loop (Caudate circuit) concerned with cognitive control of sequences of motor pattern. Basically it is concerned with motor intentions.
    - (Note: cognition means thinking process using sensory input with information
    - already stored in memory.)
- Limbic loop: involved in giving motor expression to emotions like, smiling, aggressive or submissive posture.
- Occulomotor loop concerned with voluntary eye movement [saccadic movement]

Keywords:

- Putamen circuit, Caudate circuit, Limbic loop, Occulomotor loop, Motor loop, Cognitive loop, Parkinson's disease

Title of the lecture: Physiology of brain transmitters	
Lecturer's name	Professor Laila Ayadhi
Department	Physiology
Block	Neuropsychiatry Block
Email address	Iyayadhi@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the studenst should:

- Be able to describe the main locations in the brain of the following transmitters , as well as their physiological functions ; and give examples of clinical conditions associated with their imbalance :
    - Acetylcholine (Ach) .
    - Norepinephrine (NE)
    - Glutamate .
    - GABA .
    - Serotonin .
    - Dopamine (DA)

Title of the lecture: Physiology of color vision	
Lecturer's name	Prof. Faten Zakareia
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmauil.com

Objectives of the lecture:
At the end of this lecture the students should:

- Identify and describe the mechanism of color vision and the three types of cones, including the range of spectral sensitivity and color blindness
- Describe the electrical responses produced by bipolar cells and ganglion cells and comment on the function of each
- Describe the topographic representation of the visual field within the primary and association visual cortex and describe the processing of information in the primary visual cortex

Background:

Human have three different kinds of cones that serve color vision and responding maximally to light at different wave length. Red, green and blue are called primary colors. Equal stimulation of red, green and blue cones give a sensation of seeing white.

Take home Message:

- Perception of the color of an object or light source result from a comparative assessment of the hue, saturation, and brightness of the direct or reflected light.

Title of the lecture: Physiology of motor tracts	
Lecturer's name	Prof. Faten Zakaria
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Appreciate what is upper motor neuron and lower motor neuron .
- The main differences between the pyramidal and extrapyramidal systems .
- explain the origin , course and functions of the following motor tracts ;
  - corticospinal.
  - tectospinal .
  - rubrospinal .
  - vestibulospinal .
  - reticulospinaql .
  - olivospinal.

Further reading:

- Gayton and Hall textbook of Medical Physiology 13th edition.

Title of the lecture: Physiology of the pain	
Lecturer's name	Dr. Hayam Gad
Department	Physiology
Block	Neuropsychiatry Block
Email address	hayam_gad@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- To know about the receptor of pain.
- The types of neuron responsible for conduction of impulses e.g A-delta and C- types.
- Two types of pain e.g fast and slow.
- Know the tracts involved and its functions.
- Know the role of thalamus and cortex in the perception of pain.

Background:

Pain is a protective mechanism. It is important to know pain fully in order to understand many diseases.

Key principles:

During the lecture students are required to understand about the pain e.g receptor, pathway, referred pain, neurotransmitters the required.

Take home Message:

To know about the tracts e.g lateral spinothalamic and spinoreticular tract and their functions.

Key words:

- Lateral spinothalamic tract, spino tract; culartract, phantom pain, analgesia, hyperalgesia and allodynia.

Further reading:

- Gayton and Hall textbook of Medical Physiology 12th edition.

Title of the lecture: Physiology of postural reflexes	
Lecturer's name	Prof. Faten Zakaria
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Postural reflexes are needed to keep the body in a proper position while standing, moving. When body posture is suddenly altered it is corrected by sevier reflexes. These reflexes are operating at spinal cord, medulla, mid-brain and cortical levels. To make the reflex movements smooth cerebellum, basal ganglia and vestibular apparatus are needed. Students are required to know posture-regulating parts of CNS.

Background:

For all reflexes to operate reflex are is needed and components of reflex are one receptor, afferent, spinal cord, efferent and muscle, all reflexes work when the normal tone in the muscle is present, there are supraspinal reflexes which regulates the muscle movements.

- Able to define human posture
- Explain/define the concepts of " center of gravity '' and " support base ".
- Explain what are postural reflexes and their overall function .
- Know the centers of integration of postural reflexes .
- Explain the structure and function of the vestibular apparatus (utricle, saccule & semicircular canals) in maintenance of balance
- Describe decorticate rigidity and decerbrate rigidity and explain the mechanisms underlying them .

Further reading;

- Gayton and Hall textbook of Medical Physiology 12th edition.
- Gamong's review of Medical Physiology, 23rd edition.

Title of the lecture: Physiology of speech	
Lecturer's name	Prof. Laila Al Ayadhi
Department	Physiology
Block	Neuropsychiatry Block
Email address	Iyayadhi@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- Describe brain speech areas as Broca's, Wernicke's and insula
- Explain sequence of events in speech production
- Explain speech disorders as aphasia with its types, dysarthria, and acalculia
- Explain difference between aphasia and dysarthria.

Background:

Speech is an important aspect of communication. It consist of three aspects which include sensory aspect of communication, central integration and motor aspect of communication. Speech defects are related to many diseases of nervous system.

Key Principles to be discussed:

We will discuss speech from three aspects;

- Sensory Aspects of Communication.
- Integration
- Motor Aspects of Communication.

A defect in any one of these lead to defective speech. We will also discuss difference between defective articulation and aphasia. Important speech disorders will be explained with regard to respective areas of brain like Broca's aphasia, Wernicke's aphasia, Conduction aphasia, Anomic aphasia, Global aphasia and Dyslexia.

Keywords:

- Wernicke's area, Broca's area, insula, aphasia, dysarthria, Broca's aphasia, Wernicke's aphasia, Conduction aphasia, Anomic aphasia, Global aphasia and Dyslexia.

Title of the lecture: Physiology of the brainstems	
Lecturer's name	Dr. Hayam Gad
Department	Physiology
Block	Neuropsychiatry Block
Email address	hayam_gad@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Know what is brainstem
- What are its internal structures
- What are its functions
- What will happen if damaged e.g brain death.

Background:

The brainstems is the region of the brain that connects the cerebrum with spinal cord. The pons, motor and sensory nervous travel through the brainstem allowing for the relay of signals between the brain and spinal cord.

Key principles to be discussed:

They should know the fact that the brainstem coordinates motor control signals sent from the brain to the body. The brainstem also controls life supporting autonomic functions of the peripheral nervous system. It is essential to note that the cranial nerves 3 – 12 emerge from the brainstem. The main role of brainstem has integrative functions.

Take home Message:

- It is essential for consciousness, alertness, awareness, arousal, breathing, blood pressure regulation, heart rate control, pain sensitivity control.

Key words:

- Reticular formation, respiratory center cardio vascular center, brain death.

Further reading:

- Gamong's review of Medical Physiology, 23rd edition.

Title of the lecture: Physiology of synapses and receptors	
Lecturer's name	Professor Laila Ayadhi
Department	Physiology
Block	Neuropsychiatry Block
Email address	Iyayadhi@ksu.edu.sa

Objectives of the lecturer:
After the lecture, students should be able to:

- Define a synapse and describe the structure and function of chemical and electrical synapses.
- Define what neurotransmitters are, and how they are released and act on their receptors, and how they are removed.
- Differentiate between ionotropic receptors and metabotropic receptors
- Differentiate between postsynaptic and presynaptic inhibition, and between excitatory and inhibitory postsynaptic potentials (EPSPs and IPSPs).
- Describe properties of synapses and explain the nature of temporal and spatial summation.
- Appreciate that effectiveness of neurotransmitters can be modified by drugs and diseases.

Background:

The CNS contains more than 100 billion neurons. Information is transmitted from one neuron to another in the form of nerve action potentials (APs) (also called nerve impulses) through synapses. The synapse is the junction point from one neuron to the next. Some synapses transmit signals from one neuron to the next with ease, whereas others transmit signals only with difficulty. Also, facilitatory and inhibitory signals from other areas in the nervous system can control synaptic transmission, sometimes opening the synapses for transmission and at other times closing them. In addition, some postsynaptic neurons respond with large numbers of output impulses, and others respond with only a few. In other words, each nerve impulse impulse/AP may be: (1) blocked in its transmission from one neuron to the next, (2) changed from a single AP into repetitive APs, or (3) integrated with APs from other neurons to cause highly intricate patterns of impulses in successive neurons. All these functions can be classified as synaptic functions of neurons.

Key words:

Chemical synapses; Electrical synapses; Neurotransmitters; Presynaptic inhibition; Postsynaptic inhibition; Temporal summation; Spatial summation; Synaptic vesicles; Excitatory neurotransmitters; Inhibitory neurotransmitters; Ionotropic receptors; Metabotropic receptors.

Main concepts/principles in the lecture:

During the lecture, the term ``*synapse*`` will be defined and the types of synapses (chemical and electrical) will be discussed with examples of where they occur. In addition, the following swill also be discussed in some detail: neurotransmitters; presynaptic inhibition; postsynaptic inhibition;

temporal summation; spatial summation; synaptic vesicles; excitatory neurotransmitters; inhibitory neurotransmitters; ionotropic receptors and metabotropic receptors. The followings are the main concepts that will be discussed in the lecture:

- Synapse: is a small gap, separating two neurons, that enables one neuron to pass an electrical or chemical signal to another neuron. There are 2 types: (1) chemical synapse: is a junction where the axon of a neuron terminates on the dendrites, the soma or the axon of another neuron. The cellular communication at chemical synapses is via secretion of neurotransmitters (NTs) and (2) electrical synapse: at this type of synapses the cellular communication is via current flowing through gap junctions which are communicating junctions or channels between two adjacent cells. As the name implies there is a gap between adjacent cells which are linked by small connecting tunnels formed by a protein known as connexon which is arranged in a tube like structure that extends through the thickness of the plasma membrane. The gap junction allows passage of electrical activity.
- At chemical synapses, the presynaptic neuron releases a chemical (neurotransmitter, NT) that enables the electrical signal (AP) to be transmitted to the postsynaptic neuron after binding to a specific protein receptor on the membrane of postsynaptic neuron. Thus chemical synapses enable the signal to be transmitted in one direction only (One-direction transmission). In contrast, signal transmission at electrical synapses (which are very rare in the brain and do not involve NT release from synaptic vesicles) is bidirectional.
- Although most synapses in the brain are chemical, electrical and chemical synapses may coexist and interact in the CNS. The bidirectional transmission of electrical synapses permits them to help coordinate the activities of large groups of interconnected neurons. For example, electrical synapses are useful in detecting the coincidence of simultaneous sub-threshold depolarizations within a group of interconnected neurons; this enables increased neuronal sensitivity and promotes synchronous firing of a group of interconnected neurons.
- At chemical synapses in the CNS, postsynaptic neurons can receive up to 20,000 synaptic input which is converted to a nerve impulse (AP) at axon hillock. The output signal travels by way of a single axon leaving the neuron to the axon terminal.
- The key feature of all chemical synapses is the presence of small, membrane-bounded organelles called synaptic vesicles within the presynaptic terminal. These spherical organelles are filled with one or more NTs, the chemical signals secreted from the presynaptic neuron, and it is these chemical agents acting as messengers between the communicating neurons that gives this type of synapse its name.
- The released NT binds and acts on specific receptor proteins on the post-synaptic membrane.
- There are 2 types of receptor proteins (NT receptors) on membrane of postsynaptic neurons. These are
    - Ionotropic receptors: these receptors contain two functional domains: an extracellular site that binds NTs, and a membrane-spanning domain that forms an ion channel. They directly gate ion channels and are also known as ligand-gated ion channels; they mediate rapid postsynaptic potentials (PSPs).
    - Metabotropic receptors: these are separated physically from the ion channel. They are monomeric proteins with an extracellular domain that contains a NT binding site and an

intracellular domain that binds to G-proteins. They activate channels indirectly through activation of intermediate molecules called G-proteins. They act through second messenger systems and mediate slow postsynaptic potentials (PSPs).

- A given NT may activate both ionotropic and metabotropic receptors to produce both fast and slow PSPs at the same synapse.
- More than 40 important NTs have been discovered thus far. Some of the best known are acetylcholine (Ach), norepinephrine, epinephrine, histamine, gamma-aminobutyric acid (GABA), glycine, serotonin, and glutamate. Glutamate is the most widely NT in the CNS.
- The secretion of NTs is triggered by the influx of Ca^{2+} through voltage-gated Ca^{2+}channels, which gives rise to a transient increase in Ca^{2+} concentration within the presynaptic terminal. The rise in Ca^{2+} concentration causes synaptic vesicles to fuse with the presynaptic plasma membrane and release their contents into the space (known as synaptic cleft) between the pre- and postsynaptic cells by a process known as exocytosis.
- Following exocytosis, NTs diffuse across the synaptic cleft and bind to specific receptors on the membrane of the postsynaptic neuron. The binding of NT to the receptors causes channels in the postsynaptic membrane to open (or sometimes to close), thus changing the ability of ions to flow into (or out of) the postsynaptic cells. The resulting neurotransmitter-induced current flow alters the conductance and (usually) the membrane potential of the postsynaptic neuron, increasing or decreasing the probability that the neuron will fire an action potential. In this way, information is transmitted from one neuron to another.
- The postsynaptic cell sums (or integrates) all of the EPSPs and IPSPs. Excitation of a single presynaptic terminal almost never excites the neuron because the amount of NT that is released by a single terminal to cause an EPSP is usually no greater than 0.5 to 1 millivolt, instead of the 10 to 20 millivolts normally required to reach threshold for excitation. However, many presynaptic terminals are usually stimulated at the same time and their effects can still summate;that is, they can add to one another until neuronal excitation does occur. This effect of summing simultaneous postsynaptic potentials by activating multiple terminals on widely spaced areas of the neuronal membrane is called spatial summation. Successive discharges from a single presynaptic terminal, if they occur rapidly enough, can add to one another; that is, they can "summate." This type of summation is called temporal summation.
- Inhibitory and excitatory postsynaptic potentials can be summated simultaneously. If a neuron is being excited by an EPSP, an inhibitory signal from another source can often reduce the postsynaptic potential to less than threshold value for excitation, thus turning off the activity of the neuron.
- Presynaptic Inhibition: in addition to inhibition caused by inhibitory synapses operating at the postsynaptic neuronal membrane, which is called postsynaptic inhibition, another type of inhibition often occurs at the presynaptic terminals before the signal ever reaches the synapse. This type of inhibition is called presynaptic inhibition which is caused by release of an inhibitory NT. In most instances, the inhibitory transmitter substance is GABA. This release has a specific effect of opening anion channels, allowing large numbers of chloride ions to diffuse into the axon terminal. The negative charges of these ions inhibit synaptic transmission because they cancel much of the excitatory effect of the positively charged

sodium ions that also enter the terminal when an action potential arrives. Presynaptic inhibition occurs in many of the sensory pathways in the nervous system.

- After performing their desired function, NTs are inactivated or removed by one of the following methods: a) Inactivation by enzymes, b) active pumping into synaptic knobs and c) diffusion away from the synaptic cleft.
- The effectiveness of synaptic transmission can be modified by drugs and diseases. There are many toxins, both animal and plant toxins that have potent actions on the nervous system, often interfering with synaptic transmission. Possible drug actions include:
  - Altering synthesis, storage or release of NTs.
  - Modifying NT interaction with post synaptic receptor. For example Strychnine competes with glycine; it combines with the glycine receptor & blocks it (no IPSPs).
  - Influencing NT reuptake or destruction. For example, Cocaine blocks the reuptake of Dopamine by binding competitively with dopamine reuptake transporters. This causes prolonged activation of pleasure pathway (euphoria). Prozac, an example of Selective Serotonin Reuptake Inhibitor (SSRIs) (depression), which is characterized by deficiency of serotonin (which is involved in neural pathways regulating mood & behavior).
  - Replacing a deficient NT with substitute NT. For example Levodopa (L-dopa), a precursor of dopamine (which crosses the blood-brain barrier, unlike dopamine) is used to replace the deficiency of dopamine in Parkinson's disease. Once inside the brain, it is converted to dopamine and relieves the symptoms of the disease.
- There are some diseases such as Myasthenia Gravis (autoimmune disease in which antibodies are directed against the nicotinic acetylcholine (Ach) receptors on skeletal muscle fibers) that affect synaptic transmission. The hallmark of the disorder is muscle weakness, particularly during sustained activity. This condition can be improved by treatment with inhibitors of acetylcholinesterase, the enzyme that normally degrades Ach at the neuromuscular junction.

Take home Message:

- The CNS contains more than 100 billion neurons that, each minute, receive literally millions of bits of information in the form of nerve impulses/action potentials. Transmission and integration of this information occurs mainly at and through chemical synapses. Understanding the physiology of these and electrical synapses is essential for appreciating how the nervous system processes incoming information to produce appropriate responses. It is important to note that many drugs exert their actions by modulating synaptic transmission, and that many CNS diseases (e.g. Parkinson`s disease) are due to synaptic dysfunction.

Further reading:

- Gayton and Hall textbook of Medical Physiology 13th edition. Chapter 46

Title of the lecture: Physiology of the cerebellum	
Lecturer's name	Dr. Aida Korish
Department	Physiology
Block	Neuropsychiatry Block
Email address	iaidakorish@yahoo.com

Objectives of the lecture:
At the end of this lecture the students should:

- Describe the divisions of the cerebellum
- Describe the functional divisions of the cerebellum (vestibulocerebellum, spinocerebellum and cerebrocerebellum).
- Understand cell types / nuclei of the cerebellum
- Understand the functions of cerebellum in regulation of movement, tone and balance.
- Understand the abnormalities associated with cerebellar disease: Cerebellar nystagmus , changes in muscle tone , ataxia , drunken gait , scanning speech , dysmetria (past-pointing), intention tremor , rebound phenomenon and diadochokinesia

Background:

The cerebellum provides major input to the corticospinal, rubrospinal, and other brainstem systems. Reticular and vestibular spinal systems also get an input from the cerebellum.
During this lecture, students understand about cerebellum, functional divisions, normal functions and abnormalities.

Take home messages:

- Cerebellum: Paleocerebellum, Neo cerebellum Archicerebellum and functions.
- Gross, fine movements and body balance.
- Normal functions and abnormalities of cerebellum

Key words:

- Cerebellum: Paleocerebellum, Neocerebellum Archicerebellum, Functions and
- Abnormalities

Further reading

- Guyton and Hall Textbook of Medical Physiology, 12th Edition
- Ganong's review of medical physiology, 23rd edition

Title of the lecture: Physiology of the eye and refraction	
Lecturer's name	Prof. Faten Zakareia
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Describe different components of the eye and function of each and understand the eye protection media
- Describe the refraction of light as it passes through the eye to the retina, identifying the refractive media of the eye
- Describe the refractive error that account for myopia, hypermetropia, presbyopia and astigmatism and their correction by eye glasses or contact lenses
- Know layers of retina, blind spot, and fovea centralis-explain the differing light sensitivities of the fovea, peripheral retina and optic disk

Background:

- Light is refracted at the anterior surface of the cornea and at the anterior and posterior surfaces of the lens, with total refractive power of 59 diopters when the lens is accommodated for distant vision.

Take home message:

- The bending of the light rays (refraction) allows one to focus an accurate image on to the retina.
- In hyperopia (far sightedness), the eye ball is too short and light rays come to a focus behind retina.
- In myopia (near sightedness), the eye ball is too long and light rays come to a focus in front of retina.
- Astigmatism is a common condition in which the curvature of the cornea is not uniform.
- Presbyopia is a loss of accommodation for near vision due loss of the elasticity of the lens.

Title of the lecture: Physiology of the proprioceptors in balance	
Lecturer's name	Dr. Fawzia Al Rouq
Department	Physiology
Block	Neuropsychiatry Block
Email address	falrouq@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- To know about proprioceptors its definition and its role in body balance.
- The muscle spindles and their role in stretch reflex.
- The Golgi tendon organs and analyze their function as part of a feedback system that maintain muscle tone.
- Reciprocal innervations, inverse stretch reflex, clonus and lengthening reaction.

Key principles to be discussed:

Proprioceptors are essential to regulate the physical state of the body. This includes position, tendon and muscle sensation, pressure sensations from the bottom of the feet , proprioceptors plays a essential role in keeping the body in different anatomical position,

Take home Message:

- Students should know about muscle spindle and its role in keeping the body position normal.

Key words:

- Stretch reflex, muscle spindles, Golgi tendon organs, clonus

Further reading:

- Gamong's review of Medical Physiology, 23rd edition.

Title of the lecture: Physiology of the sympathetic and parasympathetic nervous system	
Lecturer's name	Dr Fawzia Al Rouq
Department	Physiology
Block	Neuropsychiatry Block
Email address	falrouq@ksu.edu.sa

Objectives of the lecture:
Upon completion of this lecture, students should be able to understand:

- The anatomy of somatic and autonomic nervous system
- Sympathetic and parasympathetic nerves
- Pre and post ganglionic neurons
- Functions of sympathetic and parasympathetic nerves in head & neck, chest, abdomen and pelvis
- Neurotransmitters release at pre and post ganglionic sympathetic / parasympathetic nerves endings
- Various responses due to stimulation of the sympathetic / parasympathetic nervous system

Background:

The sympathetic and parasympathetic nervous system helps in the control of body's internal functions. Stress-as in the flight-or-fight response- is thought to counteract the parasympathetic system, which generally works to promote maintenance of the body at rest.

Key principles to be discussed:

During this lecture, students understand about anatomy and physiology of the sympathetic / parasympathetic nervous system, pre and post ganglionic neurons and functions of the sympathetic / parasympathetic nervous system.

Take home messages:

- Sympathetic / parasympathetic nervous system,
- Thoracolumbar
- Alarm reaction: Flight or fight response
- Cranio-sacral out flow
- Conservation of body energy

Key words:

ANS, Sympathetic nervous system, Thoracolumbar, Fight or flight response
Parasympathetic nervous system, Cranio sacral, energy restoration.

Further reading:

- Guyton and Hall Textbook of Medical Physiology, 12th Edition
- Ganong's review of medical physiology, 23rd edition

Title of the lecture: Physiology of the taste and smell	
Lecturer's name	Prof. Laila Al Ayadi
Department	Physiology
Block	Neuropsychiatry Block
Email address	Iyayadhi@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the students should:

- Appreciate the physiology of olfaction
- Describe the olfactory pathway
- Appreciate some pathophysiological conditions related to olfaction as anosmia, parosmia hypo and hyperosmia

Title of the Practical: Color vision, light and accommodation reflex	
Lecturer's name	Dr. Ola Mawlana
Department	Physiology
Block	Neuropsychiatry Block
Email address	olamawlana@gmail.com

Objectives of the Practical:

At the end of the session, the students should be able to:

- Perform the test for visual acuity using a Snellen's chart, list the common refractive errors and describe how they can be corrected.
- Perform the test for near vision using a Jaeger's chart.
- Perform the test for Astigmatism using Astigmatism chart and describe how it can be corrected.
- Determine one's near point.
- Demonstrate one's blind spot.
- Explain the mechanisms of accommodation with the help of Purkinje-Sanson images.
- Identify one's color-vision defects using the Ishihara's colored charts.

Background:

Students should be familiar with the anatomy of the human eye ball, definition of visual acuity and the role of eye ball structures like cornea and lens to achieve the best visual acuity. Students must also have basic concepts about far vision and near vision, mechanism of accommodation and role of photoreceptors in the detection of color vision.

Main concepts in the lecture:

- The students will learn how to test visual acuity for far vision using Snellen's chart.
- The students will learn how to test visual acuity for near vision using Jaegar's chart.
- The students will learn about types of refractive errors and how to diagnose and correct them.
- The students will learn how to determine one's near point using pin-head of a common pin.
- The students will learn about Astigmatism and how to diagnose and correct it.
- The students will learn how to locate and appreciate their own blind spot.
- The students will learn how to demonstrate the mechanism of accommodation using Purkinje-Sanson images.
- The students will learn how to identify the various color-vision defects with the help of Ishihara's chart.

Conclusion:

The students will learn how to test visual acuity for far vision as well as for near vision, demonstrate the mechanism of accommodation, determine one's near point, blind spot and color-vision defects and detect astigmatism.

Take home messages:

- What is visual acuity, far vision and near vision and how to test and correct any visual defects.
- What is astigmatism and how to detect and correct it.
- How to demonstrate the mechanism of accommodation and describe the concept of near point.
- How to determine color-vision defects and describe their various types.

Further reading:

- Zain's Manual of Experimental Physiology Vol. 2, Experiment 12 and 13

Title of the Practical: Electromyography (EMG) & motor nerve conduction velocity (MNCV)	
Lecturer's name	Dr Ola Mawlana
Department	Physiology
Block	Neuropsychiatry Block
Email address	olamawlana@gmail.com

Objectives of the Practical:
At the end of the session, the students should be able to:

- Acquire a skill to perform the EMG test
- Analyze the motor unit potentials and relate them to health and disease.
- Determine and calculate motor conduction velocities of peripheral nerves.

Background:

Students should be familiar with the anatomy and physiology of human motor system including upper and lower motor neurons, neuromuscular junction and the muscles innervated by those nerurons. Students must also be acquainted with some basic knowledge about action potentials, neuropathies, myopathies and neuro-muscular junction disorders and the clinical significance of calculating the motor nerve conduction velocity.

Main concepts in the lecture:

- The students will learn how to perform the procedure of EMG.
- The students will learn about the motor unit potentials (MUPs) and how they are produced.
- The students will learn to analyze the MUPs during rest, minimal stimulus and maximal stimulus.
- The students will learn the normal EMG pattern in a healthy individual.
- The students will learn how the EMG pattern changes in myopathic and neuropathic disorders.
- The students will learn how to demonstrate denervation hypersensitivity in neuropathies.
- The students will learn how to determine the motor nerve conduction velocity.
- The students will learn how the motor nerve conduction velocity helps to confirm the diagnosis.

Conclusion:

The students will learn how to perform EMG and how to interpret various EMG patterns to establish the correct diagnosis and then how to determine the motor nerve conduction velocity to further confirm the diagnosis.

Take home messages:

- What are the clinical uses of EMG and how to perform it?
- How the EMG pattern looks like in healthy individuals and in neuropathic or in myopathic individuals.
- How to calculate the motor nerve conduction velocity.
- How the motor nerve conduction velocity is affected in various neurogenic disorders.

Further reading:

- Zain's Manual of Experimental Physiology Vol. 2, Experiment 11.

Title of the Practical: Audiometry	
Lecturer's name:	Dr. Ola Mawlana
Department	Physiology
Block	Neuropsychiatry Block
Email address	olamawlana@gmail.com

Objectives of the Practical:
At the end of the session, the students should be able to:

- Determine the type, degree, and configuration of hearing loss.
- Describe the techniques of Tuning Fork tests.
- Plot the frequency-intensity recording in a procedure called audiometry and construct the audiograms.
- Interpret the audiograms.

Background:

Students should be familiar with the anatomy of the human ear, physiologic mechanism of hearing and some basic terminologies like air conduction, bone conduction, masking sound, pure tone, deafness etc. The students must also know the characteristics of sound like pitch and loudness and must have some basic concepts about the types of hearing disabilities and their causes.

Main concepts in the lecture:

- The students will learn how to perform tuning fork tests in a clinical setup.
- The students will learn the purpose of the tuning fork tests and their clinical importance.
- The students will learn how to perform the whole procedure of audiometry in a lab setup.
- The students will learn the requirements to perform an audiometry successfully.
- The students will learn how to plot an audiogram.
- The students will learn how to interpret an audiogram to reach to the correct diagnosis.
- The students will learn briefly about various conditions leading to deafness.

Conclusion:

The students will learn the purpose of the tuning fork tests and their clinical importance and how to perform the whole procedure of audiometry.

Take home messages:

- Purpose of the tuning fork tests and how to perform and relate them to the hearing loss.
- Clinical significance and the lab setup of audiometry
- How to plot and interpret an audiogram to suggest the diagnosis.

Further reading:

- Zain’s Manual of Experimental Physiology Vol. 2, Experiment 8 and 9.

Title of the lecture: Spasticity and increased muscle tone	
Lecturer's name	Prof. Faten Zakaria
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Appreciate that spasticity is an important conditions that is encountered in a broad spectrum of medical specialties such as neuropediatrics, adult neurology, orthopedics, rehabilitation medicine and others.
- Be able to define the term spasticity and understand that it occurs in medical conditions frequently encountered in the Kingdom such as stroke, multiple sclerosis, cerebral palsy, traumatic spinal cord and brain injury, cerebral and spinal tumors, spinal cord disc lesions; and in less common but important & preventable conditions such as tetanus and spinal cord infections such as tuberculosis of the spine.
- Explain the neurophysiological basis of clinical features associated with multiple sclerosis, cerebral palsy, traumatic spinal cord injury tuberculosis of the spine and tetanus.

Title of the lecture: Spinal cord functions & reflexes	
Lecturer's name	Prof. Faten Zakaria
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Describe the general structure and function of the spinal cord
- Distinguish between the functional role of gray matter and white matter
- Classify reflexes into superficial and deep, and describe the components of a monosynaptic and a polysynaptic reflex arc.
- Compare and contrast the features of a stretch reflex, a Golgi tendon reflex, a withdrawal reflex, and a cross extensor reflex.
- Appreciate the clinical importance of reflexes (their use as a diagnostic tool for assessment of nervous system function).

Background:

The spinal cord is often considered as being only a station for relaying sensory information (nerve signals) from the periphery of the body to the brain, or motor information in the opposite direction from the brain back to the body. This notion is far from the truth because without the special neuronal circuits of the spinal cord simple motor tasks cannot be carried out. For example even the most complex motor control systems in the brain could not cause movements of the legs that are required in walking. In addition to its role in voluntary movement, the spinal cord plays a pivotal role in many reflexes (which are used every day by humans and animals for survival) including: (1) reflexes that withdraw portions of the body from painful objects, (2) reflexes that stiffen the legs to support the body against gravity, and (3) autonomic reflexes that control local blood vessels, gastrointestinal movements, or urinary excretion.

Key words:

gray matter, white matter, dorsal horn, ventral (anterior) horn, monosynaptic reflex, polysynaptic reflex, withdrawal reflex, and inverse stretch reflexes

Main concepts/principles in the lecture:

A good understanding of the anatomy of the spinal cord is essential for understanding the role of the spinal cord in reflexes and integrating sensory and motor information and the topics of neural pathways. Thus an overview of the gross anatomy as well as a cross section of the spinal cord will be provided before addressing other concepts as follows:

- Cross section of the spinal cord

- The spinal cord consists of peripheral white matter and central gray matter.
- White matter is organized into columns, which are subdivided into nerve tracts, or fasciculi, which carry action potentials to and from the brain.
- Gray matter is divided into horns: (1) the dorsal horns contain sensory axons that synapse with interneurons as well as projection neurons that send sensory information to the brain, (2) the ventral horns contain the neuron cell bodies of somatic motor neurons (α-and γ-type), and (3) the lateral horns contain the neuron cell bodies of autonomic neurons.
- The gray and white commissures connect each half of the spinal cord.
- The dorsal root conveys sensory input into the spinal cord, and the ventral root conveys motor output away from the spinal cord.
- Each segment of the spinal cord (at the level of each spinal nerve) has several million neurons in its gray matter. These are: (a) the dorsal horn sensory neurons that send information to higher levels of the CNS, (b) the anterior (ventral) motor neurons, and (c) interneurons.

- Spinal nerves
  - The are 31 pairs of spinal nerves (8 cervical, 12 thoracic, 5 lumbar, 5 sacral pairs, and 1 coccygeal pair)
  - Spinal nerve are mixed nerves containing sensory nerve fibers that carry sensory information from the periphery to the spinal cord; they enter the cord through the dorsal roots, as well as motor nerve fibers that carry motor commands to the target tissue such as muscles.
  - Each spinal nerve has a specific cutaneous (skin) distribution called dermatome.
- Reflexes
  - There are two types of reflexes: (1) simple (built-in or basic), and (2) acquired (which come from practice or learning)
  - The reflex arc is the basic functional unit of the nervous system. The components of the reflex arc are: (1) Sensory receptors which respond to stimuli and produce action potentials in sensory neurons, (2) Sensory (afferent) neurons that propagate action potentials to the spinal cord, (3) Integrative center (CNS) which include interneurons between the terminal of sensory neurons and motor neurons, (4) Efferent (motor) neuron which carry action potentials from the CNS to effector organs and (5) Effector (target tissue) such as muscles or glands that respond to the action potentials arriving from motor neurons.
  - Reflexes do not require conscious thought, and they produce a consistent and predictable result. They are homeostatic and are integrated within the brain and spinal cord. Higher brain centers can suppress or exaggerate reflexes.
  - There are many types of reflexes including: (1) Stretch Reflexes in which the sensory receptor is the muscle spindle, they are also known as myotatic reflexes (this type will be covered in another lecture), (2) Golgi Tendon Reflexes in which the sensory receptor is Golgi tendon organ; they are also called inverse stretch reflexes, and (3) withdrawal reflexes which are initiated by nociceptors in the skin, muscles or the viscera; they are also called flexor reflexes.

Take home Messages:

The spinal cord is not just a relay station that relays sensory information (in the form of nerve signals) from the periphery of the body to the brain, and motor information in the opposite direction from the brain back to the body, but it plays an essential role in integrating sensory and motor information and in reflexes which are used by humans and animals to survive every day. Also autonomic reflexes keep organ systems operating smoothly. Furthermore, any voluntary movement cannot occur without an intact spinal cord.

Further reading:

- Gayton and Hall textbook of Medical Physiology 13th edition. **Chapter 55**

Title of the lecture: Stretch reflex and tendon jerks	
Lecturer's name	Prof. Faten Zakaria
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the student should:

- Describe the structure, innervation and function of the muscle spindle
- Describe the components of monosynaptic muscle stretch reflexes, including the role of alpha (α) and gamma (γ) motorneurons
- Distinguish between a static and dynamic stretch reflex.
- Describe the spinal and supra-spinal regulation of the stretch reflex.
- Describe the structure and function of the Golgi tendon organ and the inverse stretch reflex
- Appreciate the clinical importance of the stretch reflexes

Background:

As described previously (see the outline of the lecture on Spinal cord functions and Reflexes), reflexes are used every day by humans and animals to survive. There are many of such reflexes, and some of them were discussed in a previous lecture in which it was pointed out that the reflex arc (loop that mediates reflex actions) is the basic functional unit of the nervous system. In this lecture the focus is on two well studies reflexes: (1) the Stretch Reflex in which the sensory receptor is the muscle spindle, and 2) Inverse Stretch Reflex in which the sensory receptor is the Golgi tendon organ.

Key words:
α-motorneuron, γ- motorneuron, *musle spindle, Golgi tendon organ, group Ia afferent fibers, group Ib afferent fibers, monosynaptic reflex, inverse stretch reflex*

Main concepts/principles in the lecture:

A good understanding of the structure of the muscle spindle and Golgi tendon organ is essential for understanding their roles in reflexes. Thus an overview of the structure and function of these important parts of the skeletal muscles will be provided as well as a detailed discussion of other concepts as follows:

- Muscle spindles
  - Are sensory receptors (spindle shape) within the belly of a muscle. They detect changes in the length of the muscle, i.e. they convey length information to the CNS via primary sensory neurons.
  - This information is important for determining the position of body part.

- Muscle spindles are innervated by 2 types of afferent fibers:
  - primary (annulospiral) endings of type Ia afferent fibers which terminate on the non-contractile central portion of intrafusal fiber, and
  - secondary (flower-spray) endings of type II afferent fibers which terminate on the contractile end

- Muscle spindles are stimulated by stretching of their mid-portion. They can be excited in two ways: a) lengthening the whole muscle which stretches the mid-portion of the spindle and, therefore excites the receptor, b) contraction of the end portions of the spindle's intra-fusal fibers which result in stretching the mid-portions of the spindle and exciting the receptor during γ-efferent discharge.
- Group *Ia* and group II afferent fibers of a given muscle terminate directly on α-motor neurons supplying the extra-fusal fibers of the same muscle.
- Because the muscle spindle is in parallel with the extrafusal fibers of the muscle, when the muscle is passively stretched, the spindles are also stretched or the spindle is loaded. When the muscle spindle is stretched, its sensory endings are distorted and generator potentials are generated. These in turn cause generation of action potentials at a frequency proportional to the degree of stretching. This initiates reflex contraction of the extrafusal fibres in the same muscle. On the other hand, the spindle afferents stop firing when the muscle is made to contract (the spindle is unloaded).
- An important concept is the co-activation of α- and γ- motorneurons by descending pathways. The γ- motorneurons are much smaller than α-motorneuros but they are about one half as many. Activation of γ- motorneurons causes contraction of the end portions of the spindle's intra-fusal fibers which result in stretching the mid-portions of the spindle and exciting the receptor. This prevents the slackening of the spindle spindle during shortening of the whole muscle caused by α-motorneurons stimulation of the extrafusal fibers.
- Some of the afferent fibers ascend to the cerebellum (spino-cerebellum tracts) and to the cerebral cortex for conscious perception of sensation of position of limbs at joints (proprioception).
- Stretch reflex
  - Is triggered when a whole muscle is passively stretched. This triggers the contraction of the same muscle being stretched, and the response resists passive changes in muscle length. Thus, the reflex functions to oppose sudden changes in muscle length.
  - The classic example of the stretch reflex is the patellar-tendon or knee jerk reflex. During this reflex, both contraction of the muscle being stretched (quadriceps), and inhibition of the antagonistic (hamstring) muscle (through reciprocal innervation) occur simultaneously.
  - The primary function of the stretch reflex is to react to loads that tend to stretch the leg extensor muscles
  - Static stretch reflex: When the receptor portion of the muscle spindle is stretched slowly, the number of impulses transmitted from both the primary and the secondary endings increases almost directly in proportion to the degree of stretching and the endings continue to transmit these impulses for several minutes. This effect is called the static response of the spindle receptor.

    - Dynamic stretch reflex: When the length of the spindle receptor increases suddenly, the primary ending (but not the secondary ending) is stimulated powerfully. This stimulus of the primary ending is called the dynamic response, which means that the primary ending responds extremely actively to a rapid rate of change in spindle length.
- The Golgi tendon organ
    - Are netlike collections of nerve endings in a muscle tendon. There are 3-25 fibers per tendon
    - The Golgi tendon organ consists of sensory endings of fast conducting (myelinated) afferent fibers known as *Ib* fibers. These are entwined within bundles of connective tissue fibers that make up the tendon
- Inverse Stretch Reflex (Disynaptic Reflexes)
    - Up to a point, the harder a muscle is stretched, the stronger is the reflex contraction.
    - However, when the tension becomes great enough, contraction suddenly stops and muscle relaxes.
    - This relaxation in response to strong stretch is called the inverse stretch reflex or autogenic inhibition.
    - The receptor for inverse stretch reflex is in the Golgi tendon organ which consists

Take home Message:

The muscle stretch reflex is a monosynaptic reflex (where a type *Ia* proprioceptor nerve fiber synapses directly on anterior horn motor neurons that send motor nerve fibers back to the same muscle from which the muscle spindle fiber originated) is the simplest manifestation of muscle spindle function. An especially important function of the stretch reflex is its ability to prevent oscillation or jerkiness of body movements (a damping, or smoothing, function). The dynamic stretch reflex functions to oppose sudden changes in muscle length, whereas the static stretch reflex causes the degree of muscle contraction to remain reasonably constant.

Further reading:

- Gayton and Hall textbook of Medical Physiology 13th edition. Chapter 55

Title of the lecture: Upper and lower neuron lesions	
Lecturer's name	Prof. Faten Zakaria
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- What is upper motor neuron and lower motor neuron.
- What are its function.
- What effects are produced if damaged.

Background:

Motor word means movement and it requires upper and lower motor neuron to function. The example of upper motor neurons is corticospinal tract or pyramidal tract while all the peripheral motor nerves are lower motor neurons.

Key part to be discussed:

Lesion (damage) of upper motor neurons results in large loss of motor function (paralysis) increase in muscle tone, hyper reflexia, increased deep reflexes, and presence of babinski's sign.
The lower motor neuron lesion results in limented loss of power, wasting (thioning of muscle) loss of muscle tone, decreased or absent deep reflexes.

Take home Message:

- The students should be able to differentiate the effects of upper and lower motors neuron lesion. They should also know the measuring of hemiplegia, monoplegia, paraplegia.

Key words:

- Paralysis, wasting muscle Babinski's sign,

Further reading:

- Gayton and Hall textbook of Medical Physiology 12th edition.
- Gamong's review of Medical Physiology, 23rd edition.

Title of the lecture: Vision, accommodation of the light pathways & effects of lesions	
Lecturer's name	Prof. Faten Zakareia
Department	Physiology
Block	Neuropsychiatry Block
Email address	fatenz3699@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Describe visual acuity
- Contrast photopic and scotopic vision
- To know visual pathway and field of vision
- Describe the process of accommodation reflex and its pathway, contrasting the refraction of light by the lens in near vision and in far vision
- Identify and describe pupillary light reflex and its pathway and relate these to clinical situations as argyl Robertson pupil
- Identify the lateral geniculate body and visual cortex

Background:

The eye is a complex sense organ that gathers information about the environment, and the brain interprets this information to form an image of what appears within the field of vision. The eye is optically equivalent to usual photographic camera. It has lens system, a variable aperture (pupil), and retina that corresponds to the film.

Take home Message:

- The receptive field of any neuron in the visual pathway is defined as the portion of the visual field to which the neuron responds.
- Only one small region of the retina, the fovea provides high visual acuity.
- Cone photoreceptors are specialized for high-acuity color vision under bright-light (photopic)conditions, whereas rod photoreceptors are more sensitive less acuity and specialized for dim-light (scotopic vision)
- The lens system of the eye has the ability to focus an image on the retina. The image is inverted and reverses, however the brain is trained to perceive it in upright position.
- To bring the diverging rays from close objects to a focus on the retina the curvature of the lens is increased a process called accommodation.

Title of the lecture: Pain modulation	
Lecturer's name	**Dr. Hayam Gad**
Department	Physiology
Block / week	Neuropsychiaty block
Email address	Hayam_gad@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Intensity of the pain can be altered by various extrinsic and intrinsic mechanisms, extrinsic mechanism such as rubbing or shaking of an injured area. Or applying ice pack, or stimulation with an electric vibrator at the site of pain all gives some relief from pain, pain can be modulated by giving analgesic drugs e.g.morphine.

- Describe orally and evaluate critically the mechanism of pain by extrinsic and intrinsic factors as a team. (Cognitive / team work)
- Reduce the intensity of pain with two methods by applying ice pack and stimulating the site of pain by electric vibrator on a mannequins (Psychomotor)
- Prescribe independently at least one analgesic medicine with its correct route and dose (Cognitive / Psychomotor).

Background:

Pain modulation by medicines is a big developing field to control and reduce pain perception**.**

Key topics to be discussed:

Students should know gate-control hypothesis and role of body's own morphines, the opaid peptides. To know about opioid receptors and are formed in the mid brain, brainstem and spinal cord.

Take home messages:

Students are required to know about descending pain ratifying pathway from brain. This inhibits in coming pain signals at the spinal cord level. It is also required to the brain's opiate system e.g. endorphin and emkepalines**.**

Further reading:

- Gamong's review of Medical Physiology, 23rd edition.

Keywords:

- Descending analgesic system, gate-control hypothesis, morphine & enkephalins.

Title of the lecture:	Physiology of the inner ear balance
Lecturer's name	**Prof. Laila Al Ayadhi**
Department	Physiology
Block	Neuropsychiatry block
Email address	Iyayadhi@ksu.eu.sa

Objectives:

Upon completion of this lecture, students should be able to:

- Understand the sensory apparatus of the inner ear that helps the body maintain its postural equilibrium
- The mechanism of the vestibular system for coordinating the position of the head and the movement of the eyes
- The function of semicircular canals (rotational movements, angular acceleration)
- The function of the utricle and saccule within the vestibule (respond to changes in the position of the head with respect to gravity (linear acceleration)
- The connection between the vestibular system and other structure (eye, cerebellum, brain stem)

Title of the lecture:	Physiology of postural reflexes
Lecturer's name	**Prof. Faten Zakaria**
Department	Physiology
Block	Neuropsychiatry block
Email address	Fatenz3699@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Postural reflexes are needed to keep the body in a proper position while standing, moving. When body posture is suddenly altered it is corrected by sevier reflexes. These reflexes are operating at spinal cord, medulla, mid-brain and cortical levels. To make the reflex movements smooth cerebellum, basal ganglia and vestibular apparatus are needed. Students are required to know posture-regulating parts of CNS.

- Present orally the principles of postural reflexes in standing and moving positions
- Evaluate critically the reflex movements in relations to cerebellum, basal ganglia and vestibular apparatus under the supervision of a senior faculty member
- Independently seek and present information on brain parts which are involved in posture regulation at a mannequin

Background:

For all reflexes to operate reflex are is needed and components of reflex are one receptor, afferent, spinal cord, efferent and muscle, all reflexes work when the normal tone in the muscle is present, there are supraspinal reflexes which regulates the muscle movements.

- Able to define human posture
- Explain/define the concepts of " center of gravity '' and " support base ".
- Explain what are postural reflexes and their overall function .
- Know the centers of integration of postural reflexes .
- Explain the structure and function of the vestibular apparatus (utricle, saccule & semicircular canals) in maintenance of balance
- Describe decorticate rigidity and decerbrate rigidity and explain the mechanisms underlying them .

Further reading:

- Gayton and Hall textbook of Medical Physiology 12th edition
- Gamong's review of Medical Physiology, 23rd edition.

Title of the lecture:	Biochemistry of Myelin
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block	Neuropsychiatry block
Email address	sfatma@ksu.edu.sa

Objectives:

- To recognize the sphingolipids class of lipids as regard their chemical structure, tissue distribution and functions.
- To be familiar with the biochemical structure, and function of myelin.
- To describe the basics of biosynthesis and degradation of sphingolipids.
- To discuss sphingolipidosis group of diseases.

Background:

- Sphingolipds are a group of complex lipids that contains sphingosine as an alcohol.
- Sphingolipds contain either phosphate (sphingophospholipids, e.g., sphingomyelin) or carbohydrate (glycosphingolipids, e.g., cerebroside and ganglioside).
- Sphingolipds are found in greatest amounts in nervous tissue, but they are also present extra-neurally.

Main concepts in the lecture:

The precursor of sphingolipids is ceramide. Ceramide is formed of the amino alcohol sphingosine that is attached to a long-chain fatty acid by an amide linkage. For sphingomyelin, a phosphocholine is bound to the hydroxyl group at C1 of sphingosine. For glycolipids (glycosphingolipids), a monosaccharide or an oligosaccharide is attached to sphingosine by an O-glycosidic bond.

Sphingomyelin is the major structural lipid component in the membranes of nerve tissue and the only significant sphingophospholipid in humans. Glycosphingolipids are essential components of all membranes in the body with a higher amount in nerve tissue. They play a role in the regulation of cell growth, development and cell-cell interaction. They are antigenic; the carbohydrate portion of a glycolipid is the antigenic determinant. Extra-neural sphingolipids serve as cell surface receptors for cholera, diphtheria and tetanus toxins as well as for certain viruses.

Sphingomyelin of the myelin sheath contains predominately longer-chain fatty acids such as lignoceric and nervonic acids, whereas gray matter of the brain has sphingomyelin that contains primarily stearic acid.

Sphingolipds are degraded by lysosomal hydrolytic enzymes. If a specific by lysosomal hydrolase is partially or totally missing, a sphingolipid accumulates in the lysosomes, producing lipid storage diseases or sphingolipidosis. The latter are characterized by phenotypic and genotypic variability. They are progressive diseases that affect mainly nerve tissue, where neurologic deterioration can lead to early

death (acute form) or death during adulthood (chronic form), e.g., Niemann-Pick disease. Diagnosis is confirmed by measuring the enzyme activity in cultured fibroblasts or peripheral leukocytes or by detection of specific mutation in the relevant gene. Sphingolipidosis, e.g., Gaucher disease can be treated by recombinant human enzyme replacement therapy or bone marrow transplantation.

Conclusion:

Glycosphingolipids are abundant in nerve tissue, but are present in all membranes of the body.

Sphingomyelin is the major structural lipid component in the membranes of nerve tissue. It is present in myelin sheath (where it contains predominately longer-chain fatty acids) and in the brain's gray matter (where it contains primarily stearic acid).

Lipid storage diseases (sphingolipidosis) are caused by genetic defects in the lysosomal hydrolytic enzymes essential for the degradation of sphingolipids. They are progressive diseases. Diagnosis is confirmed by measuring the enzyme activity or by detection of specific mutation in the corresponding gene. Recombinant enzyme replacement therapy or bone marrow transplantation are used to treat some of these

Take home messages:

- Sphingolipids are complex lipids that include sphingo-phospholipids and glycolipids.
- Ceramide is the precursor of all sphingolipids
- Sphingolipids are present in greatest amount in nerve tissue, but they are found also extra-neural.
- Sphingolipidosis are rare genetic diseases due to the deficiency of hydrolytic enzymes with the consequence of defective degradation and accumulation of sphingolipids.

Further reading:

Lippincott Illustrated Review of Biochemistry, 6th edition, 2014, Unit 3, Chapter 17, Pages 201-218.

Keywords:

Sphingolipids, glycolipids, sphingomyelin, ceramide, ganglioside, cerebrosides, myelin, sphingolipidosis, Gaucher disease, Niemann-Pick disease.

Title of the lecture:	Vitamins B6-B12
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block	Neuropsychiatry block
Email address	sfatma@ksu.edu.sa

Objectives:

Upon completion of this lecture, the students should be able to:

- Understand the general biochemistry and functions of vitamins B_6 and B_{12}
- Recognize the role of these vitamins in maintaining the myelin sheath of nerves including their deficiency that can lead to nerve degeneration and irreversible neurological damage

Background:

Vitamins B_6 and B_{12} are members of the vitamin B complex. They are essential micronutrients in maintaining metabolism and normal functioning of nerves. Deficiency and toxicity of these vitamins can lead to nerve damage and neurological symptoms.

Main concepts in the lecture:

- Excessive intake of vitamin B_6 has been associated with neurological symptoms due to nerve damage.
- Deficiency in the absorption of vitamin B_{12} due to lack of the intrinsic factor results in pernicious anemia. Patients with vitamin B_{12} deficiency show neuropsychiatric symptoms because of irreversible CNS effects.

Take home messages:

- Vitamins B_6 and B_{12} are essential in maintaining nerve function and the central nervous system.
- Various neurological symptoms have been associated with their deficiency.

Further Reading:

Lippincott's Biochemistry. Lippincott Williams & Wilkins, New York, 2008. pp 373-374, 376

Keywords:

Vitamin B_6, vitamin B_{12}, cobalamin, functions, deficiency, toxicity, nerve damage, neurological symptoms, myelin sheath

Title of the lecture:	Vitamin A
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block	Neuropsychiatry block
Email address	sfatma@ksu.edu.sa

Objectives:

Upon completion of this lecture, students should be able to:

- To understand the function and transport of vitamin A.
- To know the role of vitamin A in vision and deficiency can lead to blindness.

Background:

Vitamin A is often used as a collective term for several related biologically active molecules. The term retinoids includes both natural and synthetic forms of vitamin A that may or

may not show vitamin A activity. The retinal a family of molecules that are related to retinol (vitamin A), are essential for vision, reproduction, growth, and maintenance of epithelial tissues. Retinal is a component of visual pigment and derived from oxidation of dietary retinol, responsible for vision.

Main concepts in the lecture:

Visual cycle: Vitamin A is a component of the visual pigments of rod and cone cells. Rhodopsin, the visual pigment of the rod cells in the retina, consists of 11-cis retinal specifically bound to the protein opsin. When rhodopsin is exposed to light, a series of

photochemical isomerizations occurs, which results in the bleaching of the visual pigment and release of all trans retinal and opsin.

This process triggers a nerve impulse that is transmitted by the optic nerve to the brain. Regeneration of rhodopsin requires isomerization of all trans retinal back to 11-cis retinal. Trans retinal, after being released from rhodopsin, is isomerized to 11-cis retinal, which spontaneously combines with opsin to form rhodopsin, thus completing the cycle. Similar reactions are responsible for color vision in the cone cells.

Take home messages:

- The main function of vitamin A in vision and its deficiency leads to blindness.
- The role of vitamin A in visual cycle and color vision.

Keywords:

- Vitamin A, Retinal, retinol, visual cycle, night blindness, Xerophthalmia, blindness

Title of the lecture:	Biochemistry of Alzheimers
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block	Neuropsychiatry block
Email address	sumbulfatma@gmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Have an overview of neurodegenerative disorders
- Understand the clinical picture and diagnostic criteria of Alzheimers disease
- Understand the different ways of processing of Amyloid precursor protein leading to amyloid generation and accumulation
- Discuss the presence of neuritic plaques and nerofibrillary tangles and the role of tau protein
- Discuss the genetics of Alzheimers
- Get an idea of the ongoing research and therapeutic approach to treat these disorders

Background:

Many diseases including Parkinson's, Alzheimer's, and Huntington's occur as a result of neurodegenerative processes. Neurodegeneration is the progressive loss of structure or function of neurons, including death of neurons. Many neurodegenerative diseases are caused by genetic mutations or by the aggregation of misfolded proteins.

Main concepts in the lecture:

- Brief description of neurodegenerative diseases
- An introduction of Alzheimers disease , its clinical picture and Diagnostic criteria
- Neritic plaques and neuritic tangles
- Role of Tau protein in amyloid angiopathy
- Pathogenesis of Alzheimers disease
- Amyloid precursor protrein processing mechanisms leading to generation of amyloid-beta peptides
- Current information about the genetics of Alzheimers
- Current management approaches and potential treatment options based on current research e.g. stem cell therapy.

Conclusion:

Alzheimer's is characterized by progressive loss of short term memory followed by general loss of cognitive and other brain functions. The hallmarks of Alzheimer's are intracellular neurofibrilliary tangles, made up in part of hyperphosphorylated forms of the tau protein and normally binds to microtubules, and extracellular senile plaques, which have a core of β-amyloid peptides (Aβ)

surrounded by altered nerve fibres and reactive glial cells. When APP is hydrolyzed by α-secretase, nontoxic peptide products are produced. But if by the others, toxic polypeptides with 40-42 amino acids are produced. The polypeptides form extracellular aggregates which can stick to AMPA receptors and calcium channels increasing calcium influx. The polypeptides also initiate an inflammatory response, with production of intracellular tangles. The damaged cells eventually die.

Take home messages:

- Neurodegeneration is the progressive loss of structure or function of neurons, including death of neurons.
- Extracellular deposition of normally soluble proteins in certain tissues in the form of insoluble fibrous aggregates known as amyloid. The deposition of amyloid interferes with normal cellular function, resulting in cell death and eventual organ failure.
- The dominant component of amyloid plaque that accumulates in Alzheimer disease is amyloid β 42(Aβ42) Peptide.

Further reading:

- Illustrated Reviews of Biochemistry by Lippincott 4th edition (pp21-22).
- Fundamentals of Biochemistry by Voett and Voett (pp 170-174)
- Stem Cell Technology for Neurodegenerative Diseases. Ann Neurol. 2011 September ; 70(3): 353–361.
- A Review: Inflammatory Process in Alzheimer's Disease, Role of Cytokines. The ScientificWorld Journal, Volume 2012, Article ID 756357,

Title of the lecture:	Biochemistry Cerebrospinal Fluid
Lecturer's name	Dr. Rana Hasanato
Department	Biochemistry
Block	Neuropsychiatry block
Email address	rhasanato@ksu.edu.sa

Objectives:

- To identify the CSF functions, formation and circulation
- To recognize the method of CSF sampling, and the procedure for specimen collection, and processing
- To identify the indications and contraindications of lumbar puncture and laboratory investigation of CSF
- To recognize and explain the normal and abnormal findings of physical and biochemical examination of CSF (with special emphasis on the glucose, protein, electrolytes and cellular content of CSF)
- To interpret CSF electrophoresis pattern
- To define expressions describing abnormal locations of CSF as otorrhea and rhinorrhea

Background:

- CSF is the liquid surrounding the brain and spinal cord.
- CSF serves several important functions, for instance physical protection and providing controlled chemical environment.
- The rate of CSF formation and excretion ensures a constant CSF volume

Main concepts in the lecture:

Obtaining a CSF sample by lumbar puncture is indicated for investigating cases of CNS infection, malignancy, hemorrhage, or demyelinating diseases. However, there are contraindications to perform lumbar puncture. The method to obtain the CSF and the steps for sample processing and analysis must follow a standardized operating procedure. Interpretation of the findings of CSF analysis (both physical and biochemical) provides important information helping in differential diagnosis of pathological conditions in the CNS. For instance, differentiating bacterial, tuberculous, and viral meningitis can be done through the distinctive appearance, predominant cell, microorganism, as well as protein, glucose, and chloride composition of the CSF. In addition, blood, hemoglobin, and excess albumin and immunoglobulin are to be reported if found in the CSF. If high CSF protein level is detected, protein electrophoresis is recommended to separate various bands of proteins and interpret the findings accordingly.

Conclusion:

CSF is the liquid surrounding the brain and spinal cord, it formed in the choroid plexus and it serves important functions. Its analysis is requested in many pathological conditions; however the method for obtaining a CSF sample and processing it has to be performed carefully following a standardized procedure. Interpretation of the physical appearance and the biochemical composition of the CSF results provide valuable information.

Take home messages:

- CSF is formed in the choroid plexus by a tightly controlled production/excretion ensuring a constant CSF volume.
- CSF is essential for the physical protection of the CNS, and for providing a controlled chemical environment for the CNS.
- The physical & Biochemical analysis of CSF is essential for diagnosis of several pathological conditions.

Further reading:

1- Lecture notes, Clinical Biochemistry, Wiley BlackWell, 8th edition, 2010, chapter 19, page 274-277

2- Clinical Chemistry, Principles, Procedures, Correlations, Lippincott Williams & Wilkins, 5th edition, 2005, chapter 27, page 560-563.

Title of the lecture:	Pathogenesis of Cerebral Infarction at Cellular and Molecular Levels
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block	Neuropsychiatry block
Email address	sumbulfatma@gmail.com

Objectives:

- To identify the cell death mechanisms implicated in the pathogenesis of ischemic brain injury.
- To recognize various factors involved in ischemia-induced metabolic stress.
- To acquire the knowledge of the important role played by oxidative stress and free radicals in the pathogenesis of cerebral infarction
- To identify neurochemical changes involved in cerebral ischemia
- To discuss the biochemical basis of potential pharmacological intervention for patients with cerebral ischemia

Background:

- Stroke is the second most common cause of mortality
- Stroke is the third most common cause of disability
- The incidence of stroke is decreasing in high-income countries & increasing in low-income countries
- The overall rate of stroke-related mortality is decreasing in high and low income countries
- The absolute number of people with stroke, stroke survivors, stroke-related deaths, and the global burden of stroke-related disability is high and increasing
- Men have a higher incidence of stroke than women at younger but not older ages
- Stroke incidence is higher in women ≥75 year old compared to men

Main concepts in the lecture:

Current knowledge regarding the pathogenesis of cerebral infarction indicates that loss of cellular integrity and tissue destruction are major contributing mechanisms.

Reactive oxygen species (ROS) are produced as by-products of many biochemical reactions. ROS have beneficial physiological roles, yet, they are considered the pathogenic mediators of a growing list of human diseases, including ischemia.

Stroke is divided into hemorrhagic (intracerebral, or subarachnoid) and ischemic (thrombotic and embolic).

The cell death mechanisms implicated in the pathogenesis of ischemic brain injury and the various factors involved in ischemia-induced metabolic stress include calcium-induced calpain-mediated

proteolysis of brain tissue. Oxidative stress plays an important role in the pathogenesis of cerebral infarction. The possible cellular/molecular aspects of cerebral ischemia and infarction will be highlighted. The central role of increased cytosolic calcium with its consequent cellular and molecular changes during ischemic injury will be described. There are several examples of potential biochemical intervention in cerebral ischemia including inhibitors of glutamate release, Ca^{2+} channel blockers, NOS inhibitors & free radical inhibition, AND Calpain inhibitors.

Conclusion:

Studying the biochemical, molecular, and cellular changes in ischemia is helpful in designing potential therapeutics targeting the defective pathways and processes.

Take home messages:

- Severe cerebral ischemic insults lead to a complex cascade of biochemical and molecular events, including:
    - Lack of oxygen supply to ischemic neurons
    - metabolic stress and ATP depletion
    - Malfunctioning of membrane ion system
    - Depolarization of neurons
    - Calcium influx and its consequences (protein kinase C activation, protein phosphorylation and proteolysis.
    - Neurotransmitter release and activation of proteases
- The above-mentioned events vary according to the severity and location of cerebral ischemia.

Further reading:

- Bramlett and Dietrich, Pathophysiology of Cerebral Ischemia and Brain Trauma: Similarities and Differences, Journal of Cerebral Blood Flow and Metabolism, 2004, 24: 133-150
- Allen and Bayraktutan, Oxidative Stress and its Role in the Pathogenesis of Ischemic Stroke, World Stroke Organization International Journal of Stroke, 2009, 4:461–470

Keywords:

- Ischemia – Cerebral infarction – Apoptosis – Necrosis – Reactive oxygen species – Oxidative stress – Metabolic stress

Title of the lecture:	Biochemistry and microbiology of CSF (Practical Sessions)
Lecturer's name	Dr. Sumbul Fatma
Department	Biochemistry
Block	Neuropsychiatry Block
Email address	sumbulfatma@gmail.com

Objectives:

By the end of this practical session, students will be able to:

- Identify the functions of CSF.
- Recognize the normal and abnormal constituents of CSF.
- Understand the role of CSF in diagnosis of different diseases of CNS.
- Interpret the microbiological and biochemical investigation results of CSF.
- Hands-on the procedure for estimation of total protein in CSF.

References:

- Presentation slides
- Lab tests online https://labtestsonline.org/understanding/analytes/csf/tab/test/

Title of the lecture: Microbiology of acute pyogenic meningitis	
Lecturer's names	Prof. Hanan Habib
Department	Microbiology
Block	Neuropsychiatry Block
Email address	hahabib@ksu.edu.sa

Objectives of the lecturer:

- Define and know important facts about pyogenic meningitis.
- Know the epidemiology of acute pyogenic meningitis.
- Know the etiologic agents of acute pyogenic meningitis and common serotypes of the three main causative bacteria pathogens, causative agents according to the age of the patient, and other common circumstances.
- Know the clinical presentation of acute pyogenic meningitis case.
- Identify the microbiology of common etiologic agents including; morphology, pathogenesis, identification tests and complications.
- Know the diagnostic approaches to a meningitis case with emphasis on the lab identification, compare the findings of normal and abnormal CSF analysis.
- Know the management of acute pyogenic meningitis case with emphasis on rapid diagnosis and selection of empirical antimicrobial therapy to most common pathogens.
- Know the prevention using vaccination and prophylaxis of contacts against common etiologic agents.

Background:

Acute Bacterial meningitis is an important cause of mortality and morbidity particularly in neonates and children throughout the world.
The introduction of vaccines against the three important pathogens (*N.meningitidis, Hib*, and *S.pneumoniae*) has changed the epidemiology of bacterial meningitis.
A case of suspected acute pyogenic meningitis is a medical emergency and needs urgent empirical antimicrobial treatment to prevent complications.
Knowledge on the pathogenesis neurological sequelae , diagnostic approaches ,treatment and prevention is needed.

Main concepts in the lecture:

Bacterial meningitis is an inflammation of the meninges affecting the Pia, Arachnoid and Subarachnoid space that happens in response to bacteria and its products. It is a worldwide diseases and an important cause of morbidity and mortality in neonates and children. The pathogens that have a special potential to cause meningitis include; *N.meningitidis, S.pneumoniae,*and *H.influenzae* type *b* (*Hib*) ,*E.oli* and *L .monocytogenes*. The pathogenesis of meningitis is due to crossing the blood brain barrier through cerebral vasculature, by the infected phagocytes and by interactions with host

receptors and release of bacterial toxins and stimulation of inflammatory responses. Host risk factors are important to be considered to know the possible etiologic agents.

Clinical presentation of meningitis differs between neonates, elderly children and adults and the most common presentation in older children and adults include; fever, headache, photophobia, nausea and vomiting, confusion or irritability.

Laboratory diagnosis is of paramount importance of a meningitis case. Lumbar puncture should be performed in patients with suspected meningitis (if no contraindications), blood culture should be performed as well .Gram stain and CSF analysis shows the causative bacteria and differential counts. Concentration of protein and glucose are helpful in the differential diagnosis of various forms of meningitis. Lab. identification of the causative bacteria is as well as the susceptibility testing must established for the selection of the optimum antimicrobial therapy recognition of pathogens with increasing resistance to antimicrobial drugs. Meningitis is a medical emergency and treatment must be empirical and parenteral against the common causative agents and the age of the patient.
Complications are common following untreated or partially treated meningitis including neurological sequelae and mortality most common following *S.pneumoniae* compared to other causative agents.
Prevention by vaccinations against the common causative bacteria and their impact on the reduction of the incidence of meningitis .Prophylaxis of contacts of *N.meningitidis* and *H.influenzae.*

The practical include microscopic appearance of *N. meningitides, H.influenze, S.pneumoniae* ,group *B Streptococcus*, and *E.coli* and the main morphologic and chemical tests used for identification.

Conclusion:

Acute pyogenic meningitis is a medical emergency that needs urgent empirical antimicrobial treatment to prevent complications.
It is an important cause of mortality and morbidity particularly in neonates and children throughout the world.
Commonly caused by the three common and pathogens (*N.meningitidis*, *Hib*, and *S.pneumoniae*) .
Lab diagnosis by rapid analysis and culture of CSF sample is required.
Management of acute pyogenic meningitis case with empirical antimicrobial therapy that cover most common pathogens is important.
Prevention using vaccination and prophylaxis of contacts against common etiologic agents.

Take home messages:

- Acute pyogenic meningitis is an important cause of morbidity and mortality especially neonates and children worldwide.
- Three main etiologic agents of acute pyogenic meningitis and common serotypes.
- The important causes of meningitis in different age groups and other medical circumstances.
- Brief on common clinical presentations of acute pyogenic meningitis.
- Microbiology of common causative agents including; Gram stain, morphology, common serotypes, identification tests.

- Lab. diagnosis of acute meningitis including; specimen collection (CSF, and blood), macroscopic examination, microscopic examination (WBC and differential counts, glucose ,and protein) in comparison to normal CSF finding.
- Meningitis is a medical emergency need empirical parenteral antimicrobial agent to cover the most common causative agents according to the age of the patients and resistance patterns.
- Prevention by vaccination against common pathogens and prophylaxis of contacts.

Further reading:

- *Sherries* Medical Microbiology, an Introduction to Infectious Diseases. Latest edition, Kenneth Ryan and George Ray. Publisher: McGraw Hill. Chapter 67, page: 873-879.

Title of the lecture: Cerebral tuberculosis and other chronic cerebral infections	
Lecturer's name	Dr. Fawzia AlOtaibi
Department	Microbiology
Block	Neuropsychiatry Block
Email address	ofawzia@ksu.edu.sa

Objectives of the lecturer:
By the end of this lecture, the students should be able to:

- Differentiate between clinical presentation of acute and chronic cerebral and meningitis infection.
- Differentiate between the cerebrospinal fluid findings in acute and chronic meningitis.
- Know generally the different microbiological causes of chronic cerebral infection and meningitis.
- Know the details of the different bacterial causes of chronic meningitis specially tuberculosis and brucellosis.
- Differentiate between the clinical presentation and laboratory findings of tuberculous and Brucella meningitis.
- Know the basis of treatment of T.B and Brucella meningitis.

Background:

Chronic inflammation of meningitis (pia, arachnoid and dura) as well as cerebral tissue can produce profound neurological disability and maybe fatal if not treated.
Chronic meningitis has an insidious onset, with progression of signs and symptoms over a period of weeks. This is different from acute pyogenic meningitis and cerebral infection which has rapid onset of symptoms and sign in a period of days.
The chronic cerebral infection and meningitis are commonly diagnosed if the characteristic neurological syndrome exists for more than 4 weeks associated with inflammatory response in (CSF).
The causes of chronic inflammation of meningitis and cerebral tissue could be Bacterial, fungal or parasitic.

Main concepts in the lecture:

Clinical features of chronic cerebral and meninges infection. A insidious gradual onset with progression of symptoms and signs over a long period of more than 4 weeks. The symptoms include chronic head ache later, signs include hydrocephalus, cranial neuropathy with chronic memory impairment. These symptoms and signs differ from those of acute pyogenic meningitis which are characterized by acute onset of fever headache, stiff neck, vomiting, irritability and neurological dysfunction in days.

The symptoms and signs of chronic meningitis can exist in two forms. In the first one, the symptoms are chronic and persistent in the other, the symptoms are recurrent, and discrete episodes of illness.

Conclusion:

Symptoms and signs of chronic cerebral infection and meningitis infection. Causes of chronic cerebral infection and meningitis addressing mainly tuberculosis and brucella infections. Laboratory findings in cerebrospinal fluid and treatment of chronic infection and meningitis stressing on the tuberculosis and brucellosis cerebral infection and meningitis infection.

Take home messages:

- The clinical presentations of chronic cerebral and meningitis infection differ from those of acute infection in being of insidious onset and of chronic nature.
- The laboratory findings of CSF mainly show increased pressure, protein and the WBC, with mainly lymphocytes predominance. It is like aseptic meningitis where organisms are not easily detected by microscopy.
- The microbiological causes of these chronic infections maybe bacterial, fungal or parasitic.
- The bacterial cause are the most common and in Saudi the most important are tuberculosis and Brucellosis. Other bacterial causes include syphilis and Leptospirosis.
- Diagnosis is by clinical features and CSF finding mentioned above. Diagnosis can also done by culturing organisms or detection of microbial components by molecular methods like PCR.
- It is important to differentiate between tuberculosis and Brucella meningitis as treatment differ.

Further reading:

- Sherris, Medical Microbiology, Introduction to infectious diseases. Latest version.

Title of the lecture: Fungal infections of the CNS	
Lecturer's name	Dr. Maha Almohizea
Department	Microbiology
Block	Neuropsychiatry Block
Email address	maha_mm990@hotmail.com

Objectives of the lecturer:

- Acquire the basic knowledge about fungal meningitis and brain abscess and there clinical features.
- Know the main fungi that affect the central nervous system.
- Identify the clinical settings of such infections.
- Know the laboratory diagnosis, and treatment of these infections.

Background:

Central nervous system infections are both, diagnostic challenges and medical emergencies. This is because the delay in diagnosis and initiation of appropriate therapy will result in the high rate of mortality or in permanent, severe neurological damage.
Several fungal agents can cause CNS infections.
CNS fungal infections may primarily involve the meninges (i.e., meningitis), or may present as brain abscess.

Main concepts in the lecture:

Clinical Settings:
Fungal CNS infections are observed particularly in leukemia, allogeneic hemopoietic stem cell transplantation patients, AIDS patients and diabetic patients. However, fungi causing brain abscess in immunocompetent people has been reported in Saudi Arabia and other Middle East countries.

Pathogenesis:
Fungi reach the central nervous system by different mechanisms. Most commonly, from hematogenous spread, and local extension from the paranasal sinuses, the ear, or the orbits. Traumatic introduction can also occur by surgical procedures, head trauma, injections and lumbar punctures.

Etiology:
There are several fungi which can cause meningitis or brain abscess. The most common fungi include:
Cryptococcus neoformans
Candida species
Zygomycetes

Aspergillus species
Fuzarium species
Ramichloridium makckenzei and other black fungi

Clinical features:

Fungal meningitis is a life threatening disease and may be caused by a variety of fungi. *Cryptococcus neoformans* is one of the most common causes of fungal meningitis in HIV patients. In CNS infection with Zygomycetes, CNS involvement is higher and this fungal infection is also characterized by a high mortality rate and seen in patients with uncontrolled diabetes with KA. Rhinocerebral diseases, caused by *Aspergillus* and *Zygomycetes* occur by direct extension of sinus disease into the orbit, eye, optic nerve, and brain parenchyma including the frontal and temporal lobes. *Ramichloridium mackenziei* is a neurotropic black mold restricted to the Middle East causing cerebral phaeohyphomycosis. Brain abscess formation is the typical form.

Diagnosis:

In these conditions, diagnosis is very difficult and diagnosis can often be performed only through aggressive procedures.

Diagnosis is usually based on the analysis cerebrospinal fluid (CSF) samples, or biopsy tissue. Fungal smear and culture is usually performed to demonstrate the fungal elements and culture in laboratory media.

Serology testing is available for some fungi including *Candida, Aspergillus* and *Cryptococcus*. For example, cryptococcal antigen detection is important tool to diagnose Cryptococcal meningitis.

Treatment:

Treatment typically involves the administration of antifungal medication such as amphotericin B, Voriconazole, posaconazole and caspofungin. Often two drugs in combination initially. In many cases surgical debridement is necessary.

Conclusion:

Fungal CNS infections are a life threatening diseases. They may present as meningitis or brain abscess. They are caused by a variety of yeast and filamentous fungi. Mortality rate is high. Early diagnosis and initiation of appropriate therapy are very important in improving the management of the patients with fungal CNS infections

Take home messages:

- Fungal CNS infections can present as meningitis or brain abscess
- These infections are usually seen in immunocompromised patients.
- The infections can be acquired through hematogenous spread, local extension from the paranasal sinuses, the ear, or the orbits and trauma
- Early diagnosis and initiation of antifungal therapy is essential.

Further reading:

- Alhedaithy, S.S., Medical Mycology Lecture slides. 2009 (2nd Edition).

Title of the lecture: Microbiology of middle ear infection	
Lecturer's names	Prof. Hanan Habib
Department	Microbiology
Block	Neuropsychiatry Block
Email address	hahabib@ksu.edu.sa

Objectives of the lecture:

- Define and know the classification of middle ear infection (otitis media (OM))
- Know the epidemiology of OM
- Explain the pathogenesis and recognize the risk factors and clinical presentation of OM
- Define the microbiology of OM and list examples of common bacterial causes of different types of OM
- Identify the diagnostic approaches and emphasize on microbiological aspect of diagnosis
- Know the management of OM
- Recall common complications of OM.

Background:

OM is very common in childhood accompanying viral upper respiratory tract infection with three classes (acute, serous & chronic)
Risk factors related to horizontal position of Eustachian tube , other anatomic abnormalities, obstruction and immune dysfunction.
Bacteria are the most common causes of acute OM.
Diagnosis and antimicrobial therapy are important to prevent serious complications.

Main concepts in the lecture:

The anatomy of middle ear is first revised and the function of Eustachian tube. Definition of OM is followed.OM is most common in children particularly in infancy period where 2/3 of cases occur during this age where it improves with age. The reason of this improvement is related to the position and function of Eustachian tube. OM is classified into acute, chronic and serous OM. Certain risk factors are associated with the development of OM related to Eustachian tube ,colonization with previous upper respiratory tract pathogens, allergy, congenital abnormalities such as cleft palate, obstruction due to adenoids or exposure to smoking.
Images of the anatomy of middle ear, acute, chronic and serous OM are shown.
The microbiology of OM is then discussed where bacteria is the most common cause of acute OM which commonly involve *S.pneumoniae, H.influenzae, S.pyogenes, S.aureus* and other Gram positive and Gram negative bacteria according to the age of the child. RSV is the common viral cause followed by Rhinovirus and Parainfluenza virus. The causative bacteria of chronic and serous OM which result due to unresolved acute infection is different where it is mixed Gram positive and Gram negative bacteria in 40% of cases which includes; coliforms and *P.aeruginosa* and anaerobic

bacteria. In serous OM ,most of the effusion is sterile with few inflammatory cells and the organisms are similar to chronic OM. An illustration of the Gram stain and culture of the common causative bacteria is given including important identification tests. Clinically most patients with OM present with fever, irritability and pain. Pus and exudative discharge appear later. The diagnostic approaches include specimen collection through tempanometry, Gram stain and culture of the causative bacteria are emphasized. Management include antimicrobial therapy according to the causative bacteria and susceptibility testing. Serious complications can arise in inappropriately treated cases, mainly hearing loss , meningitis, brain abscess or others.

Conclusion:

Acute OM is acute inflammation of middle ear. It is common in infancy due to the position and function of Eustachian tube . There are certain risk factors associated with the development of OM. OM is classified into acute, serous and chronic OM. Bacteria are the most common causes .Chronic OM is due to unresolved acute infection. Clinical diagnosis and laboratory investigation of pus aspirated from the middle ear is important for diagnosis. Appropriate antimicrobial therapy according to susceptibility testing is indicated to prevent serious complications.

Take home messages:

- OM is a common disease in children particularly infants related to Eustachian tube anatomy and function
- OM can be acute, serous or chronic
- Risk factors include position of the Eustachian tube, allergy, anatomic abnormalities, obstruction and smoking.
- Common causes of acute OM :*S.pneumniae, H.influenzae*. Mixed Gram positive and Gram negative organisms and anaerobes in chronic and serous OM.
- Clinical presentation of acute OM are fever, earache and exudative discharge
- Unresolved acute infection can progress into chronic or serous OM.
- Diagnosis is clinical and through sampling ear discharge for culture and identification of the causative organisms.
- Management according to the susceptibility testing of the causative organisms.
- Complication include hearing loss, meningitis , brain abscess and others

Further reading:

- *Sherrie's* Medical Microbiology, An Introduction To Infectious Diseases. Latest edition.
- Kenneth Ryan and George Ray. Publisher : McGraw Hill. Chapter 62, page:829-831.

Title of the lecture: Viral infections of the CNS	
Lecturer's name	Dr. Malak El-Hazmi
Department	Microbiology
Block	Neuropsychiatry Block
Email address	melhazmi@ksu.edu.sa

Objectives of the lecture:
Know the different viral neurological diseases.

- Understand the details of the different acute viral infections of the CNS. (Meningitis, paralysis and encephalitis).
- Differentiate between the clinical presentation and cerebrospinal fluid finding in the viral meningitis (aseptic meningitis) and bacterial meningitis (septic meningitis).
- Know generally the common viruses causing aseptic meningitis with the focus on the most common causes of septic meningitis (enteroviruses & polioviruses) with regard to classification, structure, epidemiology pathogenesis, infections, clinical presentation, lab diagnosis and prevention.
- Know the different viruses causing encephalitis with the focus on herpes simplex encephalitis and rabies covering structure of the virus, epidemiology, pathogenesis, clinical presentation, lab diagnosis and prevention.
- Know general information of arboviruses and giving some example of arboviruses causing CNS infection.

Background:

Viral infections of the central nervous system (CNS) are not uncommon occurrences in clinical practice; however, the incidence of these cases is not well defined. Patients usually present with clinical features of aseptic meningitis and/or encephalitis of varying degrees of severity. The outcomes are also variable, depending on unclearly defined host- and organism-specific factors, ranging from generally benign, in cases of aseptic meningitis, to severe with neurological sequelae and even death in patients with encephalitis. The initial approach to the patient with suspected CNS infection requires an early recognition of the meningitis and/or encephalitis syndrome, and needs to include a rapid diagnostic evaluation coupled with concurrent antimicrobial and adjunctive therapy (eg, corticosteroids, when indicated).
Delay in the initiation of therapy introduces the potential for increased morbidity and mortality, such as in cases of herpes encephalitis.

Main concepts in the lecture:

Viral CNS infections have a wide spectrum of causes and clinical presentations.

Viral meningitis refers to meningitis caused by a viral infection. It is sometimes referred to as "aseptic meningitis" in contrast to septic meningitis caused by bacteria.

Aseptic meningitis is inflammation of the meninges with CSF lymphocytic pleocytosis and no cause apparent after routine CSF stains and cultures. Viruses are the most common cause. Other causes may be infectious or noninfectious. Symptoms include fever, headache, and meningeal signs. Viral aseptic meningitis is usually self-limited. Treatment is usually symptomatic. Viruses: Enteroviruses, including echovirus, coxsackievirus, and enteroviruses 68 through 71, cause most cases of aseptic meningitis .The next most common causes of viral meningitis are herpes simplex virus type 2 (HSV-2), HIV, and the arthropod-borne viruses. Mumps virus & poliovirus were common causes worldwide but have been minimized by vaccination.

Differentiating bacterial meningitis from aseptic meningitis: Because bacterial meningitis requires immediate treatment and aseptic meningitis usually does not, rapid identification of bacterial meningitis is important (and sometimes difficult).

CSF findings help make the distinction. CSF glucose is usually decreased and protein is elevated in bacterial meningitis but not in aseptic meningitis. CSF WBCs are predominantly lymphocytes in aseptic meningitis; even a few CSF neutrophils (which may, however, be present in early viral meningitis) should prompt consideration of early bacterial meningitis. However, several types of bacterial meningitis have CSF characteristics that are similar to those of aseptic meningitis; they include partially treated bacterial meningitis, Listeria meningitis and TB meningitis.

Enteroviral meningitis is the commonest cause of aseptic meningitis, with both epidemic and endemic patterns of disease. They are classified into echoviruses and coxsackieviruses and polioviruses. Human enteroviruses cause a wide variety of diseases including polio, aseptic meningitis and encephalitis, most commonly affecting children and transmitted predominantly through the faecal–oral route, entering the GI tract and spreading via the bloodstream to invade the CNS. Diagnosis can be confirmed by lumbar puncture and analysis of cerebrospinal fluid (CSF). Viral meningitis is characterized by an increased WBC count with a lymphocyte predominance, slightly elevated protein, and normal glucose. but can be moderately decreased in some cases of enteroviruses .Isolation of viruses (on cell culture) from CSF or stool is the gold standard for diagnosing enteroviruses causing meningitis; however, the procedure is slow, expensive and not always sensitive .Reverse transcriptase PCR (RT–PCR) assays for enteroviruses have been shown to be more sensitive (and rapid) than cultures of CSF. Most cases of viral meningitis are self-limited and require only symptomatic treatment. Hospitalization is not usually necessary.

Control of the spread of enteroviral meningitis can be decreased through basic hygiene techniques such as hand washing. Two types of vaccines are used to prevent polio {1- Salk, Killed polio vaccine. 2-Sabin, Live-attenuated polio vaccine}.

Encephalitis is an inflammation of the brain parenchyma lead to a complex, severe, neurological syndrome that is associated with significant morbidity and mortality. It presents as an alteration in consciousness, fever, headache, seizures, and/or focal neurologic signs.

Acute viral encephalitis may be caused by a wide range of viruses but the most important is herpes simplex encephalitis (HSE) because of its severity, especially if untreated. Imaging evaluation can provide support for the diagnosis by the demonstration of temporal lobe edema /hemorrhage on MRI .The diagnostic gold standard is the detection of HSV DNA in the CSF by PCR. Acyclovir is the treatment of choice.

Rabies is a fatal viral encephalitis. It is an RNA virus that is usually transmitted to humans through bites from rabid animals. It presents with 1 of 2 clinical features, encephalitic rabies and paralytic illness. The paralytic form is much less common. Negri bodies in corneal scraping & in autopsy specimens of the brain are diagnostic of rabies. Rabies cannot be treated; therefore, efforts must be focused on preventing the disease. Prevention measures are aimed at the animals that can transmit rabies or can include Pre-exposure prophylactic immunization & post-exposure treatment of a person.
Arthropod-borne viruses, i.e., arboviruses, are viruses that are maintained in nature through biological transmission between susceptible vertebrate hosts by blood feeding arthropods (mosquitoes and ticks). The majorities of human infections are asymptomatic or may result in a nonspecific flu-like syndrome. Infection may, however, lead to meningitis or encephalitis, with a fatal outcome or permanent neurologic sequelae. Arboviral encephalitis is a worldwide problem .Some examples of these viruses are discussed. Laboratory diagnosis of human arboviral encephalitis has changed greatly over the last few years with the advent of, such as ELISA & PCR. Arboviral encephalitis can be prevented in two major ways: personal protective measures and public health measures to reduce the population of infected mosquitoes in addition to Japanese encephalitis vaccine & Tick-borne encephalitis vaccine are available.

Conclusion:

This lecture covers the most common viral infections of CNS with the focus on details of Enteroviruses, Herpes simplex virus, Rabies virus, and West Nile virus.

Take home messages:

- Viral meningitis is a central nervous system infection characterized by signs and symptoms of meningeal inflammation in the absence of positive bacterial cultures.
- The causes of viral meningitis is broad: enterovirus is the most common cause.
- Viral meningitis is usually self-limited.
- Diagnosis is by lumbar puncture and CSF analysis. Viral PCR studies of the CSF are more sensitive than routine cultures.
- Two types of polio vaccines .each has advantages, disadvantages & special situation to use.
- The viral CNS infection caused by herpes virus is treatable, others are not.
- Rabies virus is usually transmitted to humans through bites from rabid animals, causes severe encephalitis. Recovery is extremely rare, but it is prevention by vaccination.
- Arboviral encephalitis is prevalent worldwide& it is transmitted by the bite of infected vector.

Further reading:

- Medical Microbiology and Immunology.
    - By: Warren Levinson, 10th Edition, 2008.
    - Published By: McGraw-Hill Co.
- Medical Microbiology.
    - By: David Greenwood Richard C.B. Slack John F Peutherer and Mike Barer, 17th Edition, 2007. Published By: Elsevier Limited.

Title of the lecture:	Cellular injury of the nervous system
Lecturer's name	Dr. Hala Kfoury
Department	Pathology
Block	Neuropsychiatry Block
Email address	halakfoury@hotmail.com

Objectives of the lecture:
At the end of this lecture the students should:

- Understand the role of the different constituents of CNS cells in the disease status.
- Compare the "trauma" and "injury" concepts.
- Explain the basic pathological descriptive terms used in CNS cellular injury and trauma.
- Correlate the different patterns of cellular injury with some important clinical examples.
- Analyze the clinical entities that result from CNS trauma.

Background:

The central nervous system cells are unique in many pathological aspects. A good example is the CNS cellular reaction to injury.
CNS trauma is very serious and crucial subject; considering the high rate of road traffic accidents in Saudi Arabia. Trauma to the brain and the spinal cord is a significant cause of death and disability (6000 deaths in 2009/ KSA traffic police statistics). Understanding classification of the CNS trauma patterns is of particular importance for any clinician regardless of his/ her specialty.

Key principles to be discussed:

Cellular aspects of injury:
The definition of and an example for each of the following terms:
Markers of Neuronal Injury: Acute neuronal injury, red neurons, spheroids, central chromatolysis, intracellular inclusions and dystrophic neuritis
Marker of Astrocytes reaction to injury: gemistocytic astrocytes, fibrillary astrocytes and Rosenthal fibers Microglia (microglial nodules and neuronphagia)

CNS trauma:
The rule of the severity and site of injury in deciding the outcome
Traumatic parenchymal Injuries: The definition of and an example for each of the following terms: Contusion, laceration, diffuse axonal injury and concussion.
The classification of traumatic Vascular Injury entities, including the mechanisms and the clinical presentation of epidural Hematoma (with special emphasis on the lucid interval) and subdural Hematoma (with special emphasis on the pathological features).

Key principles to be covered by self-directed learning:

The definition of Corpora amylacea and its relation to CNS cellular injury.
The definition and an example for: Coup injury, countercoup injury.

Take home messages:

- The cellular constituents of the nervous system respond in different ways to various forms of injury.
- Physical injury to the brain can occur when the inside of the skull comes into forceful contact with the brain. If the head is able to move there may be contact between the skull and brain, both at the original point of contact (coup injury) and the opposite side where the brain eventually hits the skull as it moves within it (countercoup injury).
- Rapid displacement of the head and brain can lead to tearing of axons (diffuse axonal injury), which often causes immediate onset of severe and minimally reversible neurologic deficits.
- Tearing of blood vessels associated with trauma can lead to accumulation of blood in any of three spaces: epidural hematoma, subdural hematoma, or subarachnoid hemorrhage.

Key words:

- Cellular injury, neurons, glial cells, hemorrhage, epidural hematoma, subdural hematoma, subarachnoid hemorrhage, diffuse axonal injury, coup injury, countercoup injury, oligodendrocytes, ependymal cells, microglia, neuronophagia, acute neuronal injury, red neurons, spheroids, central chromatolysis, dystrophic neuritis, gemistocytic astrocyte, fibrillary astrocyte, Rosenthal fibers and Corpora amylacea.

Further reading:

- Vinay Kumar, Abul K. Abbas, Nelson Fausto, & Richard Mitchell, Robbins Basic Pathology, 8th Edition

Title of the lecture:	Congenital malformations and hydrocephalus
Lecturer's name	Dr. Hala Kfoury
Department	Pathology
Block / week	Neuropsychiatry Block
Email address	halakfoury@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Know the common types of congenital malformations of the CNS and have a basic knowledge of their pathological features.
- Correlate CNS normal development with the classification of congenital CNS malformations.
- Appreciate the role of folate deficiency as an etiological factor in neural tube defects and understand the role of Alpha feto-protein measurement and ultrasound in antenatal diagnosis of neural tube defects.
- Understand the various mechanisms that lead to the development of hydrocephalus.
- List and classify the main causes of hydrocephalus.

Background:

The incidence of CNS malformations, giving rise to mental retardation, cerebral palsy, or neural tube defects, is estimated at 1% to 2%. Malformations of the brain are more common in the setting of multiple birth defects. Prenatal or perinatal insults may either cause failure of normal CNS development or result in tissue destruction.

Hydrocephalus is abnormal buildup of cerebrospinal fluid (CSF) in the ventricles of the brain. It can result from congenital and acquired etiologies. The fluid is often under increased pressure (but not always) and can compress and damage the brain.

Key principle to be discussed:

CNS congenital malformation incidence and introduction to the basic concepts behind the pathogenesis. These include genetic and environmental factors and the role of the stage of gestation development.

- Definition and pathological changes in forebrain anomalies:
    - Megalencephaly, microencephaly and lissencephaly.
    - Microencphaly causes.

- Definition and pathological changes in neural tube defects:

    - Meningomyelocele, spina bifida, anencephaly and encephalocele.
    - Pathogenesis with special emphasis on the role of folate and alpha fetoproteins and their clinical significance.
- Definition and pathological changes in posterior fossa anomalies:
    - Arnold Chiari malformation.

Hydrocephalus:

- Definitions of normal pressure hydrocephalus, noncommunicating hydrocephalus and communicating hydrocephalus - Pathophysiology and etiology.

Key principles to be covered by self-directed learning:

- Define: meningocele.
- Define: polymicrogyria.
- What is the difference between microcephaly and microencephaly?
- Define: hydrocephalus ex vacuo.

Take home messages:

- Malformations of the brain can occur because of genetic factors or external insults.
- The timing of the injury will determine the pattern of the injury, based on the type of developmental processes occurring at the point of injury.
- Patterns of malformation include alterations in the closure of the neural tube, proper formation of the separate portions of the neural tissue, and migration of neurons to the appropriate locations.
- Hydrocephalus is an increase in CSF volume within all or part of the ventricular system.

Further reading:

- Vinay Kumar, Abul K. Abbas, Nelson Fausto, & Richard Mitchell , Robbins Basic Pathology, 8th edition
- http://www.medterms.com, for additional explanation of terms definitions.

Keywords:

- CNS malformations, hydrocephalus, noncommunicating hydrocephalus, communicating hydrocephalus, hydrocephalus ex vacuo, Arnold Chiari malformation , folate, alpha fetoproteins, meningomyelocele, meningocele, spina bifida, anencephaly, encephalocele, megalencephaly, microencephaly, lissencephaly and polymicrogyria.

Title of the lecture:	Introduction to degenerative brain disease
Lecturer's name	Dr. Hala Kfoury
Department	Pathology
Block	Neuropsychiatry block
Email address	halakfoury@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Explain the basic pathological concepts of neurodegenerative disease, using Alzheimer's and Parkinson disease as a classical example.
- Know the definition of "dementia" syndrome.
- List the possible causes of dementia.
- Explain the basic pathological concepts of a neurodegenerative disease, using Alzheimer's disease as a classical example.
- Understand the major clinic-pathological features of Alzheimer's disease.
- Hypothesize the possible etiologies of Alzheimer's disease.
- List the causes of Parkinsonism.
- Understand the major clinical and pathological feature of Parkinson disease.
- Hypothesize the possible etiologies of Parkinson disease.

Background:

Degenerative brain disease is an umbrella term for the progressive loss of structure or function of neurons, including death of neurons. Classical examples on this group of diseases are Alzheimer's disease and Parkinson's disease.

Dementia is a serious loss of cognitive ability in a previously unimpaired person, beyond what might be expected from normal aging. It has many causes. Alzheimer's disease is the most common cause of dementia in people at the age of 65 years and older.

Parkinsonism is a clinical syndrome characterized by diminished facial expression, stooped posture, slowness of voluntary movement, festinating gait, rigidity, and a "pill-rolling" tremor. This syndrome can be seen in a number of conditions that damage to dopaminergic neurons of the substantia nigra or to their projection to the striatum. Idiopathic Parkinson disease is the most common neurodegenerative disease associated with Parkinsonism; the diagnosis is made in patients with progressive Parkinsonism in the absence of a toxic or other known underlying etiology and if they show clinical response to L-DOPA.

Key principle to be discussed:

- Neurodegenerative diseases definition.
- The definition and etiology of dementia.
- Alzheimer disease:
    - Definition.
    - Clinical findings including age of onset and progression pattern.
    - Morphologic abnormalities including the gross brain changes, neurofibrillary tangles, and neuritic plaques deposition.
    - Parkinsonism: definition and etiology.
    - Parkinson disease: definition, epidemiology, pathogenesis and clinicopathological features.

Take home messages:

- Neurodegenerative diseases cause symptoms that depend on the pattern of involvement of the brain.
- Diseases that affect cerebral cortex primarily (e.g., Alzheimer disease) are more likely to cause cognitive change, alterations in personality and memory disturbance.
- Accumulation of the Aβ peptide, derived from amyloid precursor protein, is central to the pathogenesis of Alzheimer disease.
- Dementia is a non-specific illness syndrome that has many causes.
- Diseases that affect basal ganglia (e.g. Parkinson disease) have motor symptoms as prominent clinical features.
- Parkinson disease is caused by loss of dopaminergic neurons.
- Parkinsonism is not Parkinson's disease.
- Parkinson's disease is associated with abnormal aggregation of proteins, which may lead to loss of function or may trigger apoptosis. Familial forms are associated with mutations in the genes encoding these proteins.

Further reading:

- Vinay Kumar, Abul K. Abbas, Nelson Fausto, & Richard Mitchell , Robbins Basic Pathology, 8th edition

Keywords:

- Dementia, Alzheimer disease, neurofibrillary tangles, neuritic plaques, amyloid beta and phosphorylated tau. Lewy bodies, cholinergic cells, αlpha-synuclein, parkin, UCHL-1, Parkinson disease, Parkinsonism and substantia nigra.

Title of the lecture:	Pathogenesis and risk factors of cerebrovascular accidents
Lecturer's name	Dr. Hala Kfoury
Department	Pathology
Block	Neuropsychiatry block
Email address	halafoury@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Explain the concepts of brain "Hypoxia", "Ischemia" and "Infarction".
- Understand the pathogenesis of thrombotic and embolic stroke and be able to identify clinical risk factors.
- Identify the causes and consequences of subarachnoid and intracerebral hemorrhage.
- Build a list of the different causes that can lead to cerebrovascular accident.

Background:

Cerebrovascular disease is one of the leading causes of death and morbidity in Saudi Arabia. It is the most prevalent neurologic disorder in terms of both morbidity and mortality. The term cerebrovascular disease denotes any abnormality of the brain caused by a pathologic process involving blood vessels. The three basic processes are (1) thrombotic occlusion of vessels, (2) embolic occlusion of vessels, and (3) vascular rupture.

Key principle to be discussed:

- The concept of "stroke".
- Thrombotic and embolic stroke: incidence, significance of classification, causes and major clinicopathological features.
- Global Cerebral Ischemia, Border zone ("watershed") infarcts and focal Cerebral Ischemia: definition, causes and main gross and histopathological features.
- Intracerebral and subarachnoid hemorrhage: causes and major clinicopathological features.
- Vascular malformations: definition
- The main possible CNS cerebrovascular complications of hypertension including intracerebral hemorrhage, lacunar infarct, slit hemorrhages and hypertensive encephalopathy: definitions
- Vasculitis: possible causes.

Key principles to be covered by self-directed learning

- Hypoxia, Ischemia, and Infarction: revision of definitions.
- Risk factors of cerebrovascular accidents.
- Transient ischemic attacks: definition.

Take home messages:

- Stroke is the clinical term for a disease with acute onset of a neurologic deficit as the result of vascular lesions, either hemorrhage or loss of blood supply.
- Cerebral infarction follows loss of blood supply and can be widespread, focal or affect regions with the least robust vascular supply ("watershed" infarcts).
- Focal cerebral infarcts are most commonly embolic; if there is subsequent fragmentation of an embolism, a non-hemorrhagic infarct can become hemorrhagic.
- Primary intraparenchymal hemorrhages are typically due to either hypertension (most commonly in white matter, deep gray matter, or posterior fossa contents) or cerebral amyloid angiopathy.
- Spontaneous subarachnoid hemorrhage is usually caused by a structural vascular abnormality, such as an aneurysm or arteriovenous malformation.

Further reading:

- Vinay Kumar, Abul K. Abbas, Nelson Fausto, & Richard Mitchell , Robbins Basic Pathology, 8th Edition

Keywords:

- Stroke, infarction, watershed infarct, embolism, hemorrhage, hypertension, amyloid, aneurysm, arteriovenous malformation, vasculitis, lacunar infarct, slit hemorrhage, encephalopathy, hypoxia and ischemia

Title of the lecture:	Pathology and pathogenesis of multiple sclerosis
Lecturer's name	Dr. Hala Kfoury
Department	Pathology
Block	Neuropsychiatry block
Email address	halakfoury@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Appreciate the critical role of myelin in maintaining the integrity of the CNS system.
- Understand the pathogenesis and the clinic-pathological features of multiple sclerosis as the classical and the commonest example of CNS demyelinating diseases.

Background:

In general, diseases involving myelin are separated into two broad groups. Demyelinating diseases of the CNS are acquired conditions that are classically represented by multiple sclerosis. Other processes that can cause this type of disease include viral infection, drugs and other toxic agents. When myelin is not formed properly or has abnormal turnover kinetics, the resulting diseases are referred to as"dysmyelinating". These are associated with mutations affecting the proteins required for the formation of normal myelin or in mutations that affect the synthesis or degradation of myelin lipids.

Key principle to be discussed:

- Myelin function
- The differences between CNS and PNS Myelin
- Primary Demyelinating disease classification
- Multiple sclerosis: definition, epidemiology, pathogenesis and clinicopathological features; with special emphasis on CSF analysis findings, morphology and distribution of MS plaques.

Take home messages:

- In view of the critical role of myelin in nerve conduction; diseases of myelin can lead to widespread and severe neurologic deficits.
- Diseases of myelin can be grouped into demyelinating diseases (in which normal myelin is broken down for inappropriate reasons-often by inflammatory processes), and dysmyelinating diseases (which are metabolic disorders that include the leukodystrophies in which the underlying structure of the myelin is abnormal or its turnover is abnormal).
- Multiple sclerosis, an autoimmune demyelinating disease, is the most common disorder of myelin, affecting young adults often with a relapsing-remitting course and eventual progressive accumulation of neurologic deficits.

- Other less common forms of immune-mediated demyelination often follow infections and are more acute illnesses.

Further reading:

- Vinay Kumar, Abul K. Abbas, Nelson Fausto, & Richard Mitchell , Robbins Basic Pathology, 8th Edition.

Keywords:

- Multipsle sclerosis, demyelination, dysmyelination, leukodystrophy, plaques, T cell-mediated delayed type hypersensitivity, oligoclonal bands and optic neuritis.

Title of the lecture:	Pathology of brain tumors
Lecturer's name	Dr. Hala Kfoury
Department	Pathology
Block / week	Neuropsychiatry block
Email address	halakfoury@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Appreciate how the anatomy of the skull and the spinal column influences the prognosis of both benign and malignant primary CNS tumors.
- List the principal clinicopathological features of some of the main types of tumors that can arise within the central and the peripheral nervous systems.

Background:

- CNS tumors exhibit unique characteristics that make them different from tumors of the other body sites. Also childhood CNS tumors differ from those in adults, both in histologic subtypes and locations. Although histological classification and grading play a major rule in predicting the outcome a CNS tumor, the anatomic site of the neoplasm can have lethal consequences irrespective of histologic classification.

Key principle to be discussed:

- CNS tumors incidence and classification, with special consideration of the general differences between the pediatric and the adult population
- The unique characteristics that set CNS tumors apart from neoplastic processes elsewhere in the body
- The incidence, common clinical presentation, location, macroscopic appearances, microscopic features, pattern of spread and prognosis of the following neoplasms will be explained and discussed (within the context of the recommended textbook):
    - Astrocytic neoplasms: Pilocytic astrocytoma, diffuse astrocytoma, anaplastic astrocytoma and gliobastoma
    - Oligodendroglioma
    - Ependymoma
    - Medulloblastoma
    - Meningioma
    - Metastatic tumours
    - Peripheral nerve sheath tumours: schwannoma and neurofibroma

Key principles to be covered by self-directed learning:

- The inheritance pattern and the main features of:
    - Type 1 Neurofibromatosis
    - Type 2 Neurofibromatosis

Take home messages:

- Histologic distinction between benign and malignant lesions may be more subtle in comparison to other body systems.
- Even low-grade or benign tumors can have a poor clinical outcome depending on their location.
- The most aggressive and poorly differentiated glial tumor is glioblastoma; it contains anaplastic astrocytes and shows striking vascular abnormalities.
- Metastatic spread of brain tumors to other regions of the body is rare, but the brain is not comparably protected against spread of tumors from elsewhere.

Further reading:

- Vinay Kumar, Abul K. Abbas, Nelson Fausto, & Richard Mitchell , Robbins Basic Pathology, 8th Edition

Keywords:

- CNS tumors, astrocytoma, glioblastoma, oligodendroglioma, ependymoma, medulloblastoma, meningioma, metastatic tumours, peripheral nerve sheath tumours, schwannoma, neurofibroma and neurofibromatosis.

Title of the lecture:	Pathology of meningitis and its complication
Lecturer's name	Dr. Hala Kfoury
Department	Pathology
Block / week	Neuropsychiatry block
Email address	halakfoury@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Revise the spectrum of organisms that can cause meningitis.
- Explain the terms used in the description of CNS infections patterns.
- Understand the pathology of acute bacterial and tuberculous meningitis and the information that can be obtained from investigation of cerebrospinal fluid in suspected meningitis.

Background:

The brain and its coverings, as with all other parts of the body, can be affected by infections.
Damage to nervous tissue may be the consequence of direct injury of neurons or glia by the infectious agent or it may occur indirectly through the elaboration of microbial toxins, the destructive effects of the inflammatory response or the influence of immune-mediated mechanisms.

Key principle to be discussed:

Meningitis and meningoencephalitis: definition and a list of the possible infectious etiologies.

- Ports of entry of infection into the CNS.
- Pyogenic meningitis: etiology, clinic-pathological features and CSF findings.
- Viral (aseptic) meningitis: clinic-pathological features and CSF findings.
- Tuberculous Meningitis: clinic-pathological features and CSF findings.
- The definition and pathogenesis of epidural abscess, subdural empyema and brain abscess.

Take home messages:

- Different pathogens may use distinct routes to reach the brain, and will cause different patterns of disease.
- Bacterial infections may cause meningitis, cerebral abscesses or a chronic meningoencephalitis. Viral infections can cause meningitis or meningoencephalitis.
- Lumbar puncture plays an important role in the diagnostic process of some CNS infections.

Further reading:

- Vinay Kumar, Abul K. Abbas, Nelson Fausto, & Richard Mitchell , Robbins Basic Pathology, 8th Edition

Keywords:

- CNS infection, virus, bacteria, meningitis, cerebral abscesses, chronic meningoencephalitis, viral meningitis, epidural abscess, subdural empyema and tuberculosis.

Title of the lecture:	Pathology (Practical sessions)
Lecturer's name	Dr. Shaesta Zaidi / Dr. Hala Kfoury
Department	Pathology
Block	Neuropsychiatry block
Email address	snz24@yahoo.com / halakfoury@hotmail.com

Objectives:

Upon completion of this lecture, students should be able to:

- Recognize, describe and understand the morphological appearance (both macroscopic and microscopic) of some of the common diseases and disorders of the CNS.

Format

- A short mandatory power point presentation in which pictures showing both gross and microscopic sections of CNS diseases are shown and explained to the students using a computer linked to a projector.

Contents:

Study of the macroscopic and microscopic features through case studies of the following CNS diseases:

- Meningioma.
- Glioblastoma multiforme
- Multiple sclerosis.
- Schwannoma.
- Hydrocephalus
- Meningitis.
- Brain abscess
- Brain hemorrhage.
- Alzheimer's disease.

Key principles to be covered by self-directed learning:

The students should study the macroscopic and microscopic features of the following CNS diseases;

- Tuberculous meningitis.
- Parkinson's disease

Reference:

- Robbin's Basic Pathology, 8th edition by Vinay Kumar, et al.

Title of the lecture: Radiology of cerebral hemisphere	
Lecturer's name	Dr. Faten Al-Mohaideb
Department	Radiology
Block	Neuropsychiatry Block
Email address	fatenalmohideb@gmail.com

Objectives of the lecture:

- Understand the imaging planes of the brain
- Identify the anatomical structures of the cerebral hemispheres on radiological images on different planes
- Identify the location of different cerebral functions (motor/sensory/language) on radiological images on different planes
- Select the best plane for a particular cerebral anatomical structure

Background:

Gross anatomy of the cerebral hemispheres
Functional anatomy of the cerebral hemispheres

Main concepts in the lecture:

The lecture introduces the concept of cross-sectional imaging and how this helps to explore brain anatomy. The anatomical structures of the cerebral hemispheres are shown on axial, coronal, and sagittal planes then example of major anatomical landmarks are show on the three planes at the same time to give the student a sense of the 3-D imagination of cerebral anatomy. The functional anatomy is stressed all the way, including short explanation of the cerebral hemisphere blood supply.

Take home messages:

Detailed anatomical structures of cerebral hemispheres are identifiable on imaging.
Certain imaging planes show certain brain structures better.
Understanding brain anatomy in space (3-D imagination) is essential for utilization of brain imaging.

Further reading:

- Wayne State University, Radiologic Anatomy. Link: http://www.med.wayne.edu/diagRadiology/Anatomy_Modules/brain/brain.html
- W-radiology. Link: http://w-radiology.com/index.html
- Radiology Assistant. Link: http://www.radiologyassistant.nl/en/p48f4c4ccd9682/brain-anatomy.html

Title of the lecture: Medication affecting the balance system	
Lecturer's name	Dr. Aliah Alshanawani
Department	Pharmacology
Block	Neuropsychiatry Block
Email address	aalshanawani@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the student should:

- Recognize causes and symptoms of balance disorders.
- Identify the transmitters involved in vestibular transmission
- Segregate classes of drugs used in the management protocols to control or prevent vertigo
- Identify drugs that can precipitate vertigo.

Title of the lecture: Drugs used in epilepsy 1 & 2	
Lecturer's name	Prof. Yieldez Bassioni
Department	Pharmacology
Block	Neuropsychiatry Block
Email address	Yieldez@yahoo.com

Objectives of the lecture:
At the end of this lecture the student should:

- Describe types of epilepsy
- List the antiepileptic drugs
- Describe briefly the mechanism of action of and rationale of use of antiepileptic drugs.
- Enumerate the clinical uses of each drug
- Describe treatment of status epilepticus

Title of the lecture: Drugs used in anxiety & panic disorder	
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block	Neuropsychiatry Block
Email address	hananhaggar@yahoo.com

Objectives of the lecture:
At the end of this lecture the student should:

- Define different types of anxiety disorders
- Classify types of drugs used for treatment of anxiety
- Recognize the different characteristics of anti anxiety drugs

Title of the lecture: Drugs used in headache and migraine	
Lecturer's name	Dr. Aliah Alshanawani
Department	Pharmacology
Block	Neuropsychiatry block
Email address	aalshanawani@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the student should:

- Differentiate between types of headache regarding their symptoms, signs and pathophysiology.
- Recognize drugs used to prevent migraine
- Identify drugs used to rescue and abort migraine
- Elaborate on the pharmacokinetics, dynamic and toxic profile of some of these drugs.

Title of the lecture: Drugs used in management of pain	
Lecturer's name	Dr. Aliah Alshanawani
Department	Pharmacology
Block	Neuropsychiatry Block
Email address	aalshanawani@ksu.edu.sa

Objectives of the lecture:
At the end of this lecture the student should:

- Revise how pain is perceived and modulated, emphasizing on neurotransmitters, receptors, channels involved
- Classify drugs used in management of pain
- Expand on pharmacology of opiates, patterns of classification, mechanism of action,indications, ADR,…etc.
  detailing on morphine as an example.
- Compare in brief actions and indications of other opiate agonists and antagonists.

Title of the lecture: Drugs used in parkinsonism	
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block	Neuropsychiatry Block
Email address	hananhhagar@yahoo.com

Objectives of the lecture:

At the end of this lecture the student should:

- Recognize the symptoms and pathophysiology of parkinsonism
- Understand the pharmacology of drugs used for treatment of parkinsonism

Title of the lecture: Drugs used in schizophrenia	
Lecturer's name	Prof. Yieldez Bassioni
Department	Pharmacology
Block	Neuropsychiatry Block
Email address	Yieldez@yahoo.com

Objectives of the lecture:
At the end of this lecture the student should:

- List the classification of antipsychotic drugs used in schizophrenia.
- Describe briefly the mechanism of antipsychotic action of these drugs.
- Describe the pharmacological actions of antipsychotic drugs.
- Relate between pharmacological actions & adverse effects of antipsychotic drugs.
- Enumerate the clinical uses of antipsychotic drugs.
- Describe the advantages of atypical antipsychotic drugs over typical drugs.

Title of the lecture:	**Alcohol and the Brain**
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block	Neuropsychiatry block
Email address	hananhhagar@yahoo.com

Objectives:

- Describe the pharmacological actions of alcohol
- Describe the pharmacokinetic profile of alcohol
- Describe the development of intoxication symptoms of alcohol
- Describe how alcohol affects various neurotransmitters in the brain.
- Identify various toxicity of alcohols at different organs level
- Describe the addictive nature of alcohol and its mechanism
- Identify alcohol withdrawal symptoms and their management.
- Identify clinically relevant drug interactions with alcohol
- Hazards of alcohol in pregnancy

Background:

- Recognize the primary subdivisions of the brain
- Discuss the compartmentalization of functions in the brain
- Role of various neurotransmitters in the brain
- Normal autonomic physiology
- General pharmacology of drug actions

Main concepts in the lecture:

- Alcohol affects various neurotransmitters in the brain that are repsonsbile for its deleterious effects in our body
- Alcohol is toxic to the brain, cardiovascular , GIT and rebal system.
- Chronic alcohol consumption is very dangerous as it can lead to tolerance.
- Fetal alcohol syndrome is a no-reversible damage to the fetus and baby born to alcoholic mothers.
- Alcohol can lead to liver cirrhosis,
- Alcohol can cause serious toxicity with various drugs if taken concurrently such as sedating drug and drugs that are metabolized by the liver.
- Alchol withdrawal symptoms are serious but manageable conditions.

Conclusion:

- Acute or chronic use of alcohol can produce serious medical problems; It can produce serious impairment of the cardiovascular and neurological complications. Special precaution to be taken while prescribing drugs for alcoholic patients

Take home messages:

- Acute or chronic use of alcohol leads to serious medical complications. It can cause irreversible fetal Alcohol syndrome if used during pregnancy. The pharmacological mechanism of the hazards of alcohol well established in the medical field.

Further reading:

Basic and clinical Pharmacology 12th ed by B.G. Katzung ……Se E Section V: DRUGS THAT ACT IN THECENTRAL NERVOUS SYSTEM ; The Alcohols

Title of the lecture:	**Pharmacology of drugs acting on the eye**
Lecturer's name	Prof. Hanan Hagar
Department	Pharmacology
Block	Neuropsychiatry block
Email address	hananhhagar@yahoo.com

Objectives:

- Outline common routes of administration of drugs to the eye
- Discuss the pharmacokinetics of drugs applied topically to the eye
- Classify drugs used for treatment of disorders of the eye
- Elaborate on autonomic, anti-inflammatory drugs & drugs used for glaucoma
- Hint on ocular toxicity of some drugs

Background:

- The following are challenges to drug delivery to the eye:-
- Various barriers prevent high concentration of drugs passage from the blood stream into the eye e.g. blood- retinal, blood- aqueous, blood- vitreous.
- Most agents injected into the vitreous are cleared rapidly and therefore are ineffective
- Subconjunctival and intravitreal injections carry a risk of infection

Main concepts in the lecture:

Drugs are administered to the eye either topically or systemically. The advantages and disadvantages of each route is discussed. Then the absorption, distribution and metabolism of drugs of drugs applied topically to the eye are discussed. Classes of drugs studied are autonomic, antiglaucoma, anti-inflammatory drugs. Mechanism of action, pharmacodynamics effects, ADRS and clinical uses of these are discussed. Finally ocular toxicity of drugs used for other indications are hinted upon.

Conclusion:

Cholinergic drugs are used for treatment of glaucoma, to counteract the action of mydriatics and to break adhesion. While cholinergic antagonists are used to prevent adhesion in uveitis & iritis, for funduscopic examination of the eye and in measurement of refractive error. Alpha agonists are used as decongestant in minor allergic hyperemia of eye. Beta blockers are used for treatment of open angle glaucoma.

Take home messages:

- Some drugs applied systemically may induce toxic effects to the eye e.g. Chloroquine, ethambutol, sildenafil, digitalis and steroids.
- Topical corticosteroids are used postoperatively, anterior uveitis, severe allergic conjunctivitis, scleritis, prevention and suppression of corneal graft rejection

- Open angle glaucoma is treated with beta- blockers,α_2 agonists,Carbonic anhydrase inhibitors prostaglandins, adrenergic agonists (non-specific), and parasympathomimetics
- Acute angle closure glaucoma is an emergency situation that require treatment before surgery (Iridectomy). Drugs used include oral acetazolamide, topical cholinomimetics, dehydrating agents and analgesics.

Further reading:

- Basic and clinical pharmacology By B. G. Katung
- Lippincott,s pharmacology

Title of the lecture:	**Drugs used in meningitis**
Lecturer's name	Prof. Yieldez Bassioni
Department	Pharmacology
Block / week	Neuropsychiatry block
Email address	Yieldez@yahoo.com

Objectives:

- Describe briefly common types of meningitis
- Describe the principles of treatment
- List the name of antibiotics used for treatment of meningitis
- Describe the mechanism of action & adverse effects of the individual drugs

Title of the lecture:	**Pharmacology of Neurotransmitter**
Lecturer's name	Prof. Yieldez Bassioni
Department	Pharmacology
Block	Neuropsychiatry block
Email address	Yieldez@yahoo.com

Objectives:

At the end of lecture students should be able to know:

- To understand the role of neurotransmitters in the etiology and treatment of CNS diseases
- To define neurotransmitters.
- To understand neuronal circuits system for neurotranmistters.
- To compare the location, receptor subtypes, effect of release, and general physiological and pharmacological roles of the neurotransmitter systems and the dysregulation of their level
- Basic classes of neurotransmitters.
    - Cholinergic (ACh)
    - Biogenic amines (norepinephrine, epinephrine, dopamine, glutamate, and serotonin)
    - Inhibitory amino acids (GABA, glycine)
    - Opioid peptides

Title of the lecture: Depression	
Lecturer's name	Dr. Noor Modihesh
Department	Psychiatry
Block	Neuropsychiatry block
Email address	nalmodihesh@ksu.edu.sa

Objectives of the lecture:

- To be able to identify the major depressive disorder including clinical criteria for diagnosis
- To be aware of other depressive disorder that are related to primary and secondary psychiatric illness
- To identify the different etiologies for depressive disorder
- To have the basic skills for diagnosing and treating this disorder

Title of the lecture: Introduction to neuropsychiatric disorders	
Lecturer's name	Dr. Noor Al Mohidesh
Department	Psychiatry
Block	Neuropsychiatry block
Email address	nalmohidesh@ksu.edu.sa

Objectives of the lecture:

- To know the basic types of neurocognitive disorders
- To understand in concise manner their etiology
- To know the baseline management of neurocognitive disorders

Title of the lecture: Schizophrenia	
Lecturer's name	Dr. Noor Al Mohidesh
Department	Psychiatry
Block	Neuropsychiatry
Email address	Nalmohidesh@ksu.edu.sa

Objectives of the lecture:

- Appreciate that schizophrenia is a serious brain illness that needs early intervention and comprehensive management approach
- Enhance his knowledge of schizophrenia including epidemiology, etiology, diagnosis and management.

Academic Support Team

The College of Medicine and the Department of Medical Education are working on ensuring that our students receive optimal support to their learning. The list of academics shown below represents the departments involved in the teaching and learning of this block. If a student needs help in their teaching and learning they might consult one academic from the list. She might email them and arrange a time to see them if needed, otherwise email might be of help.

Name	Department	Mobile	Email
Prof. Mona Soliman	Medical Education	0505468581	Msoliman1@.ksu.edu.sa
Prof. Hamza Abdulghani	Medical Education	0505442859	hamzaabg@gmail.com
Prof. Samy Azer	Medical Education	0542307075	sazer@ksu.edu.sa
Prof. Sultan Meo	Physiology	0557640012	sultanmeo@hotmail.com
Dr. Amro Al Habib	Surgery	0506661582	amro.alhabib@gmail.com
Prof. Ahmed Fathalla	Anatomy	0501562983	ahmedfathala@gmail.com
Prof. Layla Ayadhi	Physiology	0504295974	lyayadhi@ksu.edu.sa
Dr. Sumbul Fatma	Pathology	0598245851	sumbulfatma@gmail.com
Dr. Hisham Al Khalidi	Pathology	0533408611	drhishamnaseej@hotmail.com
Dr. Ishfaq Al Bukhari	Pharmacology	0534591602	ishfaqbukhari@yahoo.com;
Dr. Fawzia Al Otaibi	Pathology	0553223309	ofawzia@ksu.edu.sa

Schedule- Female Group

WEEK 1 – NEUROPSYCHIATRY BLOCK (Female Group)				
Week (1) Starting: 17/09/2017 to 21/09/2017 **ANS, Spinal Cord & Peripheral Nerves**				
CHAIR PERSON: Prof. Sultan Ayoub Meo				
Sunday **17 September 2017**	**Monday** **18 September 2017**	**Tuesday** **19 September 2017**	**Wednesday** **20 September 2017**	**Thursday** **21 September 2017**
8:00 - 12:00 pm Student activity	8:00-9:00am Organization of the Neuropsychiatry System **(Anatomy)** **Dr. Sanaa Al Shaarawi**	8:00 - 9:00 am Sympathetic & parasympathetic nervous system **(Physiology)** Dr. Fawzia Al Roug	8:00 - 9:00 am **Clinical Skills** Dermatomes and myotomes and examination of the sensory system **Anatomy** **(Group F1)**	8:00-10:00am **Practical** Spinal cord **(Histology)** **F2** **Dr. Jamilah El Medany** **All Staff**
	9:00– 10:00am Physiology of synapses and receptors **(Physiology)** Prof. Laila Ayadhi	9:00– 10:00am Physiology of motor tracts **(Physiology)** Prof. Faten Zakaria	9:00 - 10:00 am **Clinical Skills** Dermatomes and myotomes and examination of the sensory system **Anatomy** **(Group F2)**	
	10:00 - 11:00am Anatomy of the spinal cord **(Anatomy)** **Dr. Jamilah El-Medany**	10:00 - 11:00am Physiology of pain **(Physiology)** Dr. Hayam Gad	10:00 - 11:00am Stretch reflex & Tendon jerks **(Physiology)** Prof. Faten Zakaria	10:00 - 11:00am Sensory tracts **(Anatomy)** **Dr. Jamilah El Medany**
	11:00 - 12:00pm Normal cells of the CNS **(Histology)** **Dr. Raeesa Abdultawab**	11:00 - 12:00pm Spinal cord Functions & reflexes **(Physiology)** Prof. Faten Zakaria	11:00 - 12:00pm Embryological development of the spinal cord and vertebral column **(Anatomy)** **Dr. Sanaa Al Shaarawi**	11:00 - 12:00pm **Self-Directed** **Learning**
Lunch **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**
1:00 - 2:00 pm Introduction to Neuropsychiatry System Prof. Laila Ayadhi 2:00-3:00pm Brachial plexus and lumbosacral plexus **(Anatomy)** **Dr. Sanaa Al Shaarawi**	1:00 - 3:00pm **Practical** Skull **(Anatomy)** **F1** **Dr. Jamilah El Medany** **All Staff**	1:00 - 3:00 pm **Practical** Spinal cord **(Histology)** **F1** **Dr. Jamilah El Medany** **All Staff**	1:00 - 3:00 pm **Practical** Brachial plexus and lumbosacral Plexus **(Anatomy)** **F1** **Dr. Jamilah El Medany** **All Staff**	1:00 - 3:00 pm Salam

WEEK 2 – NEUROPSYCHIATRY BLOCK (Female Group)

Week (2) Starting: 24/09/2017 to 28/09/2017

Brainstem & Related Cranial Nerves

CHAIR PERSON : Prof. Sultan Ayoub Meo

Sunday 24 September 2017	Monday 25 September 2017	Tuesday 26 September 2017	Wednesday 27 September 2017	Thursday 28 September 2017
Official Holiday for the National Day	8:00-10:00 am Small Group Learning(PBL) **Case 1 Part 1**	8:00 - 9:00 am Internal structures of the brainstem **(Anatomy)** **Dr. Sanaa Al Shaarawi**	8:00 - 9:00 am **(Clinical Skills)** Examination of cranial nerves **(Introduction to Clinical Medicine)** **(Group F1)**	8:00 - 10:00 am Small Group Learning(PBL) **Case 1 Part 2**
		9:00 - 10:00 am Biochemistry of myelin (Biochemistry) Dr. Sumbul Fatma	9:00 - 10:00 am **Clinical Skills)** Examination of cranial nerves **(Introduction to Clinical Medicine)** **(Group F2)**	
	10:00 - 11:00 am Anatomy of brainstem **(Anatomy)** **Dr. Sanaa Al Shaarawi**	10:00 - 11:00 am Anatomy of CN XI & XII **(Anatomy)** **Dr. Jamilah El Medany**	10:00 - 11:00 am Radiology of brain stem and cerebellum (Radiology) Dr. Faten AlMahaideb	10:00 - 11:00 am Pathology of brain tumors –II (Pathology) Dr. Hala Kfoury
	11:00- 12:00 pm Physiology of the brainstem (Physiology) Dr. Hayam Gad	11:00 - 12:00 pm Cellular injury of Nervous System (Pathology) Dr. Hala Kfoury	11:00- 12:00 pm Pathology of brain tumors –I (Pathology) Dr. Hala Kfoury	11:00 – 12:00 pm Pathology and pathogenesis of multiple sclerosis (Pathology) Dr. Hala Kfoury
	Lunch **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**
	1:00 -2:00 pm Anatomy of CN IX & X **(Anatomy)** **Dr. Jamilah El Medany**	1:00-3:00 pm **Practical** Neuropathology I **(Pathology)** **Dr. Shaesta/ Dr. Hala**	1:00 - 2:00 pm Spinal cord (Radiology) Dr. Faten AlMohaideb	1:00 -3:00 pm Salam
	2:00-3:00 pm Nerve supply of the face (Cranial nerves V and VII) **(Anatomy)** **Dr. Sanaa Al Shaarawi**		2:00-3:00 pm Physiology of sleep (Physiology) Dr. Nervana Bayoumi	

WEEK 3 – NEUROPSYCHIATRY BLOCK (Female Group)

Week (3) Starting: 01/10/2017 to 05/10/2017

Hearing & Special Senses

CHAIR PERSON : Prof. Sultan Ayoub Meo

Sunday 01 October 2017	Monday 02 October 2017	Tuesday 03 October 2017	Wednesday 04 October 2017	Thursday 05 October 2017
8:00-9:00am Anatomy of the ear **(Anatomy)** **Dr. Jamilah El Medany**	8:00 - 10:00 am Small Group Learning(PBL) **Case 2 Part 1**	8:00 - 10:00am **Practical** Audiometry **(Physiology)** **Dr. Ola Mawlena** **All Staff**	8:00 - 11:00 am **(Clinical Skills)** Ophthalmoscope examination and Examination of the motor system **(Introduction to Clinical Medicine)**	8:00 - 10:00 am Small Group Learning(PBL) **Case 2 Part 2**
9:00-10:00am Anatomy of the nose and olfactory nerve **(Anatomy)** **Dr Jamilah El Medany**	10:00 – 12:00 am **Practical** EMG/nerve conduction (Physiology) Dr. Ola Mawlena All Staff	10:00 – 11:00 am Anatomy of the eye globe and Cranial nerve II **(Anatomy)** **Dr. Raeesa Mohamed**		10:00 - 11:00 am Physiology of taste and smell (Physiology) Prof. Laila Al Ayadhi
11:00-12:00nn **Self-Directed Learning**		11:00- 12:00 nm Photo transduction in Light & the Dark (Physiology) Prof. Faten Zakaria	11:00- 12:00 nm Cranial nerves: III, IV, VI **(Anatomy)** **Dr. Sanaa Al Shaarawi**	11:00- 12:00 nm Physiology of the eye & refraction (Physiology) Prof. Faten Zakaria
Lunch **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**
1:00-3:00 pm **Practical** Brainstem & CNS **(Anatomy)** **F1** **Dr. Jamilah El Medany** **All Staff**	1:00 -2:00 pm Vision, Accommodation & the light pathways and effects of lesions (Physiology) Prof. Faten Zakaria 2:00-3:00 pm Mechanism of hearing (Physiology) Prof. Laila Al Ayadhi	1:00 -3:00 pm **Practical** Brainstem & CNS **(Anatomy)** **F2** **Dr. Jamilah El Medany** **All Staff**	1:00 -2:00 pm Physiology of color vision (Physiology) Prof. Faten Zakaria 2:00-3:00 pm Cranial nerve VIII **(Anatomy)** **Dr. Sanaa Al Shaarawi**	1:00 -3:00 pm Salam

WEEK 4 – NEUROPSYCHIATRY BLOCK (Female Group)

Week (4) Starting: 08/10/2017 to 12/10/2017

The Balance System

CHAIR PERSON : Prof. Sultan Ayoub Meo

Sunday 08 October 2017	Monday 09 October 2017	Tuesday 10 October 2017	Wednesday 11 October 2017	Thursday 12 October 2017
8:00 - 09:00 am Physiology of the proprioceptors in Balance (Physiology) Dr. Fawzia Al Roug	8:00 - 9:00 am Self-Directed Learning	8:00 - 10:00 am Overview and key elements of professionalism (1) & Accountability, integrity and altruism (2) Prof. Hanan Habib	8:00 - 9:00 am Self-Directed Learning	8:00 - 9:00 am Vitamin B6 and B12 (Biochemistry) Dr. Sumbul Fatma
9:00 – 10:00 am Self-Directed Learning	9:00 – 10:00 am Pharmacology of drugs acting on the eye (Pharmacology) Prof. Hanan Hagar		9:00 – 10:00am Self-Directed Learning	9:00-10:00am Self-Directed Learning
10:00 - 11:00 am Anatomy of the cerebellum and the relevant connections (Anatomy) Dr. Sanaa Al Sharawi	10:00-11:00 am Physiology of postural reflexes (Physiology) Prof. Faten Zakaria	10:00-11:00am Microbiology of middle ear infection (Microbiology) Prof. Hanan Habib	10:00- 12:00pm Practical Color Vision, light and accommodation reflex (Physiology) Dr. Ola Mawlena All Staff	10:00-11:00 am Physiology of consciousness (Physiology) Prof. Laila Ayadhi
11:00- 12:00 pm Physiology of inner ear in balanced (Physiology) Prof. Laila Ayadhi	11:00-12:00 pm Physiology of the cerebellum (Physiology) Dr. Aida Korish	11:00-12:00 pm Vitamin A (Biochemistry) Dr. Sumbul Fatma		11:00-12:00nn Medication affecting the balance system (Pharmacology) Dr. Aliah Alshanawani
Lunch 12:00- 1:00 pm	Lunch 12:00- 1:00 pm	Lunch 12:00 – 1:00pm	Lunch 12:00- 1:00 pm	Lunch 12:00- 1:00 pm
1:00 - 3:00 pm Practical Brachial plexus and lumbosacral Plexus (Anatomy) F2 Dr. Jamilah El Medany All Staff	1:00-2:00 pm Self-Directed Learning 2:00-3:00 pm Self-Directed Learning	1:00 - 3:00 pm Practical Skull (Anatomy) F2 Dr. Jamilah El Medany All Staff	1:00 - 3:00 pm Historical standards of professionalism behavior and Islamic rules of medical professionalism (Professionalism) Prof. Lulu Al-Nuaim	1:00 - 3:00 pm Salam

WEEK 5 – NEUROPSYCHIATRY BLOCK (Female Group)

Week (5) Starting: 15/10/2017 to 19/10/2017

Cerebral Hemisphere & Blood Circulation

CHAIR PERSON : Prof. Sultan Ayoub Meo

Sunday 15 October 2017	Monday 16 October 2017	Tuesday 17 October 2017	Wednesday 18 October 2017	Thursday 19 October 2017
8:00 - 10:00 am **MIDBLOCK Examination** **(Not yet confirm)**	8:00 - 10:00 am Small Group Learning(PBL) **Case 3 Part 1**	8:00 - 9:00 am Pathogenesis of cerebral infarction at cellular and molecular levels **(Biochemistry)** Dr. Sumbul Fatma	8:00 -9:00 am **(Clinical Skills)** History taking from a patient with neuropsychological problem **(Introduction to Clinical Medicine)** **(Group F1)**	8:00 - 10:00 am Small Group Learning(PBL) **Case 3 Part 2**
		9:00 – 11:00 am Embryology of the cerebral hemisphere and cerebellum **(Anatomy)** **Dr. Sanaa Al Sharawi**	9:00 – 10:00 am **(Clinical Skills)** History taking from a patient with neuropsychological problem **(Introduction to Clinical Medicine)** **(Group F2)**	
10:00 - 11:00 am Anatomy of the cerebral hemispheres **(Anatomy)** **Dr. Jamilah El Medany**	10:00 - 11:00 am Physiology of speech (Physiology) Prof. Laila Al Aadhi	10:00 - 11:00 am Pathogenesis and risk factors of cerebrovascular accidents I (Pathology) Dr. Hala Kfoury	10:00 - 11:00 am Upper and lower neuron lesions (Physiology) Dr. Fawzia Alroug	10:00 - 12:00 nn **Practical** Radiology and Anatomy of the cerebral hemisphere **F1** **(Anatomy & Radiology)** **All** **Dr. Jamilah El Medany & Dr. Faten AlMohaideb**
11:00 - 12:00 pm ***Feedback on Midterm lectures and examination** **Dr. Jamilah** **Prof. Faten Zakaria**	11:00 - 12:00 pm Cerebral blood circulation {arteries and veins} **(Anatomy)** **Dr. Jamilah El Medany**	11:00- 12:00 pm Spasticity and increased muscle tone **(Physiology)** Prof. Faten Zakaria	11:00 - 12:00 pm Radiology of cerebral hemisphere (Radiology) Dr. Faten AlMohaideb	
Lunch **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**
1:00 - 2:00 pm Functions of the cerebral hemisphere (Physiology) Dr. Fawzia A Rouq	1:00 - 3:00 pm Effective communication skills; impact on being an effective player (1) **(Professionalism)**	1:00 - 2:00 pm Pathogenesis and risk factors of cerebrovascular accidents II (Pathology) Dr. Hala Kfoury	1:00 - 3:00 pm **Practical** Radiology and Anatomy of the cerebral hemisphere **F2** **(Anatomy & Radiology)** **All** **Dr. Jamilah El Medany &** **Dr. Faten AlMohaideb**	1:00 -3:00 pm SALAM
2:00 - 3:00 pm Autoregulation of cerebral blood flow (Physiology) Prof. Faten Zakaria	**Prof. Lulu Al- Nuaim**	2:00 - 3:00 pm Ageing and changes in the brain (Physiology) Dr. Aida Korish		

WEEK 6 – NEUROPSYCHIATRY BLOCK (Female Group)

Week (6) Starting: 22/10/2017 to 26/10/2017

Neuropsychiatric Disorders and Basal Ganglia

CHAIR PERSON : Prof. Sultan Ayoub Meo

Sunday 22 October 2017	Monday 23 October 2017	Tuesday 24 October 2017	Wednesday 25 October 2017	Thursday 26 October 2017
8:00 - 9:00 am Physiology of brain transmitters (Physiology) Prof. Laila Ayadhi	8:00 – 10:00am Small Group Learning(PBL) Case 4 Part 1	8:00 - 9:00am Drugs used in parkinsonism (Pharmacology) Prof. Hanan Hagar	8:00 - 9:00am Introduction to degenerative brain diseases (Pathology) Dr. Hala Kfoury	8:00 - 10:00am Small Group Learning(PBL) Case 4 Part 2
9:00-10:00 am Physiology of basal ganglia and regulatory mechanisms (Physiology) Dr. Fawzia Al Roug		9:00-10:00am Depression (Psychiatry) Dr. Noor Modihesh	9:00 – 10:00am Introduction to neuropsychiatric disorders (Psychiatry) Dr. Abdulrahman Al-Wahibi	
10:00 – 11:00 am Anatomy of the basal ganglia and connections (Anatomy) Dr. Jamilah El Medany	10:00 - 11:00am Pain modulation (Physiology) Dr Hayam Gad	10:00 - 11:00am Drugs used in management of pain (Pharmacology) Dr. Aliah Alshanawani	10:00 - 11:00 am Drugs used in anxiety and panic disorders (Pharmacology) Prof. Hanan Hagar	10:00 – 11:00am Drugs used in depression-old (Pharmacology) Prof. Yieldez Bassioni
11:00- 12:00pm Anatomy of the limbic system and thalamus (Anatomy) Dr. Sanaa Al Sharawi	11:00- 12:00pm Alcohol and the brain (Pharmacology) Prof. Hanan Hagar	11:00- 12:00pm Schizophrenia (Psychiatry) Dr. Noor Al Modihesh	11:00 - 12:00pm Biochemistry of Alzheimer's disease (Biochemistry) Dr. Sumbul Fatma	11:00 - 12:00pm Drugs used in schizophrenia (Pharmacology) Prof. Yieldez Bassioni
Lunch 12:00- 1:00 pm	Lunch 12:00- 1:00 pm	Lunch 12:00- 1:00 pm	Lunch 12:00- 1:00 pm	Lunch 12:00- 1:00 pm
1:00 - 2:00 pm Self-Directed Learning	1:00 -3:00pm Effective communication skills; impact on being an effective player (2) (Professionalism) Prof. Lulu Al-Nuaim	1:00-2:00 pm Pharmacology of neurotransmitter (Pharmacology) Prof. Yieldez Bassioni	1:00 - 2:00pm Pathophysiology of epilepsy (Physiology) Prof. Laila Ayadhi	1:00 - 3:00pm SALAM
2:00-3:00pm Self-Directed Learning		2:00-3:00pm Self-Directed Learning	2:00-3:00pm Self-Directed Learning	

WEEK 7 – NEUROPSYCHIATRY BLOCK (Female Group)

Week (7) Starting 29/10/2017 to 02/11/2017

Cerebral & Cerebrospinal Infections

CHAIR PERSON : Prof. Sultan Ayoub Meo

Sunday 29 October 2017	Monday 30 October 2017	Tuesday 31 October 2017	Wednesday 01 November 2017	Thursday 02 November 2017
8:00 - 9:00am Anatomy of the meninges, CNS cavities, and CSF circulation **(Anatomy)** **Dr. Sanaa Al Shaarawi**	8:00 – 10:00 am Small Group Learning(PBL) **Case 5 Part 1**	8:00 – 9:00 am **Self-Directed Learning**	8:00 - 10:00am **OSCE**	8:00 - 10:00am Small Group Learning(PBL) **Case 5 Part 2**
9:00 – 10:00am Biochemistry of CSF **(Biochemistry)** **Dr. Rana Hasanato**		9:00 – 10:00 am Congenital malformations and hydrocephalus **(Pathology)** **Dr. Hala Kfoury**		
10:00 – 11:00am Cerebral TB and other chronic cerebral infections **(Microbiology)** **Dr. Fawzia Al Otaibi**	10:00 - 11:00am Viral infections of the CNS **(Microbiology)** **Dr. Malak El Hazmi**	10:00 - 11:00am Drugs used in depression-new **(Pharmacology)** **Prof. Yieldez Bassioni**	10:00 - 11:00am **Self-Directed Learning**	10:00 - 11:00am Drugs used in epilepsy-I (Pharmacology) **Prof. Yieldez Bassioni**
11:00 - 12:00pm Drugs used in meningitis **(Pharmacology)** **Prof. Yieldez Bassioni**	11:00 - 12:00pm Microbiology of acute pyogenic meningitis **(Microbiology)** **Prof. Hanan Habib**	11:00 - 12:00pm Drugs used in headache and migraine **(Pharmacology)** **Dr. Aliah Alshanawani**	11:00 - 12:00pm Pathology of meningitis and its complications **(Pathology)** **Dr. Hala Kfoury**	11:00 - 12:00pm Drugs used in epilepsy-II **(Pharmacology)** **Prof. Yieldez Bassioni**
Lunch **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**	**Lunch** **12:00- 1:00 pm**
1:00 - 2:00 pm Fungal infections of the CNS **(Microbiology)** **Dr. Maha Al Muhaizea**	1:00 - 3:00 pm **Professional and Unprofessional Behaviors (Professionalism)** **Dr. Hala Kfoury**	1:00 - 3:00 pm **(Integrated Practical)** Biochemistry and microbiology of CSF **(Biochemistry & Microbiology)** **All Staff** **Dr Fawzia / Dr. Sumbul**	1:00 - 3:00 pm **Practical** Neuropathology- II **(Pathology)** **Dr. Shaesta / Dr. Hala**	1:00 -3:00pm **Salam**
2:00-3:00 pm **Self-Directed Learning**				

*** Week 8: Consolidation week; from 05 to 09 November 2017**
*** Week 9: Examination week; from 12 to 16 November 2017**

Plagiarism

Plagiarism is a voluntary act to copy sentences and give a misleading impression that the text is created by the person whose name appears on the work. For example an assignment submitted as part of the requirements of assessment of a subject.

Plagiarism may include plagiarism of ideas and or plagiarism of text (sentences or paragraphs). It also may include the use of diagrams, tables, images, cartoons etc without acknowledging the original creator of the work.

The act of copy-and-paste writings even if the aim is to produce a good assignment with well-structured English statements is unethical and when discovered could cause serious consequences including disciplinary action. Students need to construct statements in their own words and refer to the correct references related to what they have written and included in their assignment/work. Giving credit and acknowledgement to the original authors/creators are valued by the academic community as it reflects an ethical and professional attitude.

Why is plagiarism wrong?

Universities, higher education institutes and scientific communities consider plagiarism as a major problem for a number of reasons:

- It is an act of stealing ideas and the work of original authors/creators.
- It does not represent acceptable professional, ethical or scientific behaviour.
- It raises doubts about the credibility of the person/group of people who committed such act.

How can teachers/college discover an act of plagiarism?

There are a number of software programs such as iThentcate and many others available to detect the act of plagiarism. Some of these programs are available free online.

These tools can locate the places and sentences where students have copied and the original resource (articles, manuscripts, papers, books, websites) for such statements/paragraphs or images.

What are the consequences of plagiarism?

Students who commit plagiarism will be exposed to disciplinary action including the failure of the subject concerned provided that such act has been confirmed with evidence.

Assessment of Students in the Block

In order to pass the block, you must obtain a minimum final block grade of D (the grading guide attached as appendix[1]), this grade is a composition from several block requirements, which can be subdivided as:

1. Attendance
2. Tutor assessment
3. Written Exams
4. OSPE (Objective Structured Practical Examination)

The final grade is a composition of the grades obtained for the specified block requirements, calculated as follows:

1. Continuous Assessment (Tutor Assessment and Attendance) : 15%
2. Written Examinations (MCQ) : 55%
   - Mid-Block Exam 25%
   - Final Block Exam 30%
3. OSPE : 30 %

TOTAL **: 100 %**

1. **Attendance :**

Students are required to attend not less than 75% of all educational activities during the block. These include small group teaching, lectures, practical sessions, skills training sessions and integrated clinical sessions.

Your attendance will be recorded during all sessions. Failure to meet this requirement without a valid explanation will result in exclusion from the final examination. On the other hand, your presence will be rewarded by assigned marks.

2. **Tutor Assessment in Large and Small groups (Continuous Assessment):**

During each session, your individual efforts will be evaluated by your tutor. The tutors are instructed to evaluate two aspects:

a. The extent to which you demonstrate that you study and prepare yourself thoroughly between the two sessions (i.e., preparation).

b. The extent to which you actively contribute during group discussion (i.e., participation). Your grade for each session depends upon both your preparation and your participation. The grade will be on the scale from "5", "4", "3", "2", or "1". Which have the following general descriptors:

5 = Outstanding (Excellent)
4 = Very good
3 = Good
2 = Average
1 = Poor

The block contains two sessions each week, so the maximum amount of 'participation points' you are able to obtain will be from two sessions multiplied by the number of weeks. The total participation points will be recalculated according to the weight for each participation in the total assessment.

Your tutor can give you more information about the evaluation of your participation. The details of these evaluation also given in "Tutor Assessment of Student" form.

3. **Written Examination:**

   a. Mid block exam 25% : In the form of MCQs, these are prepared mainly from sessions presented to the students in large group. This exam will consist of 50 MCQs that will assess factual knowledge.

   b. Final written exam 30%: at the end of the block in form of MCQs, that are prepared mainly from sessions and presented to the students. This exam will consist of 80 MCQs that will assess factual knowledge too.

4. **Objective Structured Practical Examination (OSPE):**

This contains 30% of the marks. It is a practical examination at the end of the block. The OSPE examination will consist of 15-20 OSPE stations. Each station will take about 5 minutes, which contains a mix of slide show and some practical sessions. The purpose of the OSPE stations is to test your deeper understanding of the basic sciences. The OSPE will take place at the end of each block.

Block Evaluation

The block evaluation uses the following three data sources:

1. Student Feedback
2. Tutor Feedback
3. Student Results

Methods of student's formative assessment:

- Self evaluation
- Peer evaluation
- Tutor evaluation (both summative & formative)
- Assignments

Assessment in Professionalism

The assessment of professionalism will be conducted separately and will be based on students' portfolio. Details regarding training students to construct their portfolios and distribution of marks are discussed in details in the professionalism booklet.

LEARNING RESOURCES

The list below comprises the key textbooks and learning resources which have been prescribed and recommended for use in the undergraduate medical course at King Saud University. It is expected that you have your own copy of prescribed textbooks and use them as one of your main resources in learning. Before making any purchases, you might carefully examine all other recommended textbooks in an area and chose the text that matches with your needs and your learning style. Although all these texts are available in the Medical Library, you might need to purchase texts that you use frequently in these years as the demand upon library texts is usually high.

Medical Dictionary

Prescribed :

Martin EA (2015). Oxford Concise Medical Dictionary.9th Ed. Oxford: Oxford University Press.

Recommended textbooks:

Dorland (2012). Dorland's Pocket Medical Dictionary with CD-ROM, 29th Edition, Elsevier, UK.

Dorland (2011). Dorland's Illustrated Medical Dictionary with CD-ROM, 32nd Edition, Elsevier, UK.

Anatomy & Embryology

Prescribed textbook:

Drake RL, Vogl W and Mitchell AWM (2014). Gray's Anatomy for Students. Philadelphia: Elsevier Churchill Livingstone.

Snell RS (2005). Clinical Anatomy for Medical Students. 7th ed. Philadelphia: Lippincott Williams & Wilkins.

Schoenwolf GC, Breyl SB, Baurer PR, Fancis-West PH. (2014). Human Embryology. New York: Churchill Livingstone.

Recommended textbooks:

McMinn RH (2004). McMinn's Color Atlas of Human Anatomy. Fifth Edition. Mosby Publisher, UK.

Moore KL and Dalley AF (2005). Clinically Oriented Anatomy. Philadelphia: Lippincott Williams & Wilkins.

Netter FH (2006). Atlas of Human Anatomy. 4th ed. Philadelphia: Saunders WB.

Agur AMR and Dalley AF (2005). Grant's Atlas of Anatomy. 11th ed. Philadelphia: Lippincott Williams & Wilkins.

More KL (2002). The Developing Human. Philadelphia: Saunders WB.

Sadler TW. (2005) Langman's Essential Medical Embryology. Philadelphia: Lippincott Williams & Wilkins.

Sadler TW. (2006) Langman's Medical Embryology. 10th ed. Philadelphia: Lippincott Williams & Wilkins.

Histology

Prescribed textbook:

Gartner LP (2016). Color Textbook of Histology. 4th ed. Philadelphia: Saunders WB.

Recommended textbooks:

Young B, O' Dowd G, Woodford P (2013). Wheater's Functional Histology. 6th ed. London: Churchill Livingstone.

Physiology

Prescribed textbook:

Rhoades R and Pflanzer R (2003). Human Physiology, 4th ed. London: Brooks/Cole.

Hall JE. Guyton and Hall Textbook of Medical Physiology (2015). 13th Edition. Churchill Livingstone, UK.

Recommended textbooks:

Koeppen BM and Stanton BA. (2010) Berne & Levy Physiology, updated Edition. 5th ed. London: Mosby

Sherwood L. (2006). Human Physiology: From Cells to Systems. 4th ed.Brooks/Cole Pub.Co: Sydney.

Fox SI. (2015). Fundamentals of Human Physiology. 14th ed. McGraw-Hill: Boston.

Saladin KS (2011). Anatomy and Physiology The Unity of FORM and FUNCTION. McGraw Hill Lange, USA

Barrett KE, Barman SM, Boitano S, Brooks HL (2015). Ganong's Review of Medical Physiology. 25th Edition. McGraw-Hill Publisher, UK.

Pharmacology

Prescribed textbook:

Rang HP, Ritter JM, Flowei RJ, Henderson G. (2016). Range & Dale's Pharmacology. 8th Edition. Churchill Livingstone, Elsevier, UK.

Recommended textbooks:

Bertram G. Katzung, Anthony J. Trevor (2014).13th Edition. Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

Medical Biochemistry

Prescribed textbook:

Gaw A, Murphy MJ, Cowan RA, O'Reilly DJ, Stewart MJ, Sheperd J, (2009). Clinical Biochemistry: An Illustrated Colour Text. 4th ed. Churchill Livingstone, Elsevier.

Ferrier D, (2014). Lippincott's Illustrated Review Biochemistry. 6th ed. Lippincott Williams & Wilkins.

Recommended textbooks:

Murray RK, Roolwell VW, Bender D, Botham KM, Weill A, Kennelly PJ (2009). Harper's Illustrated Biochemistry. 28th Editions. McGraw Hill, Lange, New York.

Baynes J and Dominiczak M (2014). Medical Biochemistry. Elsevier.

Lieberman M, (2013).4th Edition. Mark's Basic Medical Biochemistry: A Clinical Approach. Lippincott Williams & Wilkins, New York.

Champe PC, Harvey RA, Ferrier DR (2008). Lippincott's Illustrated Reviews Biochemistry. 3th ed. Philadelphia: Lippincott Williams & Wilkins.

Microbiology & Parasitology

Prescribed textbook:

Murray P, Rosenthal K, Pfaller M, (2013). Medical Microbiology: Study smart with Student Consult. 7th ed. Elsevier.

Recommended textbooks:

Goering R, DoCkrell H, Zuckerman M, Wakelin D, Riott I, Mims C (2012). Mims' Medical Microbiology. 5th Edition. Mosby, UK.

John DT, Petri Jr (2006). Markell and Voge's Medical Parasitology. Ninth Edition. Elsevier, UK.

Greenwood D, Slack RC, Peutherer JF, Barer MR (2007). Medical Microbiology. Seventh Edition. Churchill Livingstone, UK.

Strohol WA. Lippincotts Illustrated Review Microbiology (2006). Second Edition. Lippincott Williams & Wilkins, New York.

Brooks GF, Butel JS, and Morse SA. (2004). Jawetz, Melnick, and Adelberg's Medical Microbiology. 23rd ed. New York: McGraw-Hill Co and Lange Appleton.

Engleberg NC (2013). Schaechter's Mechanisms of Microbial Disease. 5th ed. Philadelphia: Lippincott Williams & Wilkins.

Neva FA, Brown HW. (1994). Basic Clinical Parasitology. 6th ed. Connecticut: Prentice-Hall International Inc.

Chamberlain NR (2008). Medical microbiology & immunology. McGraw Hill Lange Publisher, UK.

Levinson WE (2010). Review of Medical Microbiology and Immunology. Eleventh-Edition, McGraw-Hill Publisher, UK

Pathology & Genetics

Prescribed textbook:

Kumar V, Abbas A, Aster L, (2013). Robbins Basic Pathology. 9th ed. Saunders. Philadelphia Elsevier

Hoffbrand V, Moss PAH, (2016). Hoffrand's Essential Hematology. 7th ed. Wiley Blackwell.

Nusbaum RL, McInnes RR, Willar HF, (2015). Thompson & Thompson Genetics in Medicine. 8th ed. Elsevier.

Recommended textbooks:

Kumar V, Abbas AK, and Fausto N (2004). Robbins and Cotran Pathologic Basis of Disease. 7th ed. Philadelphia: Saunders WB.

Young B, Stewart W. (2009). 5th Edition. Wheaters Basic Histopathology. A Colour Atlas and Text. Churchill Livingstone, Elsevier, UK.

Immunology

Prescribed textbook:

Owen J, Punt J, Stranford S, (2013) Kuby Immunology: Kindt, kuby Immunology. 7th ed. W.H. Freeman.

Recommended textbooks:

Delves PJ, Martin SJ, Burton DR, Riott IM (2012). Riott's Essential Immunology. 8th Edition. Elsevier.

Male D, Brostoff J, Roth DB, and Roitt I. (2006). Immunology. 7th ed. Edinburgh: Mosby.

PBL and Learning Skills

Prescribed textbook:

Azer SA (2006). Core Clinical Cases in Basic Biomedical Sciences. Hodder-Arnold, UK.

Azer SA (2008). Navigating Problem-Based Learning. Elsevier Australia, Australia.

Recommended textbook:

Kushner TK and Thomasma DC (2001). Dilemmas for Medical Students and Doctors in Training. Cambridge: University Press.

Communication Skills & Introduction to Clinical Medicine

Prescribed textbook:

Lloyd M, Bor R (2009). Communication Skills for Medicine. Elsevier.

Munro JF, Campbell IW (2006). Macleod's Clinical Examination. Tenth Edition. Churchill Livingstone, UK.

Talley NJ and O'Connor S. (2006). Pocket Clinical Examination. Melbourne: Blackwell Science.

Medicine

Kumar P and Clark M (2012). Clinical Medicine. 7th ed. Edinburgh: Elsevier Saunders.

Walker B.R, Colledge Nicki.R, Ralston Stuart.H, Penman I. (2014). Davidson's Principles and Practice of Medicine. 22nd ed. Edinburgh: Churchill Livingstone.

(In the preclinical years these two textbooks may help you in the preparation of your learning issues, you will also need them in the clinical years).

Professionalism

Prescribed textbook:

Feldman MD, Christensen JF (2014). Behavioural Medicine. A Guide for Clinical Practice. McGraw-Hill Lange, UK.

Stern DT (2006). Measuring Medical Professionalism. Oxford University Press, UK.

Spandorfer J, Pohl CA, Rattner SL, Nasca TJ (2010). Professionalism in Medicine. A case-based Guide for Medical Students. Cambridge University Press, UK.

KING SAUD UNIVERSITY
College of Medicine
Department of Medical Education

Feedback to Student on PBL Performance

Year 2 (Academic Year 2016-2017)

Student's name: ……..Group number:……..

Tutor's n……...Block: NEUROPSYCHIATRY BLOCK

The feedback items are grouped under two main headings.
1= Deficient/lacking/or poor; 2= Working on it; 3= showing some improvement, 4= developed; 5=well developed (marks are allocated as follows: 1 mark for rank 1, 2 marks for rank 2, 3 marks for rank 3, 4 marks for rank 4, and 5 marks for rank 5, maximum mark is 5 for each group)

1. Learning and cognitive skills:

Ability to:	1	2	3	4	5
• Identify problems in the case					
• Generate hypotheses					
• Build mechanisms					
• Collect new information					
• Interpret findings					
• Identify learning issues					
• Apply knowledge learnt					

Mark= /5

2. Interaction and participation to group function:

Ability to:	1	2	3	4	5
• Work collaboratively with other members					
• Take active roles such as scribing					
• Communicate effectively					
• Arrive to tutorials on time					
• Demonstrate good manners					
• Keep the group focused					
• Share resources with others					

Mark = /5

Comments

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Tutor's Name: Signature: Total Mark= /10

KING SAUD UNIVERSITY
College of Medicine
Department of Medical Education

Assessment of Student in PBL

Year 2 (Academic Year 2016-2017)

Student's name: ……Group number:……..

Tutor's name: ……Block: NEUROPSYCHIATRY BLOCK

The feedback items are grouped under two main headings.
1= Deficient/lacking/or poor; 2= Working on it; 3= showing some improvement, 4= developed; 5=well developed (marks are allocated as follows: 1 mark for rank 1, 2 marks for rank 2, 3 marks for rank 3, 4 marks for rank 4, and 5 marks for rank 5, maximum mark is 5 for each group)

1. Learning and cognitive skills:

Ability to:	1	2	3	4	5
• Identify problems in the case					
• Generate hypotheses					
• Build mechanisms					
• Collect new information					
• Interpret findings					
• Identify learning issues					
• Apply knowledge learnt					

Mark= /5

2. Interaction and participation to group function:

Ability to:	1	2	3	4	5
• Work collaboratively with other members					
• Take active roles such as scribing					
• Communicate effectively					
• Arrive to tutorials on time					
• Demonstrate good manners					
• Keep the group focused					
• Share resources with others					

Mark = /5

Comments

……
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Tutor's Name: Signature: Total Mark= /10

KING SAUD UNIVERSITY
COLLEGE OF MEDICINE
MEDICAL EDUCATION DEPARTMENT

STUDENTS' EVALUATION OF THEIR PBL TUTOR

Date:_____
Tutor's Name:_____ **Group No.:**_____
Student: ☐ **Peer:** ☐ **Other:** ☐ **Name (Optional):**_____

How well did the tutor facilitate group process in the following regards? Please put a check (✓) in the box.

1.	Appropriately facilitated the brainstorming sessions.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
2.	Appropriately facilitated the hypothesis reorganization sessions.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
3.	Appropriately facilitated the reporting sessions.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
4.	Appropriately manage the time flow.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
5.	Help to keep the group focused on its task	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
6.	Provided a well balanced intervention within the group process, but avoided dominating.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
7.	Intervened when chairman or reporter needed.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
8.	Provided constructive positive and constructive feedback to the group as needed.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
9.	Encouraged positive and constructive feedback within the group about its performance	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
10.	Showed enthusiasm.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
11.	Helped to create a supportive group climate.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
12.	Encouraged logical and critical thinking.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐
13.	Overall rating of the tutor.	**1** ☐	**2** ☐	**3** ☐	**4** ☐	**5** ☐

Number Code Values:

5- EXCELLENT 4- VERY GOOD 3-GOOD 2- FAIR 1- POOR

KING SAUD UNIVERSITY
COLLEGE OF MEDICINE
MEDICAL EDUCATION DEPARTMENT

STUDENT RATING OF LECTURE

Date:_____Subject:_____Instructor:_____

Purpose:
This form is designed as an observation tool to rate the performance of each instructor in the different sessions. It is intended to provide a tool for lecturer improvement.

Directions:
Using the anchors below, check (✓) your rating for each item below. Check (✓) N/A for items that do not apply.

No.	Standard Procedure	5	4	3	2	1	N/A
1	Started and ended class on time.						
2	Presented overview of content and objectives.						
3	Presented information according to objectives.						
4	Used relevant examples and illustrations (graphs, etc.) to explain major ideas						
5	Used alternative explanations when necessary.						
6	Made efficient use of questions with students.						
7	Covered all contents/objectives.						
8	Exhibited enthusiasm.						
9	Encouraged students to express themselves.						
10	Asked questions prior to closure						
11	Summarized major points/related contents to objectives.						
12	Amount you learned in the class was:						

Mention 3 strong points in this lecture:

1. _____
2. _____
3. _____

Mention 3 points for Improvement:

1. _____
2. _____
3. _____

Your name: (optional)_____

Appendix

Medical Teacher, Vol. 26, No. 1, 2004, pp. 12–15

Taylor&Francis healthsciences

TWELVE TIPS

Becoming a student in a PBL course: twelve tips for successful group discussion

SAMY A. AZER

Faculty Education Unit, Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne, Victoria, Australia

SUMMARY *Problem-based learning (PBL) serves as an educational method to foster self-directed learning, integration across disciplines, small-group learning and decision-making strategies. The approach is student centred. During the discussion of a PBL case there are a number of important issues to be considered by students, such as keeping ground rules, knowing their roles, keeping group dynamics, becoming a purposeful learner, planning how to use tutors' feedback to enhance group discussion and boost student's learning skills, as well as striving to become a winning team. This paper provides 12 practical tips to PBL students to enhance their skills in discussing a case in their group.*

Introduction

Problem-based learning (PBL) is currently used in medical schools worldwide and also in schools of physiotherapy, nursing, pharmacy, optometry, occupational therapy and speech pathology (Davis & Harden, 1998). The approach used in PBL is student centred (Norman & Schmidt, 1992), and aims at the following educational objectives (Barrows & Tamblyn, 1980):

- Enhance students' skills to acquire principles and key concepts that should be better retained by the learners and allow them to use information learnt in other similar situations.
- Develop students' clinical reasoning skills, critical thinking and decision-making strategies.
- Develop students' skills in integrating knowledge across disciplines and better understanding of the role of a humanistic attitude towards professional performance.
- Prepare students to pursue lifelong learning.
- Promote small-group learning, the need for effective teamwork and collaborative learning.

To achieve these objectives, problem-based learning requires that students work in small groups of 8–10 students with the help of a tutor (Ledingham & Crosby, 2001). The roles of PBL tutors are to facilitate group discussion, create a healthy environment that allows all members to contribute to discussion, provide formative feedback whenever necessary and monitor group progress (Maudsley, 1999). Although successful discussion of a PBL case in a tutorial has been attributed to several factors, including authenticity of the case, flow and design of the case and PBL tutor skills, the key for successful PBL discussion remains in the hands of the students (Allen *et al.*, 1996; Engel, 1992). Students should be oriented to the philosophy of problem-based learning, the rationale for its use and their role in a tutorial. Here are 12 practical tips to be used by PBL students while discussing a case in a tutorial.

Tip 1

Keep ground rules

- Setting group norms (ground rules) early in a group's existence prevents crises from occurring in the group and allows better function.
- The tutor should discuss with the group his/her role.
- Ground rules are agreed on by group members.
- They should reflect the group's needs and principles.
- Groups should operate in keeping with the rules.

Example of ground rules agreed by a PBL group:

- Everyone has the right to express his/her views.
- We should debate issues rather than argue them.
- We should not spend too much time on one issue.
- We should respect each other and avoid personal comments.
- We need one scribe at time—2 scribes per tutorial.
- We need a recorder for each tutorial.
- We need to focus on the discussion of the case and avoid sidetracked discussion.

Some of the ground rules are non-negotiable and will be explained by your tutor, as they are important for group function:

- Attendance and punctuality are mandatory.
- All mobile phones must be turned off during the tutorial.
- Groups must use white-board as they discuss the case.
- The case should be discussed in the outlined sequence.
- The group should not take short cuts or skip a step.

Tip 2

Know your roles

- Groups function better when every member is aware of the different roles a group member should undertake.

Correspondence: Dr Samy A. Azer, Faculty Education Unit, Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne, Victoria, Parkville 3010, Australia. Tel: +61-3-83448035; fax: +61-3-83440188; email: samy@unimelb.edu.au

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- Roles should be agreed on and organized in the first tutorial of a block/semester.
- The approach is student centred.

What are the different roles available to me in a PBL tutorial? What exactly should I do?

- Be a scribe: a scribe listens to each member's input, records and organizes information discussed by the group on the white board, encourages every member to contribute and knows how to serve the group.
- Be involved in the case discussion: Every member contributes to the discussion in a way that adds new information, deepens group understanding, acknowledges others' input, focuses on the issue and avoids negative arguments.
- Be a group recorder: a group recorder summarizes all the information on the white board and makes a copy available to every member in the group after the tutorial.
- Be a word finder: look up difficult terms in the medical dictionary.
- Be the group's representative: Each semester one member is nominated by the group to represent them at faculty meetings and look after administrative issues in the group.
- Apart from the group's representative, students rotate roles every tutorial. A student may have more than one role in the same tutorial.

Tip 3

Keep group dynamics

- Ask yourself: What good qualities am I bringing to my group?
- Use individual and cultural differences as a way to empower group dynamics.
- Appreciate the values of teamwork and the need for regular evaluation of the group process.
- The last 10 minutes in tutorial two (as you complete the discussion of a case) are a good opportunity for the group to reflect on members' performance, identify specific goals that the group aims at and plan how to achieve each of these goals. Focus on one goal at a time.

What questions should the group consider as they review and reflect on group performance in the last 10 minutes of tutorial two?

- What did we achieve this week as a group?
- Have we worked effectively together?
- In what areas did we succeed?
- In what areas do we need to improve?
- As a group, what are our goals for next week?
- How can we achieve these goals?

Tip 4

Ask empowering questions

- Use of good open-ended questions could empower the discussion and keep the group focused on the issue.
- Use of good empowering questions in group discussion is vital for deep understanding and better learning.
- Avoid asking shallow questions that focus on detail.

Examples of open-ended questions that enhance group discussion:

- Normally we do not feel short of breath. What structures and functions do we need so that we breathe normally?
- What could possibly go wrong with each of these structures and cause shortness of breath?
- What are the structures in the chest that could possibly cause chest pain?
- What could possibly go wrong with each of these structures and cause chest pain?

Tip 5

Be a purposeful learner

- A powerful motivator for adult learning is keeping the learning process purposeful so that it contributes to personal growth and deep understanding.
- Your self-directed learning will be enhanced if you know exactly what questions you are trying to answer in your search.
- Shape your learning to suit the needs of your new learning environment.

A purposeful learner:

- has a continuous desire for learning;
- is focused on his/her goals;
- is a critical thinker;
- is self-motivated;
- is not afraid to ask for help;
- is able to monitor his/her progress;
- has an enquiry plan;
- is able to integrate information learnt;
- has developed reasoning skills;
- plans his/her learning.

Tip 6

Without feedback there would be no champions

- Learn how to get the best out of your tutors' feedback.
- Plan how to use feedback to enhance your input to the group discussion and boost your learning with your tutor.

How can I benefit most from my tutor's feedback?

- Focus on issues raised in the feedback and don't take it personally.
- Show interest in issues raised and explore them with your tutor.
- Negotiate an approach with your tutor to enhance your input to the group.
- Work on one issue at a time and meet with your tutor in a fortnight to further discuss your progress.
- Think about ways to keep you motivated and improve yourself.
- Keep monitoring yourself and focus on your goals.
- Record your daily progress in a journal.
- Once you have accomplished a particular skill, move on to the next area in need of improvement.
- Celebrate your successes.

Tip 7

Monitor your own progress

- One of the key elements of success is self-evaluation and motivation.
- Keep focused on your goals as you progress.
- Keep a progress journal to monitor your progress.

What issues should I address in my progress journal?

- What are my areas of strength?
- What are the areas I am still developing?
- What are the areas I need to improve?
- How can I improve myself in each of these areas?

Tip 8

Strive to be a winning team

- Effective interactions fuel the right actions.
- Focus on the issue rather than personal interest.
- Group success is the outcome of every member's contribution (Table 1).

Characteristics of winning teams:

- They define their priorities more than others do.
- They give up their personal plans more than others do.
- They appreciate the process of developing people more than others do.
- They communicate more effectively than others do.
- They encourage team members more than others do.
- They establish their common goals more than others do.
- They are committed more than others are.

Tip 9

Be a critical thinker

- Debate rather than argue an issue.
- Before making decisions, weigh evidence for and against a hypothesis.

Characteristics of critical thinkers—they:

- use their thinking abilities to the fullest extent;
- carefully analyse complex issues;
- develop a thoughtful and well-structured approach to guide their choices;
- look for supportive evidence for each of their hypotheses;
- evaluate data, synthesize information, establish links and identify areas that need further research;
- are able to discuss issues in an organized fashion;
- evaluate accuracy of their beliefs;
- have a passion for understanding and are always striving to solve problems;
- explore different aspects of an issue, e.g. scientific basis, ethical and moral issues, background and contributing factors.

Tip 10

Know the roles of your tutor

- The approach in PBL is student centred.
- Your tutor will not be the information provider.
- He/she would rather facilitate learning and put the discussion on the right track when needed.
- During one-on-one sessions, your tutor will provide you with feedback on your contribution to group discussion.
- Your group will have an opportunity to discuss ways of improving group dynamics with your tutor as you finish discussion of each problem (Maudsley, 1999).

Tip 11

Turn to the winning attitude

- Develop good habits.
- Select a model to follow.
- See opportunities for success in challenges.
- Focus on solutions.
- Have a desire to give and share resources.
- Be persistent.
- Find ways to relieve stress.
- Don't take yourself too seriously.
- Take actions to change your attitude.

Tip 12

Be a collaborative learner

- Collaboration is the critical competence for achieving and improving group performance (David *et al.*, 1999).
- To foster collaboration in the group members need to create a climate of trust.
- Ask others for help and assistance when needed.
- Listen attentively to the views of other members.
- Interact with each other on a regular basis.
- Share information and resources.
- Provide descriptive rather than evaluative or judgmental comments.
- Ask questions for clarification.
- Always say 'we'.

Conclusions

Students in a PBL course should be introduced to the educational objectives of PBL, the rationales for using PBL and the keys for successful discussion of a case. The use of

Table 1. Main differences between effective and dysfunctional groups.

Effective groups	Dysfunctional groups
• Keep their ground rules	• Failed to identify ground rules
• Members focus on group's goals	• Members do not have common goals
• Care about team achievements	• Care about personal gains
• Work in a supportive environment	• Tutor-centred or managed by a dominant student
• Continuous group monitoring	• Ignore feedback

the 12 tips provided in this paper will provide students with a clear idea of their roles in a PBL tutorial and how to make the most out of it.

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Notes on contributors

SAMY A. AZER, MB BCh Msc MEd PhD (Syd) MEd (NSW) FACG FRSM, is a Senior Lecturer in Medical Education at the Faculty of Medicine, Dentistry and Health Sciences at the University of Melbourne. He plays a major role in problem-based learning, creating cases for semesters 1 to 5, and he is the director of the PBL tutor training. His research focuses on curriculum structure and design, innovation in education, problem-based learning and the use of tools that test cognitive skills in assessment.

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Original Article

Facilitation of Students' Discussion in Problem-based Learning Tutorials to Create Mechanisms: The Use of Five Key Questions

Samy A Azer,[1] *MBBCh, MPH (NSW), PhD (Syd)*

Abstract

Without the appropriate facilitation of discussion in a problem-based learning (PBL) course and the use of specific educational tools that enhance cognitive skills, students might deprive themselves of achieving the deep learning experience expected to take place in a PBL course. One of the educational tasks in PBL is the creation of mechanisms for hypotheses made by the students, based on their knowledge of the basic sciences and the psychosocial issues raised in a particular case scenario. The whole task is student-constructed and should enhance their ability to explain the scientific basis of the symptoms and clinical signs of the patient enlisted in the case. Because students usually discuss the case without enough prior related knowledge, they might find it difficult to address different aspects of their mechanisms. These gaps in knowledge may be considered part of their "learning issues". In tutorial 2 (a PBL case is usually discussed in 2 or 3 tutorials at the maximum; each tutorial is 2 hours long), students should be able to build a comprehensive mechanism reflecting their deep understanding of the problem. However, students might not be able to integrate information learnt and their mechanisms might show a number of shortcuts and/or lack integration of information, and the flow of the pathophysiological changes may not be logical. This manuscript describes 5 key open-ended questions in PBL tutorials to facilitate students' discussions as they create their mechanisms.

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Key words: Medical education, Pathophysiology, PBL tutors

Introduction

An important aspect of problem-based learning (PBL), particularly in the early years of the undergraduate medical, physiotherapy, nursing and dental courses, is teaching basic science in a clinical format.[1,2] This approach should enhance students' skills to develop reasoning strategies, use information in relevant situations, generate hypotheses for problems identified and build mechanisms. Mechanisms are usually described as diagrammatic flowcharts illustrating a sequence of events. The main aim of including mechanisms in a PBL template is to encourage students to use knowledge learnt and information provided in the case scenario, including psychosocial issues, to explain how a hypothesis suggested by the group could explain the patient's problems.[3] During this process, the group might discover that they are unable to provide a thorough explanation and that they lack information in areas such as physiology, anatomy, pharmacology, microbiology, pathology, biochemistry or pathophysiological changes. The group may choose to include these deficiencies in their knowledge as part of their "learning issues" list.[4]

However, building mechanisms is not an easy process and the students usually find it difficult to start their mechanisms or link them back to the information provided in the case scenario. Even in tutorial 2, after they have completed their learning issues and attended a few lectures, some groups struggle to build a good mechanism that integrates related information learnt to explain the pathophysiological processes in the case, the patient's symptoms and the clinical signs elicited in the case. Several factors could have contributed to this difficulty:

- Early in a PBL course, students find it challenging to develop their mechanisms. They are usually uncertain about what exactly constitutes a good mechanism and

[1] Faculty Education Unit (FEU)
Faculty of Medicine, Dentistry and Health Sciences
University of Melbourne, Australia
Address for Reprints: Dr Samy A Azer, Faculty Education Unit (FEU), Faculty of Medicine, Dentistry and Health Sciences, University of Melbourne, Victoria, Parkville 3010, Australia.
Email: samy@unimelb.edu.au

how detailed their mechanisms should be.

- Most students are trained in high school to adopt a rote learning style rather than a learning style that encourages the application of knowledge, elaboration, reflection, critical thinking and integration.[5]
- In PBL, most tutors are not experts in the disciplines related to the case. Despite having been trained in interactive workshops on the facilitation of group discussion, some tutors find it difficult to ask appropriate open-ended questions that enhance the discussion on mechanisms.
- Textbooks, lectures, and other resources are usually discipline-based and do not help students to integrate information as they create their mechanism.[6]
- Building mechanisms requires a number of skills such as integration, a deep understanding of the basic sciences related to the case and the use of logical flow as they describe the pathophysiological changes related to the patient's problems described in the case scenario.
- The group might find it difficult to convey to the scribe where the mechanism should start and how it should progress.
- There are no guidelines in training workshops or textbooks for tutors and students to help them learn what constitutes a good mechanism.

The aims of this paper are (1) to evaluate the educational objectives of mechanisms in PBL – tutorial 1 versus those of tutorial 2, and (2) to discuss the use of 5 key open-ended questions to enhance group discussion of mechanisms.

Why are Diagrammatic Mechanisms Useful to Students' Learning in PBL Tutorial One?

Although there are a reasonable number of studies and review papers covering the rationale and educational objectives of PBL courses,[1,7,8] there are no papers in the literature discussing the educational objectives of mechanisms in a PBL curriculum and how tutors can facilitate group discussions of mechanisms. It is important that we use mechanistic tools to foster integration across disciplines, depth in learning and an understanding of the scientific basis for the patients' symptoms, clinical signs and laboratory findings, as well as the possible role of psychosocial issues in the case.

PBL cases are usually discussed in 2 or 3 tutorials, depending on the structure and design of the curriculum.[9] Each tutorial is 2 hours long. Each case usually begins with a trigger text or a scenario, which is often presented to the students without any prior preparation. A series of images, a 2- to 3- minute video clip or a cartoon may accompany the trigger text. In tutorial 1, students work in small groups of 10 students:

Step 1: Clarify key information in the trigger text and accompanied trigger images.

Step 2: Define problems in the trigger (problem formulation), and retrieve their own knowledge in relation to the identified problems.

Step 3: Generate a list of hypotheses for each problem.

Step 4: Develop an enquiry strategy on their hypotheses.

Step 5: Read further information provided with the problem of the week (e.g., medical history).

Step 6: Use the new information to support or exclude each of their hypotheses (group their hypotheses under 3 headings, less likely, most likely and to be excluded).

Step 7: Create mechanisms to explain their hypotheses.

Step 8: Identify areas of gaps in existing knowledge.

They may negotiate and refine their learning issues throughout tutorial 1.[9,10] Between tutorial 1 and tutorial 2, students work independently and look for information which addresses each of the learning issues identified in tutorial 1. Students may use resources such as textbooks, journal articles, websites and computer-aided learning (CAL) programmes in this process.[11] In tutorial 2, about 3 days after tutorial 1, the students' groups reconvene to discuss their learning issues. They discuss the knowledge they have acquired and relate the new information to issues raised in the problem. They then discuss laboratory investigations that might help to confirm their final hypothesis. They may discuss the progress provided in the case, usually cultural, ethical or psychosocial issues related to the patient. At the end of tutorial 2, students develop a comprehensive mechanism covering the patient's problem, clinical signs and laboratory findings.

It appears that the goals and educational objectives of creating diagrammatic mechanisms in tutorial 1 are not exactly the same goals and objectives of developing a comprehensive mechanism in tutorial 2 (Table 1). The scenario below summarises key information from the trigger and history findings from a PBL case.

An Example of a PBL Case Scenario

Ms Linda Hart, a 42-year-old librarian, is brought to the emergency department of a local hospital by ambulance at 4 am. She is pale and vomiting fresh blood. Although drowsy, she is oriented and able to answer your questions. Linda gives a history of vomiting large amounts of fresh blood at her house, before arriving at the hospital. Last night she started vomiting repetitively after binge drinking. Thirty minutes later, the vomitus became bloody. Immediately on arrival to the emergency department, the nurse tells you that Ms Hart's blood pressure is 100/60 mm Hg (on lying flat), her pulse rate is 105/min and regular, her respiratory rate is 20 per minute and her temperature is

Table 1. Aims and Objectives of Mechanisms in PBL in Tutorial 1 versus Tutorial 2

Tutorial 1	Tutorial 2
No prior knowledge about the case and its contents.	Students collect information from textbooks, computer-aided programmes, lectures and practical classes related to their learning issues and the case.
Building a mechanism helps students to identify areas of deficiencies in their knowledge.	Building a mechanism helps students to integrate information learnt, apply knowledge and address a patient's presentation and clinical signs.
Mechanism may be broad and may include several hypotheses.	Mechanism is usually focused around the final hypothesis.
Mechanism is usually superficial, not detailed and may contain short cuts.	Mechanism should be comprehensive, detailed, and reflect integration of knowledge with no short cuts.

36.5° C. The registrar inserts a large intravenous line in her forearm vein and she is commenced on Haemaccel intravenously. Ms Hart gives you a history of recurrent headaches, for which she takes aspirin tablets from time to time. Recently, she has experienced abdominal pain and indigestion. Her bowels are regular but she noticed that her stools have become black and soft over the last few hours. She has 2 tattoos on her back. She has been drinking a bottle of white wine a day for the last 10 years, but has increased her consumption since the death of her husband and son in a motor car accident under a year ago. Due to her alcohol problem and feelings of depression, she was seen by a psychologist 6 months ago and was advised to attend counselling sessions regarding her alcohol problem. However, she refused to attend.

Students identified haematemesis (vomiting blood) as one of the main problems: Their hypotheses for the problem were:

- Bleeding from an ulcer in the stomach (possibly caused by aspirin).
- Bleeding from oesophagus (oesophageal varices).
- Bleeding from a tear in the oesophagus (caused by repeated vomiting).
- Bleeding from a cancer of the oesophagus/stomach (cancers may ulcerate and bleed).

Due to uncertainty and lack of information, students might find it difficult to develop a mechanism explaining their hypotheses. Students may tend to develop a "backward reasoning" approach.[12] Using this approach, students begin by asking what could possibly cause Ms Hart to vomit blood. They might suggest, "Bleeding from oesophagus, stomach and duodenum". They then consider a new question: "What caused bleeding from these structures? Was it mechanical damage to the lining tissues? Was it bleeding from abnormal blood vessels in the oesophagus or the stomach?". The scribe adds this new information to the whiteboard. As the group places these items in their mechanisms and thinks about the preceding question they continue to develop their mechanism (Fig. 1). During this process, the group discovers that they lack information in

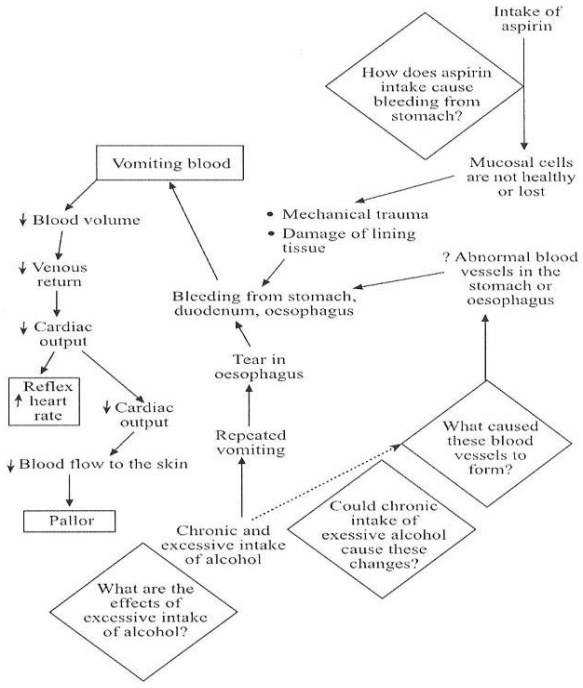

Fig. 1. An outline of a mechanism developed by students in tutorial 1 explaining vomiting blood, increased heart rate and pallor.

4 areas and they add them to their "learning issues" list: (1) What are the effects of excessive intake of alcohol? (2) Could chronic intake of excessive alcohol cause these changes? (3) How does aspirin intake cause bleeding from the stomach? (4) What caused these blood vessels to form? (These are illustrated as diamonds in Figure 1.) It is important to note that groups vary in their approaches and

not all groups will necessarily use "backward reasoning".

Thus, the development of diagrammatic mechanisms in tutorial 1 is useful and facilitates the achievement of these educational goals:

- Enhance students' reasoning skills.
- Encourage students to apply previously learnt information to a novel case.
- Enable students to identify areas of gaps in their knowledge and define their learning issues.
- Prompt students to realise the need for a grasp of the basic sciences to better understand a clinical context.
- Foster communication skills, peer-peer interaction, and the ability to make links, use logic and clarify areas of confusion.

Why is Creating Mechanisms Useful to Students' Learning in Tutorial 2?

At this stage, students have researched their learning issues using textbooks, journal articles and appropriate web sites, attended 4 or 5 lectures and possibly used a CAL programme related to the case. They should be able to use and integrate information to build a comprehensive mechanism (Fig. 2). Therefore, in contrast with tutorial 1, the goals for developing mechanisms in tutorial 2 are:

- The integration of knowledge across disciplines and the consideration of psychosocial issues in the case scenario.
- To allow students to appreciate the role of contributing factors and any risk factors mentioned in the case history.

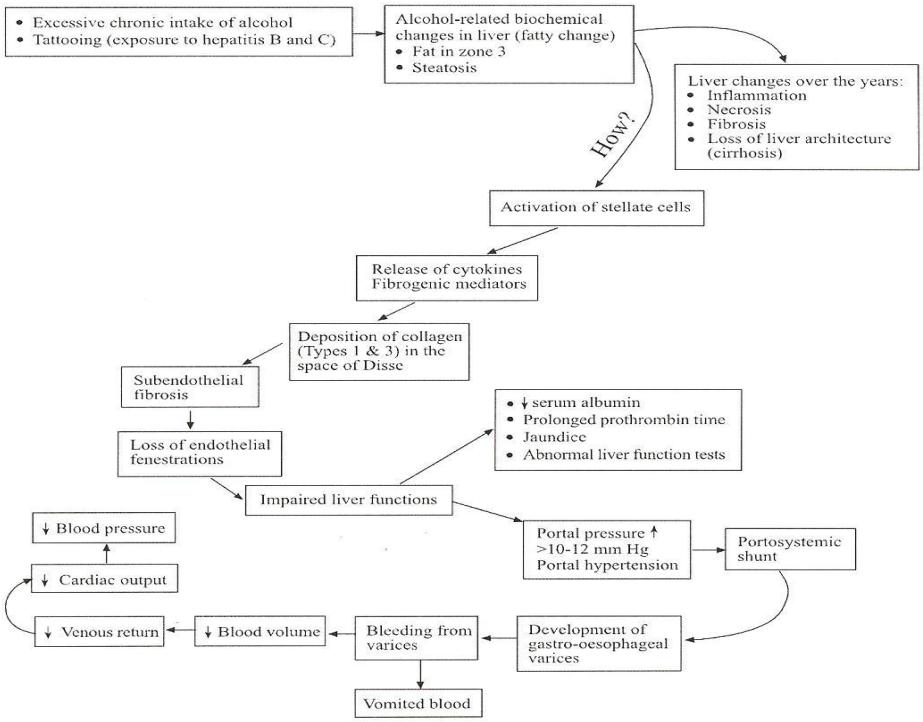

Fig. 2. Outline of mechanism created in tutorial 2.

- To encourage students to reflect on the scientific basis of a patient's presenting problems, clinical signs and laboratory changes.
- To enhance students' skills in organising pathophysiological changes in body systems and at the cellular level, with no short cuts or missed steps.

These goals cannot be achieved unless tutors are trained in facilitating group discussion so that students realise the significance of reviewing their mechanisms and utilising the information gained from different resources, such as textbooks, web sites, lectures, practical classes and CAL programmes.

Training PBL Tutors to Facilitate the Discussion of Mechanisms

In the context of major curriculum change, a staff development programme has a crucial role to play.[13] Hands-on training workshops present an opportunity for tutors to explore new skills in teaching and how to use group facilitation effectively. Successful group facilitation necessitates that tutors possess several skills including active listening, critical reflection, and the ability to create a healthy environment that allows every member in the group to participate in the discussion and to ask open-ended questions that enhance group discussion.[14] Open-ended questions should have a purpose, allow depth in learning, enable the group to work on a task and add new information to the whiteboard, motivate students to discover new challenges and foster their deep understanding of concepts discussed. However, most PBL tutors are not experts in the disciplines related to cases. They usually find it difficult to ask good open-ended questions that can drive the students' discussions and help them to build sound mechanisms. In order to boost the tutors' skills in this area, tutors need to be trained in refresher workshops, particularly in these areas:[16,17]

- How to encourage groups to keep their ground rules;
- How to explain the different roles members in the group may undertake;
- How to guide their groups to practise debating of issues rather than arguing;
- How to help the group work as a team;
- How to use open-ended questions effectively to facilitate discussion; and
- How to provide constructive feedback to the group.

The use of open-ended questions may help groups to work as a team on a task and construct their mechanisms (Table 2). This approach can be introduced in the workshop with the aims of teaching the PBL tutors to:

- Use mechanisms in PBL;
- Understand the uses of mechanisms in tutorial 1 and tutorial 2;

Table 2. Five Key Open-ended Questions to Enhance Students' Ability in Creating Their Final Mechanism

Q1. Have you considered contributing factors for your hypotheses?
- Possible environmental factors.
- Genetic background and family history.
- Possibility of exposure to infectious agents.
- Risk factors for vascular problems e.g., obesity, high blood pressure, high blood cholesterol/triglycerides, family history, diabetes.
- Medications, allergies.
- Psychosocial issues.

Q2. Have you considered explanations using your knowledge in basic sciences?
- Anatomical, biochemical or physiological explanations, microbiological information and pathological changes.
- Does your mechanism cover key issues at body system and cellular levels?

Q3. Does the flow of the mechanism explain the patient's problems and clinical signs with no short cuts?
- Pathophysiological changes are placed in order.
- No shortcuts.
- The flow is logical.

Q4. Does the mechanism reflect the information provided in the case?
- Age and background of the patient.
- Medications.
- Previous illness.

Q5. Have you addressed the target of your mechanism?
- E.g., abdominal pain (as the problem) caused by a peptic ulcer (your hypothesis).

- Understand the different components of a mechanism;
- Realise that there is no one way for creating a mechanism but that students might need their support during this process;
- Practise how to use the 5 key open-ended questions to facilitate the discussion of mechanisms; and
- Practise how to give constructive feedback to students using the 5 key questions.

Such sessions in a workshop should be carried out by a PBL educator. The educator should:

- Provide participants with clear objectives for the session;
- Encourage participants to work in small groups and create a mechanism;
- Encourage participants to discuss the main challenges they face (as non-experts) in the construction of a mechanism;
- Use role-play in implementing the 5 key open-ended questions; and
- Provide feedback to participants on their performance.

Over the last 2 years, I have run 4 refresher workshops titled "Challenges Facing PBL Tutors" at the Faculty of Medicine, Dentistry and Health Sciences. Each workshop was attended by 12 to 14 tutors from a wide range of

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King Saud University

College of Medicine

Medical Education Department