AL-FARABI KAZAKH NATIONAL UNIVERSITY
Faculty of Medicine and Healthcare, Higher School of Medicine
Department of Fundamental Medicine

PO2204 General pathology

Instructions for tutorials

№	Tutorial topic, content, tasks	Max point
	GENERAL PATHOLOGY	
1-2	**General aspects of disease:** Introduction to Pathology. Discipline of Pathology. 1. *Define and use in proper context following terms:* 2. *brain death, diagnosis, differential diagnosis, disease, etiology, exacerbation, factitious, functional, abnormality, iatrogenic, idiopathic, idiopathic, lesion, morphology, mortality rate, natural history, nosocomial, pathogenesis, pathognomonic, prognosis, psychosomatic, remission, sign, somatic death, structural abnormality, symptom, syndrome.* 3. *Distinguish between disease and non-disease.* 4. *Outline a classification of causes of disease, basic responses of the body to injury, and manifestations of disease; and classify common examples in each category.* 5. *State the three most common causes of death in your country.* **Cellular Responses to Stress and Toxic Insults 1:** Cause of cell injury. Overview of cell injury and cell death. Cellular adaptations to stress. <u>General questions</u>: Categories of cause of cell injury. Mechanism of cell injury. Reversible cell injury. Mechanisms of adaptation. Pathogenesis of Hyperplasia, Hypertrophy, Atrophy, Metaplasia. 1. *Define and use in proper context: agenesis, anthracosis, aplasia, apoptosis, atrophy, autolysis, autophagy, bilirubin, cellular swelling, (hydropic change), Dysplasia, Gangrene, heat-shock protein, hemosiderin, hemosiderosis, heterophagy, homeostasis, hyaline (hyalin), hyperplasia, hypertrophy, hypoplasia, hypoxia, infarct, ischemia, karyolysis, karyorrhexis, lipofuscin, melanin, metaplasia, necrosis, neoplasia, pyknosis, steatosis,* 2. *Compare cell and tissue adaptation, reversible cell injury, and irreversible cell injury (cell death) on the basis of: etiology, pathogenesis, morphologic appearance (ultrastructural and histologic);* 3. *Compare and contrast cell death and somatic death, on the basis of: causes, pathogenesis, histologic appearance;* 4. *Outline the relationships between: biochemical, light microscopic, ultrastructural changes in the processes of cell injury and death* 5. *Compare: coagulative (coagulation) necrosis, liquefactive (liquefaction) necrosis, gangrenous nec by rosis, caseous necrosis, fat necrosis, fibrinoid necrosis, apoptosis* *in terms of: -common sites or tissues involved and reasons for this; -common causes or causative mechanisms; -gross and microscopic appearance; -types and extent of healing* 6. *Compare and contrast the following types of cell injury: reperfusion, free radical-induced, chemical, in terms of biochemical and molecular mechanisms* **Images:** Hypertrophy of myocyte – 099, 108 Atrophy of myocyte (Lipofuscin granules in cardiac myocytes) – 043, 189 Metaplasia of columnar to squamous epithelium in a bronchus - 004, Fatty liver – 052, 160	10

	Benign prostatic hyperplasia – 003, 163, Swelling of proximal convoluted tubule – 077, Nodular hyperplasia in cortex of adrenal glands – 063, 208 Hyperplasia of pancreatic islets – 197 Myocardial infarction - 001, 162 Necrosis of proximal convoluted tubule – 006, 247 Necrosis of adipose tissue - 194 Lung infarction– 008, 087 Renal infarct – 225 Brain infarct - 100 Gangrene of the small intestine – 213 Recommended Articles for Discussion: 7. Review Article: Cellular and molecular mechanisms of muscle atrophy Paolo Bonaldo, Marco Sandri Disease Models & Mechanisms 2013 6: 25-39; doi: 10.1242/dmm.010389 8. Review Article: Mechanisms and Strategies to Counter Muscle Atrophy Elisabeth Barton, Carl Morris The Journals of Gerontology: Series A, Volume 58, Issue 10, October 2003, Pages M923–M926, https://doi.org/10.1093/gerona/58.10.M923 9. Review Article: Muscle Atrophy Induced by Mechanical Unloading: Mechanisms and Potential Countermeasures Yunfang Gao and all. Front. Physiol., 20 March 2018 | https://doi.org/10.3389/fphys.2018.00235 10. Brain Atrophy Is Associated with Disability Progression in Patients with MS followed in a Clinical Routine. E. Ghione, N. Bergsland, M.G. Dwyer, J. and R. Zivadinov American Journal of Neuroradiology November 2018, DOI:https://doi.org/10.3174/ajnr.A5876 11. Contribution of normal aging to brain atrophy in MS. Christina J. Azevedo, Steven Y. Cen. Neurology Neuroimmunology Neoroinflammation November 2019; 6 (6) DOI: https://doi.org/10.1212/NXI.0000000000000616 12. *Review Article*: Cell death: Apoptosis versus necrosis. International Journal of Oncology 21(1):165-70 · July 2002. DOI: 10.3892/ijo.21.1.165 13. Fat necrosis in the Breast: A systematic review of clinical. Narges Vasei, Azita Shishegar, Forouzan Ghalkhani. *Lipids in Health and Disease* volume 18, Article number: 139 (2019)	
3-4	**Cellular Responses to Stress and Toxic Insults II:** Pathogenesis and morphologic patterns of Intracellular accumulation. General questions: Mechanism of extracellular and intracellular accumulation. Morphologic patterns of Pathologic calcification: dystrophic calcification, metastatic calcification. 1. *Define and use in proper context: lipofuscin, melanin, metaplasia, necrosis, neoplasia, pyknosis, steatosis,* 2. *List the types of subcellular alterations that can occur in cell injury, with respect to the following organelles: lysosomes, endoplasmic reticulum, mitochondria, cytoskeleton* 3. *Discuss the significance of intracellular accumulations of: lipids, proteins, glycogen, pigments (exogenous and endogenous)* 4. *Compare fatty change (steatosis) and fatty infiltration on the basis of: causes, pathogenesis, organs commonly involved, histologic appearances* 5. *Compare dystrophic and metastatic calcification in terms of: definition, etiology and pathogenesis, morphologic appearance, sites and associated diseases, clinical significance* **Images for I:** Lipofuscin granules in cardiac myocytes – 043, 189 Fatty liver - 052, 160	10

	Fatty change in heart – 044, 140, 228, Hemosiderin granules in liver cells – 031 Hemosiderin granules in lung - 085 Hemosiderin granules in brain – 179, Cholestasis - 250 **Images for II:** Nephrocalcinosis – 013, Atherosclerosis – 014, 126, 249 Cartilage calcinosis – 064, 088 Calcinosis of mitral valve – 062 Calcinosis in brain – 080, 157 Calcinosis of the cerebral meninges – 089, Coronary artery calcification - 251 Coal pigment aggregates in pulmonary parenchyma – 012, 047 Recommended Articles for Discussion: 1. An Overview of the Role of Lipofuscin in Age-Related Neurodegeneration Alexandra Moreno-García, Alejandra Kun, [...], and Miguel Calero. Frontiers in Neuroscience, 2018: 12: 464. 2. Specificity and Sensitivity of Hemosiderin-Laden Macrophages in Routine Bronchoalveolar Lavage in Children. Zeynep N. Salih, MD, Afreen Akhter, BA, and Javeed Akhter, MD. Archives of Pathology & Laboratory Medicine , Volume 130, Issue 11 (November 2006). 3. Iron homeostasis in the liver. Anderson ER, Shah YM. Iron homeostasis in the liver. *Compr Physiol*. 2013;3(1):315–330. doi:10.1002/cphy.c120016 4. REVIEW ARTICLE: The Involvement of Iron in Traumatic Brain Injury and Neurodegenerative Disease. Maria Daglas Front. Neurosci., 20 December 2018 | https://doi.org/10.3389/fnins.2018.00981	
5-6	**Hemodynamic Disorders** 1: Pathophysiologic categories of the oedema. Pathogenesis and morphologic patterns of hyperemia and congestion. Pathogenesis of hemorrhage. Pathophysiologic categories of hemorrhage. Classification of hemorrhage. General questions: Hemodynamic disorders. Oedema. Increased hydrostatic pressure. Lymphatic obstruction. Sodium and water retention. Hyperemia and congestion. Pathogenesis and morphologic patterns of hemorrhage. 1. *Define and use in proper context: hyperemia, congestion, congestive heart failure, edema, inflammatory, noninflammatory, renal, lymphedema, anasarca, effusion, ascites, exudate, transudate* 2. *Compare and contrast active hyperemia and passive congestion, in terms of:* • *mechanisms of development* • *clinically important examples* 3. *Describe chronic passive congestion of:* • *lungs* • *liver* • *kidneys* • *spleen* 4. *in terms of:* • *morphologic features* • *functional alterations* 5. *Discuss the pathogenesis of edema, giving examples associated with the following mechanisms:* • *altered plasma oncotic pressure* • *inflammation* • *venous obstruction/stasis* • *lymphatic obstruction* 6. *and classify each in terms of localized vs. generalized*	10

	7. *Compare edema of:* • *subcutaneous tissue* • *lungs* • *brain* • *kidneys* 8. *on the basis of:* • *pathogenesis* • *morphologic changes* • *clinical effects* 9. *Compare acute and chronic hemorrhage in terms of:* • *common causes* • *clinical manifestations* • *compensatory mechanisms* 10. *Compare and contrast bleeding due to:* • *vascular defect (localized or generalized)* • *platelet defect* • *coagulation defect* 11. *in terms of:* • *etiologic/precipitating factors* • *common sites of occurrence* • *organs commonly involved* • *type and size of vessels involved* • *results, complications, and fate of lesions* • *clinical features* • *laboratory findings* **Images:** Pulmonary edema – 226, 240 Acute pneumonia – 058, 161, 178 Chronic passive congestion in lung – 085, 091 Acute paranephritis – 081 Acute meningitis - 153 Fibrosis of liver – 175 Hemorrhage in brain – 007, Hemorrhage in stomach – 074 An organized trombus - 128 Gastric Ulcer - 130 Aortic laceration – 156, 188 Recommended Article for Discussion: 1. TOPICAL REVIEW. Local control of blood flow during active hyperaemia: what kinds of integration are important? Coral L. Murrant. The Journal of Physiology 593.21 (2015) pp 4699-4711	
7	**Hemodynamic** disorders 2: Pathogenesis of Haemostasias and thrombosis. Embolism. Shock. <u>General questions:</u> Normal hemostasis. Antithrombotic properties. Prothrombotic properties. Platelets. Coagulation cascade. Thrombosis. Embolism. Infarction. Shock. Major types of shock. Stages of shock. 1. *Describe thrombi in terms of:* • *types of thrombotic material* • *factors conditioning the development of thrombi* • *possible fate of thrombi* 1. *Distinguish between venous thrombi and arterial thrombi on the basis of:* • *etiologic and precipitating factors*	10

- *common sites of occurrence*
- *type and size of vessel involved*
- *morphologic appearance*
- *organs commonly involved*
- *local and distant effects*
- *fate of lesions and prognosis*
- *clinical and laboratory features*

2. *Compare the following types of emboli:*
   - *arterial thrombotic*
   - *venous thrombotic*
   - *paradoxical*
   - *fat*
   - *bone marrow*
   - *atheromatous*
   - *air tumor*
   - *amniotic fluid*
   - *foreign body*
2. *in terms of:*
   - *defining morphologic features*
   - *etiologic/precipitating factors*
   - *common sites of occurrence*
   - *organs commonly involved*
   - *type and size of vessels involved*
   - *complications*
   - *fate of lesion*
   - *common clinical manifestations*
3. *Compare and contrast arterial and venous infarcts on the basis of:*
   - *location*
   - *pathogenesis*
   - *morphology*
   - *clinical manifestations*
4. *Describe the morphologic appearance and natural history of infarcts of:*
   - *heart kidney*
   - *lung spleen*
   - *bowel brain*
3. *Describe the following stages of shock:*
   - *non-progessive (compensated)*
   - *progressive (decompensated)*
   - *irreversible*
4. *in terms of:*
   - *pathophysiology*
   - *morphologic changes*
   - *prognosis*
5. *Compare and contrast the following types of shock:*
   - *neurogenic*
   - *normovolemic*
   - *hypovolemic*
   - *hemorrhagic*
   - *septic*
   - *cardiogenic*
   - *anaphylactic*
6. *in terms of:*
   - *pathogenic mechanism*
   - *common causes*
   - *structural changes*
   - *functional changes*

	• *clinical features and prognosis* 7. *List the morphologic changes and functional effects of shock on:* • *lungs* • *kidneys* • *adrenals* • *brain* • *gastrointestinal tract* **Images:** Red thrombus – 116, 120, 149 Thrombus (organization) - 128 Thrombosis of small vessels in system of pulmonary - 143 Pulmonary embolism – 010, 252 Pulmonary infarction - 008, 087 Pulmonary tumor embolism – 236, 237 Amniotic fluid emboli - 009 Tumor embolism – 246 Myocardium infarction – 001 Pulmonary infarction - 008, 060, 087 Kidney infarction - 225 Recommended Articles for Discussion: 1. Acute pulmonary embolism: a concise review of diagnosis and management. Hepburn-Brown M, Darvall J, Hammerschlag G. Intern Med J. 2019 Jan;49(1):15-27. doi: 10.1111/imj.14145. 2. Thrombosis: a major contributor to global disease burden. Raskob GE, Angchaisuksiri P et al. Arterioscler Thromb Vasc Biol. 2014; 34: 2363-2371 3. The economic burden of incident venous thromboembolism in the United States: a review of estimated attributable healthcare costs. Grosse SD, Nelson RE, Nyarko KA, Richardson LC, Raskob GE. *Thromb Res.* 2016; 137: 3-10. 4. Epidemiology of venous thromboembolism. Heit JA. *Nat Rev Cardiol.* 2015; 12: 464-474 5. Epidemiology of cancer-associated venous thrombosis. Timp JF, Braekkan SK, Versteeg HH, Cannegieter SC *Blood.* 2013; 122: 1712-1723 6. Genetics of venous thrombosis**:** update in 2015. Morange P.E, Suchon P, Trégouët D.A *Thromb Haemost.* 2015; 114: 910-919 7. Silent pulmonary embolism in patients with deep venous thrombosis: a systematic review. Stein PD, Matta F, Musani MH, Diaczok B. *Am J Med.* 2010; 123: 426-431	
8	**Acute inflammation** I: Overview of inflammation. Stimuli of inflammation. Vascular changes. Cellular events: leukocyte recruitment and activation. Leukocyte-induced tissue injury. Morphologic patterns of acute inflammation General questions: Pathogenesis and Morphologic patterns of Serous inflammation and Fibrinous inflammation. **Acute inflammation** II: Overview of inflammation. Stimuli of inflammation. Vascular changes. Cellular events: leukocyte recruitment and activation. Leukocyte-induced tissue injury. Morphologic patterns of acute inflammation. General questions: Pathogenesis and Morphologic patterns of purulent inflammation. 1. *Define and use in proper context: abscess, autocrine, cellulitis, chemotaxis, cytokine, edema, effusion, emigration, endocrine, erosion, exudate, fibrinous, granulation tissue, granuloma, inflammation, margination, paracrine, phagocytosis, purulent, pus, pyogenic, resolution, serosanguineous, serous, suppurative, transudate, ulcer* 2. *Describe the classic vascular changes and cellular events of the inflammatory reaction.*	5

3. *Discuss the five cardinal signs of inflammation in terms of pathogenesis and underlying morphologic changes.*
4. *Discuss the following chemical mediators of inflammation, in terms of origin (cells vs. plasma) and chief in vivo functions:*
    - *vasoactive amines*
    - *nitric oxide*
    - *proteases of clotting, kinin, complement systems*
    - *lysosomal granule contents*
    - *arachidonic acid metabolites*
    - *oxygen-derived free radicals*
    - *platelet activating factor*
    - *neuropeptides*
    - *cytokines/chemokines*
5. *Discuss each of the following in terms of the associated type of inflammation and their role therein:*
    - *platelets*
    - *lymphocytes*
    - *giant*
    - *cells*
    - *mast cells/basophils*
    - *plasma cells*
    - *fibroblasts*
    - *neutrophils*
    - *eosinophils*
    - *cell adhesion molecules*
    - *endothelial cells*
    - *monocytes/macrophages/histiocytes*
6. *Compare and contrast acute, chronic, and granulomatous inflammation in terms of:*
    - *etiology*
    - *pathogenesis*
    - *histologic appearance*
    - *laboratory findings*
    - *characteristic cells involved*
    - *outcome*
    - *systemic effects*
7. *Compare and contrast resolution and organization with respect to the termination of an inflammatory response.*
8. *Develop and utilize the nomenclature used to describe inflammation in the various tissues and organs*

Images:

Serous pneumonia - 015, 219
Serous meningitis - 153
Fibrinous pericarditis – 042
Fibrinous perisplenitis - 076
Acute pneumonia – 058, 139, 161, 174, 178
Acute perisplenitis – 076
Acute pericarditis – 104, 107, 111
Acute nephrinis – 039, 081
Purulent meningitis - 261
Peritonitis – 082, 215, 218, 244, 245
Cerebral abscess - 176
Renal abscess – 039
Lung abscess, acute bronchitis, acute pleuritis – 069
Acute pneumonia – 058, 139, 161, 174, 178

	Recommended Articles for Discussion: 1. Inflammatory responses and inflammation-associated diseases in organs Linlin Chen, Huidan Deng, [...], and Ling Zhao. Oncotarget. 2018 Jan 23; 9(6): 7204–7218. 2. SEROUS INFLAMMATION. W. H. WASHBURN, M.D. *JAMA.* 1898; XXX(20):1159-1161. doi:10.1001/jama.1898.72440720023001g 3. Resolution of Inflammation: What Controls Its Onset? Michelle A. Sugimoto, Lirlândia P. Sousa, [...], and Mauro M. Teixeira. Front Immunol. 2016; 7: 160. 4. Acute fibrinous and organizing pneumonia: two case reports and literature review Jingjing Lu, Qi Yin… & Qiang Li. *BMC Pulmonary Medicine* volume 19, 141 (2019) 5. Clinical and microbiological characteristics of purulent and non-purulent cellulitis in hospitalized Taiwanese adults in the era of community-associated methicillin-resistant *Staphylococcus aureus.* Chun-Yuan Lee, Hung-Chin Tsai, [...], and Yao-Shen Chen. BMC Infect Dis. 2015; 15: 311. 6. Modern exudate management: a review of wound treatments 7. Bacterial Brain Abscess Kevin Patel, MD and David B. Clifford, MD Neurohospitalist. 2014 Oct; 4(4): 196–204. doi: 10.1177/1941874414540684 8. A current review of brain abscess Duke S. Samson, Kemp Clark, M.D. The American Journal of Medicine, February 1973 Volume 54, Issue 2, Pages 201–210 DOI: https://doi.org/10.1016/0002-9343(73)90224-6 9. The Evolving Nature of Hepatic Abscess: A Review Marianna G. Mavilia, Marco Molina, and George Y. Wu. J Clin Transl Hepatol. 2016 Jun 28; 4(2): 158–168. doi: 10.14218/JCTH.2016.00004 10. Lung abscess-etiology, diagnostic and treatment options Ivan Kuhajda, Konstantinos Zarogoulidis, [...], and Danijela Kuhajda. Ann Transl Med. 2015 Aug; 3(13): 183. doi: 10.3978/j.issn.2305-5839.2015.07.08	
10	**Colloquium 1** Review and quiz on all micro-images of previous classes (from 1 seminar to 9) and recommended articles for Discussion. **Images:** Hypertrophy of cardiac myocytes Atrophy of cardiac myocytes Metaplasia of columnar to squamous epithelium in bronchus Swelling of proximal convoluted tubule Fatty liver Fatty change in the heart Benign prostatic hyperplasia Nodular hyperplasia in cortex of adrenal glands Myocardial infarction Necrosis of adipose tissue Kidney Infarction Brain Infarction Hemosiderin granules in liver, in lung, in brain Cartilage calcinosis Vascular Calcification Calcification of mitral valve Brain Calcinosis Calcinosis of the cerebral meninges Coal pigment aggregates in pulmonary parenchyma Pulmonary edema Chronic passive congestion in lung, in liver Serous pneumonia Serous meningitis Fibrinous pericarditis Fibrinous perisplenitis Purulent pericarditis Purulent nephrinis	39

	Purulent peritonitis Cerebral abscess Lung abscess Brain Hemorrhage Organized trombus Gastric Ulcer Red thrombus Pulmonary infarction Pulmonary tumor embolism Amniotic fluid embolism	
11-12	**Chronic inflammation** I: Systemic effect of inflammation. General questions: Pathogenesis and Morphologic patterns of Chronic inflammation. **Chronic inflammation II**: Pathogenesis of Granulomatous inflammation. General questions: Pathogenesis of granulomatous inflammation. Morphologic patterns of granulomatous inflammation 1. *Define and use in proper context: abscess, autocrine, cellulitis, chemotaxis, cytokine, edema, effusion, emigration, endocrine, erosion, exudate, fibrinous, granulation tissue, granuloma, inflammation, margination, paracrine, phagocytosis, purulent, pus, pyogenic, resolution, serosanguineous, serous, suppurative, transudate, ulcer* 2. *Describe the classic vascular changes and cellular events of the inflammatory reaction.* 3. *Discuss the five cardinal signs of inflammation in terms of pathogenesis and underlying morphologic changes.* 4. *Discuss the following chemical mediators of inflammation, in terms of origin (cells vs. plasma) and chief in vivo functions:* • *vasoactive amines* • *nitric oxide* • *proteases of clotting, kinin, complement systems* • *lysosomal granule contents* • *arachidonic acid metabolites* • *oxygen-derived free radicals* • *platelet activating factor* • *neuropeptides* • *cytokines/chemokines* 5. *Discuss each of the following in terms of the associated type of inflammation and their role therein:* • *platelets* • *lymphocytes* • *giant* • *cells* • *mast cells/basophils* • *plasma cells* • *fibroblasts* • *neutrophils* • *eosinophils* • *cell adhesion molecules* • *endothelial cells* • *monocytes/macrophages/histiocytes* 6. *Compare and contrast acute, chronic, and granulomatous inflammation in terms of:* • *etiology* • *pathogenesis* • *histologic appearance* • *laboratory findings*	10

- *characteristic cells involved*
- *outcome*
- *systemic effects*

7. *Compare and contrast resolution and organization with respect to the termination of an inflammatory response.*
8. *Develop and utilize the nomenclature used to describe inflammation in the various tissues and organs*

Images:

Chronic endocarditis - 151
Chronic bronchitis – 090, 129, 137
Chronic hepatitis – 092, 127, 136, 160, 211
Chronic nephritis – 113, 132,134
Atherosclerosis – 086
Chronic sialadenitis – 257
Chronic tonsillitis - 258
Granulation tissue – 020
Tuberculous granuloma - 017.
Tuberculous lymphadenitis - 248
Actinomycosis - 019
Syphilitic aortitis - 018
Trichinellosis - 223
Echinococcosis of liver – 079
Cystic echinococcosis – 266, 267

Recommended Articles for Discussion:

1. REVIEW ARTICLE: Activation of Resolution Pathways to Prevent and Fight Chronic Inflammation: Lessons From Asthma and Inflammatory Bowel Disease. Front. Immunol., 23 July 2019 https://doi.org/10.3389/fimmu.2019.01699
2. Chronic diseases, inflammation, and spices: how are they linked? Ajaikumar B. Kunnumakkara, Bethsebie L. Sailo, Kishore Banik, *Journal of Translational Medicine* volume 16, Article number: 14 (2018)
3. From Pathogenesis, Clinical Manifestation, and Diagnosis to Treatment: An Overview on Autoimmune Pancreatitis Ou Cai and Shiyun Tan Gastroenterology Research and Practice 2017 https://doi.org/10.1155/2017/3246459
4. Resolution of chronic inflammatory disease: universal and tissue-specific concepts Georg Schett, Markus F. Neurath *Nature Communications* volume 9, Article number: 3261 (2018)
5. Histopathologic review of granulomatous inflammation Kabeer K. Shah, Bobbi S. Pritt, Mariam P. Alexander Journal of Clinical Tuberculosis and Other Mycobacterial Diseases 7 (2017) 1–12
6. Contents lists available at ScienceDirect journal homepage: www.elsevier.com/locate/jctube Kabeer K. Shah Journal of Clinical Tuberculosis and Other Mycobacterial Diseases Volume 7, May 2017, Pages 1-12 https://doi.org/10.1016/j.jctube.2017.02.001
7. Granulomatous inflammation--a review. G T Williams and W J Williams J Clin Pathol. 1983 Jul; 36(7): 723–733. doi: 10.1136/jcp.36.7.723
8. A clinicopathological classification of granulomatous disorders D Geraint James. BMJ
9. Granulomatous Lung Disease: An Approach to the Differential Diagnosis Sanjay Mukhopadhyay, MD and Anthony A. Gal, MD Archives of Pathology & Laboratory Medicine Volume 134, Issue 5 (May 2010)
10. Differential diagnosis of granulomatous lung disease: clues and pitfalls Shinichiro Ohshimo, Josune Guzman, Ulrich Costabel, Francesco Bonella European Respiratory Review 2017 26: 170012; DOI: 10.1183/16000617.0012-2017

13-14	**Tissue Renewal, Regeneration, and Repair** I: Pathogenesis of Regeneration. Proliferative capacities of tissue. Granulation tissue. General questions: Proliferative capacities of tissue. Pathogenesis and morphologic patters of granulation tissue. 1. *Define and use in proper context:* 1. *angiogenesis (neovascularization)* 2. *cicatrix* 3. *contact inhibition* 4. *contracture* 5. *dehiscence* 6. *fibrosis (fibroplasia)* 7. *granulation tissue* 8. *haptotaxis* 9. *keloid* 10. *organization* 11. *regeneration* 12. *repair* 13. *scar* 14. *stricture* 2. *Distinguish between labile, stable, and permanent cells, and place each of the following cell/tissue* 1. *types into the appropriate category:* 2. *hematopoietic muscular (smooth, skeletal, cardiac)* 3. *glandular parenchymal neuronal* 4. *epithelial glial* 5. *osseous and chondroid connective* 3. *Discuss the basic aspects of collagen synthesis, degradation, and function, and state the tissue(s) in which collagen types I-IV are predominantly localized.* **Images**: Glial cells reaction after brain stroke – 179, 190 Regeneration of the ulcer base - 022 The scar tissue after myocardial infarction - 122 Organizing thrombus – 128 Carnification of lung – 053 Cystic fibrosis – 078 Splenic arteriolar hyalinosis – 117 Granulation tissue– 020 1. Granulation tissue formation and remodeling Lari Häkkinen, Hannu Larjava, Leeni Koivisto https://doi.org/10.1111/etp.12008 2. Skin Acute Wound Healing: A Comprehensive Review Luis Cañedo-Dorantes International Journal of Inflammation 2019 https://doi.org/10.1155/2019/3706315 3. Regeneration of injured skeletal muscle after the injury Tero AH Järvinen, Markku Järvinen, and Hannu Kalimo Muscles Ligaments Tendons J. 2013 Oct-Dec; 3(4): 337–345.	10

15-16	**Tissue Renewal, Regeneration, and Repair** II: Control of cell proliferation. Pathologic aspects of repair: Sclerosis, Fibrosis, Cirrhosis.
	General questions: Proliferative capacities of tissues – continuously dividing tissues, stable tissues, permanent tissues.
	1. *Compare and contrast:*
	  1. *resolution*
	  2. *regeneration*
	  3. *repair*
	  4. *organization*
	2. *in terms of:*
	  1. *type of antecedent injury*
	  2. *tissue involved*
	  3. *cellular response*
	  4. *time course*
	  5. *ultimate outcome*
	  6. *classic/common examples of each*
	3. *Describe the four steps of tissue repair, including the cell types and growth factors involved, and the approximate timetable for the tissue repair process.*
	4. *Discuss the role of each of the following in the repair reaction:*
	  1. *cell migration*
	  2. *integrins*
	  3. *growth factors*
	5. *Describe the role of each of the following in the process of wound healing:*
	  1. *myofibroblasts*
	  2. *endothelial cells*
	  3. *fibroblasts*
	  4. *macrophages*
	  5. *collagen*
	6. *Compare healing by first intention (primary union) and second intention (secondary union) in terms of time, sequence of events, morphologic changes, and final outcome.*
	7. *Describe the local and systemic factors that influence wound healing, stating whether each of these influences accelerates or retards the rate of healing.*
	8. *List the complications of wound healing.*
	
	Images:
	
	Splenic arteriolar hyalinosis – 117
	Granulation tissue– 020
	Scar -021
	Cardiosclerosis – 099, 108, 109, 216, 263
	Myocardium - 075
	Pancreosclerosis- 051,125
	Pleural sclerosis – 222
	Sclerosis of liver - 175
	Nephroscltrosis– 113, 127, 132
	Cirrhosis of liver – 147
	Glomerulosclerosis– 158
	
	Recommended Articles for Discussion:
	
	1. Wound healing - A literature review Ana Cristina de Oliveira Gonzalez An Bras Dermatol. 2016 Sep-Oct; 91(5): 614–620. doi: 10.1590/abd1806-4841.20164741
	2. Skeletal muscle regeneration is modulated by inflammation WenjunYangPingHu Journal of Orthopaedic Translation Volume 13, April 2018, Pages 25-32 https://doi.org/10.1016/j.jot.2018.01.002
		10

3. Foreign Body Granuloma After Cranial Surgery: A Systematic Review of Reported Cases. Akhaddar A[1], Turgut AT[2], Turgut M[3]. World Neurosurg. 2018 Dec;120:457-475. doi: 10.1016/j.wneu.2018.09.143.
4. The Living Scar – Cardiac Fibroblasts and the Injured Heart. Eva A Rog-Zielinska, Russell A Norris, Peter Kohl, and Roger Markwald Trends Mol Med. 2016 Feb; 22(2): 99–114. doi: 10.1016/j.molmed.2015.12.006
5. Characterization of Electrical Activity in Post-myocardial Infarction Scar Tissue in Rat Hearts Using Multiphoton Microscopy. Front. Physiol., October 2018 | https://doi.org/10.3389/fphys.2018.01454

17-18	**General Pathology of Infectious diseases 1:** Mechanism of bacterial Injury. Mechanism of Viral Injury. General questions: Etiology, pathogenesis and morphologic pattern of Tuberculosis, Syphilis. Actinomycosis, Echinococcosis, Trichinellosis. 1. *List three general ways in which infectious agents damage tissues* 2. *Discuss the different mechanisms of dissemination and transmission of microbial organisms.* 3. *Discuss the different mechanisms of bacterial-induced cellular and tissue injury including mechanisms of adhesions, exotoxins, and endotoxins.* 4. *Compare endotoxins and exotoxins based on:* 1. *sources* 2. *effects* 3. *immunologic response* 5. *Discuss the significance of:* 1. *pyogenic inflammation* 2. *granulomatous inflammation* 3. *caseous necrosis* 4. *gangrene* 5. *liquefactive necrosis* 6. *lymphocytic reaction* 7. *plasmacytic reaction* 8. *eosinophil reaction* 9. *pseudomembranous reaction* 10. *cytopathic/cytoproliferative reaction* 6. *in terms of:* 1. *possible causative agents* 2. *mechanism of reaction* 3. *morphologic features* 7. *Identify granulomatous inflammation and enumerate special stains needed to differentiate infectious etiologies thereof* 8. *Compare and contrast the following types of infectious diseases:* 1. *bacterial* 2. *mycobacterial* 3. *fungal* 4. *rickettsial* 5. *viral* 6. *protozoan* 7. *helminthic* 8. *prion* 9. *in terms of:* 1. *histologic reaction* 2. *organ and tissue distribution* **Images:** Tuberculous granuloma – 017 Tuberculous lymphadenitis - 248 Syphilitic aortitis - 018 Actinomycosis - 019 Trichinellosis - 223 Echinococcosis of liver – 079 Cystic echinococcosis – 266 Echinococcosis of lung – 267 Echinococcus osteomyelitis - 265 Recommended Articles for Discussion:	10

	1. Prachi B Tripathi, Anjali D Amarapurkar Morphological spectrum of gastrointestinal tuberculosis Tropical Gastroenterology DOI: http://dx.doi.org/ 2. Mihai Raul Popescu, Iancu Emil Pleşea, Marian Olaru Morphological aspects in tuberculosis of oral cavity – our experience and a review of the literature attempt. Rom J Morphol Embryol 2015, 56(3):967–987 3. Mann, K. J. Lung Lesions in Skeletal Tuberculosis. Review of 500 Cases. Lancet 1946 pp.744-9 ref.14 4. Ameeta E. Singh and Barbara Romanowski. Syphilis: Review with Emphasis on Clinical, Epidemiologic, and Some Biologic Features Clin Microbiol Rev. 1999 Apr; 12(2): 187–209. 5. Rebecca E. LaFond, Sheila A. Lukehart Biological Basis for Syphilis. Clinical Mycrobiology Reviews. DOI: 10.1128/CMR.19.1.29-49.2006 6. João Carlos Regazzi Avelleira; Giuliana Bottino. Syphilis: diagnosis, treatment, and control. An Bras Dermatol. 2006;81(2):111-26. 7. Rebecca E. LaFond, Sheila A. Lukehart Biological Basis for Syphilis. Clinical Mycrobiology Reviews. DOI: 10.1128/CMR.19.1.29-49.2006 8. João Carlos Regazzi Avelleira; Giuliana Bottino. Syphilis: diagnosis, treatment, and control. An Bras Dermatol. 2006;81(2):111-26. 9. Suk Hee Heo and all Imaging of Actinomycosis in Various Organs: A Comprehensive Review. Radiographics 34(1):19-33 · January 2014 DOI: 10.1148/rg.341135077 10. Alessandra Siracusano, Antonella Teggi and Elena Ortona· Human Cystic Echinococcosis: Old Problems and New Perspectives. Interdisciplinary Perspectives on Infectious Diseases, 2009, https://doi.org/10.1155/2009/474368 11. Greg D. Appleyard, Alvin A. Gajadhar. Review of Trichinellosis in People and Wildlife in Canada. Canadian Journal of Public Health. July 2000, Volume 91, Issue 4, pp 293–297.	
19-20	**Environmental and Nutritional Diseases:** Air Pollution, Effect of Tobacco, Effect of Alcohol, Obesity General questions: Pathogenesis and morphological patterns of Air Pollution, Effect of Tobacco. Effect of Alcohol, Obesity 1. *List the various substances found in cigarette smoke and their health effects.* 2. *Discuss the effects of:* • *active tobacco smoke* • *passive (sidestream) tobacco smoke* • *smokeless tobacco* 3. *in terms of:* • *magnitude of problem* • *resultant diseases* 4. *Outline the basic pathogenesis of pneumoconioses.* 5. *Compare and contrast the following pneumoconioses:* • *coal workers' pneumoconiosis* • *silicosis* • *asbestosis* • *berylliosis* 6. *in terms of:* • *types of occupational exposure* • *pathogenesis* 7. *morphologic pulmonary reactions* • *clinical course* • *complications* 8. *Compare coal workers' pneumoconiosis with simple asymptomatic anthracosis.* 9. *Give examples of different forms of silica and differentiate between silicoproteinosis and classic nodular silicosis* 10. *Discuss ethanol in terms of:* • *effects ethanol on society* • *blood alcohol levels and their effects*	5

	• *metabolism and systemic effects of:* • *acute alcohol ingestion* • *chronic ethanol abuse* 11. *Discuss the following:* • *fetal alcohol syndrome* • *association of ethanol with cancer* 12. *List the five main categories of nutritional disorders and discuss main malnutrition states regarding:* • *nomenclature* • *incidence* • *specific etiologic entities* • *pathogenesis* • *morphologic changes* 13. *Discuss obesity in terms of:* • *epidemiology* • *clinical measurements* • *etiology* • *types of obesity* • *pathogenesis* • *morphologic changes* • *complication* **Images:** Coal pigment aggregates in pulmonary parenchyma – 012, 047 Chronic bronchitis – 090, 137, 264 Pneumosclerosis – 144 Pulmonary emphysema – 054, 094 Squamous metaplasia of respiratory epithelium - 004 Liver – 050 Lung – 071 Trachea – 145 Bronchus – 072 Fatty liver, chronic hepatitis - 160, Chronic hepatitis – 092, 136 Cirrhosis of the liver - 147 Pulmonary emphysema – 054, 094 Fatty infiltration of the myocardium – 044 Recommended Articles for Discussion: 1. The Effects of Air Pollution on the Brain: a Review of Studies Interfacing Environmental Epidemiology and Neuroimaging. Paula de Prado Bert, Elisabet Mae Henderson Mercader, Jesus Pujol, Jordi Sunyer and Marion Mortamais. Curr Environ Health Rep. 2018; 5(3): 351–364. doi: 10.1007/s40572-018-0209-9 2. Air pollutants and early origins of respiratory diseases. Dasom Kim, Zi Chen, Lin-Fu Zhou, and Shou-Xiong Huang. Chronic Dis Transl Med. 2018 Jun; 4(2): 75–94. doi: 10.1016/j.cdtm.2018.03.003.	
20	**Colloquium 2** Review and quiz on all micro-images of previous classes (from 11 seminar to 19) and discussion of recommended articles. **Images**: Chronic endocarditis Chronic bronchitis Chronic hepatitis Chronic nephritis Atherosclerosis	39

	Granulation tissue Tuberculous granuloma Actinomycosis Syphilitic aortitis Trichinellosis Echinococcosis of liver Cystic echinococcosis Glial cells reaction after brain stroke Regeneration of the ulcer base The scar tissue after myocardial infarction Organizing thrombus Carnification of lung Cystic fibrosis Splenic arteriolar hyalinosis Coal pigment aggregates in pulmonary parenchyma Fatty liver, chronic hepatitis Chronic hepatitis Cirrhosis of the liver Chronic bronchitis Pneumosclerosis Pulmonary emphysema Squamous metaplasia of respiratory epithelium Fatty infiltration of the myocardium Pancreatic lipomatosis	
21	**Clinical correlation – case-study, body proof**: *Cell injury* (President and the Alzheimer's disease, the last queen of Egypt, The Medici, the Golden Mask) Hemodynamic disorders (The Sun King, the Mystery of the Painting, the Death of the Poet, the Inventor of Penicillin, the Death of the Genius), *Acute Inflammation* (the Ruler of Athens, Father of evolution theory, The Roman Emperor, Curse of the mummy). *Chronic inflammation* **(**the Lady of the Camellias, Another Rembrandt, the Story of a Kingor a Commander), *Regeneration and tissue repair* (the Story of the Titan, the Terminator, British Icon, German composer, Sting Like a Bee, the Ancient Artifact and Sir Walter Scott). 1. *Apply knowledge of the pathogenesis and morphogenesis of typical pathological processes* 2. *Demonstrate analytical skills in the integration of knowledge on pathophysiology, pathological anatomy, and immunology in the formation of judgments regarding general pathology.* 3. *Demonstrate the ability to identify learning gaps and create strategies to enhance one's own knowledge and skills.* 4. *Effectively communicate with other students and teachers regarding medical and scientific information, articulate their opinions clearly when discussing pathophysiological processes and their impact, and work effectively as a member of the team*	10
22-23	**Diseases of the Immune System**: Morphologic patterns of immune disorders. General questions: Pathogenesis and morphological patterns of Rheumatic heart disease. 1. *Discuss different mechanisms by which immune tolerance is lost in the general pathogenesis of autoimmune diseases.* 2. *Compare and contrast the mechanisms of the immune reactions with respect to the situations in which each is triggered, mechanisms of injury, resulting pathologic effects on tissue, and the ultimate clinical consequences.* 3. *Discuss the Rheumatic heart disease in terms of: terminology, relative frequency, pathogenesis, morphology (light, immunofluorescent, and electron microscopic), clinical features, prognosis* **Images:** Rheumatic heart disease, acute rheumatic endocarditis – 042, 104,	10

	Rheumatic heart disease, cardiosclerosis - 263 Rheumatic heart disease, purulent pneumonia - 172 Chronic rheumatic endocarditis – 151, Reduction of spleen follicles - 097 Mitral valve – 135 Myocardium and epicardium – 040 Myocardium – 041 Endocardium, myocardium and epicardium - 103 Recommended Articles for Discussion: 1. Diagnostic Testing and Interpretation of Tests for Autoimmunity Christine Castro, D.O. and Mark Gourley, M.D. J Allergy Clin Immunol. 2010 Feb; 125(2 Suppl 2): S238–S247. doi: 10.1016/j.jaci.2009.09.041 2. Rheumatoid arthritis: Disease or syndrome? Jessica A Stanich, John D Carter, Judith Whittum-Hudson, and Alan P Hudson. Open Access Rheumatol. 2009; 1: 179–192. doi: 10.2147/oarrr.s7680 3. Inflammatory lesions in the bone marrow of rheumatoid arthritis patients: a morphological perspective Serena Bugatti, Antonio Manzo, Roberto Caporali, Carlomaurizio Montecucco *Arthritis Research & Therapy* volume 14, Article number: 229 (2012) 4. Nailfold Capillaroscopy in Rheumatic Diseases: Which Parameters Should Be Evaluated? Mahnaz Etehad Tavakol, Alimohammad Fatemi, Abdolamir Karbalaie, Zahra Emrani, and Björn-Erik Erlandsson**.** BioMed Research International https://doi.org/10.1155/2015/974530	
24-25	**Neoplasia**. Components of a tumor. Benign neoplasms. Malignant neoplasms. Etiology of cancers. Anaplasia. Dysplasia. Carcinoma in situ. Anaplasia. Dysplasia. Carcinoma in situ. Metastasis. Tumors of epithelial origin. General questions: Characteristics of Benign and Malignant neoplasms. Clinical aspects of neoplasia. 1. *Discuss the following: anaplasia hyperplasia, aplasia hypoplasia, atrophy, metaplasia, dysplasia, neoplasia* 2. *in terms of:* • *etiology* • *pathogenesis* • *morphology* • *functional sequelae* • *specific examples* 3. *Outline the classification and nomenclature for benign and malignant neoplasms, using appropriate prefixes and suffixes and indicating specific exceptions to rules of nomenclature.* 4. *Compare and contrast the following in terms of tissue of origin, gross and microscopic features, and mode of spread:* • *normal vs. neoplastic tissue* • *adenoma vs. carcinoma* • *carcinoma vs. sarcoma* 5. *List the general cytologic changes found in neoplastic cells.* 6. *List the most common sites of origin of:* • *adenocarcinoma* • *squamous cell carcinoma* • *melanoma* • *cystadenoma* • *adenoma* • *papilloma* 7. *Describe the metaplasia-dysplasia-carcinoma-in-situ-invasive carcinoma sequence.*	10

	Images: Neurofibroma – 036 Melanoma - 035 Brain tissue – 093 Meningeal tissue – 048 Lateral ventricle of brain – 164 Skin tissue – 256 Recommended Articles for Discussion: 1. The Role of Large-Format Histopathology in Assessing Subgross Morphological Prognostic Parameters: A Single Institution Report of 1000 Consecutive Breast Cancer Cases Tibor Tot. International Journal of Breast Cancer, 2012 https://doi.org/10.1155/2012/395415 2. Micropapillary urothelial carcinoma: Clinico-pathologic review Aleksandr M.PerepletchikovAnil V.Parwani. Pathology - Research and Practice. Volume 205, Issue 12, 15 December 2009, Pages 807-810. https://doi.org/10.1016/j.prp.2009.07.016 3. W. GlennMcCluggage Morphological subtypes of ovarian carcinoma: a review with emphasis on new developments and pathogenesis. Pathology, Volume 43, Issue 5, August 2011, Pages 420-43 https://doi.org/10.1097/PAT.0b013e328348a6e7 4. Recommended Articles for Discussion: 5. Eble JN, Young RH Carcinoma of the urinary bladder: a review of its diverse morphology. Seminars in Diagnostic Pathology, 30 Apr 1997, 14(2):98-108 PMID: 9179971	
26-27	**Neoplasia: Soft tissue tumours** (fibrous, fatty, bone, synovial tumours, skeletal muscle, smooth muscle tumours, blood vessels tumours). General questions: Benign and malignant tumors of connective tissue and derivatives. Hemangioma, angiosarcoma: Lymphangioma, lymphangiosarcoma. Synovial sarcoma. Mesothelioma. Meningioma, invasive meningioma. Leomioma, leomiosarcoma. Rhabdomioma, rhabdomyosarcoma. 1. *Describe the essential morphologic features of neoplasms and indicate how these can be used to diagnose, classify, and predict biological behavior of cancers.* 2. *Discuss the cellular capabilities of neoplasms that enable them to invade tissues and to metastasize and recognize how this differentiates benign from malignant neoplasms.* 3. *Discuss the dependence of cancers on stromal elements and ability to generate their own blood supply to maintain growth and explain how this information can be used to treat cancers.* 4. *Define and provide examples of paraneoplastic syndromes and describe how substances produced by cancers can produce systemic effects in the host.* 5. *Discuss the mechanism by which neoplasms produce each of the following, listing neoplasms that are commonly associated with each effect:* • *anemia* • *jaundice* • *ischemia* • *obesity* • *fever* • *masculinization* • *leukocytosis* • *episodic flushing* • *leukopenia* • *hypercalcemia*	10

	• *infection* • *hemorrhage* • *obstruction* • *thrombophlebitis* • *pain* • *endocrine effects* • *itching* • *fracture* 6. *Compare and contrast the basic grading and staging of neoplastic diseases and describe the tumor, (lymph) nodes, metastasis (TNM) classification for common tumors.* **Images**: Multiple myeloma, pleural empyema - 214 Multiple myeloma, diffuse effacement of kidney by neoplastic cells – 193,198 Multiple myeloma, diffuse effacement of pancreas by neoplastic cells - 199 Multiple myeloma, diffuse effacement of liver by neoplastic cells – 184 Multiple myeloma, duodenal ulcers - 171 Recommended Articles for Discussion: 1. Enoch M. Sanders Jr., Virginia A. LiVolsi, James Brierley, Jennifer Shin, Gregory W. Randolph An evidence-based review of poorly differentiated thyroid cancer World Journal of Surgery May 2007, Volume 31, Issue 5, pp 934–945 2. Jae Hoon Lim Cholangiocarcinoma: Morphologic Classification According To Growth Pattern And Imaging Findings American Journal of Roentgenology 2003, Volume 181, Issue 3 3. McCormick D1, Mentzel T, Beham A, Fletcher CD Dedifferentiated liposarcoma. Clinicopathologic analysis of 32 cases suggesting a better prognostic subgroup among pleomorphic sarcomas. The American Journal of Surgical Pathology, 30 Nov 1994, 18(12):1213-1223 DOI: 10.1097/00000478-199412000-00004 4. Gastrointestinal Stromal Tumors: Review on Morphology, Molecular Pathology, Prognosis, and Differential Diagnosis Markku Miettinen, MD and Jerzy Lasota, MD Archives of Pathology & Laboratory Medicine Volume 130, Issue 10 (October 2006) 5. Carolina Reyes, Yevgeniy Karamurzin, Norma Frizzell. Uterine smooth muscle tumors with features suggesting fumarate hydratase aberration: detailed morphologic analysis and correlation with S-(2-succino)-cysteine immunohistochemistry. Modern Pathology volume 27, pages1020–1027(2014)	
28-29	**Leukaemia. Lymphomas 1**: Etiologic and pathogenetic factors in white cell neoplasia. Leukemia/Lymphoma. Myeloid neoplasms. General questions: Neoplastic proliferation of white cells: Lymphoid neoplasm. Myeloid neoplasm. 1. *Describe the essential morphologic features of white cell neoplasms and indicate how these can be used to diagnose, classify, and predict biological behavior of them.* 2. *Define and provide examples of paraneoplastic syndromes and describe how substances produced by neoplasms can produce systemic effects in the host.* 3. *Cite local and general mechanisms which are believed to affect the rate of tumor growth.* 4. *Discuss how tumor growth rates can be evaluated using mitotic rate and cell proliferation markers.* 5. *List four major pathways by which neoplasms spread.*	5

	6. *Discuss metastasis of malignant neoplasms, in terms of:* *molecular genetics* *cellular adhesion* *mechanisms of invasion of extracellular matrix* *mechanisms of vascular dissemination and homing of tumor cells* *tissues and organs in which metastases are:* • *Common* • *Uncommon* 7. *and cite possible reasons for lack of metastases in some instances when cancer cells are spilled into the blood stream.* **Images ,**: Leukemia, diffuse effacement of lymphatic nodule by neoplastic lymphoid infiltrate - 168 Leukemia, diffuse effacement of spleen by neoplastic lymphoid infiltrate – 207 Leukemia, diffuse effacement of bone marrow by neoplastic lymphoid infiltrate – 206 Leukemia, diffuse effacement of kidney by neoplastic lymphoid infiltrate – 169 Leukemia, diffuse effacement of liver by neoplastic lymphoid infiltrate – 170 Leukemia, diffuse effacement of pancreas by neoplastic lymphoid infiltrate - 037 Leukemia, purulent pneumonia – 166 Recommended Article for Discussion: Estella Matutes Aaron Polliack Morphological and Immunophenotypic Features of Chronic Lymphocytic Leukemia https://doi.org/10.1046/j.1468-0734.2000.00002.x David P.Steensma[a]AyalewTefferi[a]Chin-YangLi[b] Splenic histopathological patterns in chronic myelomonocytic leukemia with clinical correlations: reinforcement of the heterogeneity of the syndrome. Leukemia Research Volume 27, Issue 9, September 2003, Pages 775-782 https://doi.org/10.1016/S0145-2126(03)00006-7	
30	**Colloquium** 3. Review and quiz on all micro-images of previous classes (from 21 seminar to 29) and discussion of recommended articles. **Images**: Adenoma of thyroid gland Papilloma Squamous cell carcinoma Cystadenoma ovary Adenoma liver Carcinoma kidney Carcinoma liver Colonic adenocarcinoma Metastatic cancer to the liver Metastatic cancer to the pancreas Metastatic cancer to the lung Invasive squamous cell breast carcinoma Prostate cancer Cavernous hemangioma Chondroma Leiomyomatous uterus Neurofibroma Leukemia, diffuse effacement of lymphatic nodule by neoplastic lymphoid infiltrate Leukemia, diffuse effacement of spleen by neoplastic lymphoid infiltrate Leukemia, diffuse effacement of bone marrow by neoplastic lymphoid infiltrate Leukemia, diffuse effacement of kidney by neoplastic lymphoid infiltrate Leukemia, diffuse effacement of pancreas by neoplastic lymphoid infiltrate Leukemia, diffuse effacement of liver by neoplastic lymphoid infiltrate Multiple myeloma, diffuse effacement of kidney by neoplastic cells	39

	Multiple myeloma, diffuse effacement of pancreas by neoplastic cells Multiple myeloma, diffuse effacement of liver by neoplastic cells	

	BASICS OF IMMUNOPATHOLOGY	
1	**Laboratory Techniques in Immunology. Antigen-Antibody Interactions: Principles and Applications** The main provisions of the immunodiagnostics. Material for the research of immune status. Rules of conducting immunological survey. The generally accepted methods of estimating the state of immunity; Strength of Antigen-Antibody Interactions. Cross reactivity. Precipitation reactions. Agglutination reactions. 1. *list the main provisions of the immunodiagnostics.* 2. *name the first level tests.* 3. *describe second level tests.* 4. *list major uses of serologic (Antibody-Based) tests.* 5. *explain the procedures for quantification of antigen-antibody complexes.* 6. *draw and explain the general steps and applications of agglutination.* 7. *draw and explain main steps of precipitation reactions.* 8. *explain how to interpret data from these tests to diagnose immunologic or microbial diseases.*	2
2	**Methods of the evaluation of innate immunity** Evaluation of phagocyte activity: tests of spontaneous and stimulated phagocytosis, Nitro blue tetrazolium test; Complement Fixation, determination of the concentration of circulating immune complexes 1. *identify the main stages tests of spontaneous and stimulated phagocytosis;* 2. *explain differences between spontaneous and stimulated phagocytosis* 3. *draw and explain principles of Nitro blue tetrazolium test;* 4. *compare and contrast tests of phagocytosis and NBT-test;* 5. *explain methods for detecting complement components in blood serum;* 6. *describe the main stages of determining the level of circulating immune complexes;* 7. *explain how to interpret data from these tests to diagnose immunologic or microbial diseases.*	2
	Antibody-Based Methods Modern methods of determining the number of lymphocytes: Immunofluorescence. Flow cytometry and Fluorescence; Radioimmunoassay. Enzyme-Linked Immunosorbent Assay. Variants of ELISA. Western Blotting (Immunoblot). 1. *explain the general stages and applications of fluorescence method;* 2. *describe the direct fluorescent antibody test;* 3. *draw and explain the indirect fluorescent antibody test;* 4. *justify the use of flow cytometry for analysis and separate different cell types;* 5. *list main steps of Radioimmunoassay;* 6. *draw and explain stages of Enzyme-Linked Immunosorbent Assay;* 7. *list variants of ELISA;* 8. *compare and contrast Radioimmunoassay and ELISA;* 9. *explain how HIV diagnosed using Western blot;* 10. *explain how to interpret data from these tests to diagnose immunologic or microbial diseases.*	2

	Tests for evaluation of cell-mediated immunity Evaluation functional activity of T-lymphocytes: blast transformation reactions, mixed lymphocyte reaction, Macrophage Migration Inhibitory Factor. Skin Tests for the Presence of Delayed-Type Hypersensitivity. *1. explain principle of blast transformation reactions;* *2. name groups of stimulants are which applied during blast transformation reactions;* *3. draw and explain principles of mixed lymphocyte reaction;* *4. give examples of using mixed lymphocyte reaction in clinical practice;* *5. explain the role of macrophage migration inhibiting factor in the assessment of the T-system of immunity;* *6. describe main steps of inhibiting migration macrophages reaction;* *7. summarize the role of these tests in detection of functional activity of T-cells;* 8. *explain how to interpret data from these tests to diagnose immunologic or microbial diseases.*	2
5	**Colloquium -1** Quiz on topics 1-4	8
6	**Cancer and Immune System** Cancer: Origin and Terminology. Malignant transformation of cells. Oncogenes and cancer induction. Cellular transformation and cancer. The cancer problem from an immune perspective. Classes of tumor antigens. Tumor-specific transplantation antigens. Tumor-associated transplantation antigens. Oncofetal tumor antigens. *1. define the terms: carcinoma, sarcoma;* *2. explain induction of cellular proliferation;* *3. describe inhibition of cellular proliferation;* *4. draw and explain regulation of programmed cell death* *5. name and explain tumor antigens:* *7. tumor-specific transplantation antigens* *8. tumor-associated transplantation antigens* *6. know differences chemically and virally induced tumor antigens;* *7. list oncofetal tumor antigens.*	2
7	**Tumor Immunity** Tumor and Immune system. Types of immune response. NK cells and macrophages in tumor recognition. Mechanisms of Antibody Attack. Antitumor antibodies can enhance tumor growth. Killing by Cytotoxic T Lymphocytes. Tumor evasion of the immune system. Cancer Immunotherapy: enhancement of APC activity, cytokine therapy, monoclonal antibodies, and cancer vaccines *1. explain mechanism of innate immune response to tumor cells;* *2. describe features of recognition of tumors by NK-cells;* *3. draw and describe non-specific cytotoxicity of NK-cell;* *4. draw and explain antibody-Mediated Elimination of tumor cells by NK cells;* *5. draw and explain antibody-Mediated Elimination of tumor cells by complement;* *6. describe killing of tumor cells by cytotoxic T-lymphocytes;* *7. explain mechanisms of Tumor evasion of the immune system;* 8. *list main methods of cancer immunotherapy.*	2
8	**Major Histocompatibility Complex & Transplantation** MHC proteins. Biologic Importance of MHC. The polymorphism of the human HLA system. Genetic control of transplantation antigens. The different types of tissue	2

	transplantation performed in medicine. The mechanisms and timing of graft rejection phenomena. 1. *draw and explain the structure of MHC complex;* 2. *explain roles of MHC gene products in immune response;* 3. *describe structure and function of MHC I and MHCII molecules;* 4. *explain principle of inheritance of the MHC;* 5. *explain types of graft;* 6. *describe of types of rejection: Hyperacute rejection, Acute rejection, and Chronic rejection;* 7. *draw and explain mechanism of primary allograft rejection;* 8. *describe mechanism of secondary allograft rejection;* 9. *explain consequences of MHC incompatibility.*	
	Transplantation Immunology Matching the donor and recipient. Testing for tissue compatibility. Immunologic basis of graft rejection. Clinical Manifestations of graft rejection. Clinical transplantation. The pathogenesis of graft-versus-host disease. Prevention and treatment of allograft rejection. 1. *list HLA tissue typing methods;* 2. *describe purpose and main steps of microcytotoxicity test;* 3. *name methods of identify class II MHC-molecules;* 4. *explain mixed lymphocyte reaction;* 5. *identify The role of antibody in allograft rejection;* 6. *explain the role of T cells in allograft rejection;* 7. *name and describe stages of cell-mediated graft rejection:* 9. *sensitization stage* 10. *effector stage* 8. *list and describe prevention and treatment of allograft rejection* 9. *explain mechanism of Graft versus host disease;* 10. *explain how to prevent Graft versus host disease.*	2
10	**Colloquium -2** Quiz on topics 6-9	8
11	**Primary Immunodeficiency** Defects of humoral immunity. Primary T-cell deficiency. Severe combined immunodeficiency. Diagnosis and treatment of primary immunodeficiency. 1. *explain the molecular defects, signs, and symptoms associated with defects of X-linked agammaglobulinemia;* 2. *explain the molecular defects, signs, and symptoms associated with defects of X-linked hyper-IgM syndrom;* 3. *describe selective deficiencies of immunoglobulin classes;* 4. *name and explain defective thymic development:* 11. *DiGeorge syndrome* 12. *arrest of early T-cell differentiation* 5. *explain the molecular defects, signs, and symptoms associated with defects of severe combined immunodeficiency:* 13. *Wiskott-Aldrich syndrome* 14. *Interferon-gamma-receptor defect* 15. *Absence of adenosine deaminase (ADA) and purine nucleoside phosphorylase (PNP)* **16.** *Ataxia teleangioectasia.*	2
12	**Primary Immunodeficiency of Innate Immunity. Secondary Immunodeficiency. AIDS**	2

	Phagocytic cell defects. Deficiencies of complement or its regulation. The receptors and coreceptors of HIV. The immunologic results of systematic TH-cell eradication. The mechanisms through which HIV evades the immune response. *1. name and describe Phagocytic cell defects:* *17. chronic granulomatous disease* *18. Chediak-Higashi syndrome* *19. Job's Syndrome* *2. explain mechanism of hereditary angioedema.* *3. list main factors that may nonspecifically depressed Immune responsiveness;* *4. draw and explain mechanisms of Immune System Destruction by the Human Immunodeficiency Virus;* *5. explain how HIV destroys cell-mediated and humoral immune responses;* *6. explain mechanism of evasion HIV the immune response.*	
13	**Hypersensitivity. IgE-mediated hypersensitivity. Antibody-mediated hypersensitivity (type I and II)** Gell and Coombs classification. Atopic allergy. Anaphylaxis. Reaginic antibody. Mast cells and basophils. Pharmacologic agents mediated type I reactions. The immunologic mechanisms involved in the type II of hypersensitivity reactions. Transfusion reactions. Maternal antibodies. Autoimmune type II hypersensitivity reactions. *1. define the terms: allergy, allergen, hypersensitivity;* *2. draw and explain pathogenesis of atopic allergy;* *3. describe the role of mast cells and basophils;* *4. explain mechanism of dysregulation between Th1 and Th2-lymphocytes;* *5. name clinical examples of atopic allergy;* *6. explain immunologic mechanisms of type II hypersensitivity;* *7. describe clinical examples of Antibody-mediated hypersensitivity:* *20. Transfusion reaction* *21. Hemolytic disease of the newborn* **22.** *Drug induced hemolytic anemia*	2
14	**Hypersensitivity. Immune complex-mediated hypersensitivity. Delayed type hypersensitivity (DTH)** Disease resulting from circulating complexes. Deposition of immune complexes at other sites. Inflammatory lesions due to locally formed Complexes. The Arthus reaction. The cellular basis of type IV hypersensitivity. Tissue damage produced by type IV reactions *1. explain immunologic mechanisms of type III hypersensitivity* *2. name examples of Type III Hypersensitivities;* *3. explain how is immune complexes damage joints, blood vessels and kidney;* *4. draw and explain pathogenesis of Delayed type hypersensitivity;* *5. name and explain phases of the DTH response;* *6. name and explain cytokines that are participate in the DTH reactions;* *7. describe important clinical aspects of delayed hypersensitivities.*	2
15	**Colloquium -3** Quiz on topics 11-14	8

Methodical instruction for tutorials

Aim: integrated knowledge of pathophysiological processes and morphological changes in organs, tissues, cells, ultrastructures in the event of the development and

outcomes of pathological processes and diseases; understanding of immunopathogenesis of diseases developing due to inadequacy of the immune system; principles of immunization, immunodiagnostics and immunotherapy; to provide a stimulating and challenging learning environment where teaching is informed and enhanced by research, and to provide training in scientific principles and experience in the evaluation and practice of research.

Learning outcomes:

1. Demonstrate understanding of the basic concepts of general nosology, sanogenesis, thanatogenesis;
2. Demonstrate understanding of the pathogenesis and morphogenesis of typical pathological processes (hypoxia, impaired peripheral circulation, inflammation, regeneration, wound healing, sclerosis, fibrosis, allergic reactions, oncoprocess).
3. Value and possibilities of modeling pathological processes and experimental therapy in animals in the study of human diseases. General principles of construction of biomedical experiments.
4. Demonstrate analytical skills in the integration of knowledge on pathophysiology, pathological anatomy, and immunology in the formation of judgments regarding general pathology.
5. To be able to interpret the results of specific immunodiagnostics and apply knowledge of immunodiagnostics and immunoprophylaxis
6. Understand the pathogenesis of the development of immunodeficiency states and primary immunodeficiencies, immunopathological states (inflammation, oncopathology, autoimmune and allergic diseases)
7. Understand the principles of histocompatibility and interaction of the graft and host (graft and host)
8. Demonstrate the ability to identify learning gaps and create strategies to enhance one's own knowledge and skills.
9. Effectively communicate with other students and teachers regarding medical and scientific information, articulate their opinions clearly when discussing pathophysiological processes and their impact, and work effectively as a member of the team

Plan of preparation work for each lesson

1. Work with the basic and additional literature, use textbooks, the syllabus and present instructions, Internet resources to prepare for tutorials.
2. Be prepared for class and participate actively on case-discussion and problem-solving group activities.
3. Work carefully with microscope and micro-slides and prepare your descriptions.
4. Use the examples (in this number cases and your own experience studied before) for illustration of theoretic material.
5. Use different tools for studying, discussion and visualization of thoughts - drawing, mind maps, 3d-modelling.
6. Use the group work with cases for the development of team work skills, communication, problem solving and self-studying.

№	Grading Criteria	level			
		4 excellent 90-100	3 good 75-89	2 satisfied 50-74	**1** fall 49 - 0
1.	Level of understanding of the pathological process	20	17	14	0
2.	Understanding of changes in the tissue	20	17	14	0
3.	Knowledge of relevant theories regarding pathological process	20	17	14	0
4.	Choice of examples	20	17	14	0
5.	Knowledge of professional terminology	20	17	14	0
	Total **Maximum score – 100**				

Oral Survey (Immunopathology)

№	Grading Criteria (Immunopathology)	level			
		4 excellent 90-100	3 good 75-89	2 satisfied 50-74	**1** fall 49 - 0
1.	Level of understanding of the topic	20	17	14	0
2.	Ability to make informed conclusions	20	17	14	0
3.	Compliance of the material with the questions posed	20	17	14	0
4.	Confirmation of the answer with concrete examples and facts	20	17	14	0
5.	Knowledge of professional terminology	20	17	14	0
	Total **Maximum score - 100**.				

References:

1. Robbins and Cotran Pathologic Basis of Disease [Electronic resource] : textbook / ed.: V. Kumar, A. Abbas, J. Aster. - Philadelphia : Elsevier Saunders, 2015. - 1392 p. - ISBN 978-1-4557-2613-4 : 0.00
2. Pathophysiology of Disease [Electronic resource] : An Introduction to Clinical Medicine / ed.: G. Hammer, S. McPhee. - 7th ed. - New York ; Chicago San ;

Francisco : McGraw-Hill, 2014. - 779 p. - ISBN 978-0-07-180601-5. - ISBN 978-0-07-180600-8 : 0.00
3. Basic Immunology [Electronic resource] : Functions and Disorders of the Immune System / A. K. Abbas, A. H. Lichtman, S. Pillai [et al.]. - 5th ed. - St. Louis : Elsevier, 2015. - 327 p. - ISBN 978-0-323-39082-8 : 0.00
4. Roitt's Essential Immunology [Electronic resource] : textbook / P. J. Delves, S. J. Martin, D. R. Burton [et al.]. - 13th ed. - Pondicherry : Garamond by SPi Global, 2017. - 576 p. - ISBN 978-1-118-41577-1 : 0.00

On line resources:

1. Set of video-lectures on Univer system kazNU
2. https://webpath.med.utah.edu/GENERAL.html
3. https://www.webpathology.com/
4. https://web.duke.edu/pathology/
5. https://alf3.urz.unibas.ch/pathopic/e/intro.htm

SOME TIPS ON TEAMWORK AND LEARNING[1]

The medical profession involves working in multidisciplinary teams, so these skills are identified as key in the competence of the doctor and other health professionals in all countries.

Therefore, group work is included as an essential component in the practical exercises of our course. In addition, it aims to provide a safe environment in which you can try out new ideas and practices and acquire relevant group skills. These can be tasks for performance in pairs, triples or small groups of 4-6 people (work with cases, tasks of the ISW, etc.).

When you are working on a project or task in a team, you have the opportunity to use the various strengths of the group members to create a wider and better project or task than if you were working independently.

Group training means you need to share your knowledge and ideas with other students. There are two benefits to this: you need to think carefully about your own ideas in order to explain them to others, and you expand your own understanding, taking into account the knowledge and ideas of others.

Interpersonal Communication and Discussion

Take some time to chat and get to know each of your group mates. The better you know each other and the more convenient you communicate, the more effective you can work together.

Create a culture of mutual respect in your group. You probably had little choice or no choice at all when forming training groups and small teams in the classroom. Therefore, you will have to learn to overcome the differences between people. In addition, you will not have the opportunity to choose employees in the workplace, and at work, you will experience much greater pressure to be a productive member of the team.

[1] adapted from UNSW Guide to Group Work https://student.unsw.edu.au/groupwork)

For effective communication and discussion in a team: you should not be shy to express your opinion and it is important to feel that these opinions will be heard; it is necessary to feel that all members of the group make a feasible contribution to solving problems, observing agreed rules and plans, performing work efficiently and on time; it is important to know that everyone's feelings are taken into account by team members, but the goals and objectives of the group are not compromised, in favor of the whims or desires of individual members;

Try to express your opinion and listen to others. There is nothing wrong with disagreeing with your classmates, no matter how confident they are. When you disagree, be constructive and focus on the problem, not the person. Similarly, when someone disagrees with you, respect what he says and the risk that he takes upon himself to express his opinion. Try to find a way that everyone can agree with, and this is not necessarily the opinion of the loudest or smartest member of the team.

Below we provide some examples of constructive and destructive group behavior[2]

Constructive group behavior - a person who:

Unites - interest in the views and opinions of others and willingness to adapt to interest

Clarifies - clearly defines the problems for the group by listening, summarizing, focusing the discussion

Inspires - encourages the group, stimulates participation and progress

Harmonizes - stimulates group unity and teamwork. For example, uses humor as a relaxation after difficult situations.

Take the risk - willingness to take risks at the expense of oneself for the success of the group or project

Manages the process - organizes a group on the issues of the process: for example, plan, schedule, timeline, topic, solution methods, and use of information

Destructive group behavior:

Domination - takes a lot of time expressing your opinion and views. Trying to take control by capturing energy, time, etc.

Fussiness - hastens the group to move quickly before the task is completed. Impatient in listening to other opinions and working together.

Suspension - removes itself from a discussion or decision. Opt out

Ignoring - does not respect or belittle the ideas and suggestions of the team or individuals. An extreme manifestation of ignoring is an insult in the form of ridicule.

Distraction - excessive talkativeness, tells stories and leads groups away from the goal

Blocking - prevents group progress by denying all ideas and suggestions. "It will not work because ..."

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[2] adapted from Brunt (1993): https://tle.wisc.edu/solutions/engagement/constructive-and-destructive-groupbehaviors

Effective group work does not arise by itself. A conscious and planned effort is needed, and since many people participate in it, one cannot rely on memory; need to make notes. **The following steps** will help you and your team work together effectively.

1. Define clear objectives. At each stage, you should try to coordinate the tasks. They include a timeline for the project, as well as more specific tasks (such as "agree on an approach to the task before Friday"). Each meeting or discussion should also begin with a specific goal (for example, make a list of tasks that need to be completed). Tasks should be broken down into smaller parts and planned. Sometimes one part cannot be started until the other part is finished, so you may need to draw a simple temporary map.

· discuss the resources that you have and those that you will need to find.
· formulate the desired result.
· consider how you know when you did it well enough?
· split tasks between the team and
· set deadlines for subtasks and time for future meetings.

2. Set the basic rules. Discussions can become erratic and can prevent more modest group members from participating if you do not have rules to stimulate discussion, resolve disagreements, and make decisions without repetition. Set the rules from the start and change them as needed. For example: an interesting rule that was developed by one group - anyone who missed a meeting would buy the rest of the group coffee in a coffee shop. No one ever missed a meeting after that.

3. Communicate effectively. Make sure you regularly communicate with group members. Try to be clear and positive in what you say without repeating.

4. Find consensus. People work together most effectively when they work towards a goal with which they have agreed. Make sure everyone has their own opinion, even if you need time to get more participants to say something. Make sure you listen to everyone's ideas and then try to come to an agreement that everyone shares, and everyone has contributed.

5. Define the roles. Divide the work that needs to be done into separate tasks, for which you can use the strengths of individual team members. Define roles for both fulfilling your tasks and for meetings / discussions (for example, Aran is responsible for summarizing the discussions, Joseph is for everyone to express their opinions and make decisions, etc.).

Examples of roles and functions:

Facilitator or *leader* (depending on context) - to clarify the goals of the meeting and to summarize the discussions and decisions; ensures that the meeting takes place, continues and the basic rules are respected.

Secretary - keep a record of the ideas discussed and decisions made and who does what.

Time Manager - to make sure that you discuss everything that you need in the time allotted for the meeting.

Controller - to ensure that work is completed by an agreed time, and to solve problems if they are not being performed.

A process observer is someone who monitors the process, not the content, and can bring problems to the attention of the team. In this role, it is important to be positive, not condemning.

Editor - bring all materials together, identify gaps or matches and ensure consistency in the final presentation.

6. Make it clear. When a decision is made, it should be explained in such a way that it is clear to everyone that it was decided, including the time frame.

7. Keep good notes. Always summarize the discussions and document the decisions and publish them (for example in WhatsApp chat) so you can always get back to them. This includes lists of those who agreed what to do.

8. Stick to the plan. If you agreed to do something as part of the plan, do it. Your group relies on you to do what you agreed to do, and exactly in this way, not in the way you would like. If you think the plan should be reviewed, discuss it.

9. Keep track of progress and keep up to date. Discuss progress together regarding your schedule and deadlines. Make sure you meet deadlines personally, so you do not let your group down.

Co-writing a document / report

Joint writing is one of the most difficult parts of group work. There are many ways to do this, and your group must decide how to separate the work of writing, comparing, editing, and finalizing your work. Writing in a group (six people crowd around the keyboard) is a recipe for conflict and lack of progress. The other extreme - when one person assumes all responsibility and ultimately does most of the work - is also unproductive and contributes to conflict.

Three approaches are possible when working on a common document:

1 - One person writes the most part - this means that a narrow circle of ideas is used, and the rest of the team does not learn (and will not learn) to write reports and documents.

2 - Each person writes one section - then it is difficult to make a single consistent report, and you will not know about the rest, except for your own section.

3- Co-writing. This is the most productive way to solve group problems and provides the greatest benefit from collaboration. For example: in each section, there is a writer and at least one reviewer, and each team member is the author of a section and a reviewer of another one.

All team members before finalization by **the editor** must review the final product. Alternatively, you can have one author with others, editors, add and review, and someone tidies the finished report.

Try to divide the writing of source documents into tasks and solve them individually or in pairs. After the first draft of the sections are written, send out all the components and read them. You will probably need to come together to discuss how to combine them so that they fit together. Any participants who were not involved in preparing the drafts can do part of this work. Then edit, improve, and polish the draft. It is convenient to collaborate on documents in Google documents.

When preparing a report / final document, regularly check the following:

- Is the purpose of the project clear from the report?

- Are the conclusions or recommendations clear?
- Do conclusions follow from the main part of the report?
- Do sections fit well?
- Does the report achieve goals (and evaluation criteria)?
- Are the necessary components sufficiently covered?

Whatever method you use, all group members must agree on the process and how they are going to maximize the collaborative approach to writing the final document.

Monitoring team performance and coping

Below is a checklist that includes a list of common problems that arise in a group work. Use it regularly to identify problems before they get out of hand. If serious problems and tensions arise, use it to determine where something might go wrong. First answer each question about yourself, and then give answer to this **question** about the group. Then gather a group and discuss where, in your opinion, problems may arise, and think about how you can overcome these problems.

Each participant must complete this checklist. You should do this exercise regularly to track and improve your team's performance.

1. Answer each question regarding your teamwork.
2. Answer each question regarding the rest of the team.
3. Get together with your entire team and discuss where, in your opinion, any problems arise.
4. Discuss what you are going to do to overcome these problems.

Checklist for self-assessment of team effectiveness.

You	I personally	Group as a whole	Comments
Effectively clarify your tasks and tasks at each stage?			
Evaluate the progress of work?			
We clarify and document everything that the group decided?			
We clarify who will do what and how?			
We clarify by what date each task should be done?			
Setting meeting management rules?			
Adhere to agreed rules?			
Listening to each other?			
Allow some team members to dominate?			
Allow some team members to refuse / withdraw?			

We sacrifice personal desires for the success of the team?			
Recognize the feelings of other team members?			
Making equal contributions to team progress?			
Adhere to agreed rules for writing and naming files?			

Points and Grade

Group tasks and assignments mean that grades are given to the whole group based on the results of the work of the whole group. Everyone should be interested in ensuring the effective contribution of all members of the group and ensuring the high quality of the assignment. Sometimes, to assess the relative contribution of each to the group process, a form of peer-to-peer or peer review and a team assessment form will be used. This can be used to moderate assignment grades, or simply as a way to give feedback on your work in a group. The following are examples of student assessment criteria for team training.

№	**Student assessment criteria in practical classes**
1	*Preparation for classes:* He studies information focused on the case and problematic issues, uses various sources, and supports the statements with relevant links.
2	*Group skills and professional attitude:* Demonstrates excellent attendance, reliability, responsibility Takes the initiative, takes an active part in the discussion, helps the teammates, willingly takes on tasks
3	*Communication skills:* Actively listens, shows emotions according to the situation, is susceptible to non-verbal and emotional signals, shows respect and correctness in relation to others, helps to resolve misunderstandings and conflicts
4	*Feedback Skills:* Demonstrates a high level of introspection, critically evaluates oneself and colleagues, provides constructive and objective feedback in a friendly manner, accepts feedback without opposition
5	*Skills of critical thinking and effective learning:* Effectively participates in generating hypotheses and formulating problematic questions, gives relevant examples from life, skillfully applies knowledge to the problem / case under consideration, critically evaluates information, draws conclusions, explains and substantiates statements, draws diagrams and drawings, demonstrates a constant interest in the material being studied

6	*Theoretical knowledge and skills on the topic of the lesson:* All key aspects are presented logically; accuracy, relevance of answers to the questions posed without redundancy; integration of theoretical issues; Use of relevant examples proper use of professional terminology