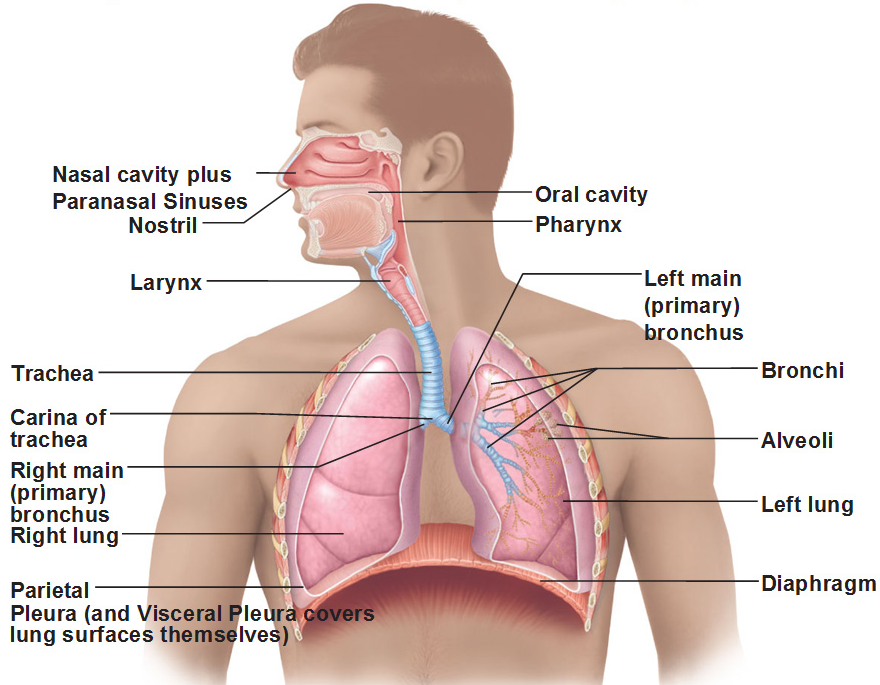
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**COLLEGE OF MEDICINE**

**Department of Medical Education**

**Curriculum Development & Research Unit**

****

**STUDENT'S BOOK**

**RESPEIATORY**

**BLOCK**

**YEAR 1**

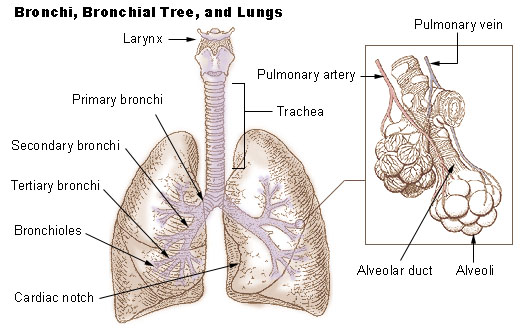
**2016-2017**

**(1437-1438)**

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**College of Medicine**

**Department of Medical Education**

**[](http://upload.wikimedia.org/wikipedia/commons/d/db/Illu_bronchi_lungs.jpg)**

**BLOCK BOOK and STUDENT GUIDE**

**THE RESPIRATORY BLOCK**

**YEAR ONE**

****

**College of Medicine**

**Department of Medical Education**

**THE RESPIRATORY SYSTEM BLOCK**

**Year ONE**

**BLOCK BOOK AND STUDENT GUIDE**

**(5 February to 9 March 2017)**

**Group – Male A**

**1437 - 1438**

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**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.

**Prof. Khalid Fouda**

**Dean, College of Medicine and the Supervisor of University Hospitals**

**A Message from the Respiratory System Block Chair**

I would like to welcome you, our future physicians and medical educators, to the Respiratory Block as part of you first year reformed curriculum. We should be looking forward to an exciting, rich and smooth running four weeks.

The block is designed in such way to introduce you to the respiratory system in a weekly theme stepwise fashion, where all basic sciences and clinical aspects are arranged in a logical and systematic way in order to increase your interest and enhance your understanding of how the body use the respiratory system to work and function and what happen if it falls ill. Hence you will start the anatomy, physiology and biochemistry followed by the pathology and clinical medicine aspects of the part of the respiratory system in each of the weekly themes.

Lectures, lab practical’s, introduction to clinical medicine practical’s and small group discussions will all be combined to enhance your learning experience in this block. The respiratory block contain beside others 3 of the main clinical conditions facing human race complication of smoking, asthma and tuberculosis affecting 1/3 of human race, infecting 8 millions and killing 3 million each year.

Also, I would like to extend my deepest gratitude and respect to our teachers for their enormous hard work and dedication, the Medical Education Department for their endless support and coordination. And finally to the college’s administration lead by the dean for their relentless efforts to continuously improve, modernize and refine the educational process.

I wish you all the best, and look forward to have you as students during the block, and as colleagues as you graduate in the near future.

**Dr. Malak El-Hazmi.**

**Chairman**

**Respiratory Block**

|  |  |
| --- | --- |
| **TABLE OF CONTENTS** | |
| General Information | 7 |
| Teaching staff | 8 |
| List Of Problem – based Learning Cases | 10 |
| Objectives of theRespiratory Block | 11 |
| Objectives of the lectures | 12-85 |
| Academic Support Team | 86 |
| Schedule of the Block | 87-90 |
| Plagiarism | 91 |
| Assessment of the Student in the block | 92-93 |
| Learning Resources | 94-98 |
| Feedback to students on PBL Performance | 99 |
| Assessment of student’s Performance in PBL | 100 |
| Students’ evaluation of their PBL tutor | 101 |
| Student Rating of lecture | 102 |

**GENERAL INFORMATION:**

Block Title : Respiratory Block

Block Code & Number : Resp 112

Credit Hour : 4

Block Duration : 4 Weeks

Block Dates : 5 February to 9 March 2017

Block Chairman : Dr. Malak El-Hazmi

Block CO-Chair : Dr. Sami Al Nasser

**Tutors Information**

**Year 1 Male Group A**

**Respiratory Block**

|  |  |  |  |
| --- | --- | --- | --- |
| Respiratory Block (Male Group) | | | |
| Name | **Department** | **Extension** | **E-mail** |
| Prof. Saeed Abuelmakarem | Anatomy | 71307 | [sabuelmakarem@ksu.edu.sa](mailto:sabuelmakarem@ksu.edu.sa)  [saaedmakarem@hotmail.com](mailto:saaedmakarem@hotmail.com) |
| Dr. Essam Eldin Salama | 99330 | [esalama@ksu.edu.sa](mailto:esalama@ksu.edu.sa) [/essamco58@yahoo.co.uk](mailto:/essamco58@yahoo.co.uk)  [essamco58@gmail.com](mailto:essamco58@gmail.com) |
| Prof. Ahmed Fatalla | 71314 | [ahmedfathala@hotmail.com](mailto:ahmedfathala@hotmail.com) |
| Dr. Mohammed Vohra | 99329 | [vohra@ksu.edu.sa](mailto:vohra@ksu.edu.sa) |
| Dr. Usman Ghani | Biochemistry | 90140 | [ugresearch@hotmail.com](mailto:ugresearch@hotmail.com) |
| Dr. Ahmed Mujamammi | 9512 | [mujamammi@gmail.com](mailto:mujamammi@gmail.com)  amujamammi@ksu.edu.sa |
| Dr. Ali AlHazmi | Family Medicine | - | ali1hazmi@yhaoo.com |
| Dr. Aly Mohamed | Histology | 79034 | [alymahmed53@hotmail.com](mailto:alymahmed53@hotmail.com) |
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| Dr. Ahmed Al-Barrag | 90818 | [aalbarrag@ksu.edu.sa](mailto:aalbarrag@ksu.edu.sa) /  aalbarraq2@hotmail.com |
| Dr. Abdulkareem Al Hetheel | 71523 | [aalhetheel@ksu.edu.sa](mailto:aalhetheel@ksu.edu.sa)  [abdulkarimfahad@hotmail.com](mailto:abdulkarimfahad@hotmail.com) |
| Prof. Ammar Al-Rikabi | Pathology | 71893 | [Ammar\_rikabi12@yahoo.com](mailto:Amma_rikabi12@yahoo.com) |
| Prof. Mohammad Al Humayyd | Pharmacology | 71350 | humayyd@yahoo.com |
| Dr. Ishfaq Bukhari | 71325 | bukharirph@yahoo.com |
| Dr. Osama Yousif | 71327 | [oymiaharsoul@hotmail.com](mailto:oymiaharsoul@hotmail.com) |
| Dr. Abulrahman Al Howaikan | Physiology | 70848 | [amalhowikan@gmail.com](mailto:amalhowikan@gmail.com) |
| Prof. Sultan Meo | 71604 | [sultanmeo@hotmail.com](mailto:sultanmeo@hotmail.com) |

**WELCOME**

**Dear Students,**

**We are pleased to welcome you in the College of Medicine, Respiratory Block Attachment. We hope you will find this block both useful and enjoyable.**

**Prof. Mona Soliman**

**ccccc**

**Chairman of**

**List of the Problem-Based Learning Cases**

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

|  |  |  |
| --- | --- | --- |
| **Week** | **Case No.** | **Case title** |
| Week 1 | **No case** | |
| Week 2 (Sunday & Wednesday) | Case 1 | “.. I am short of breath” |
| Week 3 (Sunday & Wednesday) | Case 2 | “…still coughing” |
| Week 4 | **No case** | |

**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 Saturday and Tuesday. 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, he/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, students should be able to :**

* Understand the relationship between the anatomical structure of the different components of the respiratory system and their functions.
* Discuss the pathology, microbiology, pathogenesis and factors contributing to the development of most common diseases affecting and respiratory system
* Use basic science to explain patient’s sign and symptoms; interpret investigation results, and provide justification for their views.
* Develop communication skills and explore biopsychosocial and ethical issues in their assessment of the case.
* Use clinical cases to apply knowledge learnt, generate hypotheses, build an enquiry plan, and use evidence to refine their hypothesis, justify different views.
* Design a management plan, and understand the pharmacological basis of drugs used in the management of common diseases affecting the respiratory system.
* Enhance their communication skills, and practice with the help of simulation patients to improve their communication in relation to respiratory case scenarios.

**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
* Practical classes.
* Clinical skills
* Independent learning
* Problem based learning

**Objectives of the lectures:**

|  |
| --- |
| **Title of the lecture: Muscles Involved in Respiration** |

Lecturer’s name : Prof. Ahmed Fathalla - Dr. Sanaa AlShaarawi

Department : Anatomy

Block / week : Respiratory/W1

Email address : [ahmedfathala@gmail.com](mailto:ahmedfathala@gmail.com) - [salsharawi@ksu.edu.sa](mailto:salsharawi@ksu.edu.sa)

=========================================================

**Objectives of the lecture:**

* Describe the components of the thoracic cage and their articulations.
* Describe in brief the respiratory movements.
* List the muscles involved in inspiration and in expiration.
* Describe the attachments of each muscle to the thoracic cage and its nerve supply.
* Describe the origin, insertion, nerve supply of diaphragm.

==========================================================

**Keywords:**

(The thoracic cage, The respiratory movements, Muscles of inspiration**,** Muscles of Expiration, The Diaphragm, Movements of the Ribs, Anterior abdominal wall, Innervations of respiratory muscles,)

**==========================================================**

**Background:**

* The components of the thoracic cage.
* The muscles involved in inspiration.
* The muscles involved in Expiration.

**==========================================================**

**Main concepts in the lecture:**

The thoracic cage Has 2 apertures ;superior (thoracic outlet): narrow, open, continuous with neck and inferior outlet : wide, closed by diaphragm. It is formed of : Sternum & costal cartilages anteriorly ; Twelve pairs of ribs laterally; and twelve thoracic vertebrae posteriorly.

Inspiratory muscles are the Diaphragm (most important muscle) ; Rib elevators: external intercostal muscles and Accessory muscles (only during forced inspiration) as : Muscles attaching cervical vertebrae to first & second rib: scalene muscles and Muscles attaching thoracic cage to upper limb: pectoralis major.

The diaphragm is a musculotendinous partition between thoracic & abdominal cavity; attached to: sternum, costal cartilages,12th rib & lumbar vertebrae; Fibers converge to join the central tendon;

its action: contraction and descent to increase vertical diameter of thoracic cavity , so it is essential for normal breathing .Expiratory muscles act only during forced expiration as rib depressors (Internal intercostal ,Innermost intercostal ,Subcostals, Transversus thoracis) and Anterior abdominal wall muscles ( External oblique,Internal oblique,Transversus abdominis,Rectus abdominis).

**==========================================================**

**Conclusion:**

Inspiratory muscles are the Diaphragm; Rib elevators: external intercostal muscles and Accessory muscles (only during forced inspiration) as : scalene muscles and pectoralis major. Expiratory muscles Act only during forced expiration as rib depressors and anterior abdominal wall muscles.

==========================================================

**Take home messages:**

* Role of inspiratory muscles during breathing.
* Role of expiratory muscles during breathing.
* Anatomy and importance of the diaphragm in breathing.

==========================================================

**Further reading:**

*Clinical Anatomy for Medical Students by Richard S. Snell- Latest Edition.*

*Grant’s Atlas of Anatomy by Anne and Arthur –Latest Edition.*

…………………………………………………………………………………………………………………………………

|  |
| --- |
| Title of the lecture: Functions and organization of the respiratory system |

Lecturer’s name: DR. Aida Korish & DR Abdulrahman Al Howaikan

Department: Physiology departement

Block / week: Respiratory Block / Week 1 to Week 4

Email address: [ahowikan@ksu.edu.sa](mailto:ahowikan@ksu.edu.sa), [akorish@ksu.edu.sa](mailto:akorish@ksu.edu.sa)

========================================================================

**Objectives of the lecture:**

* Describe the structures and respiratory zones functions of the conductive and of airways.
* Understand the difference between internal and external respiration.
* Understand the functions of the respiratory system, including non-respiratory functions,
* Understand clearance mechanism by mucus and cilia, production of surfactant and its physiological significance.

========================================================================

**Background:**

* student already they know the concept of respiratory gas exchange ( taking O2 and Produce CO2. from this knowledge we start to illustrate conductive zone and respiratory zone
* student know the meaning of internal external respiration, based on this background we taught them pulmonary ventilation, diffusion of O2 & Co2, transport of O2 & Co2
* background the alveoli should keep open, from this knowledge we taught them surface tension tends to oppose alveoli expansion and pulmonary surfactant reduces surface tension

========================================================================

**Main concepts in the lecture:**

Functions of the respiratory system

* clearance mechanism by mucus and cilia
* production of surfactant
* Gas exchange
* Phonation
* Pulmonary defense

conductive zone and respiratory zone, internal and external respiration, surface tension pulmonary surfactant

========================================================================

**Conclusion:**

The functions of respiratory system and they differentiate between respiratory zones and conductive zone.

Student recognized the difference between internal and external respiration. Finally they identify the function of surface tension opposite to pulmonary surfactant

========================================================================

**Take home messages:**

* functions of respiratory system
* difference between respiratory zones

The respiratory bronchioles and the alveolar ducts are responsible for the gas exchange

* conductive zone

their function is to filter, warm, and moisten air and conduct it into the lungs

* meaning of internal respiration

Internal respiration or tissue respiration/cellular respiration refers to a metabolic process in which oxygen is released to tissues or living cells and carbon dioxide is absorbed by the blood.

* external respiration

External respiration is basically the transfer of gas between respiratory organs such as lungs and the outer environment.

* function of surface tension and pulmonary surfactant

========================================================================

**Further reading:**

1. [Guyton & Hall: Textbook of Medical Physiology 12E](http://www.studentconsult.com/content/default.cfm?ISBN=9781416045748&home=true)
2. Physiology 5th Edition by linda s costanzo

|  |
| --- |
| Title of the lecture: Mechanics of pulmonary ventilation. |

Lecturer’s name: DR. Aida Korish & Dr Abdulrahman Al Howaikan

Department : Physiology departement

Block / week : Respiratory Block / Week 1 to Week 4

Email address : [ahowikan@ksu.edu.sa](mailto:ahowikan@ksu.edu.sa), [akorish@ksu.edu.sa](mailto:akorish@ksu.edu.sa)

========================================================================

**Objectives of the lecture:**

* List the muscles of respiration and describe their roles during inspiration and expiration.
* Understand the importance of the following pressures in respiration: atmospheric, alveolar, intrapleural, and transpulmonary.
* Explain why intrapleural pressure is always subatmospheric under normal conditions, and the significance of the thin layer of the intrapleural fluid surrounding the lung.
* Define lung compliance and list the determinants of compliance.

========================================================================

**Background:**

* Breathing in is known as inspiration and breathing out is known as expiration
* Breathing consists of two phases, inspiration and expiration. During inspiration, the diaphragm and the intercostal muscles contract. The diaphragm moves downwards increasing the volume of the thoracic (chest) cavity, and the intercostal muscles pull the ribs up expanding the rib cage and further increasing this volume
* Inspiration, during expiration the diaphragm and intercostal muscles relax. This returns the thoracic cavity to it's original volume, increasing the air pressure in the lungs, and forcing the air out.
* Both inhalation and exhalation depend on pressure gradients between the lungs and atmosphere
* Elastic recoil of the lungs after having been stretched by inhalation or relaxed by exhalation to prevent the lungs from collapsing and overstretched

========================================================================

**Main concepts in the lecture:**

We discuss the function of inspiratory and expiratory muscles during rest and force forced expiration and inspiration and the role different pressures inrespiratory system : atmospheric, alveolar, intrapleural, and transpulmonary. Finally we explain the lung compliance which is the magnitude of the change in lung volume produced by a change in pulmonary pressure. It is express the distensible of the lung and we discus how to calculate it mathematically

========================================================================

**Conclusion:**

muscles of respiration and describe their roles during inspiration and expiration, and the role different pressures the concept of distensible of the lung and how to calculate mathematically

========================================================================

**Take home messages:**

* What are inspiratory muscles and expiratory muscles during (resting- forced)
* What is the different types of pressure in the lungs during breathing

========================================================================

**Further reading:**

1. [Guyton & Hall: Textbook of Medical Physiology 12E](http://www.studentconsult.com/content/default.cfm?ISBN=9781416045748&home=true)
2. Physiology 5th Edition by linda s costanzo

|  |
| --- |
| Title of the lecture: Globular Proteins |

Lecturer’s name : Dr. Ahmed Mujamammi and Dr. Sumbul Fatma

Department :Pathology

Block / week :Respiratory/ week 1

Email address : [amujamammi@ksu.edu.sa](mailto:amujamammi@ksu.edu.sa)

[sumbulfatma@gmail.com](mailto:sumbulfatma@gmail.com)

========================================================================

**Objectives of the lecture:**

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

1. Understand the types and functions of globular proteins in the body.

2. Understand the structure-function relationship of globular proteins.

3. Recognize the importance of globular proteins in various body functions including hemoglobin, myoglobin and antibodies, and in disease

========================================================================

**Background:**

* From the Foundation Block lectures the students already have an idea of different shapes attained by polypeptide chains by folding process.
* They know that to be soluble, proteins will have polar residues on the surface and non-polar in the interior of the protein.
* The students have been introduced to the concept that structure translates into function.

========================================================================

**Main concepts in the lecture:**

* Amino acid chains fold into shapes that resemble spheres are called globular proteins.
* Fibrous proteins are mainly insoluble, while globular proteins are soluble structural proteins.
* Hb, Myoglobin, globulines and enzymes are examples of globular proteins.
* Functionally, Hb is for O2 transport.
* HbA, HbA2 and HbF are examples of normal Hb, in which the tetrameric structure is composed of 2α constant subunits with 2 changeable β subunits according to Hb type.
* Glu6 in HbS is replaced by Val, while it is replaced by Lys in HbC.
* Methemoglobinemia is caused by oxidation of Hb, inhibiting O2 binding leading to chocolate cyanosis.
* Thalassemia is caused by a defect in synthesis of either α- or β-globulin chain, as a result of gene mutation.
* α-Thalassemia causes less severe anemia than β-Thalassemia.
* Myoglobin is a globular hemeprotein, which stores and supplies O2 to the heart and muscle only.
* Hb is composed of 4 chains (subunits), while Myoglobin is composed of a single chain.
* Myoglobinuria is a specific marker for muscle injury and may cause acute renal failure.
* Immunoglobulins are defense proteins produced by the B-cells.
* Immunoglobulins consist of 5 types: IgA, IgD, IgE, IgG and IgM.

========================================================================

**Conclusion:**

One of the major globular proteins in the body is hemoglobin. There are several structural forms of hemoglobin that perform oxygen transport. Hemoglobinopathies are a number of abnormalities in the structure of hemoglobin due to genetic mutations. The abnormal hemoglobin molecules are mainly defective in one amino acid that leads to abnormal structure and function. Other globular proteins include myoglobin and immunoglobulins that carry oxygen supply and immune functions respectively.

========================================================================

**Take home messages:**

Globular proteins perform important functions in the body. Abnormality in their structure affects their normal function that leads to a number of diseases.

========================================================================

**Further reading:**

Lippincott’s Biochemistry 6th Edition, pp. 25–42.

|  |
| --- |
| Title of the lecture: Histology of the upper respiratory tract |

Lecturer’s name : Dr. Aly Mohamed Ahmed, Dr. Raeesa Abdel Twab

Department : Anatomy

Block / week : Respiratory

Email address : [alymahmed53@hotmail.com](mailto:alymahmed53@hotmail.com) – drraeesama@gmail.com

========================================================================

**Objectives of the lecture:**

By the end of this lecture the student should be able to describe the microscopic structures of:

1. Vestibule of the nasal cavity.
2. Respiratory mucosa of the nasal cavity.
3. Nasal septum.
4. Olfactory mucosa of the nasal cavity.
5. Mucosa of the paranasal sinuses.
6. Pharynx: with special emphasis on Nasopharynx.
7. Larynx.

========================================================================

**Keywords:**

(microscopic anatomy, respiratory nasal mucosa, olfactory mucosa, paranasal sinuses, nasopharynx, oropharynx, laryngopharynx, larynx)

========================================================================

**Background:**

Studying the microscopic structure of the nasal cavity and paranasal sinuses enable the students to understand the physiological functions of them, and to interpret the histopathological changes in allergic and infectious nasal diseases.

========================================================================

**Main concepts in the lecture:**

Nasal cavity:

It is formed of:

1. Vestibule.
2. Respiratory portion.
3. Olfactory portion.

The microscopic structure of the wall of posterior nasal cavity is formed of:

a. respiratory mucosa and, b. olfactory mucosa. The nasal sinuses are lined with respiratory mucosa similar to that of the nasal cavity.

Pharynx: e.g. Nasopharynx.

Larynx: It is formed of a group of hyaline and elastic cartilage which are connected together by ligaments and muscles. The larynx is lined with mucous membrane.

========================================================================

**Conclusion:**

The lining respiratory epithelium is formed of pseudostratified ciliated columnar epithelium with goblet cells. The wall of the respiratory passages is mainly supported by hyaline cartilage.

========================================================================

**Take home messages:**

Microscopic structure of respiratory epithelium and olfactory epithelium.

========================================================================

**Further reading:**

* Color Textbook of Histology. Gartner L. and Hiatt J. Latest ed.
* Basic Histology. Junqueira et al. Latest ed.

|  |
| --- |
| Title of the lecture: Pulmonary function in health and diseases |

Lecturer’s name : DR. Aida Korish & Dr.Abdulrahman Al Howaikan

Department : Physiology Department

Block / week : Respiratory/ Week 1-4

Email address : [ahowikan@ksu.edu.sa](mailto:ahowikan@ksu.edu.sa) , [akorish@ksu.edu.sa](mailto:akorish@ksu.edu.sa)

========================================================================

**Objectives of the lecture:**

* Describe the structure of the spirometry.
* Identify the physiological factors that influence the pulmonary function tests (PFTs).
* List the different indications of pulmonary function tests (PFTs).
* Compare between PFTs in obstructive and restrictive pulmonary diseases.
* Interpret the changes in PFTs in smokers in comparison to nonsmokers.

========================================================================

**Background:**

|  |
| --- |
| * Static volumes of the lung are measured with a spirometer. * The four lung volumes are tidal volume (Vt), inspiratory reserve volume, expiratory reserve volume and the residual volume (RV) * There are several lung capacities; each lung capacity includes two or more lung volumes * The inspiratory capacity (IC), the functional residual capacity (FRC), the vital capacity (VC), the total lung capacity (TLC).   ======================================================================== |

**Main concepts in the lecture:**

* PFTs differ between people according to physiology conditions: Age, gender, height, weight, ethnic group and pregnancy.
* Indications of spirometry could be based on:

1. Symptoms: Dyspnea, cough, sputum production, chest pain
2. Signs: Cyanosis, clubbing, chest deformity, diminished chest expansion, hyperinflation, diminished breath sounds, Prolongation of expiratory phase & crackles
3. Arterial blood gas analysis: Hypoxemia, hypercapnia
4. Abnormal chest x ray:
5. Describe the course of diseases affecting PFTs: e.g neuromuscular diseases, pulmonary diseases, Obstructive airway diseases, interstitial lung diseases
6. To access the Adverse reactions of drugs with known pulmonary toxicity [Pulmonary fibrosis]
7. To assess the therapeutic interventions e.g. Bronchodilator therapy, Steroid treatment for asthma
8. To differentiate between chronic obstructive lung disease and interstitial lung disease.
9. Pre operative indications:
10. To determine the suitability for and management during and after anesthesia
11. To assess the risk for surgical procedures known to affect lung function

========================================================================

**Conclusion:**

* Spirometry is a widely used, effort depended basic lung function test
* Assess the lung performance
* Assess physiological parameters; lung volumes, capacities & flow rate.
* Differentiate between the obstructive and restrictive lung conditions.
* Play a critical role in the diagnosis, differentiation and management of respiratory illness.

========================================================================

**Take home messages:**

* In normal healthy nonsmoker subject after the age of 30 the expected decline in Lung function parameter [FEV1] is 25–30 ml/ year.
* The average rate of decline of lung function in smokers as measured by Forced Expiratory Volume in 1 sec [FEV1] is 60-70 ml / year.

========================================================================

**Further reading:**

1. Text Book of Medical Physiology. Arthur C. Guyton, M.D. 11th Edition Unit VII, Chapter 41 Pages 514- 523.
2. Physiology. Linda. S. Costanzo 3rd edition Page 223-2026.

|  |
| --- |
| Title of the lecture: Functions and organization of the respiratory system |

Lecturer’s name: DR. Aida Korish & DR Abdulrahman Al Howaikan

Department: Physiology departement

Block / week: Respiratory Block / Week 1 to Week 4

Email address: [ahowikan@ksu.edu.sa](mailto:ahowikan@ksu.edu.sa), [akorish@ksu.edu.sa](mailto:akorish@ksu.edu.sa)

=======================================================================

**Objectives of the lecture:**

* Describe the structures and respiratory zones functions of the conductive and of airways.
* Understand the difference between internal and external respiration.
* Understand the functions of the respiratory system, including non-respiratory functions,
* Understand clearance mechanism by mucus and cilia, production of surfactant and its physiological significance.

**========================================================================**

**Background:**

* student already they know the concept of respiratory gas exchange ( taking O2 and Produce CO2. from this knowledge we start to illustrate conductive zone and respiratory zone
* student know the meaning of internal external respiration, based on this background we taught them pulmonary ventilation, diffusion of O2 & Co2, transport of O2 & Co2
* background the alveoli should keep open, from this knowledge we taught them surface tension tends to oppose alveoli expansion and pulmonary surfactant reduces surface tension

==========================================================================

**Main concepts in the lecture:**

* Functions of the respiratory system
* clearance mechanism by mucus and cilia
* production of surfactant
* Gas exchange
* Phonation
* Pulmonary defense
* conductive zone and respiratory zone, internal and external respiration, surface tension pulmonary surfactant

=====================================================================

**Conclusion:**

The functions of respiratory system and they differentiate between respiratory zones and conductive zone.

Student recognized the difference between internal and external respiration. Finally they identify the function of surface tension opposite to pulmonary surfactant

========================================================================

**Take home messages:**

* functions of respiratory system
* difference between respiratory zones
* The respiratory bronchioles and the alveolar ducts are responsible for the gas exchange
* conductive zone
* their function is to filter, warm, and moisten air and conduct it into the lungsmeaning of internal respiration
* Internal respiration or tissue respiration/cellular respiration refers to a metabolic process in which oxygen is released to tissues or living cells and carbon dioxide is absorbed by the blood.
* external respiration

External respiration is basically the transfer of gas between respiratory organs such as lungs and the outer environment.

* function of surface tension and pulmonary surfactant

**Further reading:**

1. [Guyton & Hall: Textbook of Medical Physiology 12E](http://www.studentconsult.com/content/default.cfm?ISBN=9781416045748&home=true)
2. Physiology 5th Edition by linda s costanzo

|  |
| --- |
| Title of the lecture: Nose, Nasal cavity, Paranasal Sinuses & Pharynx |

Lecturer’s name :Dr. Jamila EL medany & Dr. Essam Eldin Salama

Department :Anatomy

Block / week :Respiratory / W1

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**Objectives of the lecture:**

* Describe the boundaries of the nasal cavity.
* Describe the nasal conchae and meati.
* Demonstrate the openings in each meatus.
* Describe the paranasal sinuses and their functions
* Describe the pharynx, its parts, and the related structures.

=====================================================================

**Keywords:**

(Nasal cavity. nasal conchae …, meati. paranasal sinuses …, pharynx,)

========================================================================

**Background:**

* The nasal cavity; extension, divisions , its relation, blood and nerve supply , lymphatic drainage
* Paranasal sinuses, anatomy and function
* Pharynx; extension, divisions , its relation, blood and nerve supply , lymphatic drainage

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**Main concepts in the lecture:**

* The nasal cavity extends from the external (anterior) nares to the posterior nares (choanae). Divided into right & left halves by the nasal septum. Each half has a roof , lateral wall medial wall (septum) and floor
* The roof is narrow & formed (from behind forward) by the: Body of sphenoid.Cribriform plate of ethmoid bone. Frontal bone.Nasal bone & cartilage
* The floor separates it from the oral cavity. It is formed by the hard (bony) palate.
* Medial Wall (Nasal Septum) Osseocartilaginous partition, formed by: perpendicular plate of ethmoid bone. vomer, and septal cartilage.
* Lateral Wall shows three horizontal bony projections, the superior, middle & inferior conchae, the cavity below each concha is called a meatus and are named as superior, middle & inferior corresponding to the conchae the small space above the superior concha is the sphenoethmoidal recess, the conchae increase the surface area of the nasal cavity, the recess & meati receive the openings of the paranasal sinuses, and nasolacrimal duct.
* Nerve supply of the nasal cavity; olfactory mucosa supplied by olfactory nerves, nerves of general sensation are derived from; ophthalmic, and maxillary division of the trigeminal nerve, anterior ethemoidal nerve, nasal, nasopalatine and palatine branches of the pterygopalatine ganglion.
* Arterial Supply: Branches of theMaxillary; sphenopalatine artery Facial; superior labial & Ophthalmic; ethmoidal arteries.The arteries make a rich anastomosis in the region of the vestibule, and anterior portion of the septum.Venous Drainage:Submucosal plexus by veins accompany the arteries which drain into the facial, ophthalmic, and spheno-palatine veins.
* Lymphatic drainage; the lymphatics from the vestibule drains into the submandibular lymph nodes, rest of the cavity drains into the upper deep cervical lymph nodes.
* The paranasal sinuses; air filled cavities located in the bones around the nasal cavity: ethmoid, sphenoid, frontal bones & maxillae. They are lined by respiratory mucosa which is continuous with the mucosa of the nasal cavity, drain into the nasal cavity. Functions of the paranasal sinuses; lighten the skull, act as resonant chambers for speech, air conditioning. The respiratory mucosal lining helps in warming, cleaning and moistening the incoming air.
* The pharynx; is a muscular tube lying behind the nose, oral cavity & larynx, it extends from the base of the skull to level of the 6th cervical vertebra, where it is continuous with the esophagus, its anterior wall is deficient and shows (from above downward), the posterior nasal apertures, opening of the oral cavity, laryngeal inlet, its muscles are arranged in circular layer; Superior constrictor, Middle constrictor & Inferior constrictor, they overlap each other. The longitudinal layer; Stylopharyngeus Salpingopharyngeus and Palatpharyngeous. The pharynx is divided into three parts:Nasopharynx, Oropharynx, and Laryngopharynx.
* The naso pharynx Extends from the base of skull to the soft palate.communicates with the nasal cavity through posterior nasal aperturesPharyngeal tonsils (adenoides) present in the submucosa covering the roof.Lateral wall shows:Opening of auditory tube.Tubal elevation (produced by posterior margin of the auditory tube).Tubal tonsil. Salpingopharyngeal fold (raised by salpingo-pharyngeus muscle).Paryngeal recess
* The oropharynx; Lies behind the mouth cavity, communicates with the oral cavity through theoropharyngeal isthmusExtends from soft palate to upper border of epiglottis.Lateral wall shows:Palatopharyngeal folds.Palatoglossal foldPalatine tonsil located between them in a depression called the ‘tonsillar fossa’.
* The laryngopharynx; Lies behind the laryngeal inlet & the posterior surface of larynx communicates with the larynx through the laryngeal inletExtends from upper border of epiglottis to lower border of cricoid cartilage.A small depression situated on either side of the laryngeal inlet is called ‘piriform fossa’.It is a common site for the lodging of foreign bodies.Branches of *internal laryngeal & recurrent laryngeal nerve* lie deep to the mucous membrane of the fossa and are vulnerable to injury during removal of a foreign body.
* Nerve supply; Sensory: Nasopharynx, Maxillary nerve, Oropharynx: Glossopharyngeal nerve Laryngopharynx: Vagus nerve, Motor Nerve Supply; all the muscles of pharynx are supplied by the pharyngeal plexus. except ; the Stylopharyngeus is supplied by the glossopharyngeal nerve
* Arterial supply; from branches of ascending pharyngeal artery, ascending palatine artery, facial artery, maxillary artery, and Lingual artery, the Veins drain into pharyngeal venous plexus, which drains into the internal jugular vein The lymphatics drain into the deep cervical lymph nodes either directly, or indirectly via the retropharyngeal or Para tracheal lymph nodes

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**Conclusion:**

* The nasal cavity; extension, divisions , its relation, blood and nerve supply , lymphatic drainage
* Paranasal sinuses, anatomy and function
* Pharynx; extension, divisions , its relation, blood and nerve supply , lymphatic drainage

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**Take home messages:**

* The nose
* The paranasal sinuses
* The pharynx

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**Further reading:**

Richard S. Snell, Clinical anatomy, 7th edition

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| --- |
| Title of the lecture: Embryology of the respiratory system |

Lecturer’s name : Dr. Muhammad Saeed Vohra – Dr. Sanaa Al Shaarawi

Department : Anatomy

Block / week : Respiratory

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**Objectives of the lecture:**

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

* Describe the development of the larynx
* Describe the development of the trachea
* Describe the development of the lungs

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**Keywords:**

(embryology, development of the larynx., development of the trachea, respiratory system, laryngotracheal tube, stages of lung development)

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**Background:**

* The respiratory system is one of the systems that develop from endoderm.

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**Main concepts in the lecture:**

* Development of larynx and trachea:
* Laryngotracheal groove; Laryngotracheal diverticulum; tracheo-esophageal septum, primordium for trachea, bronchi and lungs
* Development of the laryngeal cartilages
* Recanalization of laryngeal lumen
* Development of bronchi and lungs:
* Endodermal lung bud, splanchnic mesoderm
* Visceral and parietal pleura
* Maturation of lung
* Pseudoglandular period
* Canalicular period
* Terminal sac period
* Alveolar period
* Postnatal maturation of lung
* Breathing movements
* Factors important for normal development of lung
* Common anomalies: Tracheoesophageal Fistula

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**Conclusion:**

The respiratory tract mainly develops from an endodermal outgrowth called the respiratory diverticula derived from the floor of the primitive pharynx. The diverticulum gives rise to the larynx, trachea and bronchial tree. Development of the lungs follows several stages before birth and continues after birth.

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**Take home messages:**

The mucous lining of the respiratory system is derived from the endoderm of the floor of the primitive pharynx. Development of the lungs continues postnatally.

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**Further reading:**

Moore and Persaud: The developing human – Clinically oriented embryology - Latest edition

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| --- |
| Title of the lecture: Respiratory ventilation |

Lecturer’s name: DR. Aida Korish & DR Abdulrahman Al Howaikan

Department: Physiology department

Block / week: Respiratory Block / Week 1

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**Objectives of the lecture:**

* Define the various lung volumes and capacities and provide typical values for each.
* Define ventilation rate, their typical values, and their measurement.
* Describe FEV1 and its role in differentiating obstructive and restrictive lung diseases.
* Describe the types of dead space. State a volume for the anatomical dead space.
* Define the term minute ventilation and state a typical value.
* Distinguish minute ventilation from alveolar ventilation.

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**Background:**

movement of air into and out of the lungs, can be recorded by a method called spirometry

accessory muscles functioning during forced breathing

two common respiratory abnormalities This ratio differentiate obstructive and restrictive lung diseases

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**Main concepts in the lecture:**

Students learned the normal standard values of various lung volumes and capacities in addition, they do some mathematical calculation ; ventilation rate, minute ventilation and FEV1/FVC ratio . they differentiate between obstructive and restrictive lung diseases. And how to estimate dead space.

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**Conclusion:**

Student learned movement of air into and out of the lungs, can be measured by spirometry, values of various lung volumes and capacities and they differentiate between obstructive and restrictive lung diseases

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**Take home messages:**

The different between male and female in the values of various lung volumes and capacities

Commen obstructive and restrictive lung diseases

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**Further reading:**

1. [Guyton & Hall: Textbook of Medical Physiology 12E](http://www.studentconsult.com/content/default.cfm?ISBN=9781416045748&home=true)
2. Physiology 5th Edition by linda s costanzo

|  |
| --- |
| Title of the lecture: Lung and Pleura |

Lecturer’s name; Dr. SanaaAlShaarawi, Prof. Saeed Abuel Makarem

Department: Anatomy

Block / week: Respiratory Block

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**Objectives of the lecture:**

* Describe the anatomy of the pleura regarding parietal & visceral pleurae.
* List the parts of parietal pleura and its recesses.
* Describe the surface anatomy of both pleurae and lungs.
* Describe the anatomy of lungs : shape, relations, nerve supply & blood supply.
* Describe the difference between right & left lungs.
* Describe the formation of bronchopulmonary segments and the main characteristics of each segment in the lung.

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**Keywords:**

( Pleural subdivisions, Pleural Recesses, Relations of the Lungs, Innervations of lung, Blood supply of lung, Surface Anatomy of Lung, Surface Anatomy of Pleura, Difference between the lungs, Bronchopulmonary segments)

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**Background:**

* Importance of the anatomy of both lung and pleura.
* Clinical importance related to both lung and pleura.
* Important relations of both lungs.
* Differences between both lungs.

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**Main concepts in the lecture:**

…The pleura has two layers; Parietal layer, which lines the thoracic walls and Visceral layer, which covers the surfaces of the lung. The costodiaphragmatic is a slit like space between costal and diaphragmatic pleurae.

The costomediastinal recess is a slit like space between costal and mediastinal pleurae.Pleural Effusion is an abnormal accumulation of pleural fluid about 300 ml, in the *Costodiaphragmatic pleural recess* , (normally 5-10 ml fluid) ……

… Each lung has apex and base: identify the top and bottom of the lung, respectively;

Costal surface: surrounded by the ribs from front & back) and Medial surface where the bronchi, blood vessels, and lymphatic vessels enter the lung at the hilum. Anterior border of left lung presents a cardiac notch at its lower end.

The right lung has 2 fissures and 3 lobes but the left one has one fissure and 2 lobes. At the root of lung; the right lung has 2 bronchus; but the left one has one bronchus only. Inervations of the lung by autonomic nervous system through pulmonary plexus. Bronchopulmonary segments are the anatomic, functional, and surgical units of the lungs.

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**Conclusion:**

The pleura has two layers; Parietal layer, which lines the thoracic walls and Visceral layer, which covers the surfaces of the lung. Costodiaphragmatic pleural recess is a slit like space between costal and diaphragmatic pleurae.

The right lung has 2 fissures and 3 lobes but the left one has one fissure and 2 lobes and cardiac notch at its anterior border. Innervations of the lung by autonomic nervous system and blood supply by bronchial and pulmonary vesseles.

========================================================================

**Take home messages:**

* The parts of parietal pleura and its recesses.
* Describe the surface anatomy of both pleurae and lungs.
* Describe the anatomy of lungs.
* Formation of bronchopulmonary segments and the main characteristics of each segment in the lung.

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**Further reading:**

*Clinical Anatomy for Medical Students by Richard S. Snell- Latest Edition.*

*Grant’s Atlas of Anatomy by Anne and Arthur –Latest Edition.*

|  |
| --- |
| Title of the lecture: Histology of trachea, bronchi and lung |

Lecturer’s name : Dr. Aly Mohamed Ahmed, Dr. Raeesa Abdel Twab

Department : Anatomy

Block / week : Respiratory

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**Objectives of the lecture:**

By the end of this lecture, the student should be able to describe:

1. The microscopic structures of the walls of:

* Trachea.
* Primary or extrapulmonary bronchi.
* Intrapulmonary (secondary and tertiary) bronchi.
* Bronchioles.

1. The microscopic structures of:

* Interalveolar septum.
* Alveolar phagocytes.
* Pleura.

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**Keywords:**

(microscopic anatomy, extrapulmonary bronchus, intrapulmonary bronchus, terminal bronchioles, respiratory bronchioles, alveolar epithelium, trachea)

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**Background:**

Studying the microscopic structure of the wall of different respiratory passages enable the students to understand the physiological processes occurring through the walls of trachea and bronchi and interpret the histopathological changes in their wall especially in cases of bronchial asthma and bronchitis.

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**Main concepts in the lecture:**

The microscopic structure of the wall of the trachea and the bronchi is formed of 3 coats; mucosa, Submucosa and adventitia that housing hyaline cartilage. The structural character of the trachea and bronchi is essential for their functions. The wall of the trachea and bronchi is formed of:

1. Mucosa: pseudostratified columnar ciliated epithelium with goblet cells.
2. Submucosa: formed of loose vascular connective tissue with lymphoid elements.
3. Adventitia: formed of fibrocartilagenous connective tissue.

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**Conclusion**:

The lining respiratory epithelium is formed of pseudostratified ciliated columnar epithelium with goblet cells. The wall of the respiratory passages is mainly supported by hyaline cartilage. The alveolar epithelium is formed of type I pneumocytes and type II pneumocytes. The interalveolar septum is rich in continuous blood capillaries (pulmonary capillaries) and elastic fibers.

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**Take home messages:**

* Microscopic structure of respiratory epithelium and olfactory epithelium.
* Microscopic structure of the wall of trachea and bronchi.
* Microscopic structure of the wall of bronchioles.
* Microscopic structure of interalveolar septum in correlation with function.

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**Further reading:**

(Color Textbook of Histology. Gartner L. and Hiatt J. Latest ed.

Basic Histology. Junqueira et al. Latest ed.

|  |
| --- |
| Title of the lecture: Effects of exercise on the respiratory system |

Lecturer’s name: DR. Aida Korish & DR Abdulrahman Al Howaikan

Department: Physiology departement

Block / week: Respiratory Block / Week 1 to Week 4

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**Objectives of the lecture:**

* Describe the effects of moderate and severe exercise on oxygen consumption, and ventilation volumes.
* Describe the effects of exercise on arterial PO2, PCO2 and H+ ions.
* Define the diffusing capacity of the respiratory membrane, and its typical values at rest, and explain its changes in exercise.
* Explain causes of hyperventilation in exercise.

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**Background:**

* students know the differences between exercise intensities ( low , moderate and vigorous exercise)
* students know rest breathing fequency ,oxygen consumption and pulmonery ventilation
* influence of exercise on the ratio of alveolar ventilation to pulmonary blood flow per minute (V/Q ratio)
* control of breathing by respiratory center during rest and forced breathing
* CO2 carried mainly by post anaerobic threshold extra CO2 production over that produced therefore hyperventilation increased to washout the extra CO2 .
* Different between energy of carbohydrate and fat

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**Main concepts in the lecture:**

oxygen consumption influenced by exercise intensities Diffusing capacity for oxygen at rest 21ml/min/mmHg differ during exercise 65ml/min/mmHg . Respiration is stimulated mainly by neurogenic mechanisms during exercise and sensory signals transmitted into the respiratory center from the contracting muscles and moving joints finally buffering H+ ions by bicarbonate results in extra CO2 production over that produced by aerobic metabolism. Finally ,the different energy sources during exercise.

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**Conclusion:**

low, moderate and vigorous exercise influence the amount of oxygen consumption

Diffusing capacity for oxygen at rest lower than diffusing capacity at efforts

neurogenic mechanisms during exercise factor control hyperventilation during exercise

amount of energy produce by different energy sources

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**Take home messages:**

oxygen consumption of different sports activities

noninvasive method for determined anaerobic threshold break point.

Accumulation of lactic acid post vigorous exercise

energy produce by carbohydrate and fat during rest and maximal efforts

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**Further reading:**

[Guyton & Hall: Textbook of Medical Physiology 12E](http://www.studentconsult.com/content/default.cfm?ISBN=9781416045748&home=true)

Physiology 5th Edition by linda s costanzo

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| --- |
| Title of the lecture: Transport of Oxygen and carbon dioxide |

Lecturer’s name : DR. Aida Korish & Abdulrahman Al Howaikan

Department : Physiology Department

Block / week : Respiratory/ Week 1-4

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**Objectives of the lecture:**

* Identify the forms of oxygen transport in the blood, the importance of each.
* Contrast between O2 capacity, O2 content and O2 saturation.
* Interpret the relation of PO2 and %HbO2 (Oxygen- hemoglobin dissociation curve)
* Define the P50 and justify its significance.
* Interpret the effect of DPG, temperature, H+ ions and PCO2 on the affinity of O2 to Hemoglobin.
* Describe the three forms of carbon dioxide that are transported in the blood, and the chloride shift.

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**Background:**

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| --- |
| * Hemoglobin is a globular protein consisting of four subunits. Each subunit contains a heme moiety, which is an iron-binding porphyrin. * Adult hemoglobin (hemoglobin A) is called α2β2; each subunit can bind one molecule of O2, for a total of four molecules of O2 per molecule of hemoglobin. * When hemoglobin is oxygenated, it is called oxyhemoglobin; when it is deoxygenated, it is called deoxyhemoglobin. For the subunits to bind O2, iron in the heme moieties must be in the ferrous state (i.e., Fe2+). * There are several variants of hemoglobin. Methemoglobin, Fetal hemoglobin and Hemoglobin S   ======================================================================== |

**Main concepts in the lecture:**

* O2 is carried in two forms in blood: dissolved and bound to hemoglobin.
* Dissolved O2 accounts for approximately 2% of the total O2 content of blood. It O2 is the *only form* of O2 that produces a partial pressure, which, in turn, drives O2 diffusion.
* The remaining 98% of the total O2 content is reversibly bound to hemoglobin inside the red blood cells.
* The O2 content of blood is primarily determined by the hemoglobin concentration and by the O2-binding capacity of hemoglobin.
* The O2-binding capacity is the maximum amount of O2 that can be bound to hemoglobin per volume of blood.
* The O2 content is the actual amount of O2 per volume of blood. The O2 content can be calculated from the O2-binding capacity of hemoglobin and the percent saturation of hemoglobin, plus any dissolved O2.
* Percent saturation of hemoglobin is a function of the Po2 of blood, as described by the O2-hemoglobin dissociation curve.
* The curve is sigmoid shape so the percent saturation of heme sites does not increase linearly as Po2 increases.
* P50 is the Po2 at which hemoglobin is 50% saturated. A change in the value of P50 is used as an indicator for a change in affinity of hemoglobin for O2. An increase in P50 reflects a decrease in affinity, and a decrease in P50 reflects an increase in affinity.
* The O2-hemoglobin dissociation curve can shift to the right or shift to the left. Such shifts reflect changes in the affinity of hemoglobin for O2 and produce changes in P50.
* Shifts of the O2-hemoglobin dissociation curve to the right occur when there is decreased affinity of hemoglobin for O2
* Shifts of the O2-hemoglobin dissociation curve to the left occur when there is increased affinity of hemoglobin for O2.
* CO2 is carried in the blood in three forms: dissolved CO2, carbaminohemoglobin (CO2 bound to hemoglobin), and as bicarbonate (HCO3-). *HCO3-* *is quantitatively the most important* of these forms.
* In the tissues, CO2 generated from aerobic metabolism is added to systemic capillary blood, converted to HCO3- by carbonic anhydrase, and transported to the lungs. In the lungs, HCO3- is reconverted to CO2 and expired.

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**Conclusion:**

* O2 is mostly transported in the blood bound to haemoglobin.If the PO2 increases Hb binds O2.
* If PO2 decreases Hb releases O2.
* The O2 content of blood is primarily determined by the hemoglobin concentration and by the O2-binding capacity of that hemoglobin.
* CO2 is carried in the blood in three forms. *HCO3-* is quantitatively the most important of these forms

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**Take home messages:**

* Oxygen is carried to the tissues in both physical solution and a bind forms to Hb. The greater form is the oxyhemoglobin.
* Carbon dioxide is transported in three forms, of which *HCO3 is* the greatest form formed mainly inside the red blood vessels.
* The amount of O2 delivered to tissues is determined by blood flow and the O2 content of blood.
* O2 delivery = cardiac output X O2 content of the blood = cardiac output x (Dissolved O2 + O2- hemoglobin).

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**Further reading:**

1. Text Book of Medical Physiology. Arthur C. Guyton, M.D. 11th Edition Unit VII, Chapter 40 Pages 502- 513.
2. Physiology. Linda. S. Costanzo 3rd edition Page 209-2017.

|  |
| --- |
| Title of the lecture: Gas Transfer (Diffusion of O2 and CO2) |

Lecturer’s name :Dr. Aida Korish & Dr.Abdulrahman Al Howaikan

Department : Physiology Department

Block / week : Respiratory/ Week 1-4

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**Objectives of the lecture:**

* Define the partial pressure of a gas and explain how it is influenced by altitude.
* Explain how the pressure exerted by each gas in a mixture of gases is independent of the pressure exerted by the other gases (Dalton's Law)
* Interpret that gases in a liquid diffuse from higher partial pressure to lower partial pressure (Henry’s Law)
* Describe the components of the alveolar-capillary membrane andAnalyze the factors that determine the concentration of a gas in a liquid.
* Correlate the various factors determining gas transfer: -Surface area, thickness, partial pressure difference, and diffusion coefficient of gas
* Compare between the partial pressures of oxygen and carbon dioxide in the atmosphere, alveolar gas, at the end of the pulmonary capillary, in systemic capillaries, and at the beginning of a pulmonary capillary

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**Background:**

* Gas exchange in the respiratory system refers to diffusion of O2 and CO2 in the lungs and in the peripheral tissues.
* O2 is transferred from alveolar gas into pulmonary capillary blood and, ultimately, delivered to the tissues, where it diffuses from systemic capillary blood into the cells.
* CO2 is delivered from the tissues to venous blood, to pulmonary capillary blood, and is transferred to alveolar gas to be expired.

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**Main concepts in the lecture:**

* The pulmonary capillaries are perfused with blood from the right heart.
* Gas exchange then occurs between alveolar gas and the pulmonary capillary.
* Transfer of gases across cell membranes or capillary walls occurs by simple diffusion.
* The rate of transfer of gases by diffusion (Vx) is directly proportional to the driving force, a diffusion coefficient, and the surface area available for diffusion and is inversely proportional to the thickness of membrane barrier.
* The values for Po2 and Pco2 (mmHg) show differences between the dry inspired air, humidified tracheal air, alveolar air, and pulmonary capillary blood. The PaO2 in the arterial blood actually is slightly less than 100 mm Hg because of the physiologic shunt.
* Oxygen concentration in the atmosphere is 21%, so PO2 in atmosphere = 760 mmHg x 21% = 160 mmHg.
* This mixes with “old” air already present in alveolus to arrive at PO2 of 104 mmHg in alveoli.
* Carbon dioxide concentration in the atmosphere is 0.04%, so PCO2 in atmosphere =760 mmHg x 0.04% = 0.3 mmHg. This mixes with high CO2 levels from residual volume in the alveoli to arrive at PCO2 of 40 mmHg in the alveoli.
* At resting condition 250 ml of oxygen enter the pulmonary capillaries/min at ventilator rate of 4.2 L/min.
* During exercise 1000 ml of oxygen is absorbed by the pulmonary capillaries per minute, the rate of alveolar ventilation must increase 4 times to maintain the alveolar PO2 at the normal value of 104 mmHg.
* Normal rate of CO2 excretion is 200 ml/min, at normal rate of alveolar ventilation of 4.2 L/min.

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**Conclusion:**

* The driving force for diffusion of a gas is the partial pressure difference of the gas (ΔP) across the membrane, *not* the concentration difference.
* If the Po2 of alveolar air is 100 mm Hg and the Po2 of mixed venous blood that enters the pulmonary capillary is 40 mm Hg, then the partial pressure difference is 60 mm Hg (100 mm Hg - 40 mm Hg).
* The diffusion coefficient of a gas (D) is a combination of the usual diffusion coefficient, which depends on molecular weight and the solubility of the gas.

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**Take home messages:**

* Diffusion of the important respiratory gases across the respiratory membrane is according to the pressure difference.
* The higher the diffusion coefficient of the gas the better will be its diffusion.
* The diffusion coefficient of the gas has enormous implications for its diffusion rate, as illustrated by differences in the diffusion rates of CO2 and O2.
* The diffusion coefficient for CO2 is approximately 20 times higher than the diffusion coefficient for O2; as a result, for a given partial pressure difference, CO2 diffuses approximately 20 times faster than O2.

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**Further reading:**

1. Text Book of Medical Physiology. Arthur C. Guyton, M.D. 11th Edition Unit VII, Chapter 39 Pages 491- 501.
2. Physiology. Linda. S. Costanzo 3rd edition Page 202-2010.

|  |
| --- |
| Title of the lecture:Control of Breathing |

Lecturer’s name : DR. Aida Korish & Dr.Abdulrahman Al Howaikan

Department : Department of Physiology

Block / week : Respiratory/ Week 1-4

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**Objectives of the lecture:**

* Explain the role of the medulla oblongata in determining the basic pattern of breathing.
* List some factors that can modify the basic breathing pattern like e.g.

1. The Hering-Breuer reflexes,
2. The proprioreceptor reflexes,
3. The protective reflexes, like the irritant, and the J-receptors.

* Understand the respiratory consequences of changing PO2, PCO2, and PH.
* Describe the locations and the role of the peripheral and central chemoreceptors.
* Compare and contrast metabolic and respiratory acidosis and metabolic and respiratory alkalosis.

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**Background:**

* The volume of air inspired and expired per unit time is tightly controlled.
* Breathing is controlled both with respect to frequency of breaths and to tidal volume.
* Breathing is regulated so the PaO2 and PaCO2 remain within the normal range, even in exercise.

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**Main concepts in the lecture:**

* Breathing is controlled by centers in the brain stem. There are four components to this control system: (1) chemoreceptors for O2 or CO2, (2) mechanoreceptors in the lungs and joints, (3) control centers for breathing in the brain stem (medulla and pons), and (4) the respiratory muscles, whose activity is directed by the brain stem centers .
* Voluntary control can also be exerted by commands from the cerebral cortex (e.g., breath-holding or voluntary hyperventilation), which can temporarily override the brain stem.
* Breathing is an involuntary process that is controlled by the medulla and pons of the brain stem.
* The medullary respiratory center is located in the reticular formation and is composed of two groups of neurons that are distinguished by their anatomic location: the inspiratory center (dorsal respiratory group) and the expiratory center (ventral respiratory group).
* Stimulation of the apneustic center in the lower pons excites the inspiratory center in the medulla, prolonging the period of action potentials in the phrenic nerve, and thereby prolonging the contraction of the diaphragm.
* The pneumotaxic center turns off inspiration, limiting the burst of action potentials in the phrenic nerve.
* The brain stem controls breathing by processing sensory (afferent) information and sending motor (efferent) information to the diaphragm. Of the sensory information arriving at the brain stem, the most important is that concerning PaO2, PaCO2, and arterial pH.
* The brain stem chemoreceptors are highly sensitive to changes in the pH of cerebrospinal fluid (CSF). Decreases in the pH of CSF produce increases in breathing rate (hyperventilation), and increases in the pH of CSF produce decreases in breathing rate (hypoventilation)
* There are peripheral chemoreceptors for O2, CO2, and H+ in the carotid bodies located at the bifurcation of the common carotid arteries and in the aortic bodies above and below the aortic arch.

Information about arterial Po2, Pco2, and pH is relayed to the medullary inspiratory center via CN IX and CN X, which orchestrates an appropriate change in breathing rate.

* In addition to chemoreceptors, several other types of receptors are involved in the control of breathing, including lung stretch receptors, joint and muscle receptors, irritant receptors, and juxtacapillary (J) receptors.

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**Conclusion:**

* The main function of the control mechanisms of respiration is to maintain a normal PO2, PCO2. And PH even in conditions of exercise or abnormal surrounding barometric pressures (high and Low altitudes).
* The control of respiration is accomplished by neural and chemical mechanisms.
* Neural mechanisms involves pontine centers) and Medullary centers.
* The chemical regulation involves a peripheral chemoreceptors, and PH) and central chemoreceptors. Take home messages:
* Breathing is an involuntary process that is controlled by the medulla and pons of the brain stem.
* Commands from the cerebral cortex can temporarily override the automatic brain stem centers.
* When PO2 is VERY low (Hypoxia), ventilation increases
* The most important regulator of ventilation is PCO2, small increases in PCO2, greatly increases ventilation
* As hydrogen ions increase (acidosis), alveolar ventilation increases.

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**Further reading:**

1. Text Book of Medical Physiology. Arthur C. Guyton, M.D. 11th Edition Unit VII, Chapter 41 Pages 514- 523.
2. Physiology. Linda. S. Costanzo 3rd edition Page 223-2026.

|  |
| --- |
| Title of the lecture: Hypoixia and cyanosis |

Lecturer’s name : Dr. Aida Korish & Dr. Abdulrahman Al Howaikan

Department : Physiology Department

Block / week : Respiratory/ Week 1-4

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**Objectives of the lecture:**

* Identify hypoxia and list its various physiological and pathological causes.
* Compare between hypo and hyper-ventilation in terms of arterial PCO2 and PO2.
* Identify cyanosis and its clinical presentation.
* Interpret the relation between ventilation/perfusion (V͎/Q) and its normal values.

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**Background:**

* Some respiratory diseases result from inadequate ventilation and others result from abnormal diffusion. Almost any of the respiratory problems can cause serious degrees of body wide cellular hypoxia.
* The oxygen therapy is of different degrees of importance in different types of hypoxia.
* Therefore, it is important to understand the different types of hypoxia and the physiologic principles of oxygen therapy.

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**Main concepts in the lecture:**

* Hypoxia s defined as deficiency of oxygen in the tissue cells.

The following is a descriptive classification of the causes of hypoxia:

* Hypoxic or arterial hypoxia: due to Alveolar hypoventilation, Diffusion abnormalities, Right to left shunt or Ventilation-perfusion imbalance.
* Anemic hypoxia: It is caused by reduction in the oxygen carrying capacity of the blood, due to decreased amount of Hb or abnormal type of Hb.
* Stagnant hypoxia: Caused by reduced blood flow through the tissues.
* Histiotoxic hypoxia: This is inability of the tissues to use oxygen due to inhibition of the oxidative enzyme activity.
* Oxygen Therapy in Different Types of Hypoxia

Is by giving oxygen therapy in a tent or high oxygen tension mask.

* This is useful in hypoxic hypoxia, but of less value in other types of hypoxia .Histiotoxic hypoxia will not benefit from O2 therapy.
* The term *cyanosis* means blueness of the skin, and its cause is excessive amounts of deoxygenated hemoglobin in the skin blood vessels especially in the capillaries.
* Ventilation perfusion ration is the ratio of alveolar ventilation to pulmonary blood flow per minute.

The alveolar ventilation at rest (4.2 L/min) and the pulmonary blood flow is equal to right ventricular output per minute (5L/min)

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**Conclusion:**

* The symptoms of hypoxia varies from impairment of judgment to even coma and death.
* Oxygen therapy is useful in hypoxic hypoxia, but of less value in other types of hypoxia.
* Histiotoxic hypoxia will not benefit from O2 therapy.

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**Take home messages:**

* Hypercapnia usually occurs in association with hypoxia only when the hypoxia is caused by *hypoventilation* or *circulatory deficiency*.
* A person with *anemia* almost never becomes cyanotic because there is not enough hemoglobin for 5 grams to be deoxygenated in 100 milliliters of arterial blood.
* Conversely, in a person with excess red blood cells, as occurs in *polycythemia vera*, the great excess of available hemoglobin that can become deoxygenated leads frequently to cyanosis.
* COPD is the most prevalent cause of pulmonary disability today, lung effectiveness as a gas exchange organ may decrease.

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**Further reading:**

1. Text Book of Medical Physiology. Arthur C. Guyton, M.D. 11th Edition Unit VII, Chapter 42 Pages 524- 533.
2. Physiology. Linda. S. Costanzo 3rd edition Page 217-2022.

|  |
| --- |
| Title of the lecture: Effects of low and high gas pressure on the body |

Lecturer’s name: DR. Aida Korish & DR Abdulrahman Al Howaikan

Department: Physiology departement

Block / week: Respiratory Block / Week 1 to Week 4

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**Objectives of the lecture:**

* Describe the effects of exposure to low and high barometric pressures on the body
* Describe the body acclimatization to low barometric pressure.
* Define decompression sickness and explain how it can be avoided.
* Understand the effects of high nitrogen pressure, and nitrogen narcosis.

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**Background:**

Students know the partial pressure of a gas and how it is influenced by altitude. In addition they know the partial pressures of oxygen and carbon dioxide in the atmosphere, the partial pressure of gas inside the alveolar and at the end of the pulmonary capillary . Students know also factors affecting gas diffusion between alveolar and capillaries membrane

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**Main concepts in the lecture:**

Under certain limits these high pressures cause tremendous alterations in the physiology of the body. Nitrogen dissolve freely in the fats of the body under high pressure This leads to narcosis develops. However, decrease in barometric pressure cause of all the hypoxia problems and remaining at high altitudes for days , weeks or years acclimatized the body to low PO2.

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**Conclusion:**

high and low pressures cause tremendous alterations in the physiology of the body . Students understand the body acclimatization to low barometric pressure and what physiological changes will be achieved due to reduction of barometric pressure. on other hand students know ascending quickly for deep sea will cause decompression sickness, which might will cause nitrogen narcosis.

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**Take home messages:**

* read about factors that influence solving and dissolving of gases in liquid
* influence of different levels of high barometric pressures on human body
* impact of hyperbaric chamber on decompression sickness
* sign and symptom of nitrogen narcosis the recommendation that should be given to divers to avoid nitrogen narcosis

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**Further reading:**

1. [Guyton & Hall: Textbook of Medical Physiology 12E](http://www.studentconsult.com/content/default.cfm?ISBN=9781416045748&home=true)
2. Physiology 5th Edition by linda s costanzo

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| --- |
| Title of the lecture: Pathology and Pathogenesis of Bronchial Asthma. |

Lecturer’s name : DR. AMMAR AL-RIKABI and DR. MAHA ARAFAH

Department : Pathology

Block / week : Respiratory/ week 2

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**Objectives of the lecture:**

* Understand asthma as an episodic, reversible bronchoconstriction caused by increased responsiveness of the tracheobronchial tree to various stimuli.
* Know that asthma is divided into two basic types: extrinsic or atopic allergic and intrinsic asthma.
* Understanding the morphological changes (gross and microscopic) seen in the lungs in cases of severe asthma.

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**Keywords:**

(Bronchial Asthma, bronchoconstriction, extrinsic asthma, atopic asthma, intrinsic asthma, status asthmaticus)

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**Background:**

* Asthma is characterized by reversible bronchoconstriction.
* Atopic asthma is caused by TH2 and IgE mediated immunologic reaction.
* Major basic protein is an eosinophils product responsible for airway damage.
* Airway remodeling adds an irreversible component to the obstructive disease.

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**Main concepts in the lecture:**

* Definition of asthma as one of the chronic obstructive airway diseases.
* Types and pathogenesis of extrinsic (immune) asthma and intrinsic (non-immune) asthma.
* Clinical presentation and pathological changes seen in the bronchial tree in cases of asthma.
* Complications of asthma: superimposed infection, chronic bronchitis and pulmonary emphysema.
* Definition and manifestations of status asthmaticus as an acute medical emergency.

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**Conclusion:**

Asthma is an increased irritability of the bronchial tree with paroxysmal narrowing of the airways, which may reverse either spontaneously or after treatment with bronchodilators.

Asthma can be 1. Extrinsic asthma (atopic)—early-onset asthma triggered by environmental allergens with family history of allergic disorders and raised IgE levels. 2. Intrinsic asthma (non-atopic) associated with chronic bronchitis, cold or exercise. IgE levels are normal with no family history of allergic disorders.

The main structural changes Immune cell infiltration—the bronchial mucosa is infiltrated by eosinophils, mast cells, lymphoid cells and macrophages, mucosal oedema, mucus hypersecretion, hypertrophy of bronchial smooth muscle due to recurrent bronchoconstriction, focal necrosis of the airway epithelium, caused by prolonged inflammation and deposition of collagen beneath the bronchial epithelium in long-standing cases.

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**Take home messages**:

* Asthma: episodic and reversible airway disease of bronchi and bronchioles
* Atopic asthma is caused by a TH2 and IgE-mediated immunologic reaction to environmental allergens and is characterized by acute-phase (immediate) and late-phase reactions. The TH2 cytokines IL-4, IL-5, and IL13 are important mediators.
* Triggers for nonatopic asthma are less clear but include viral infections and inhaled air pollutants, which can also trigger atopic asthma. It is also associated with chronic bronchitis, cold or exercise
* Eosinophils are key inflammatory cells found in almost all subtypes of asthma; other inflammatory cells include mast cells, neutrophils and T lymphocytes.
* Clinical: expiratory wheezing; nocturnal cough; anterior posterior diameter of chest wall
* Severe form, status asthmaticus, the paroxysm persists for days and even weeks, sometimes causing airflow obstruction that is so extreme that marked cyanosis or even death ensues.

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**Further reading:**

Robbin’s Basic Pathology 9th Edition pages. 468 to 470

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| --- |
| Phospholipids of Clinical Significance |

Lecturer’s name : Dr. Usman Ghani / Dr. Sumbul Fatma

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Block / week : Respiratory/ Week 2

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**Objectives of the lecture:**

By the end of this lecture the First Year students will be able to:

* Identify types and functions of phospholipids
* Discuss the physiological importance of phospholipids
* Understand the role of glycerphospholipids in lung surfactant and their clinical implications in respiratory distress syndrome (RDS)
* Identify the classes and physiological functions of phospholipase enzymes

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**Keywords:**

Phospholipid, surfactant, respiratory distress syndrome, phospholipases

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**Background:**

* Lipids are heterogeneous group of compounds that are relatively water-insoluble.
* Example of simple lipids: fatty acid, TG, ketone bodies, cholesterol.
* Example of complex lipids: Phospholipids, Lipoproteins.
* Lipids have important physiological functions.
* Lipid disorders are the basis for common human diseases.

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**Main concepts in the lecture:**

Phospholipids are either membrane-bound; the functions of which are structural, anchoring, signaling, or insulation and speeding up transmission of nerve impulses; or non-membrane-bound; the functions of which are structural, solubilizing, or emulsification.

Lung surfactant is an example of non-membrane-bound PL that is essential for re-inflation of alveoli by air. The major lipid component of lung surfactant is dipalmitoylecithin; other components include other phospholipids, cholesterol & proteins. Congenital Respiratory distress syndrome (RDS) results from insufficient production of lung surfactant (especially in pre-term babies). Pre-natal diagnosis, prevention and treatment of the RDS are based on the biochemical defect in premature babies.

Calcium/phosphatidyl inositol system as an intracellular signaling pathway for hormones and neurotransmitters such as acetylcholine, ADH, and catecholamines is a tightly regulated pathway.

Examples of anchored proteins that utilize PL for their attachment to cell membranes include alkaline phosphatase and acetylcholine esterase.

Different classes of phospholipases include glycerophospholipids phospholipases A1, A2, C and D, and lysosomal phospholipase for sphingophospholipids. Phospholipases play a role in the degradation and the remodeling processes of PL.

**Conclusion:**

Phospholipids are complex lipids that have important physiological functions. They are either membrane-bound or non-membrane bound. Among their roles, they have structural, signaling, membrane anchoring, alveolar re-inflation, and detergent effects. Enzyme degrading phospholipids are termed phospholipases. Several classes of phospholipases exist, including phospholipases A1, A2, C and D; and lysosomal phospholipases as sphingomyelinase. These enzymes play important roles in PL(s) degradation and remodeling. Insufficient production of lung surfactant is the cause for congenital respiratory distress syndrome. Lecithin/sphingomyelin ratio in amniotic fluid is used for the pre-natal diagnosis of RDS.

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**Take home messages:**

* Phospholipids are complex lipids that perform important physiological functions in the body
* Membrane-bound phospholipids are involved in cell signaling, protein anchoring and myelin protective functions
* Nonmembrane-bound phospholipids function as lung surfactant and as detergent in the bile
* Phospholipases are enzymes that degrade phospholipases
* They are important for remodeling of phospholipids

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**Further reading:**

Lippincott’s Illustrated Reviews, Biochemistry, 6th Edition, Denise R. Ferrier, Lippincott Williams & Wilkins, USA, pp 201-207.

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| --- |
| Title of the lecture: Radiologcial anatomy of the chest |

Lecturer’s name : Prof. Saeed Abuel Makraem – Dr. Jamila Al Medany

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Block / week :Respiratory

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**Objectives of the lecture:**

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

* Identify the important structures in a postero-anterior chest radiograph the following:
* Identify the bronchial tree in a postero-anterior bronchogram of the chest.
* Identify the esophagus in a lateral radiograph of the chest, after barium swallow.
* Identify the coronary arteries in a coronary angiogram.

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**Keywords:**

(bronchogram, radiograph, barium swallow, angiogram, borders of mediastinum, aortic knuckle, costophrenic angle, cardiophrenic angle.)

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**Background:**

Radiology is an important clinical diagnostic tool. Radiological anatomy helps students to identify the different structures using radiography.

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**Main concepts in the lecture**:

* Identification in a postero-anterior chest radiograph the following:

1. Bones: clavicle, ribs, thoracic vertebrae and medial border of scapula.
2. Diaphragm: right and left domes, cardiophrenic angles, costodiaphragmatic angles.
3. Trachea: normal position.
4. Lungs and bronchi: lung shadow, hilum and root of lung, pulmonary blood vessels.
5. Heart: apex and borders.
6. Great vessels: aorta, pulmonary trunk, superior vena cava.
7. Constituents of right border of the mediastinum: right brachiocephalic vein, superior vena cava, right atrium and inferior vena cava.
8. Constituents of left border of the mediastinum: aortic knuckle (aortic arch), pulmonary trunk, left auricle and left ventricle.

* Identification of the bronchial tree in a postero-anterior bronchogram of the chest.
* Identification of the esophagus in a lateral radiograph of the chest, after barium swallow.
* Identification of the coronary arteries in a coronary angiogram.

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**Conclusion:**

Radiology is an important clinical diagnostic tool. Radiological anatomy helps students to identify the different structures using radiography.

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**Take home messages:**

Radiological Anatomy is an important branch that helps to identify important structures using specific techniques.

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**Further reading:**

Clinical Anatomy for Medical Students by Richard S. Snell- Latest Edition.

Grant’s Atlas of Anatomy by Anne and Arthur –Latest Edition.

|  |
| --- |
| Title of the lecture: Immunology of Asthma |

Lecturer’s name : Prof. Zahid Shakoor

Department : Pathology (Immunology)

Block / week : Respiratory block, Week 2

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**Objectives of the lecture:**

* Identify differences between extrinsic and intrinsic asthma
* Identify types of allergens and their role in allergic sensitization
* Explain the inflammatory processes operating in allergic asthma
* Explain the airway remodeling

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**Keywords:**

(Extrinsic asthma, intrinsic asthma, outdoor Allergens, indoor allergens, Th2 cytokines, eosinophils, mast cells, early allergic response, late allergic response, inflammation, remodeling.

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**Background**:

* Students will understand how the interplay of the innate and the adaptive immunity and the polarization of the immune system towards a Th2 cytokine response.
* Understand the pathogenesis of allergic asthma, clinical manifestations and the outcome of long standing inflammation in terms of airway remodeling.

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**Main concepts in the lecture:**

Asthma is characterized by reversible airway obstruction and the manifestations include cough, breathlessness, wheezing or chest tightness. Asthma is caused by constriction of airways and the symptoms may occur for a certain period of time during the year or may be perennial. Asthma is usually classified as either intrinsic (non-atopic or extrinsic (atopic). Intrinsic asthma is considered not to be due to allergy and the cause is seldom known. It is frequently observed in older population without any detectable evidence of allergy. The underlying pathology of atopic or allergic asthma is the allergic inflammation of the airways among atopic individuals mediated by IgE class of antibodies. Allergic sensitization is usually due to production of allergen specific IgE antibodies against inhaled allergens (aeroallergens) such as house dust mite, animal dander, pollens and fungal spores.

Allergen specific IgE antibodies bind to receptors on the surface of mast cells located in the submucosa. Mast cell degranulation occurs subsequent to antigen antibody interaction on the surface of the mast cells leading to release of mast cell inflammatory mediators resulting in bronchospasm and the clinical manifestations of allergic asthma. Release of cytokines such as IL-4, IL-5, IL-9 & IL-13 tilt the immune response in allergic asthma to Th2 type humoral immune responses. This is believed to due to dysregulation of T regulatory cell function. In addition IL-13 has been implicated in induction of airway remodeling which is a characteristic feature of long standing inadequately controlled asthma.

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**Conclusion:**

Asthma is classified in 2 types intrinsic and extrinsic. Allergens trigger extrinsic asthma and polarize the immune response towards Th2 response and initiate a chronic inflammation. Chronic inflammation may lead to irreversible changes and remodeling of the respiratory tract.

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**Take home messages:**

* Asthma is characterized by episodic reversible airway obstruction
* Classified in 2 types: intrinsic & extrinsic
* In the extrinsic type allergens drive T-cells into Th2 pattern
* Airway inflammation is a hallmark finding in the asthmatic lung
* Inflammatory cells lead to increased bronchial reactions & airway remodeling which is not revisable.

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**Further reading:**

Kuby Immunology, 7th edition 2013, Chapter 15 page 494-496, page 498.

|  |
| --- |
| Title of the lecture: Tuberculosis |

Lecturer’s name : Prof. Hanan Habib & Prof. Ali Somily

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Block / week : Respiratory /week 3

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**Objectives of the lecture:**

* Define tuberculosis as a chronic disease mainly affecting the respiratory system.
* Recognize roughly the epidemiology of tuberculosis worldwide and in the kingdom of Saudi Arabia
* Understand the methods of transmission of tuberculosis and the people at risk.
* Know the causative agents and their characteristic and classification and methods of detection.
* Understand the pathogenesis of tuberculosis.
* Differentiate between primary and secondary tuberculosis and the clinical features of each.
* Understand and describe and explain the methods of tuberculin test, tuberculin skin test (TST) and its different results.
* Know the radiological and laboratory diagnostic methods.
* Know the chemotherapeutic and other methods of management of tuberculosis cases.
* Describe the methods of prevention and control of tuberculosis.

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**Background:**

Tuberculosis ( TB) is an ancient (old disease) that affect humans and is a major cause of death world wide.

It is caused by the *Mycobacterium tuberculosis complex species* of *mycobacteria*. It usually affects the lung although other organs can be affected in one third of cases. If properly treated, tuberculosis caused by susceptible strains is curable, if untreated it is fatal in most cases. It is transmitted from respiratory infected cases by airborne droplets spreading during coughing, singing, talking and sneezing.

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**Main concepts in the lecture:**

Tuberculosis as a chronic ancient (old) disease shall be explained indicating that tuberculosis is the most killing single infectious disease in the world. It mainly affects the lungs. Other organs in the body that can be affected will be stated.

Epidemiology of the disease will be explained in general. The following points shall be stressed. TB is affecting one third of human race( 2 billion people) as latent (dormant) tuberculosis infection making it second only to Helicobacter pylori which infect 50% of human race .TB infect 8 million people each year with 2-4million deaths in KSU ,the incidence is 32-64/100,000 in USA 5.2 /100,000. In South Africa it is 290/100,000. This is due to coupling with HIV infection which is common in South Africa. This is a factor that increases susceptibility to T.B infection.

Transmission of the diseases by airborne droplet nuclei of (<5µ m ) from pulmonary diseased cases. It affects children and young adults and people, whose occupation subjects them to the diseases e.g. poor people, nurses, doctors (from cases) and microbiology technicians from cultures of the organism.HIV infected people are prone to the diseases as clear from the association between HIV& TB in sub-Saharan African countries.

Characteristics, description, classification of the causative agent (*M.tuberculosis*) shall be addressed under the following. It is a member of the genus mycobacteria. *M.tuberculosis, M.bovis, M.aftricanum*, BCG strains all these are called *mycobacterium tuberculosis* complex.

Like other mycobacteria all are acid fast bacilli that resist staining by gram stain and are only stained by Zeihl-Neelson stain (Z-N) as they resist decolorization by acid due to the mycolic acid in their cell wall. These bacteria (*M. tuberculosis*) need a long time to grow (2-8 weeks) on solid medium called Loweinstein Jensen medium (L.J)

Pathogenesis of the disease will be explained as a disease acquired by airborne droplet or ingestion of milk from infected cattle. Which then reach the alveolar macrophages of the lung where they are able to survive in macrophages (this is their main virulence factor) this will start a cell mediated immune response which control the multiplication of the organism but not killing it. This forms a granuloma and the organisms lives in a dormant state the infection is then called latent tuberculosis infection. (LT infection).The patient will show evidence of delayed cell mediated immunity. The later results of the disease are due to the destructive effect of cell mediated immunity. Clinically the disease is divided in to Primary infection when the patient first encounter the organism or Secondary, when the latent infection is activated due to immunosuppression by malignancy, diabetes, corticosteroids or old age.

Clinical features of the diseases are usually due to secondary tuberculosis which can be due to:

1. Reactivation of an old primary TB , or
2. Reinfection: both in a previously sensitized patients who has previous encountered with the organism. It mainly affects the lung with formation of cavities with features include granuloma and caseation. Clinical feature are:

a-Fever

b-Cough

c- Haemoptysis

d- Weight loss

e- Weakness

TUBERCULIN SKIN TEST (TST): It is elicited by type 4 delayed hypersensitivity reaction in people who has previous contact with the *mycobacteria*. It is done by injection intra dermally of purified protein derivative (PPD) of mycobacterium origin that actives synthesized lymphocytes to produce cell mediated immunity which appears on the skin as an induration. The size of this induration will determine, the positively or negatively of the test. The positive result indicates previous contact with mycobacteria.If this happens recently in a person who has been in contact with an active case of tuberculosis, it will show that this person is infected by this case. Another test in which sensitized lymphocytes are challenged by same mycobacteria proteins will produce interferon gamma which has an even more specific significance than TST.

DIAGNOSIS OF TUBERCULOSIS

Laboratory diagnosis : sputum or other respiratory secretions are examined by Z-N stain for AFB. This is usually indicates open infectious TB. If microscopy for AFB is negative. The specimen should be cultured. The culture is the gold standard for diagnosis of TB. Specimens from other organs than respiratory can be tested similarly. This isolated M*ycobacterium* can be identified and differentiated from other mycobacteria by same biochemical test like niacin and Nitrate test or DNA detection by tests like Prob tec test.

Imaging is of great importance in the diagnosis of TB specially pulmonary as it can show the primary complex and Ghon focus in primary TB or cavitation in secondary TB of the lung or chronic granuloma in other organs.

MANAGEMENT OF TUBERCULOSIS CASES

Therapy should be for minimum of 6 months. There are differences between primary and secondary drugs. The drugs used are:

Primary drugs

1. Rifampicin
2. Isonizide(INH)
3. Ethambutol
4. Pyrazinamide and some times Streptomycin.

Therapy for first two month drug 1, 2, 3 and 4 then for further 4-6 month by the first two drugs ( Rifampicin &INH).

Control and prevention

1. Case finding and treatment
2. Isolation of infectious cases for 14 post treatment
3. Vaccination for children in KSA at birth to prevent primary infection.
4. Pasteurization of milk to prevent bovine tuberculosis from cattle.

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**Conclusion:**

Tuberculosis is worldwide chronic disease that mainly affects the lungs caused by M.tuberculosis complex. TB is transmitted through airborne droplets. TB can be a primary or secondary and stimulate cell mediated immunity. Lab diagnosis by Z-N stain of sputum or other respiratory samples and culture . Patients with active TB should be isolated .Treatment of TB is by a combination of antituberculous drugs for a minimum of 6 months with follow up of treatment compliance . Prevention is by ,case finding and vaccination and pasteurization of milk.

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**Take home messages:**

1. TB is a chronic ancient disease that affect one third of human race causing 8 millions infections each year and 2 million deaths each year
2. The disease is caused by M*ycobacterium tuberculosis complex* group of the *genus mycobacteria*
3. Pathogenesis of the disease is mainly by airborne droplet from pulmonary infected cases or in the past by ingestion of milk from infected cattle. The lungs are the mainly infected organs in the body although other organs can be affected.
4. Two types of syndromes are known latent infection and TB disease.
5. Two types of diseases are known primary diseases characterized by development of Ghon focus and primary complex Secondary tuberculosis which can be from reactivation of primary infections or exogenously re-infection.
6. Cell mediated immunity can be tested by TST.
7. Treatment of TB is by use of first line drugs e.g. Rifampicin, Isonizide Ethambutol and Pyrazinamide. secondary drugs e.g. PAS, Ethionamide,Cycloserene Kanamycin for resistant organisms
8. Prevention is by case finding, and treatment as well as isolation of infection cases and BCG for children.
9. Pasteurization of cow milk.

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**Further reading:**

*Sherries* Medical Microbiology, an Introduction to Infectious Diseases. Latest edition, Kenneth Ryan and George Ray. Publisher : McGraw Hill .

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| --- |
| Title of the lecture: Chronic Obstructive Airway Diseases (COPD): Chronic Bronchitis, Emphysema and  Bronchiectasis. |

Lecturer’s name : DR. AMMAR AL-RIKABI and DR. MAHA ARAFAH

Department : Pathology

Block / week : Respiratory/ week 3

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**Objectives of the lecture:**

* Understand that COPD group of disorders is characterized by an increase in resistance to airflow, owing to partial or complete obstruction at any level of the bronchial/bronchiolar.
* Know that the major obstructive disorders are chronic bronchitis, emphysema, asthma and bronchiectasis.
* Is aware that the symptom common to all these disorders is "dyspnea" (difficulty in breathing) but each have their own clinical and anatomical characteristics.
* Define chronic bronchitis, describe its pathogenesis and the morphologic changes. Describe the mechanism of airway obstruction in a patient with chronic bronchitis. Understand that when severe obstruction is present in chronic bronchitis, significant emphysema is nearly always present.
* Define emphysema and the following forms of emphysema: panacinar emphysema, centriacinar emphysema, compensatory hyperinflation, obstructive overinflation and interstitial emphysema.
* Describe the pathophysiologic mechanisms of airway obstruction.
* Define bronchiectasis. Describe the gross anatomic lesion, and list the conditions that predispose to its development.

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**Keywords:**

(Chronic Obstructive Airway Diseases, Chronic Bronchitis, Emphysema, Bronchiectasis, dyspnea, cor pulmonale, centrilobular, panacinar, alpha one antitrypsin, pneumothorax, kartagener's syndrome)

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**Background:**

1. Chronic bronchitis is defined as persistent productive cough for at least 3 consecutive months in at least 2 consecutive years. Cigarette smoking is the most important risk factor; air pollutants also contribute. The dominant pathologic features are mucus hypersecretion and persistent inflammation. Histologic examination demonstrates enlargement of mucous-secreting glands, goblet cell hyperplasia, chronic inflammation, and bronchiolar wall fibrosis.
2. Emphysema is a chronic obstructive airway disease characterized by permanent enlargement of air spaces distal to terminal bronchioles. Subtypes include centriacinar (most common, smoking related), panacinar (seen in α1-antitrypsin deficiency), distal acinar and irregular. Smoking and inhaled pollutants cause ongoing accumulations of inflammatory cells, releasing elastases and oxidants, which destroy the alveolar walls. Most patients with emphysema also have some degree of chronic bronchitis, which is to be expected since cigarette smoking is an underlying risk factor for both.
3. Bronchiectasis is a disorder in which destruction of smooth muscle and elastic tissue by chronic necrotizing infections leads to permanent dilation of bronchi and bronchioles.

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**Main concepts in the lecture:**

In chronic bronchitis, constant irritation by cigarette smoke causes chronic inflammation of the respiratory bronchioles (bronchiolitis) with increased mucous secretion and extensive mucus plugging leading to the clinical obstructive features of the disease. Patient have cough and sputum production. In later stages of the disease, progressive obstruction may result in patients with hypercapnia, hypoxaemia and cyanosis.

Emphysema is a permanent dilatation of any part of the air spaces distal to the terminal bronchiole, occurring with tissue destruction but without fibrosis. Causes include cigarette smoking, occupational dusts or chemicals and atmospheric pollution. The inherited disorder α1-antitrypsin deficiency predisposes to early emphysema. The lungs are hyperinflated, the trachea is often descended (i.e. there is decreased cricosternal distance between the cricoid cartilage and the sternal notch) and the accessory muscles may be hypertrophied.

Bronchiectasis is irreversible dilatation of the bronchi or their branches. Its causes are: congenital including cystic fibrosis, primary ciliary dyskinesia and Kartagener’s syndrome (bronchiectasis, situs inversus and sinusitis) or acquired such as infection (especially whooping cough, necrotising pneumonia or measles in childhood) and obstruction (either by an inhaled foreign body or by tumour). Widened bronchi are more prone to infections, Haemophilus influenzae and Pseudomonas aeruginosa being the most common pathogens. Patients often cough up purulent sputum, which may contain blood.

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**Conclusion:**

Chronic obstructive pulmonary disease (COPD) is a chronic, slowly progressive disease of airflow limitation caused by an abnormal inflammatory response of the lungs to noxious substances (usually from cigarette smoking).

COPD classically involves three overlapping pathological processes:

1. Chronic bronchitis: this causes hyperpalsia of the bronchial submucosal glands, leading to an increased Reid index.
2. Emphysema: abnormal diltation of air spaces with destruction of alveolar walls.
3. Bronchiectasis: permanent dilation bronchi/bronchioles caused by destruction of the muscle and the supporting elastic tissue resulting from or associated with chronic necrotizing infections.

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**Take home messages:**

* Chronic bronchitis is persistent cough for at least three consecutive months in at least two consecutive years.
* Emphysema is a COPD characterized by permanent enlargement of air spaces distal to terminal bronchioles leading to functional obstruction with dyspnea.
* Bronchiectasis is the permanent dilation of bronchi and bronchioles leading to repeated episodes of airway infection and inflammation

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**Further reading:**

Robbin’s Basic Pathology 9th Edition pages: 463 to 472

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| --- |
| Title of the lecture: Restrictive Lung Diseases. |

Lecturer’s name : DR. AMMAR AL-RIKABI and DR. MAHA ARAFAH

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Block / week : Respiratory/ week 3

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**Objectives of the lecture:**

* Understand the structure and constituents of the lung interstitium as well as the restrictive changes which occur in these diseases and lead to the development of symptoms of progressive breathlessness and cough in affected patients.
* Appreciate the pathogenesis of interstitial lung diseases regardless of their type. This pathogenesis include the influx of inflammatory cells into the alveoli and alveolar walls, distortion of the normal structure of alveoli, release of chemical mediators and promotion of fibrosis (honey-combed lung).
* Become aware of the classification of interstitial lung diseases.

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**Keywords:**

(Restrictive pulmonary diseases, Adult and neonatal respiratory distress syndromes, Pneumoconiosis, Anthracosis, Silicosis, Asbestosis,honeycomb lung Hypersensitivity, pneumonitis, Goodpasture syndrome, Eosinophilic granuloma, Idiopathic pulmonary fibrosis, Sarcoidosis)

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**Background:**

Restrictive lung diseases are characterized by reduced lung compliances and reduced forced vital capacity (FVC). The ratio of FEV to FVC is normal.

Pathogenesis of restrictive pulmonary diseases which include abnormalities in the chest wall or neuromuscular diseases that restrict lung expansion or interstitial lung fibrosis

The diseases that cause interstitial lung fibrosis are heterogeneous. The underlying cause is injury to the alveoli with activation of macrophages and release of fibrogenic cytokines such as TGF Beta. Idiopathic pulmonary fibrosis will lead to honey combed lung.

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**Main concepts in the lecture:**

Restrictive lung diseases are disorders characterized by reduced total lung capacity (TLC) in the presence of a normal or reduced expiratory flow rate. It can be acute or chronic. Acute interstitial lung disease (e.g., ARDS). ARDS is a clinical syndrome of progressive respiratory insufficiency caused by diffuse alveolar damage in the setting of sepsis, severe trauma, or diffuse pulmonary infection. Chronic interstitial lung disease is eitherfibrosing disorders (e.g., idiopathic pulmonary fibrosis and pneumoconiosis) or granulomatous disease (e.g., sarcoidosis)/

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**Conclusion:**

Chronic interstitial pulmonary diseases are a heterogeneous group of disorders characterized predominantly by inflammation and fibrosis of the pulmonary inter­stitium. Many of the entities are of unknown cause and pathogenesis, and some have an intra-alveolar as well as an interstitial component and characterized by reductions in diffusion capacity, lung volume, and lung compliance.

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**Take home messages:**

* Diffuse interstitial fibrosis of the lung gives rise to restrictive lung diseases characterized by reduced lung compliance and reduced forced vital capacity (FVC). The ratio of FEV1 to FVC is normal.
* Idiopathic pulmonary fibrosis is prototypic of restrictive lung diseases. It is characterized by patchy interstitial fibrosis fibroblastic foci and formation of cystic spaces (honeycomb lung). This histologic pattern is known as usual interstitial pneumonia.
* The cause of idiopathic pulmonary fibrosis is unknown, but analyses point to role of alveolar epithelium stress. The resulting injury to alveolar epithelial cells leading to increase local production of fibrogenic cytokines such as TGF-β.
* The other diseases that cause diffuse interstitial fibrosis are heterogeneous poorly understood, but most have better prognoses that idiopathic pulmonary fibrosis.
* Pneumoconioses encompass a group of chronic fibrosing diseases of the lung resulting from exposure to organic and inorganic particulates, most commonly mineral dust. Coal dust-induced disease varies from asymptomatic anthracosis to simple coal workers’ pneumoconiosis (coal macules or nodules, and centrilobular emphysema), to progressive massive fibrosis (PMF), manifested by increasing pulmonary dysfunction, pulmonary hypertension, and cor pulmonale. Silicosis is the most common pneumoconiosis in the world, and crystalline silica (e.g., quartz) is the usual culprit. The lung disease is progressive even after exposure stops.The manifestations of silicosis can range from asymptomatic silicotic nodules to large areas of dense fibrosis; persons with silicosis also have an increased susceptibility to tuberculosis. There is two-fold increased risk of lung cancer. Asbestos fibers come in two forms; the stiff amphiboles have a greater fibrogenic and carcinogenic potential than the serpentine chrysotiles. Asbestos exposure is linked with six disease processes: (1) parenchymal interstitial fibrosis (asbestosis); (2) localized pleural plaques (asymptomatic) or rarely diffuse pleural fibrosis; (3) recurrent pleural effusions; (4) lung cancer; (5) malignant pleural and peritoneal mesotheliomas; and (6) laryngeal cancer.
* Sarcoidosis is a multisystem disease of unknown etiology; the diagnostic histopathologic feature is the presence of noncaseating granulomas in various tissues.
* Hypersensitivity pneumonitis result s from the inhalation of organic dust containing antigens made up of the spores of thermophilic bacteria, fungi, animal proteins, or bacterial products. Most patients have specific antibodies against the causative antigen in their serum. The presence of noncaseating granulomas in two thirds of the patients suggests that T-cell–mediated (type IV) hypersensitivity reactions against the implicated antigens are also common and have a pathogenic role.

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**Further reading:**

­­­­­­­­­­­­­­­­­­­­­­­ Robbin’s Basic Pathology 9th Edition pages: 472 to 481

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| --- |
| Respiratory Chain |

Lecturer’s name : Dr. Usman Ghani / Dr. Sumbul Fatma

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Block / week : Respiratory Block

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**Objectives of the lecture:**

* Understand how energy-rich molecules including glucose are metabolized by a series of oxidation-reduction reactions ultimately yielding CO2 and water.
* Explain the process of electron transport chain that releases free energy, which is used for ATP synthesis and heat production.
* Recognize the reactions of electron transport chain taking place in mitochondria that are coupled to oxidative phosphorylation.

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**Keywords:**

Respiratory chain, electron transport chain, oxidative phosphorylation, coenzymes, oxidation, reduction, mitochondria, oxidative phosphorylation, ATP synthase, ATP synthesis, heat, electrons, protons, uncoupling proteins

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**Background:**

* The electron transport chain (ETC) is present in the inner mitochondrial membrane
* It is the final common pathway by which electrons derived from different fuels of the body flow to oxygen.
* The components of ETC include mobile and immobile enzymes and proteins that transfer electrons in the chain.
* Electron transport and ATP synthesis by oxidative phosphorylation are coupled and is taking place continuously in all tissues that contain mitochondria.

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**Main concepts in the lecture:**

The metabolic intermediates undergo a series of oxidation—reduction reactions donating electrons to specific coenzymes – nicotinamide adenine dinucleotide (NAD+) and flavin adenine dinucleotide (FAD) – to form the energy-rich reduced coenzymes, NADH and FADH2. Each of these reduced coenzymes can, in turn, donate a pair of electrons to a specialized set of electron carriers, collectively called the electron transport chain. As electrons are passed down the electron transport chain, they lose much of their free energy. Part of this energy can be captured and stored for the production of ATP from ADP and inorganic phosphate (Pi). This process is called oxidative phosphorylation. The remainder of the free energy not trapped as ATP is released as heat through the uncoupling proteins.

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**Conclusion:**

* Energy-rich molecules undergo a series of oxidation-reduction reactions and ultimately yielding CO2 and water.
* Electron transport chain coupled with ATP synthesis by oxidative phosphorylation continuously supply all body tissues with ATP and heat.
* The free energy released during the electron transport chain is utilized for ATP synthesis as well as for heat production.

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**Take home messages:**

* ETC is a common pathway of transferring energy-rich electrons from metabolism finally yielding CO2 and water.
* The energy of the electrons transferred is used for ATP synthesis and heat production.

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**Further reading:**

Lippincott’s Illustrated Reviews, Biochemistry, 6th Edition, Denise R. Ferrier, Lippincott Williams & Wilkins, USA, pp. 53-68.

|  |
| --- |
| Title of the lecture: Upper Respiratory Tract Infection |

Lecturer’s name : Prof. Ali Somily & Dr. Fawzia Alotaibi

Department : Microbiology

Block / week : Respiratory Block

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**Objectives of the lecture:**

* To learn the epidemiology
* To learn various clinical presentation of URTI.
* To identify the common etiological agents causing these syndromes.
* To study the laboratory diagnosis of these syndromes.
* To determine the antibiotic of choice for treatment.

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**Background:**

* Upper respiratory tract infection is considered one of the important and very common infections particularly among children.
* Knowing the causative agents, clinical presentation, and diagnosis and management is very important.

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**Main concepts in the lecture:**

Student should know the different pathogens causing upper respiratory tract infection. Of major important is group A streptococci as a major and most common cause of tonsillitis. The clinical presentation, diagnosis and management of tonsillitis must be emphasized. Differentiation between viral and bacterial infection is also be mentioned. At the end of the lecture students must be aware of the complications and of mismanagement of patients.

The student should be able to recognize the symptoms and signs od Acute bacterial rhinosinusitis In children, who present with sever onset persistent nasal discharge (any type) or cough lasting 10 days or more without improvement and define the causative agents and how to treatment.

The student should be able to define the Laryngotracheitis and laryngotracheobronchitis which Nasopharyngitis often precedes laryngitis and tracheitis by difficulty in Swallowing may be difficult or painful hoarseness or loss of voice is a key manifestation of laryngeal involvement

Student should able to recognized serious conditions like epiglottitis, diphtheria and whooping cough. He should know that epiglottitis is more often found in children aged 1-5 years, who present with a sudden onset of Sore throat, drooling, difficulty or pain during swallowing, globus sensation of a lump in the throat Muffled dysphonia or loss of voice, dry cough or no cough, dyspnea, fever, fatigue or malaise (may be seen with any URI). The management is critical and the most importance avoidance of nasopharyngeal examination and immediate staring of appropriate antibiotics covering the most common known causative agents. Method of diagnosis microbiological and radiological should be understood.

The student should know that presence of a whitish membrane in the pharynx of unvaccinated child might suggest serious condition like diphtheria. Drooling of saliva and difficulty in breathing might be present. Understanding the epidemiology pathogenesis of this microorganism is important. The student should recognize that this organism will not grow on routine media rather wise specifically requesting culturing this organism is required. Appropriate antibiotics should be started immediately. Recommended methods of prevention and complication should be defined by the students.

The student should understand that chronic cough is one of the clinical feature of whooping cough (pertussis). The classic whoop sound is an inspiratory gasping squeak that rises in pitch, typically interspersed between hacking coughs. The whoop is more common in children which is often comes in paroxysms of a dozen coughs or more at a time and is often worst at night. The student should be able determine the specific way of diagnosis of whooping cough ie nasopharyngeal swab and lymphocytosis. Infection control measure and medical treatment and prevention should be understood by the student.

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**Conclusion:**

* Upper respiratory tract infection (URI) represents the most common acute illness evaluated in the outpatient setting.
* URIs range from the common cold—typically a mild, self-limited, catarrhal syndrome of the nasopharynx—to life-threatening illnesses such as epiglottitis.

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**Take home messages:**

* Most URIs is viral in origin. Diagnosis is mainly based on clinical manifestations in some cases pharyngeal rapid streptococcal antigen detection test before considering antimicrobial therapy.
* Sinus puncture and sinus CT are not recommended for the diagnosis of uncomplicated sinusitis.
* A nasopharyngeal swab used mainly for viral diagnosis of respiratory infection, some bacteria like Bordetella pertussis diagnosed by nasopharyngeal swab.
* Antibiotics should be avoided in patients with a common cold, mild acute rhinosinusitis, or acute bronchitis, but should be prescribed to patients with GABHS pharyngitis and to moderate or severe suspected acute bacterial rhinosinusitis.
* Penicillin is the recommended treatment for GABHS pharyngitis.

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**Further reading:**

1. Medical microbiology by Patrick Murray Ken Rosenthal Michael Pfaller (2015), 8th Edition. Elsevier ISBN: 978-0-323-29956-5.
2. Medical Microbiology. 4th edition; Chapter 93Infections of the Respiratory System

Clinical Guidelines Diagnosis and Treatment Manual for Curative Programmes in Hospitals and Dispensaries Guidance for prescribing 2016 Edition

|  |
| --- |
| Title of the lecture: Mediastinum |

Lecturer’s name : Prof. Ahmed Fathalla – Dr. Jamila El Medany

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Block / week :Respiratory

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**Objectives of the lecture:**

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

* Define the word “Mediastinum”.
* Subdivide the mediastinum and describe the boundaries of each subdivision.
* List the contents of each mediastinum and describe in brief the relations between them.

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**Keywords:**

( mediastinum, thorax, sternum, diaphragm, thoracic vertebrae, esophagus, aorta, brachiocephalic veins, superior vena cava, vagi, phrenic nerves, thoracic duct )

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**Background:**

* The mediastinum is the median partition of the thoracic cavity.
* The mediastinum is subdivided into parts. Each part contains several important structures. The relations between the different structures in each part of the mediastinum must be understood.

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**Main concepts in the lecture:**

The mediastinum is the median partition, in the thoracic cavity, between the pleurae & lungs.

Subdivisions: It is subdivided into superior and inferior, by an imaginary plane passing between sternal angle and lower border of 4th thoracic vertebra.

Inferior mediastinum is further subdivided, by the pericardium and heart into:

* Middle mediastinum: containing pericardium and heart.
* Anterior medisatinum: between sternum and pericardium
* Posterior mediastinum: between pericardium and thoracic vertebrae from fourth to twelfth.

Main contents:

* *Superior mediastinum*: esophagus, thoracic duct, trachea, aortic arch and its branches, brachiocephalic veins, superior vena cava, phrenic nerves, vagus nerves, lymph nodes and remains of thymus gland.
* *Posterior mediastinum:* esophagus, thoracic duct, vagus nerves, azygos vein, hemiazygos veins, descending aorta, sympathetic trunks and lymph nodes.
* *Middle mediastinum:* heart, pericardium, superior and inferior vena cava, ascending aorta, pulmonary trunk, pulmonary veins and phrenic nerves.
* *Anterior mediastinum:* remains of thymus gland and lymph nodes.

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**Conclusion:**

The mediastinum is the median partition of the thoracic cavity. The mediastinum is subdivided into parts. Each part contains several important structures. The relations between the different structures in each part of the mediastinum are important clinically.

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**Take home messages:**

* The boundaries and contents of each mediastinum.
* The relations between the structures in each mediastinum.
* The clinical importance of the mediastinum

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**Further reading:**

* Clinical Anatomy for Medical Students by Richard S. Snell- Latest Edition.
* Grant’s Atlas of Anatomy by Anne and Arthur –Latest Edition.

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| --- |
| Title of the lecture: Immunology of TB |

Lecturer’s name : Prof. Zahid Shakoor

Department : Pathology (Immunology)

Block / week : Respiratory Block (Week 3)

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**Objectives of the lecture:**

* To know how M. tuberculosis infection is contracted and its initial encounter with the immune system
* To understand delayed type of hypersensitivity reaction against M. tuberculosis
* To identify possible outcomes of the infection with M. tuberculosis in immuno-competent and immuno-compromised hosts.
* To know the basis of interferon gamma release assay and its potential to detect latent tuberculosis.
* To identify the basis of tuberculin test and its importance in gauging immunity against M. tuberculosis

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**Keywords:**

(Pulmonary TB, Primary TB, Ghon complex, Granuloma, Latent TB, Reactivation of TB, Cytokines, Tuberculin test, Interferon gamma release assay)

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**Background:**

* To know that Mycobacterium TB (MTB) causes chronic infection and is handled by mononuclear cells
* Exposure to MTB generates specific cell mediated immunity by antigen presentation resulting in granuloma formation
* The possible outcomes of MTB interaction with the host immune system

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**Main concepts in the lecture:**

Pulmonary TB is described in reference to mode of transmission along with mycobacterial virulence and host factors responsible for predisposition to development of pulmonary TB. The innate and adaptive immune responses play a key role in protecting individual from acquiring the infection. Breaches in immune responses may allow mycobacterium to survive and grow in about 10% of humans resulting in disease manifestations. Depending upon the immune status of the host the infection can either be cleared completely, cause a latent infection, result in primary disease or may manifest as reactivation of previous infection. Primary infection by mycobacterium TB typically presents as Ghon complex comprising of the involvement of lung parenchyma and lymph nodes. Latent TB occurs when immune status of the individual fails to clear mycobacterial infection with simultaneous containment of florid mycobacterial infection. Reactivation of old mycobacterial infection may occur in immune-compromised individuals. The characteristic feature of TB infection is the formation of caseating granuloma. The primary focus of granuloma is the presentation of mycobacterial antigen to T lymphocytes and its evolution with the passage of time. Exposure to mycobacterium TB usually results in a positive tuberculin test. The interpretation of tuberculin tests provides valuable information about the immune status of the individual and the disease severity. Interpretation of interferon of the results of interferon release assay is discussed with reference to the ability of this test to differentiate between active and latent mycobacterial infection.

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**Conclusion:**

Only about 10% of the population exposed to MTB is likely to acquire infection depending upon the host immunity and microbial virulence factors. Vast majority of the humans are protected because of the acquired immunity due to BCG vaccination. Immune compromised individuals are more likely to suffer from reactivation of past infection or conversion of latent TB to active infection. Whereas TB granuloma may contain the spread of infection it may however serve as possible source of future infection.

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**Take home messages:**

* After exposure to M. tuberculosis immune handling of the infection determines the final outcome.
* Relatively small proportion of individuals develop primary disease
* Reactivation of tuberculosis can occur in patients who are immuno-compromised
* Tuberculin test should be interpreted with caution as it may be difficult to differentiate between DTH against M. tuberculosis and latent disease

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**Further reading:**

Kuby Immunology 7th Edition Page 564-565

Robbins & Cotran Pathologic Basis of Disease 8th Edition, Page 366-372.

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| --- |
| Title of the lecture: Community Acquired Pneumonia |

Lecturer’s name : Prof. Ali Somily & Dr. Fawzia Alotaibi

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Block / week : Respiratory Block

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**Objectives of the lecture:**

* Define the term pneumonia, community acquired pneumonia ( CAP ) and healthcare associated pneumonia ( HCAP ), hospital acquired pneumonia ( HAP ) and ventilator associated pneumonia ( VAP ).
* Name their different causative agents.
* Describe their epidemiology and pathogenesis.
* Classify and describe their types.
* Describe their different chemotherapeutic antimicrobial agents.
* Evaluate response to treatment and recognize reasons for failure of treatment.
* Recognize the methods for prevention.

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**Background:**

* General classification of bacteria
* Anatomical structure of the lower respiratory tract
* Knowledge about the major classes of antimicrobial agents

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**Main concepts in the lecture:**

Recognize the epidemiology of pneumonia

Understand the pathogenesis of lower respiratory infections especially community acquired pneumonia (CAP) including the host and pathogens factors contributing in that.

Enumerate the pathogens attribute in causing of lower respiratory tract infections and CAP

Recognize the major symptoms, signs and complications of lower respiratory tract infections and radiological and laboratory investigations for the diagnosis of lower respiratory tract infections especially CAP

Understand the fundamental of management and prevention of lower respiratory tract infections and CAP

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**Conclusion:**

Pneumonia is a major syndrome in clinical practice, understanding epidemiology pathogenesis and risk factors help in prevention of this disease. Ability to recognize the signs and symptoms and major pathogens causing this disease determine the appropriated antimicrobial agents to be used.

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**Take home messages:**

* Importance of understanding classification and causative agents in understanding the community acquired pneumonia
* There difference in the epidemiology and pathogenesis of various types of community acquired pneumonia
* The recognition of the severity of the clinical presentation and diagnosis of community acquired pneumonia affect management plan
* Immediate management and future prevention of community acquired pneumonia is critical.

**Further reading:**

1. Medical microbiology by Patrick Murray Ken Rosenthal Michael Pfaller (2015), 8th Edition. Elsevier ISBN: 978-0-323-29956-5.
2. Clinical Guidelines Diagnosis and Treatment Manual for Curative Programmes in Hospitals and Dispensaries Guidance for prescribing. 2016 Edition; Medecins Sans Frontieres. 2016 Edition ISBN 987-2-37585-001-5

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| --- |
| Title of the lecture: Health Care Associated Pneumoniae |

Lecturer’s name : Prof. Hanan Habib & Prof. Ali Somily

Department : Pathology (Microbiology)

Block / week :Respiratory /3

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**Objectives of the lecture:**

* Define the terms commonly acquired pneumonia, hospital acquired, health care pneumonia and ventilator associated pneumonia
* Describe the pathogenesis of hospital acquired pneumonia, health care and ventilator associated pneumonia.
* Describe the classification of health care associated (hospital associated pneumonia) according to the time of onset.
* Name the different causative bacterial agents as per the time of onset.
* Classify and describe the type of ventilator associated pneumonia (VAP)
* Describe the different chemotherapeutic anti-microbial agents for the treatment of health care associated pneumonia.
* Evaluate response to treatment and recognized reasons for failure of treatment.
* Recognize the ways by which (VAP) is prevented.

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**Background:**

Pneumonia is the infection of the pulmonary parenchyma although a common cause of morbidity and mortality it is often misdiagnosed. It is classified as commonly susceptible to antimicrobial agents. The commonest causative organism is *S.* *pneumoniae* and health care associated pneumonia i.e. is caused by organisms found in the hospital and other health care institutions. These types of pneumonia are mostly caused by organisms resistant to antimicrobial agents. The commonest causative organisms these days are *Pseudomonas aeruginosa* and *Acinetobacter* species. These pneumonias mostly affected patients who are immunocompromised or having mechanical ventilation in intensive care unit. It is then called ventilator associated pneumonia (VAP).

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**Main concepts in the lecture:**

Definition of pneumonia is an infection of the lung parenchyma it is a common infection with high mortality and morbidity.

The differences between communities acquired pneumonia caused by organism (bacteria) in the community outside the hospital. The names of the most common bacteria will be mentioned. Special stress will be put on *S. pneumoniae* as the most important cause of community acquired pneumonia. Organisms like *H.Influenzae* and the atypical cause like *Chlamydia pneumoniae* and *Mycoplasma pneumoniae* shall be mentioned as well.

The definition and description of health care associated pneumonia will be clarified. The relationship and similarity between health care associated ( HCAP), hospital associated and ventilator associated pneumonia (HAP)& (VAP) will be explained. The importance of this pneumonia as a cause of morbidity and mortality will be explained.

The pathogenesis of HCAP & HAP as well VAP will be explained as being a consequence of spreading of organism from the oropharynex down to the lung. As these organisms are usually found in the hospital environment, they are resistant to many antibiotics and may be more virulent. The condition of the patients as being immunocompromised and on mechanical ventilation in case of VAP reduce the patient host defenses can be easily overcomed by the large numbers of pathogenic bacteria found and transmitted by micro-aspiration of oropharyngeal secretions.

The interaction of host factors, prior antimicrobial therapy which generate resistant organism, invasive devices and contaminated water medication as mediators of pathogenesis through inhalation and aspiration will be explained.

The classification of HCAP on the basis of time of onset <4 day or >4 days and the effect of this on the type of causative organism. The importance of *P.aeruginosa* and *Acinetobacter* species as causes of late onset will be discussed.

The preventive measures and treatment of (VAP) will be discussed. The importance of empirical therapy to cover all possible resistant organisms like vancomycin for MRSA, broad spectrum antipseudomonal agents will be stressed. Non response to therapy of VAP and the other possible differential diagnosis will be considered and explained in details.

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**Conclusion:**

HAP &VAP are commonly caused by organism commonly found in the hospital after 4 days of admission and most are resistant to antibiotics. Most patients have impaired host defense mechanisms. Empirical therapy should include antimicrobial agents against MRSA and *P.aeruginosa* and *Acinetobacter* species.

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**Take home messages:**

1. Pneumonia is classified in to:

* Community acquired and
* Health care acquired (HCAP)

1. VAP is hospital acquired and when related to mechanical ventilator to assist breathing.
2. All type of HCAP are mostly caused organisms found in the hospital and mainly resistant to antibiotics.
3. The pathogenesis of HCAP is mainly due to impaired host defenses and aspiration or inhalation of the organisms from oropharyngeal secretion.
4. The type of organisms depends on the time of onset of pneumonia in the long term. *P.aeruginosa* *Acinetobacter* species and other gram negative rods are the commonest causes as well as MRSA.
5. Empirical therapy should include antibiotic agents against MRSA, and broad spectrum antibiotic agents against P.aeroginosa and *Acinetobacter* species.

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**Further reading:**

Sherries Medical Microbiology, an Introduction to Infectious Diseases. Latest edition by Kenneth Ryan and George Ray. Publisher: McGraw Hill.

|  |
| --- |
| Title of the lecture: Viruses Causing Respiratory Infections 1 |

Lecturer’s name: Dr. Abdulkarim Alhetheel & Dr. Mona Badr

Department: Pathology/Microbiology

Block / week: Respiratory/ W4

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**Objectives of the lecture:**

At the end of the lecture students must be aware of the main viral causes of respiratory tract infections. In the 1st lecture, Influenza viruses, Parainfluenza viruses, Respiratory syncytial virus (RSV) and human metapneumovirus (HMPV), Measles virus, and Mumps Virus will be covered.

Upon completion of lectures, students should be able to:

* Acquire the basic knowledge about structure and classification of these viruses infecting the respiratory system.
* Describe their epidemiology and pathogenesis
* Identify the respiratory infections and the clinical features of URTI and LRTI.
* Describe their epidemiology and pathogenesis
* Know the laboratory diagnosis, and treatment of these infections.
* Recognize the methods for prevention.

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**Background:**

* Respiratory infections are the commonest of human infections and cause a large amount of morbidity and loss of time at work (sick leave).
* Respiratory infections are common in both children and adults.
* Respiratory infections are mostly caused by viruses.
* Respiratory infections are mostly mild and confined to the upper respiratory tract (URT).
* Respiratory infections are mostly self-limiting disease.
* Respiratory infections may spread to other organs causing more severe infection and death.

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**Main concepts in the lecture:**

1. Influenza viruses:Influenza viruses belong to the family Orthomyxoviridae. There are three types of influenza viruses, known as type A, B and C. Influenza A viruses undergo major (shift) and minor (drift) antigenic variations, influenza B undergoes minor antigenic variation only, whereas influenza C is antigenically stable. The main symptoms of influenza are fever, malaise, chills, sore throat, headache, cough and generalized aches. Prognosis is good, recovery is usual. Complications include primary influenza pneumonia and secondary bacterial pneumonia. Lab diagnosis is not needed, diagnosis depends mainly on symptoms. Anti-viral drugs are available for treatment and prevention. There are two types of vaccines; both vaccines contain the current influenza A viruses and the current influenza B strains.
2. Parainfluenza viruses, Respiratory syncytial virus (RSV), human metapneumovirus (HMPV), Measles virus, and Mumps Virus belong to the family Paramyxoviridae. Croup is an acute respiratory disease, mostly caused by viruses, mainly parainfluenza virus’s types 1 and 2, influenza virus. RSV, HMPV and parainfluenza virus type 3 are the major causes of Bronchiolitis and pneumonia in infants and young children: The clinical presentation, diagnosis and management of croup, bronchiolitis and pneumonia will be explained.
3. Both Measles and Mumps are highly contagious diseases caused by the measles virus and mumps virus respectively. They infect first epithetical cells of respiratory tract then virus spread to the blood causing viremia the virus widely disseminated to the skin causing maculopopular rash (measles) or to parotid gland mainly causing parotitis (mumps). About one out of 10 children with measles also gets an ear infection, and up to one out of 20 gets pneumonia. Both measles and mumps are diagnosed by serology test for the detection of measles IgM and mumps IgM. No specific treatments for measles or mumps, both diseases are prevented by vaccine

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**Conclusion:**

Influenza viruses, parainfluenza viruses, RSV, and HMPV usually cause confined lower and upper respiratory illnesses while Measles and Mumps viruses cause systemic infection. All transmitted though inhalation of infectious respiratory droplets. Diagnosis is done by using several methods such as DIF, ELISA, or PCR.

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**Take home messages:**

* Influenza A virus is the only zoonotic influenza virus.
* Influenza is only viral respiratory disease that is treated by specific antiviral therapy and is prevented by vaccine, but should be given annually.
* Parainfluenza viruses type 1 and 2 are the major causes of croup in young children
* RSV, HMPV and Parainfluenza viruses -3, the major cause of bronchiolitis and pneumonia in infants and young children.
* Diseases caused by Influenza, Parainfluenza viruses, and RSV are diagnosed in the lab by detection of their antigens in NPA.
* Measles and Mumps viruses are transmitted though inhalation of infectious respiratory droplets, but causing systemic infection. So they are diagnosed by the detection of specific IgM in the serum.
* Mumps and measles are preventable by vaccination.

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**Further reading:**

1. Medical Microbiology and Immunology. 10th Edition, 2008. By: Warren Levinson.

Published By: McGraw-Hill Co.

1. Medical Microbiology. 17th Edition, 2007, By: David Greenwood, Richard C.B. Slack, John F Peutherer and Mike Barer.

Published By: Elsevier Limited

|  |
| --- |
| Title of the lecture: Respiratory Fungal Infections |

Lecturer’s name : Dr. Ahmed Albarrag & Dr. Maha Almohizea

Department : Pathology (Microbiology)

Block / week : Respiratory Block (Week 4)

Email address : [aalbarrag@ksu.edu.sa](mailto:aalbarrag@ksu.edu.sa) & maha\_mm990@hotmail.com

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**Objectives of the lecture:**

Upon completion of lectures, students should be able to:

* Acquire the basic knowledge about fungal infections of the respiratory system.
* Know the main fungi that affects the respiratory system.
* Identify the clinical settings of such infections.
* Know the laboratory diagnosis, and treatment of these infections.

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**Keywords:**

Fungi, lung, Sinusitis, Invasive fungal infections, Allergy.

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**Background:**

Fungal infections of the respiratory system is can be divided into those that occur in generally healthy individuals and those that occur opportunistically in immunosuppressed patients. In the first group organisms such as *Histoplasma capsulatum*, *Coccidioides immitis* and *Blastomyces dermatitidis* are frequent pathogens and they are endemic in certain regions. Fungi which affect immunosuppressed individuals are frequently species of *Aspergillus* spp. and *Candida* spp. as well as *Zygomycetes* and *Cryptococcus neoformans*. The other uncommon fungi are*, Fusarium* spp., *Penicillium marneffii* and *Pseudallescheria boydii* and others.

Rates of invasive fungal infections have increased during recent decades, largely because of the increasing size of the population at risk. This population includes patients who are immunosuppressed because of diseases, such as cancers, and those with human immunodeficiency virus (HIV) infection. It also includes patients taking immunosuppressive drugs.

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**Main concepts in the lecture:**

Fungal infections of the respiratory system is caused by either primary fungal pathogens or opportunistic pathogens. They can be divided into those that occur in generally healthy individuals and those that occur in immunosuppressed patients.

The main risk factors include:

* Bone marrow/ organ transplantation
* Cancer: Leukemia, lymphoma etc
* Drugs: Cytotoxic drugs, steroids, etc
* Endocrine related: Diabetes
* AIDS

Primary systemic infections:

Infections of the respiratory system, acquired through Inhalation. Mild disease but dissemination seen in immunocompromised hosts. Common in North America and South America but not seen in Saudi Arabia in patients with no travel history.

Etiologies are dimorphic fungi and considered as primary pathogens and are highly infectious. They include: Histoplasmosis, Blastomycosis, Coccidioidomycosis, and Paracoccidioidomycosis

Aspergillosis:

Aspergillosis is a spectrum of diseases of humans and animals caused by members of the genus *Aspergillus*.

These include: (1) Mycotoxicosis (2) Allergy (3) Colonization (without invasion and extension) in preformed cavities

(4) Invasive, inflammatory, granulomatous, necrotizing disease of lungs, (5) disseminated disease.

The type of disease and severity depends upon the immunological and physiologic state of the host and the species of *Aspergillus* causing the disease.

Aetiological Agents: Common species are *A. fumigatus,* *A. flavus, A. terreus, A. niger*

Zygomycosis:

Can be pulmonary zygomycosis or rhinocerebral zygomycosis. The main risk factors include: Diabetic ketoacidosis,

Malignancy, HSCT, granulocytopenia, and many others. Uncontrolled diabetes is a common host factor.

Etiology: is Zygomycetes fungi which have Non-septate hyphae e.g. *Rhizopus, Mucur, Absidia*

The disease present with Angioinvasion, pulmonary infractions and hemorrhage.

Pneumocystosis (PCP):

Opportunistic fungal pneumonia, It is interstitial pneumonia of the alveolar area. Affect compromised host especially common in AIDS patients. Etiology is *Pneumocystis jiroveci*. Does not grow in laboratory media e.g. SDA. Naturally found in rodents (rats), other animals (goats, horses), Humans may contract it during childhood. For Laboratory Diagnosis, Bronchoscopic specimens (B.A.L.), or lung biopsy tissue. Histological sections or smears stained by GMS stain or immunuofluorescence (has better sensitivity) will show cysts of hat-shape, cup shape, crescent.

Treatment is Trimethoprim – sulfamethoxazole or combination with Dapsone.

Pulmonary Candidiasis:

Primary pneumonia is less common and could be a result of aspiration pneumonia. Can be seen with hematogenous candidiasis (dissemination) in immunocompromised patients. Isolation of *Candida* from sputum is not always significant.

Other yeast causing pulmonary infections and *Trichosporon* and *Geotrichum*.

Diagnosis of respiratory fungal infections:

Type of Specimens could one of respiratory specimens such as sputum, BAL, lung biopsy, or other sample based on the type of infection. Blood can be collected for serology. Microbiological Lab. Investigations: Direct Microscopy: Smear stained with Giemsa, Grecott methenamine silver stain (GMS). Will show fungal elements e.g. yeast, septate hyphae or non-spetate hyphae based on the etiology. Samples are cultured on SDA. Identification of fungi will be based on macroscopic and microscopic characteristics and biochemical tests. Serology is available for Aspergillosis, Candidiasis, and PCP.

Management of respiratory fungal infections:

Choice of antifungal agents is based on the type of fungi and clinical features, and condition of the patient. For systemic treatment, Amphotericin B, Fluconazole, Voriconazole, or Caspofungin can be selected.

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**Conclusion:**

Invasive fungal infections of the respiratory system are opportunistic infections in immunosuppressed patients. Risk factor are cancer, AIDS, transplantation, diabetes and others. The type of disease and severity depends upon the immunological and physiologic state of the host and the fungi causing the disease. Diagnosis is made by selection of appropriate clinical sample and microbiological investigations. Radiology and other investigations are necessary in many cases. Choice of antifungal agents is dependent on identification of fungi and also considering the clinical features of the disease and condition of the patient.

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**Take home messages:**

* Invasive fungal infections of the respiratory system are opportunistic infections in immunosuppressed patients
* Fungi frequently cause this infections are *Aspergillus* spp. *Zygomycetes* and *Candida* spp .in addition to other less common fungi.
* Rates of invasive fungal infections have increased mainly because of the increase in the population at risk.
* Patients who are at risk include cancer, human immunodeficiency virus (HIV) infection, transplant patients, diabetes.
* A spectrum of diseases is caused by different fungi ranging from allergy, chronic disease, invasive, inflammatory, granulomatous, disease of lungs, and disseminated disease.
* The type of disease and severity depends upon the immunological and physiologic state of the host and the fungi causing the disease.
* Microbiological Lab. Investigations include direct Microscopy and culture on SDA.
* Choice of antifungal agents is based on the type of fungi , clinical features, and condition of the patient
* Begin antifungal therapy early.
* Control the underlying disease

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**Further reading:**

Sherries Medical Microbiology, an introduction to Infectious Diseases. Latest edition, Kenneth Ryan and George Ray. Publisher: Mc Graw Hill.

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

|  |
| --- |
| Title of the lecture: Viruses Causing Respiratory Infections 2 |

Lecturer’s name: Dr. Abdulkarim Alhetheel & Dr. Mona Badr

Department: Pathology/Microbiology

Block / week: Respiratory block / W4

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**Objectives of the lecture:**

In the 2nd lecture, Coronavirus (SARS & Middle East Respiratory Syndrome - Coronavirus COV (MERS-CoV), Rhinovirus, Enteroviruses, Adenovirus, and EBV will be covered for the following aspects:

* Structure and classification of these viruses.
* Pathogenesis.
* Target group and modes of transmission
* Clinical manifestations.
* Laboratory diagnosis.
* Prevention and treatment.

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**Background:**

* Most respiratory viral infections are mild and confined to the upper respiratory tract (URT).
* Viruses like Coronavirus (MERS-COV), Enteroviruses, Adenovirus and EBV start with respiratory illnesses but may spread to other organs causing systemic infections and complications.

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**Main concepts in the lecture:**

1. Rhinoviruses and coronaviruses other than MERS CoV are the major cause of Common cold:Rhinoviruses have been classified within the family Picornaviridae. They are unenveloped small spherical particles with ss-RNA genome of positive polarity. On the other hand, Coronaviruses are pleomorphic, enveloped, with large glycoprotein spikes and have ss-RNA genome with positive polarity. Diagnosis usually based on the clinical features, therefore lab diagnosis not needed. Common cold is mild self-limiting disease, therefore, there is no treatment or vaccine is available.
2. MERS-CoV:Middle East Respiratory Syndrome (MERS) is a respiratory illness caused by a coronavirus called MERS-CoV. Clinical features vary from asymptomatic to severe acute respiratory illness. Some develop severe complications such as pneumonia and kidney failure. People with comorbidities such as diabetes, cancer, and chronic lung, heart, and kidney disease may be more likely to become infected with MERS, or have a severe case. So far, all the cases have been linked to countries in and near the Arabian Peninsula. This virus has spread from ill people to others through close contact. However, there is no evidence of sustained spreading in community settings. Lab diagnosis: detection of the viral nucleic acid by PCR. Currently, there is no specific antiviral treatment and no vaccine available to prevent MERS-CoV infection. People are advised to protect themselves from respiratory illnesses by taking preventive actions.
3. Enteroviruses, including coxsackievirus and echovirus cause herpangina and pharyngitis. They are classified under the Picoronaviridae. Human enteroviruses cause a wide variety of diseases including respiratory diseases and other illnesses, most commonly affecting children and transmitted through the inhalation of respiratory aerosols, faecal–oral route and spreading via the bloodstream to invade different organs. Diagnosis is confirmed by isolation of viruses (on cell culture) or detection of viral nucleic acid by Reverse transcriptase PCR (RT–PCR). Most cases are self-limited.
4. Adenoviruses:Belong to the family Adenoviridae. They are unenveloped viruses with ds- DNA genomes. They cause different diseases including acute respiratory diseases, Pharyngoconjunctival fever and other non-respiratory illnesses. Adenoviral respiratory diseases are diagnosed by direct detection of their antigens in NPA, or isolation of these viruses in on cell culture, or detection of viral nucleic acid by PCR.
5. Epstein-Barr Virus (EBV): EBV belongs to Herspesviridae. It is an enveloped virus with ds-DNA genome. It is a lymphotropic virus and has oncogenic properties. The virus is transmitted mostly by saliva. It causes tonsillitis, pharyngitis, infectious mononucleosis, hepatitis, Burkett’s lymphoma, nasopharyngeal carcinoma. It causes lymphoproliferative disease, and oral hairy leukoplakia in immunocompromised. EBV is also diagnosed by detection viral specific antibody (IgM) or viral nucleic acid. There is no specific treatment or vaccine.

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**Conclusion:**

Rhinovirus and Coronavirus other than MERS cause common cold, but MERS-COV, Enterovirus, Adenovirus, and EBV may start with upper respiratory illnesses and spread to other organs. These viruses are transmitted though inhalation of infectious respiratory droplets and/or close contact. Adenovirus is diagnosed by DIF, but Rhinovirus, MERS-COV, Enteroviruses, and EBV are diagnosed by PCR. EBV is also diagnosed by serology and detection viral specific antibody (IgM) using ELISA.

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**Take home messages:**

* Rhino and coronaviruses are the major cause of common cold and their laboratory diagnosis not needed.
* MERS -CoV causes severe acute respiratory illness and complications such as pneumonia and kidney failure.
* MERS mostly seen in elderly people, low immune individuals, and individuals with comorbidities ,endemic in Middle East
* Enteroviruses, Adenoviruses and EBV cause respiratory tract infection and other diseases.

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**Further reading:**

1. Medical Microbiology and Immunology. 10th Edition, 2008. By: Warren Levinson.

Published By: McGraw-Hill Co.

1. Medical Microbiology. 17th Edition, 2007, By: David Greenwood, Richard C.B. Slack, John F Peutherer and Mike Barer.

Published By: Elsevier Limited

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| --- |
| Title of the lecture: Lung Tumors |

Lecturer’s name : DR. AMMAR AL-RIKABI and DR. MAHA ARAFAH

Department : Pathology

Block / week : Respiratory/ week 3

Email address : [ammar\_rikabi@hotmail.com](mailto:ammar_rikabi@hotmail.com) [marafah@hotmail.com](mailto:marafah@hotmail.com)

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**Objectives of the lecture:**

* Understand the incidence, age group of affected patients and predisposing factors of bronchogenic carcinoma.
* Is aware of the new classification of bronchogenic carcinoma which include: squamous carcinoma, adenocarcinoma, small cell and large cell (anaplastic) carcinomas.
* Understands the clinical features and gross pathology of bronchogenic carcinoma. Know the precursors of squamous carcinoma (squamous dysplasia) and adenocarcinoma (adenocarcinoma in situ and atypical adenomatous hyperplasia).
* Have a basic knowledge about neuroendocrine tumours with special emphasis on small cell carcinoma and bronchial carcinoid.
* Is aware that the lung is a frequent site for metastatic neoplasms.

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**Keywords:**

(Lung tumours, adenocarcinoma, squamous carcinoma, large cell and small cell carcinomas, metastatic, carcinoid).

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**Background:**

Primary lung cancer is the most common fatal cancer in both men and women worldwide. Cigarette smoking most common cause. Classified as small cell or non–small cell (most common) cancers. Primary lung cancer by specific type in decreasing incidence: Adenocarcinoma (most common primary cancer), Squamous cell carcinoma, Small cell lung carcinoma, Large cell carcinoma and Bronchial carcinoid. Histological types and the stage of lung cancer determine the outcome and its likely response to treatment. Survival is better for early stage disease, except for small cell carcinoma (very early metastases).

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**Main concepts in the lecture:**

The major histologic subtypes are adenocarcinomas (most common), squamous cell carcinoma, large cell carcinoma, and small cell carcinoma. Each of these is clinically and genetically distinct. Small cell lung carcinomas are best treated by chemotherapy, because almost all are metastatic at presentation. The other carcinomas may be curable by surgery if limited to the lung. Combination chemotherapy also is available along with tyrosine kinase inhibitors for those with EGFR, ALK, ROS, and c-MET mutations. Smoking is the most important risk factor for lung cancer; in women and nonsmokers, adenocarcinomas are the most common cancers. Precursor lesions include squamous dysplasia for squamous cancer and atypical adenomatous hyperplasia and adenocarcinoma in situ (formerly bronchioloalveolar carcinoma) for adenocarcinomas. Tumors 3 cm or less in diameter characterized by pure growth along preexisting structures (lepidic pattern) without stromal invasion are now called adenocarcinoma in situ.. Lung cancers, particularly small cell lung carcinomas, can cause paraneoplastic syndromes.

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**Conclusion:**

Lung cancer is one of the most insidious and aggressive neoplasms. in patients in their 50s or older whose symptoms are of several months’ duration. Symptoms of metastases depend on the site, for example, back pain in bone metastases, headache, hemiparesis, cranial nerve damage, and seizures in brain metastases.

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**Take home messages:**

* The four major histopathological subtypes of lung cancer are: squamous, large cell, small cell and adenocarcinomas. Each of these tumours are genetically and clinically distinct.
* Smoking is the most important risk factor for lung cancer. The women and non-smokers adenocarcinomas are the most common cancers.
* Lung cancers, particularly SCLC’s can cause paraneoplastic syndromes.

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**Further reading:**

Robbin’s, Basic of Pathology, 9th Edition pages 505-512.

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| --- |
| Title of the lecture: Pneumonia |

Lecturer’s name : DR. AMMAR AL-RIKABI and DR. MAHA ARAFAH

Department : Pathology

Block / week : Respiratory/ week 4

Email address : [ammar\_rikabi@hotmail.com](mailto:ammar_rikabi@hotmail.com) [marafah@hotmail.com](mailto:marafah@hotmail.com)

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**Objectives of the lecture:**

* Understand that pneumonia is an inflammatory condition of the lung characterized by consolidation (solidification) of the pulmonary tissue.
* Is aware of the pathogenesis of pneumonia and its classification which principally include bronchopneumoniae, lobar pneumonia and atypical pneumonia.
* Is able to appreciate the aetiology and pathogenesis of lung abscess.

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**Keywords:**

(Bronchopneumoniae, lobar pneumonia, S. pneumoniae, atypical pneumonia, Mycoplasma pneumoniae, Lung abscess)

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**Background:**

* Respiratory tract infections are more frequent than infections of any other organ and account for the largest number of workdays lost in the general population. The vast majority are upper respiratory tract infections caused by viruses (common cold, pharyngitis), but bacterial, viral, mycoplasmal, and fungal infections of the lung (pneumonia) still account for an enormous amount of morbidity and are the eighth leading cause of death
* S. pneumoniae is the most common cause of community acquired acute pneumonia. The distribution of inflammation is usually lobar.
* Other causes of acute pneumoniaes include H. influenza and m.catarrhalis. S.aureus is usually secondary to viral infections.
* Atypical pneumoniaes are characterized by inflammation that is predominantly confined to alveolar septae with generally clear alveoli. The most common causes are viruses and mycoplasma pneumoniae.

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**Main concepts in the lecture:**

Pneumonia is defined as the infection of alveolar tissue resulting in the consolidation of lung tissue with an intra-alveolar inflammatory exudate. It is most common in the very young and the elderly. Predisposing factors include suppressed cough reflex, impaired mucociliary clearance, pulmonary oedema, impaired alveolar macrophages, retention of secretions, immunosuppression, instrumentation and prior viral respiratory tract infection. Pneumonia can be classified into lobar pneumonia and bronchopneumonia. Lobar pneumonia is a uniform or homogenous consolidation of part of a lobe or of the whole lobe caused by infection in bronchopneumonia, infection is centred on the bronchi but with the extension of the inflammatory exudate into the alveoli, causing a patchy consolidation of the lung (lobular distribution). Complications and sequelae include resolution—complete resolution occurs only if treatment is instituted early, before the onset of structural damage, bronchial damage—imperfect repair of the bronchial mucosa results in scarring of the bronchial wall, with increased predisposition to further infection and to bronchiectasis, lung fibrosis—inflammatory exudate is often not completely absorbed but is organized with residual fibrous scarring, lung abscesses—single or multiple areas of suppurationa, empyema—pus in the pleural cavity as a result of extension of infection into the pleural cavity, pericarditis—direct extension of infection to the pericardium and death—very common cause of death, particularly as a terminal manifestation of debilitating diseases.

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**Conclusion:**

Pneumonia is classified as community-acquired or nosocomial (hospital-acquired). Community-acquired pneumonia is further subdivided into typical and atypical pneumonia. Typical community-acquired pneumonia is most often due to Streptococcus pneumoniae (50%–75% of cases. Atypical pneumonia are usually caused by Mycoplasma pneumoniae Other pathogens include Chlamydophila pneumoniae, Viruses and Chlamydia trachomatis in newborns. Nosocomial pneumonia are commonly caused by Gram-negative bacteria such as Pseudomonas aeruginosa (respirators) and Escherichia coli. Pneumonia in immunocompromised hosts, occur as a complication of AIDS and bone marrow transplantation, are caused by opportunistic infections including Cytomegalovirus, Pneumocystis jiroveci and Aspergillus fumigatus. Lung abscesses are most often due to aspiration of oropharyngeal material (e.g., tonsillar material). Risk factors include alcoholism, loss of consciousness, recent dental work, microbial pathogens (aerobic and anaerobic streptococci, staphylooccus, Prevotella, Fusobacterium and anaerobes in 60% of cases). Lung abscess can occur as a complication of bacterial pneumonia such as Staphylococcus aureus or Klebsiella pneumoniae infection, septic embolism from infective endocarditis and from obstructive lung neoplasia (From 10% to 15% of abscesses are behind a bronchus obstructed by cancer).

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**Take home messages:**

* S. pneumoniae (the pneumococcus) is the most common cause of community-acquired acute pneumonia; the distribution of inflammation is usually lobar.
* Lobar pneumonias evolve through four stages: congestion, red hepatization, gray hepatization, and resolution.
* Other common causes of acute pneumonias in the community include H. influenzae and M. catarrhalis (both associated with acute exacerbations of COPD), S. aureus (usually secondary to viral respiratory infections), K. pneumoniae (observed in patients who are chronic alcoholics), P. aeruginosa (seen in persons with cystic fibrosis, in burn victims, and in patients with neutropenia), and L. pneumophila, seen particularly in organ transplant recipients.
* The term atypical organisms is used for Mycoplasma pneumoniae, Chlamydophila pneumoniae, Coxiella burnetii, and viruses (influenza viruses types A and B, human metapneumovirus) since they are not detectable on Gram stain nor do they grow on the standard bacteriologic culture media.
* Bacterial pneumonias are characterized by predominantly intra-alveolar neutrophilic inflammation while viral pneumonia shows interstitial lymphocytic inflammation. Characteristic viral inclusions may be seen.
* Lung abscess is often caused by anaerobic organisms or by mixed infections and frequently occur in debilitated individuals following aspiration of oral flora

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**Further reading:**

Robbin’s Basic Pathology 9th Edition pages. 472 to 481

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| Title of the lecture: (Dynamic Spirometry) |

Lecturer’s name : Dr. Aurangzeb T. Halepota, Dr. Mustafa Kamal, Dr. Muhammad Yehya

Department : Physiology

Block / week : Respiratory Practicals

Email address : [amalhowikan@gmail.com](mailto:amalhowikan@gmail.com)

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**Objectives of the lecture:**

* To be able to use a spirometer and determine the lung volumes and capacities.
* Compare and evaluate the obtained values with the normal.
* Recognize the factors responsible for modifying the lung function.

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**Keywords:**

(Volumes, Capacities, Obstructive and Restrictive Airway Disease)

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**Background:**

* The various pressure changes in the Pleura and the lungs during breathing.
* The student should know the mechanics of breathing.
* Factors that Increase and Decrease the work of breathin

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**Main concepts in the lecture:**

By the end of this sitting the student should be able to diagnose Obstructive and Restrictive Airway diseases based on the analysis of the various graph and calculations performed.

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**Conclusion:**

* The recognition of various volumes and capacities and their changes.
* Diagnostic points of COPD and CRPD
* Their differences.

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**Further reading:**

* Guyton’s Textbook of Medical Physiology by John Hall
* Review of Medical Physiology by Ganong

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| --- |
| Title of the lecture: (Simple Spirometry) |

Lecturer’s name : Dr. Aurangzeb T. Halepota, Dr. Mustafa Kamal, Dr. Muhammad Yehya

Department : Physiology

Block / week : Respiratory Practicals

Email address : [amalhowikan@gmail.com](mailto:amalhowikan@gmail.com)

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**Objectives of the lecture:**

* To be able to use a spirometer and determine the lung volumes and capacities.
* Compare and evaluate the obtained values with the normal.
* Differentiate between a volume and a capacity.

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**Keywords:**

(Volumes, Capacities, VT, IRV, IC, VC, ERV and Residual Volume)

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**Background:**

* The various pressure changes in the Pleura and the lungs during breathing.
* The student should know the mechanics of breathing.
* To be able to measure all the volumes and capacities.

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**Main concepts in the lecture:**

By the end of this sitting the student should be able do Simple Spirometry and interpret the various volumes and Capacities.

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**Conclusion:**

* The recognition of various volumes and capacities and their changes.
* Their differences.
* How to measure and understand the basic changes in the spirogram.

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**Further reading:**

* Guyton’s Textbook of Medical Physiology by John Hall
* Review of Medical Physiology by Ganong

**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 represent 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. He/she might email them and arrange a time to see them if needed, otherwise email might be of help.

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| --- | --- | --- | --- |
| **CHAIRPERSON :** Dr. Malak El-Hazmi  Pathology Department - Microbiology  Email : [melhazmi@ksu.edu.sa](mailto:melhazmi@ksu.edu.sa) | | **CO-CHAIR :** Dr. Sami Al Nassar  Medical Education Department  Extension : 70191  Email : [dralnassar@hotmail.com](mailto:dralnassar@hotmail.com) | |
| **MEMBERS** | **DEPARTMENT** | **HOSP. EXT.** | **E-MAIL ADDRESS** |
| Professor Samy Azer | Medical Education Department | 99178 | [sazer@ksu.edu.sa](mailto:sazer@ksu.edu.sa) |
| Prof. Ahmed Fathalla | Anatomy | 71314 | [ahmedfathala@hotmail.com](mailto:ahmedfathala@hotmail.com) |
| Dr. Abdulrahman AlHowaikan | Physiology | 71613 | [amalhowikan@gmail.com](mailto:amalhowikan@gmail.com) |
| Dr. Ahmed Mujamammi | Biochemistry | 71339 | Mujamammi@gmail.com |
| Dr. Maha Arafa | Pathology | 71067 | [marafah@ksu.edu.sa](mailto:marafah@ksu.edu.sa) |
| Dr. Ishfaq Bukhari | Pharmacology | 71325 | ishfaqbukhari@yahoo.com |
| Prof. Zahid Shakoor | Immunology | 71229 | [shakoor\_zahid@yahoo.com](mailto:shakoor_zahid@yahoo.com) |

**Schedule of the Block**

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| **WEEK 1 – RESPIRATORY BLOCK ( Male – A )** | | | | |
| **Week ( 1 ) Starting: 05/02/2017 to 09/02/2017 (08/05/1438) to (12/05/1438)**  **Normal Breathing and Respiratory Function** | | | | |
| **CHAIR PERSON: Dr. Malak El-Hazmi** | | | | |
| **CO-CHAIR: Dr. Sami Al-Nassar** | | | | |
| **Sunday**  **05 February 2017** | **Monday**  **06 February 2017** | **Tuesday**  **07 February 2017** | **Wednesday**  **08 February 2017** | **Thursday**  **09 February 2017** |
| **8:00 -9:00am**  Introduction to the Respiratory Block  **Prof. Ali Somily** | **8:00-10:00am**  **(Practical)**  Muscles involved in normal respiration  **(Anatomy/Prof. Ahmed Fathala)** | **8:00 - 9:00am**  Lung function in health and disease  **(Physiology)**  **Prof. Sultan Meo** | **8:00 – 9:00am**  Globular proteins  **(Biochemistry)**  **Dr. Ahmed Mujamammi** | **8:00 – 9:00am**  **Self – directed learning** |
| **9:00 - 10:00am**  Functional organization of the respiratory system  **(Physiology)**  **Dr. Abdulrahman Alhowikan** | **9:00 - 10:00am**  Embryology of the Respiratory System  **(Anatomy)**  **Dr. Mohammed Vohra** | **9:00 - 10:00am**  Histology of the lung and bronchial tree  **(Histology)**  **Dr. Aly Mohamed** | **9:00 - 11:00am**    **(Practical)**  Lung volumes and capacity  **(Physiology)** |
| **10:00 - 11:00am**  Muscles involved in normal respiration  **(Anatomy)**  **Prof. Ahmed Fathalla** | **10:00 - 11:00am**  Anatomy of larynx, trachea & bronchi  **(Anatomy)**  **Prof. Saeed Abuelmakarem** | **10:00 - 11:00am**  Anatomy of lungs and pleura  **(Anatomy)**  **Prof. Saeed Abuelmakarem** | **10:00 - 12:00nn**  **(Practical)**  Anatomy and histology of lung and pleura  **(Anatomy/Prof. Ahmed Fathala & Histology)** |
| **11:00- 12:00nn**  Anatomy and histology of the nasal cavity & pharynx  **(Anatomy)**  **Dr. Essam Salama**  **Dr. Aly Mohamed** | **11:00- 12:00nn**  Mechanics of the breathing  **(Physiology)**  **Dr. Abdulrahman Alhowikan** | **11:00 - 12:00nn**  Anti-cholinergic drugs  **(Pharmacology)**  **Dr. Osama Yousef** | **11:00 - 12:00nn**  **Self – directed learning** |
| **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** |
| **1:00-2:00pm**  Measuring peak expiratory flow in a normal subject  **Clinical Skills**  **(A1)** | **1:00 -2:00pm**  Respiratory ventilation  **(Physiology)**  **Prof. Sultan Meo** | **1:00 - 3:00pm**  **(Practical)**  Anatomy & Histology of upper respiratory tract  **(Anatomy/Prof. Ahmed Fathala & Histology)** | **1:00-2:00pm**  Effects of exercise on the respiratory system  **(Physiology)**  **Dr. Abdulrahman Alhowaikan** | **1:00 - 3:00pm**  **Salam** |
| **2:00-3:00pm**  Measuring peak expiratory flow in a normal subject  **Clinical Skills**  **(A2)** | **2:00-3:00pm**  **Self – directed learning** | **2:00-3:00pm**  **Self – directed learning** |

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| **WEEK 2 – RESPIRATORY BLOCK ( Male – A )** | | | | | | | | | |
| **Week (2 ) Starting: 12/02/2017 to 16/02/2017 (15/05/1438) to (19/05/1438)**  **Bronchial Asthma and Allergy** | | | | | | | | | |
| **CHAIR PERSON: Dr. Malak El-Hazmi** | | | | | | | | | |
| **CO-CHAIR: Dr. Sami Al-Nassar** | | | | | | | | | |
| **Sunday**  **12 February 2017** | | **Monday**  **13 February 2017** | **Tuesday**  **14 February 2017** | | **Wednesday**  **15 February 2017** | | **Thursday**  **16 February 2017** | | |
| **8:00 - 10:00am**  **Problem-based**  **Learning**  **Case 1 Tutorial 1** | | **8:00-9:00am**  Control of breathing  **(Physiology)**  **Dr. Abdulrahman Alhowikan** | **8:00 - 9:00am**  Treatment of acute and chronic rhinitis and cough  **(Pharmacology)**  **Dr. Saeed Ahmed** | | **8:00 -10:00am**  **Problem-based**  **Learning**  **Case 1 Tutorial 2** | | **8:00 - 9:00am**  Drugs used in anaphylaxis  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | | |
| **9:00 - 10:00am**  Hypoxia and cyanosis  **(Physiology)**  **Prof. Sultan Meo** | **9:00 - 10:00am**  Pathology of bronchial asthma  **(Pathology)**  **Prof. Ammar Al Rikabi** | | **9:00 - 10:00am**  Low and high altitude  **(Physiology)**  **Dr. Abdulrahman Alhowaikan** | | |
| **10:00 - 11:00am**  Oxygen-carbondioxide transport  **(Physiology)**  **Prof. Sultan Meo** | | **10:00- 11:00am**  **Self- Directed**  **Learning** | **10:00 - 11:00am**  **Self- Directed**  **Learning** | | **10:00- 11:00am**  **Self- Directed**  **Learning** | | **10:00 - 11:00am**  Adrenergic agonist  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | | |
| **11:00- 12:00nn**  Gas exchange and gas transfer  **(Physiology)**  **Prof. Sultan Meo** | | **11:00- 12:00nn**  Immunology of bronchial asthma  **(Immunology)**  **Prof. Adel Almogren** | **11:00- 12:00nn**  Pharmacology of drugs used in bronchial asthma  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | | **11:00- 12:00nn**  Phospholipids of clinical significance    **(Biochemistry)**  **Dr. Usman Ghani** | | **11:00- 12:00nn**  **Self- Directed**  **Learning** | | |
| **Lunch**  **12:00 – 1:00pm** | | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | | **Lunch**  **12:00 – 1:00pm** | | **Lunch**  **12:00 – 1:00pm** | | |
| **1:00-2:00pm**  Taking a medical history related to respiratory signs and symptoms  **Clinical Skills**  **(A1)** | | **1:00 -2:00pm**  Radiological anatomy of the chest  **(Anatomy)**  **Prof. Saeed Abuelmakarem** | **1:00 - 3:00pm**  **(Practical)**  Dynamic spirometry  **(Physiology)** | | **1:00 -2:00pm**  **Self- Directed**  **Learning** | | **1:00 - 3:00pm**  **Salam** | | |
| **2:00-3:00pm**  Taking a medical history related to respiratory signs and symptoms  **Clinical Skills**  **(A1)** | | **2:00-3:00pm**  **Self- Directed**  **Learning** | **2:00-3:00pm**  **Self- Directed**  **Learning** | |
| **WEEK 3 – RESPIRATORY BLOCK ( Male – A )** | | | | | | | | |
| **Week (3 ) Starting: 19/02/2017 to 23/02/2017 (22/05/1438) to (26/05/1438)**  **Chronic Obstructive Pulmonary Diseases and Respiratory Infections** | | | | | | | | |
| **CHAIR PERSON: Dr. Malak El-Hazmi** | | | | | | | | |
| **CO-CHAIR: Dr. Sami Al-Nassar** | | | | | | | | |
| **Sunday**  **19 February 2017** | **Monday**  **20 February 2017** | | | **Tuesday**  **21 February 2017** | | **Wednesday \***  **22 February 2017** | | **Thursday**  **23 February 2017** |
| **8:00 - 10:00am**  **Problem-based**  **Learning \*\***  **Case 2 Tutorial 1** | **8:00 - 9:00am**  Pharmacology of drugs used in COPD  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | | | **8:00 - 9:00am**  **Tuberculosis**  **(Microbiology)**  **Prof. Ali Somily** | | **Progress Test** | | **8:00 - 9:00am**  Bacteria causing upper respiratory tract infection  **(Microbiology)**  **Prof. Ali Somily** |
| **9:00 - 10:00am**  Mediastinium  **(Anatomy)**  **Prof. Ahmed Fathalla** | | | **9:00 - 10:00am**  Pathology of Tuberculosis  **(Pathology)**  **Prof. Ammar Al Rikabi** | | **9:00 - 10:00am**  Tumours of the lung  **(Pathology)**  **Prof. Ammar Al Rikabi** |
| **10:00 - 11:00 am**  Introduction to COPD including bronchiectasis, chronic bronchitis & emphysema.  **(Pathology)**  **Prof. Ammar Al Rikabi** | **10:00 - 12:00nn**  **Practical**  Mediastinium  **(Anatomy)**  **Prof. Ahmed Fathalla**  **All staff** | | | **10:00 - 11:00am**  Pharmacology of drugs used in tuberculosis  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | | **10:00 - 12:00nn**  **(Practical)**  Cancer of the lung  **(Pathology)**  **Prof. Ammar Al Rikabi** |
| **11:00- 12:00nn**  Respiratory Chain  **(Biochemistry)**  **Dr. Usman Ghani** | **11:00- 12:00nn**  Immunology of T.B  **(Immunology)**  **Prof. Zahid Shakoor** | |
| **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | | | **Lunch**  **12:00 – 1:00pm** | | **Lunch**  **12:00 – 1:00pm** | | **Lunch**  **12:00 – 1:00pm** |
| **1:00- 2:00pm**  Examination of the respiratory system and mucus membrane for cyanosis  **Clinical Skills**  **(A1)** | **1:00- 2:00pm**  Pathology of restrictive lung disease including allergic alveolitis  **(Pathology)**  **Prof. Ammar Al Rikabi** | | | **1:00 - 3:00pm**  **( Practical)**  Chronic obstructive lung disease  **(Pathology)**  **Prof. Ammar Al Rikabi** | | **1:00- 2:00pm**  **Self- Directed**  **Learning** | | **1:00 - 3:00pm**  **Salam** |
| **2:00-3:00pm**  Examination of the respiratory system and mucus membrane for cyanosis  **Clinical Skills**  **(A2)** | **2:00-3:00pm**  Tobacco consumption, problems and solutions  **(Family Medicine)**  **Dr. Ali AlHazmi** | | | **2:00-3:00pm**  **Self- Directed**  **Learning** | |

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| **WEEK 4 – RESPIRATORY BLOCK ( Male – A )** | | | | |
| **Week (4 ) Starting:: 26/02/2017 to 02/03/2017 (29/05/1438) to (03/06/1438)**  **Respiratory Infections** | | | | |
| **CHAIR PERSON: Dr. Malak El-Hazmi** | | | | |
| **CO-CHAIR: Dr. Sami Al-Nassar** | | | | |
| **Sunday**  **26 February 2017** | **Monday**  **27 February 2017** | **Tuesday**  **28 February 2017** | **Wednesday**  **01 March 2017** | **Thursday**  **02 March 2017** |
| **8:00 - 10:00am**  **Problem-based**  **Learning**  **Case 2 Tutorial 2** | **8:00-9:00am**  Pathology of Lobar pneumonia& broncho pneumonia  **(Pathology)**  **Prof. Ammar Al Rikabi** | **8:00-9:00am**  **Self- Directed**  **Learning** | **Consolidation** | **Consolidation** |
| **9:00 - 10:00am**  **Self- Directed**  **Learning** | **9:00 - 10:00am**  Respiratory fungal infection  **(Microbiology)**  **Dr. Ahmed Al Barrag** |
| **10:00 - 11:00am**  Community acquired pneumonia  **(Microbiology)**  **Prof. Ali Somily** | **10:00 - 11:00am**  Antibiotics  **(Pharmacology)**  **Prof. Mohammad Al Humayyd** | **10:00 - 11:00am**  Treatment of Respiratory tract infection  **(Pharmacology)**  **Prof. Mohammad Al Humayyd** |
| **11:00 - 12:00nn**  Viruses causing respiratory (1)    **(Microbiology)**  **Dr. Abdulkareem Al Hetheel** | **11:00- 12:00nn**  Viruses causing respiratory (2)  **(Microbiology)**  **Dr. Abdulkareem Al Hetheel** | **11:00- 12:00nn**  Hospital acquired pneumonia  **(Microbiology)**  **Prof. Ali Somily** |
| **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** |
| **1:00-3:00pm**  **Practical**  Bacteriological diagnosis of respiratory infection  **(Microbiology)**  **Prof.. Ali Somily** | **1:00-2:00pm**  **Self- Directed**  **Learning** | **1:00-2:00pm**  **Self- Directed**  **Learning** |
| **2:00-3:00pm**  **Self- Directed**  **Learning** | **2:00-3:00pm**  **Self- Directed**  **Learning** |

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| **WEEK 2 – RESPIRATORY BLOCK B ( Male – B )** | | | | |
| **Week (2 ) Starting: 12/02/2017 to 16/02/2017 (15/05/1438) to (19/05/1438)**  **Bronchial Asthma and Allergy** | | | | |
| **CHAIR PERSON: Dr. Malak El-Hazmi** | | | | |
| **CO-CHAIR: Dr. Sami Al-Nassar** | | | | |
| **Sunday**  **12 February 2017** | **Monday**  **13 February 2017** | **Tuesday**  **14 February 2017** | **Wednesday**  **15 February 2017** | **Thursday**  **16 February 2017** |
| **8:00 - 10:00am**  **Problem-based**  **Learning**  **Case 1 Tutorial 1** | **8:00-10:00am**  **(Practical)**  Dynamic spirometry  **(Physiology )** | **8:00 - 9:00am**  Control of breathing  **(Physiology)**  **Dr. Abdulrahman Alhowikan** | **8:00 -10:00am**  **Problem-based**  **Learning**  **Case 1 Tutorial 2** | **8:00 - 9:00am**  **Self- Directed**  **Learning** |
| **9:00 - 10:00am**  Hypoxia and cyanosis  **(Physiology)**  **Prof. Sultan Meo** | **9:00 - 10:00am**  Radiological anatomy of the chest  **(Anatomy)**  **Prof. Saeed Abuelmakarem** |
| **10:00 - 11:00am**  Phospholipids of clinical significance  **(Biochemistry)**  **Dr. Usman Ghani** | **10:00 - 11:00am**  Oxygen and carbon dioxide transport  **(Physiology)**  **Prof. Sultan Meo** | **10:00 - 11:00am**  **Self- Directed**  **Learning** | **10:00 - 11:00am**  Pathology of bronchial asthma  **(Pathology)**  **Prof. Ammar Al Rikabi** | **10:00 - 11:00am**  Low and high altitude  **(Physiology)**  **Dr. Abdulrahman Alhowaikan** |
| **11:00- 12:00nn**  Immunology of bronchial asthma  **(Immunology)**  **Prof. Adel Almogren** | **11:00 - 12:00nn**  Gas exchange and gas transfer  **(Physiology)**  **Prof. Sultan Meo** | **11:00 - 12:00nn**  Treatment of acute and chronic rhinitis and cough  **(Pharmacology)**  **Dr. Saeed Ahmed** | **11:00 - 12:00nn**  Adrenergic agonist  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | **11:00 - 12:00nn**  **Self- Directed**  **Learning** |
| **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** | **Lunch**  **12:00 – 1:00pm** |
| **1:00-2:00pm**  Effects of exercise on the respiratory system  **(Physiology)**  **Dr. Abdulrahman Alhowikan** | **1:00 - 2:00pm**  Taking a medical history related to respiratory signs and symptoms  **Clinical Skills**  **(B1)** | **1:00 - 2:00pm**  Pharmacology of drugs used in bronchial asthma  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | **1:00-2:00pm**  Drugs used in anaphylaxis  **(Pharmacology)**  **Dr. Ishfaq Bukhari** | **1:00 - 3:00pm**  **Salam** |
| **2:00-3:00pm**  **Self- Directed**  **Learning** | **2:00-3:00pm**  Taking a medical history related to respiratory signs and symptoms  **Clinical Skills**  **(B2)** | **2:00-3:00pm**  **Self- Directed**  **Learning** | **2:00-3:00pm**  **Self- Directed**  **Learning** |

**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 behavior.
* 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 authenticate 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**

Dear Student, in order to pass the block, you must obtain a minimum final block grade of D (the grading guide attached as appendix¹), this grade is a composition from several block requirements, which can be subdivided as:

1. Attendance
2. Tutor assessment
3. Written Exams (MCQs, SAQs)
4. OSPE (Objective Structured Practical Examination)
5. Clinical Skills

* **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.

* **Marks distribution for Blocks with SHORT duration**

(Musculoskeletal, Respiratory, CVS & Renal)

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

* Continuous Assessment (Tutor Assessment in PBL sessions) : 5%
* Written Examinations:
  + SAQs : 20%
  + MCQ (Final Block Exam) : 40%

* OSPE : 30 %
* Clinical Skills : 5%

**TOTAL 100%**

1. **Tutor Assessment in Small groups (Continuous Assessment):**

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

1. The extent to which you demonstrate that you study and prepare yourself thoroughly between the two sessions (i.e., preparation).
2. 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.

1. Written Examination:
2. **MCQ**

Final written exam 40%: 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 80-100 MCQs that will assess factual knowledge too.

1. **Short Answer Questions (SAQs)**

This comprises 20% of the marks. It contains 3 to 5 cases which include 12-20 integrated SAQs. The time allocated for each question is 3 minutes. The purpose of SAQs is to test the knowledge and its application in integrated manner and to increase the validity and reliability of the written exam. The SAQ will take place at the end of each block

1. **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. 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

**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 (2010). Oxford Concise Medical Dictionary. Oxford: Oxford University Press.

*Recommended textbooks:*

Dorland (2010). Dorland’s Pocket Medical Dictionary with CD-ROM, Twenty-eighth Edition, Elsevier, UK.

Dorland (2007). Dorland’s Illustrated Medical Dictionary with CD-ROM, Thirty-first Edition, Elsevier, UK.

**Anatomy & Embryology**

*Prescribed textbook:*

Drake RL, Vogl W and Mitchell AWM (2005). Gray’s Anatomy for Students. Philadelphia: Elsevier Churchill Livingstone.

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

Larson WJ (2001). 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 and Hiatt JL (2002). Color Textbook of Histology. 2nd ed. Philadelphia: Saunders WB.

*Recommended textbooks:*

Young B, Lowe JS, Stevens A and Heath JW (2006). Wheater’s Functional Histology. 5th 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 (2010). Twelfth Edition. Churchill Livingstone, UK.

*Recommended textbooks:*

Berne RM, Levy MN, Koeppen BM and Stanton BA. (2005) Physiology. 5th ed. London: Mosby

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

Fox SI. (2008). Fundamentals of Human Physiology. 9th ed. McGraw-Hill: Boston.

Saladin KS (2009). Anatomy and Physiology. McGraw Hill Lange, USA

Barrett KE, Barman SM, Boitano S, Brooks HL (2009). Ganong’s Review of Medical Physiology. Twenty Third Edition. McGraw-Hill Publisher, UK.

**Pharmacology**

*Prescribed textbook:*

Rang HP, Dale MM, Ritter JM, Moore PK (2007). Pharmacology. Six Edition. Churchill Livingstone, Elsevier, UK.

*Recommended textbooks:*

Katszing BG (2008). Basic and Clinical Pharmacology. New York: McGraw Hill/Appleton & Lange.

**Medical Biochemistry**

*Prescribed textbook:*

Lieberman M, Marks AD (2008). Mark’s Basic Medical Biochemistry: A Clinical Approach. Lippincott Williams & Wilkins, New York.

Champe PC, Harvey RA, Ferrier DR (2005). Lippincott’s Illustrated Reviews Biochemistry. 3rd ed. Philadelphia: Lippincott Williams & Wilkins.

*Recommended textbooks:*

Murray RK, Roolwell VW, Bender D, Botham KM, Weill A, Kennelly PJ (2009). Harper’s Illustrated Biochemistry. Twenty -eighth Editions. McGraw Hill, Lange, New York.

Baynes J and Dominiczak M (2005). Medical Biochemistry. 2nd ed. London: Mosby.

Bhagavan NV (2002). Medical Biochemistry. Fourth-Edition, Elsevier, UK.

**Microbiology & Parasitology**

*Prescribed textbook:*

Goering R, DoCkrell H, Zuckerman M, Wakelin D, Riott I, Mims C (2008). Mims’ Medical Microbiology. Fourth Edition. Mosby, UK.

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

*Recommended textbooks:*

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, DiRita V, and Dermody TS. (2007). Schaechter’s Mechanisms of Microbial Disease. 4th 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**

*Prescribed textbook:*

Kumar V and Cotran RS (2007). Robbins Basic Pathology. 8th ed. Philadelphia: Saunders WB.

*Recommended textbooks:*

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

Stevens A, Lowe JS, Young B (2008). Wheaters Basic Histopathology. A Colour Atlas and Text. Churchill Livingstone, Elsevier, UK.

**Immunology**

*Prescribed textbook:*

Delves PJ, Martin SJ, Burton DR, Riott IM (2006). Riott’s Essential Immunology. Eleventh Edition. Blackwell Publishing, UK.

*Recommended textbooks:*

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 (2006). Communication Skills for Medicine. Churchill Livingstone. UK.

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 (2010). Clinical Medicine. 7th ed. Edinburgh: Elsevier Saunders.

Edwards C and Bouchier IA. (2003). Davidson’s Principles and Practice of Medicine. 14th 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).*



KING SAUD UNIVERSITY

College of Medicine

Department of Medical Education

**Feedback to Students on PBL Performance**

**Respiratory Block**

**Year 1 (Academic Year 2016-2017)**

**Student’s ID no : …………………………………………………Group number:…………**

**Student’s name:………………………………………………………………………………..**

**Tutor’s name……………………………………………………………………………….......**

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You will receive feedback on your performance in PBL tutorials from your tutor. After completing the 2nd PBL case, your tutor will meet with each student in your group on individual basis. He or she will use the following criteria for providing feedback on your performance. 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 for rank 1, 2 mark for rank 2, 3 marks for rank 3, and 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 participate to the 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

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

Comments

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KING SAUD UNIVERSITY

College of Medicine

Department of Medical Education

**Assessment of student’s Performance in PBL**

**Respiratory Block**

**Year 1 (Academic Year 2016-2017)**

**Student’s ID no.: ............................................................................ Group number:…………………**

**Student’s name: ………………………………………….......................................................................................**

**Tutor’s name: …………………………………………….........................................................................................**

**1=Unsatisfactory ; 2=Poor; 3=Good, 4=Very good; 5=Excellent**

**1. Preparation and participation:**

Ability to:

* Contribute actively to discussion 1 2 3 4 5
* Use evidence when debate an issue 1 2 3 4 5
* Demonstrate critical analysis skills 1 2 3 4 5
* Integrate knowledge 1 2 3 4 5
* Demonstrate deep understanding 1 2 3 4 5

**Total Marks = 25**

**2. Professional behaviour:**

Ability to:

* Come to tutorials on time 1 2 3 4 5
* Communicate effectively 1 2 3 4 5
* Demonstrate good manners 1 2 3 4 5
* Keep the group focused 1 2 3 4 5
* Give and receive feedback 1 2 3 4 5

**Total marks = 25**

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**Tutor’s Name: Signature: Total maximum Marks for the case = 50 /10 = 5 marks**

**Comments**

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**KING SAUD UNIVERSITY**



**COLLEGE OF MEDICINE**

**MEDICAL EDUCATION DEPARTMENT**

**STUDENT’S 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** |
| 1. Appropriately facilitated the hypothesis reorganization sessions. | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately facilitated the reporting sessions. | **1** | **2** | **3** | **4** | **5** |
| 1. Appropriately manage the time flow. | **1** | **2** | **3** | **4** | **5** |
| 1. Help to keep the group focused on its task | **1** | **2** | **3** | **4** | **5** |
| 1. Provided a well balanced intervention within the group process, but avoided dominating. | **1** | **2** | **3** | **4** | **5** |
| 1. Intervened when chairman or reporter needed. | **1** | **2** | **3** | **4** | **5** |
| 1. Provided constructive positive and constructive feedback to the group as needed. | **1** | **2** | **3** | **4** | **5** |
| 1. Encouraged positive and constructive feedback within the group about its performance | **1** | **2** | **3** | **4** | **5** |
| 1. Showed enthusiasm. | **1** | **2** | **3** | **4** | **5** |
| 1. Helped to create a supportive group climate. | **1** | **2** | **3** | **4** | **5** |
| 1. Encouraged logical and critical thinking. | **1** | **2** | **3** | **4** | **5** |
| 1. 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 LECTURES**

**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)\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_\_­­­**