

**Instructional Objectives / Learning Outcomes**  
**DMP 775, Veterinary Clinical Pathology**  
**Department of Diagnostic Medicine/Pathobiology**  
**College of Veterinary Medicine, Kansas State University**

Chapter 1: Introductory concepts

1. Define *pathology* and use the noun appropriately in written and oral language.
2. Explain why laboratory tests are used to evaluate samples from patients.
3. Contrast and compare qualitative and quantitative laboratory assays.
4. Convert common units that use the prefixes including *milli*, *micro*, *nano*, *pico*, *femto*, and *deci*.
5. Define *normal* in the context of describing clinical laboratory data. Explain why a “*normal*” animal may have laboratory data outside of reference intervals. Explain why a sick animal may have laboratory data within reference intervals.
6. Define *reference individual*, *reference population*, *reference sample group*, *reference value*, *reference distribution*, *reference limit*, *reference interval*, and *observed value* and use the terms appropriately.
7. Describe how reference intervals are established and their purposes.
8. Explain what *reference intervals* represent and when parametric or non-parametric methods should be used to establish reference limits.
9. Explain factors or procedures that determine the quality of laboratory test results and give one example when erroneous results are produced if that factor or procedure is poor.
10. Explain why reference intervals published in textbooks may not be adequate for evaluating your patient.
11. Contrast and compare the analytical properties of assays (precision, accuracy, specificity, detection limit, and sensitivity), explain how these properties influence our interpretation of assay results, and use the terms appropriately to explain assay results that are outside of reference intervals.
12. Identify and classify the appropriate analytical property in clinical situations. For example:
  - a. Results of repeated analysis of the same sample do not give the same results. The different results represent \_\_\_\_\_.
  - b. Result of assay A was 100 mg/dL but that of assay B on the same sample was 80 mg/dL. If assay A was the “gold standard” assay, then assay B is \_\_\_\_\_.
  - c. Lipemia in a sample can increase the measured concentration of same analytes. Accordingly, this assay is \_\_\_\_\_.
  - d. Results of assay C indicate the analyte’s concentration is 10 µg/dL, but assay D indicates the concentration is 0 µg/dL. This difference may be due to \_\_\_\_\_.
  - e. Results of assay E indicate the analyte’s concentration is 100 mg/dL on both day 1 and day 2 samples. However, assay F values were 92 mg/dL and 105 mg/dL on the two samples. The differences in results may be due to \_\_\_\_\_.
13. Explain how knowledge of an assay’s analytical precision is used to determine if changes in laboratory data are due to biologic variation or analytical variation.
14. Contrast and compare a control solution and a standard solution.
15. Define coefficient of variation (CV) and explain why a CV of 10% may be very acceptable for one assay but very unacceptable for another assay.
16. Contrast *random error* and *systematic error* (bias).

17. In the context of predictive values, define *true positive*, *true negative*, *false positive*, and *false negative*. List or describe the two factors that must be known to classify data into the four categories.
18. Contrast and compare diagnostic sensitivity, diagnostic specificity, diagnostic accuracy, and predictive value of positive or negative test and explain how the concepts influence our interpretation of assay results.
19. Contrast and compare diagnostic sensitivity and PV(+). Contrast and compare diagnostic specificity and PV(-).
20. Given necessary clinical and laboratory information about a group of animals, calculate diagnostic sensitivity, diagnostic specificity, diagnostic accuracy, and predictive value of positive or negative test.
21. Explain how the following alter the calculated values for the diagnostic sensitivity, diagnostic specificity, and diagnostic accuracy of a test.
  - a. Prevalence of disease
  - b. Poor “gold standard”
  - c. Increasing or lowering the cut-off value
  - d. Using a healthy animal group as the “disease absent” group
  - e. Using a sick animal group with similar clinical signs but without the disease as the “disease absent” group
22. Recognize and list the diagnostic property that is most important for a:
  - a. Screening test
  - b. Confirmation test
23. Explain the purpose of constructing ROC curves for laboratory assays.
24. State the data that are plotted on the y-axis and x-axis of a ROC curve. Based on those facts, explain why the best ROC curve approaches the upper left corner of the graph.

#### Chapter 2: Basic hematologic assays

25. Contrast and compare blood, plasma, and serum.
26. Contrast and compare the anticoagulant properties of EDTA, citrate, and heparin.
27. Define a CBC and describe each component including what the units of measurement represent.
28. Explain the basic purpose or potential value of each component of a CBC.
29. Explain how or why poor sample collection or handling may lead to erroneous CBC results.
30. For impedance cell counters, recognize or list the parts of the CBC that are measured and the parts that are calculated from measured values. Describe the basic principles used in the measurements.
31. Explain what an electronic cell counter measures and why such instruments may not provide accurate results for all animal species.
32. Recognize and list the major advantages of an optical counter over an impedance counter. Describe how the advantages help us.
33. Recognize and explain how a QBC VetAutoread™ determines the following: Hct, Hgb, MCHC, WBC concentration, platelet concentration.
34. For the typical CBC results, explain how a neutrophil concentration is determined.
35. Contrast and compare the Hct that is determined by centrifugation and electronic hematologic instruments.
36. Describe the general types of abnormalities that may be found in a blood film evaluation.

37. Define reticulocyte percentage (RP), corrected reticulocyte percentage (CRP), and reticulocyte concentration (RC) and describe the diagnostic value of information obtained from them.
38. Explain when we expect to find the following.
  - a. reticulocytosis in a dog, cow, or horse
  - b. punctate or aggregate reticulocytosis in a cat
39. Explain the clinical significance if we expect reticulocytosis and it is not present.
40. Given appropriate information, calculate and interpret a corrected WBC concentration, nRBC concentration, RP, CRP, and RC.
41. Explain the clinical significance of:
  - a. Positive Coombs' test in an animal with a regenerative anemia
  - b. Positive Coombs' test in an animal with a nonregenerative anemia
  - c. Positive Coombs' test in an animal without an anemia
  - d. Negative Coombs' test in an animal with a regenerative anemia
  - e. Negative Coombs' test in an animal with a nonregenerative anemia

### Chapter 3: Leukocytes

42. Given leukograms and reference intervals for domestic mammals with common clinical disorders:
  - a. List and classify abnormalities using appropriate terms.
  - b. Propose appropriate diseases, syndromes, pathologic states or conditions that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
43. Define and contrast the following or similar states (information not in FVCP, 1<sup>st</sup> ed.).
  - a. Absolute neutrophilia
  - b. Relative neutrophilia
44. Recognize and explain the situations or conditions when the following are true (information not in FVCP, 1<sup>st</sup> ed.).
  - a. There is a relative lymphocytosis but there is not a lymphocytosis.
  - b. There is a relative lymphopenia but there is a lymphocytosis.
  - c. There is a normal leukocyte differential count but there is a neutropenia.
45. Define, in the context of blood neutrophils: left shift, right shift, regenerative left shift, degenerative left shift.
46. List and recognize these diseases or conditions that may cause a right shift: glucocorticoid hormones, Poodle marrow dyscrasia syndrome, and *in vitro* aging. For each, explain the pathogenesis of the right shift (if known).
47. List and recognize these 4 categories or types of neutrophilia: inflammatory, glucocorticoid-associated, physiologic, neoplastic. For each, explain the processes that produce neutrophilia (if known). List the types of diseases/disorders that may create an inflammatory neutrophilia.
48. List and recognize these 4 categories or types of neutropenia: inflammatory, peripheral destruction, granulocytic hypoplasia, ineffective production. For each, explain the processes that produce neutropenia (if known). List the types of diseases/disorders that may create an inflammatory neutropenia, destruction neutropenia, granulocytic hypoplasia, and ineffective-production neutropenia.
49. List and recognize these 4 categories or types of lymphocytosis: inflammatory, physiologic, neoplastic, hypoadrenocorticism. For each, explain the processes that

- produce lymphocytosis (if known). List the types of diseases/disorders that may create an inflammatory lymphocytosis.
50. List and recognize these 4 categories or types of lymphopenia: inflammatory, glucocorticoid-associated, depletion, lymphoid hypoplasia/aplasia. For each, explain the processes that produce lymphopenia (if known). List the types of diseases that may create an inflammatory lymphopenia or glucocorticoid-associated lymphopenia.
  51. List and recognize the categories or types of disorders that cause these findings.
    - a. Monocytosis
    - b. Eosinophilia (see Table 3.9, p. 71)
    - c. Eosinopenia
    - d. Basophilia
    - e. Mastocytemia
  52. Explain the species variations in leukocyte kinetics which cause the following.
    - a. Inflammatory neutropenia in cattle with relatively mild inflammatory diseases
    - b. Leukocytosis, neutrophilia, or lymphocytosis in frightened cats.
  53. List and recognize the categories or types of disorders that cause these findings.
    - a. Leukocytosis (major concepts)
    - b. Leukopenia (major concepts)
  54. Explain the clinical significance of the following if found in an animal's blood.
    - a. Toxic neutrophils
    - b. Giant neutrophils
    - c. Hypersegmented neutrophils
    - d. Reactive lymphocytes
  55. For morphologic examination, identify abnormal leukocytes or leukocyte inclusions of the following and explain their clinical significance.
    - a. Erythrophage
    - b. Ehrlichial morulae
    - c. *Hepatozoon americanum*
    - d. *Histoplasma capsulatum*
    - e. *Leishmania* spp..
    - f. *Mycobacterium* spp.
    - g. Pelger-Huët neutrophils or eosinophils
  56. Extra credit (potential) - For morphologic examination, identify abnormal leukocytes or leukocyte inclusions of the following and explain their clinical significance.
    - a. Hereditary disorders that have leukocyte inclusions (p. 79)
    - b. Bone marrow dyscrasia of poodles (p. 80)
    - c. Idiopathic hypersegmentation of horses (p. 80)
    - d. Pseudo-Pelger-Huët neutrophils (p. 80)

#### Chapter 4: Erythrocytes

57. Given erythrograms and reference intervals of domestic mammals with the major anemia disorders:
  - a. List and classify abnormalities using appropriate terms.
  - b. Classify anemias as regenerative or nonregenerative if reticulocyte percentages are available.
  - c. Propose appropriate diseases, syndromes, pathologic states, or conditions that could cause the defined abnormalities.

- d. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
58. Explain the clinical significance of the following if found in an animal's blood; be able to identify those followed by (*ID*).
- a. Rouleau, rouleaux (*ID*)
  - b. Agglutination (*ID*)
  - c. Rubricytosis (*ID*) (also, see later objective)
  - d. Increased central pallor
  - e. Decreased central pallor
  - f. Ghost cell (*ID*)
  - g. Hypochromic erythrocyte (*ID*)
  - h. Hypochromasia (*ID*)
  - i. Polychromatophilic erythrocyte (polychromatophil) (*ID*)
  - j. Increases polychromasia (*ID*)
  - k. Reticulocyte (*ID*)
  - l. Reticulocytosis
  - m. Organisms: *Anaplasma*, *Babesia*, *Cytauxzoon*, *Eperythrozoon*, *Mycoplasma (Haemobartonella)* (*ID*)
  - n. Basophilic stippling (*ID*)
  - o. Heinz body (*ID*)
  - p. Howell-Jolly body (*ID*)
  - q. Siderotic granules (Pappenheimer bodies)
  - r. Anisocytosis (*ID*)
  - s. Macrocytes (macrocytosis) (*ID*)
  - t. Microcytes (microcytosis) (*ID*)
  - u. Acanthocyte (spur cell) (acanthocytosis) (*ID*)
  - v. Codocyte (target cell, Mexican hat cell) (codocytosis) (*ID*)
  - w. Eccentrocyte (bite cell, cross-bonded cell, hemighost cell) (eccentrocytosis) (*ID*)
  - x. Echinocyte (echinocytosis) (*ID*)
  - y. Keratocyte (helmet cell) (keratocytosis) (*ID*)
  - z. Leptocyte (leptocytosis) (*ID*)
  - aa. Poikilocyte (poikilocytosis) (*ID*)
  - bb. Pyknocyte (pyknocytosis) (*ID*)
  - cc. Schizocyte (schistocyte or RBC fragment) (schizocytosis) (*ID*)
  - dd. Spherocyte (spherocytosis) (*ID*)
  - ee. Stomatocyte (stomatocytosis) (*ID*)
59. Contrast and compare appropriate and inappropriate rubricytosis including rule-outs and pathogeneses. (p. 95)
60. List the information that is needed to use each of the three anemia classification systems.
61. If a reticulocyte percentage or concentration is not available, list or explain other information that would suggest there is an effective marrow response to the anemia. When considered individually, state why such information would not be as reliable as establishing the presence or absence of reticulocytosis.
62. List the basic pathologic states or processes that are probably present if an animal has a regenerative (responsive) anemia.
63. Explain why:
- a. Most nonregenerative anemias are normocytic normochromic anemias.

- b. Nearly all hemorrhagic and hemolytic anemias are normocytic normochromic anemias during the first few days after the hemolytic or hemorrhagic episodes.
  - c. Most regenerative anemias are either macrocytic normochromic or macrocytic hypochromic anemias.
64. State major rule-outs (diseases, pathologic states, etc) for each of the following; for each, explain the process by which the MCV or MCHC changes. In which of the states do you expect the MCH to change (either increase or decrease)?
- a. Normocytic normochromic anemia
  - b. Macrocytic normochromic anemia
  - c. Macrocytic hypochromic anemia
  - d. Microcytic normochromic anemia
  - e. Microcytic hypochromic anemia
65. Wintrobe's erythrocyte indices typically reflect erythrocyte changes that can also be seen during the microscopic examination of erythrocytes, but not always. List reasons why the following may be found.
- a. MCV is WRI but macrocytes are seen in a blood film.
  - b. MCV is increased but macrocytes are not seen in a blood film.
  - c. MCV is decreased but microcytes are not seen in a blood film.
66. List and explain the reasons for increased MCHC values, both factitious and pathologic.
- a. Factitious
  - b. Pathologic
67. List the three major pathophysiologic processes that produce anemias and are the basis of the pathophysiologic classification of anemias. For each process, explain how it produces an anemia.
68. Recognize and list the four major types of disorders or conditions that cause nonregenerative anemia. For each, recognize and explain the pathologic processes that cause anemias in these disorders. Compare and contrast these processes.
69. Explain the pathogenesis of the anemia of:
- a. Inflammatory disease (3 major components)
  - b. Renal disease (3-4 major components)
  - c. Marrow hypoplasia or aplasia
  - d. Erythroid hypoplasia or ineffective erythropoiesis
70. List types of diseases or disorders that cause marrow hypoplasia or aplasia (4 major types).
71. List types of diseases or disorders that cause erythroid hypoplasia or ineffective erythropoiesis (5 types).
72. Explain how blood loss results in:
- a. Regenerative anemia
  - b. Iron deficiency anemia
73. Define hemolysis; contrast and compare intravascular hemolysis and extravascular hemolysis including clinical manifestations and sites of erythrocyte destruction.
74. Explain how hemolysis produces:
- a. Anemia
  - b. Hemoglobinemia and hemoglobinuria
  - c. Icterus, hyperbilirubinemia, and bilirubinuria
75. Explain why a thorough examination of erythrocytes in a blood film is indicated if laboratory test results suggest or indicate the presence of a hemolytic anemia.

76. List the major mechanisms or processes that cause hemolytic anemias in the hemolytic disorders. For each type, list the major diseases and other conditions for domestic animals.
77. Describe pathologic events or physiologic responses that lead to the development of:
  - a. Immune hemolytic anemias
  - b. Heinz body hemolytic anemias
  - c. Eccentrocytic hemolytic anemias
  - d. Parasitic hemolytic anemias
  - e. Fragmentation hemolysis
  - f. Hemolytic disorders of bacterial infections
78. Red urine can indicate the presence of three pathologic states; list them and explain how the one caused by hemolysis is differentiated from the other two.
79. Describe situations when bone marrow examinations may be indicated for an animal with a persistent normocytic normochromic nonregenerative anemia.
80. Describe how the following findings might help determine the cause of an animal's anemia.
  - a. Hyperproteinemia
  - b. Hypoproteinemia
  - c. Hyperbilirubinemia and bilirubinuria
  - d. Hemoglobinemia and hemoglobinuria
  - e. Positive Coombs' test
  - f. Hypoferremia
81. Define, compare, and contrast erythrocytosis, hemoconcentration, polycythemia, "relative polycythemia," and "spurious polycythemia."
82. List the 5 major types of erythrocytotic (erythrocytosis) disorders.
83. Describe the pathologic events or physiologic responses which cause erythrocytosis in the following:
  - a. Hemoconcentration
  - b. Splenic contraction
  - c. Secondary appropriate erythrocytotic disorders
  - d. Secondary inappropriate erythrocytotic disorders
  - e. Primary erythrocytotic disorders
84. Recognize or explain the pathologic or physiologic processes that produce the following. For each, briefly explain how the process created the abnormality (if known).
  - a. Hyperferremia
  - b. Hypoferremia
  - c. ↑ TIBC
  - d. ↓ TIBC
  - e. ↑ iron storage
  - f. ↓ iron storage
  - g. Hyperferritinemia
  - h. Hypoferritinemia
85. If provided with clinical information and results of an iron profile, differentiate the following disorders.
  - a. Fe deficiency from inflammation
  - b. Inflammation from iron overload
  - c. Inflammation from hemolysis

- d. Hepatic insufficiency from Fe deficiency
- 86. Extra credit material: explain the pathogenesis (fact or theory) of the erythrocyte abnormalities associated with or due to:
  - a. Erythrocyte enzyme deficiencies: PK, PFK, G6PD, cytochrome b<sub>5</sub> reductase
  - b. Erythrocyte FAD deficiency
  - c. Hereditary stomatocytosis
  - d. Hereditary elliptocytosis
  - e. Megaloblastic anemia
  - f. Nutritional deficiencies: copper, folate, cobalamin
  - g. Hypophosphatemia
  - h. Porphyria
  - i. Heparin
  - j. Infusion of hypotonic fluid
  - k. Envenomation

#### Chapter 5: Hemostasis

- 87. Given pertinent historical or physical findings, platelet concentration, PTT, PT, ACT, bleeding time, FDP concentration and/or fibrinogen concentration (*see class cases*):
  - a. List and classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
- 88. Explain why or when platelet clumps are found on a blood film. Explain why detecting the presence of platelet clumps is an important aspect of routine blood examinations.
- 89. List the three major pathologic mechanisms (or combinations) that produce a thrombocytopenia. For each, list common diseases or conditions that produce thrombocytopenia through that process/mechanism.
- 90. List and recognize the three reasons for a pseudothrombocytopenia.
- 91. List and recognize the types of disorders or conditions that may cause a “production failure” thrombocytopenia.
- 92. List and recognize the types of disorders or conditions that may cause a “decreased platelet survival” thrombocytopenia.
- 93. Compare and contrast destruction thrombocytopenia and consumption thrombocytopenia.
- 94. List and recognize the mechanisms that may contribute to:
  - a. Infectious thrombocytopenia
  - b. Neoplasia-associated thrombocytopenia
- 95. Compare and contrast the pathogeneses of the following immune-mediated thrombocytopenias, especially the source of or stimulus for the offending antibody: idiopathic IMT, drug-induced IMT, infection-associated IMT, neonatal alloimmune thrombocytopenia, systemic immune-mediated illness.
- 96. List and recognize the nonimmunologic causes of decreased platelet survival.
- 97. List and recognize the two changes in platelet kinetics that can result in a thrombocytosis.
- 98. Compare or contrast a clonal thrombocytosis and a reactive thrombocytosis.
- 99. List the common causes of thrombocytosis and recognize other causes of all thrombocytoses.
- 100. Contrast and compare bleeding time procedure with clotting time assays.

101. Describe the special sample collection and handling that is required for the plasma that is used for PTT and PT assays.
102. List the clotting factors or hemostasis processes that are evaluated by a:
  - a. PTT
  - b. ACT
  - c. PT
  - d. TT
  - e. FDP
  - f. Fibrinogen concentration
103. List the possible defects in the hemostasis system that result in the following abnormalities.
  - a. Prolonged PTT
  - b. Prolonged ACT
  - c. Prolonged PT
  - d. Prolonged TT
  - e. Increased FDP
  - f. Decreased fibrinogen concentration
104. State the major pathologic process that results in increased FDP or D-dimer concentrations.
105. Explain why knowledge of FDP concentration helps with the interpretation of PT, PTT, TT, ACT, and BMBT results.
106. Explain how the following disorders result in a hemostasis defect.
  - a. Diseases that cause thrombocytopenia
  - b. Hepatic disease
  - c. Rodenticide or other anticoagulant toxicosis
  - d. Cholestasis
  - e. Malabsorptive or maldigestive disorders
  - f. Fulminant DIC
107. Explain the following concurrent findings.

Case	Platelet concentration	BMBT	PTT	PT
A	↓	WRI	WRI	WRI
B	↓	↑	WRI	WRI
C	WRI	↑	WRI	WRI
D	WRI	WRI	↑	WRI
E	WRI	WRI	WRI	↑
F	↓	WRI	↑	↑
G	↓	↑	↑	↑

108. List diseases, disorders, or conditions that might cause the following.
  - a. Prolonged TT and hypofibrinogenemia
  - b. Prolonged TT and WRI fibrinogen concentration
109. Extra credit material
  - a. MPV: p. 173-175
  - b. Reticulated platelets: p. 177
  - c. PSAIg and related tests: p. 177-178
  - d. vWf: p. 178-181
  - e. Lee-White clotting time: p. 188

- f. INR: p. 191-192
- g. Other specific coagulation factors: p. 193-194
- h. PIVKA: p. 195
- i. RVVT: p. 195
- j. Endogenous anticoagulants: p. 195-197
- k. Thrombosis: p. 210-211

Chapter 6: Bone marrow and lymph node

110. Explain the possible clinical significance of finding the following. That is, when these states are detected, list the types of possible disorders or conditions that are present in the animal.
  - a. Regenerative anemia and erythroid hyperplasia
  - b. Nonregenerative anemia and erythroid hyperplasia
  - c. Nonregenerative anemia and erythroid hypoplasia
  - d. Erythrocytosis and erythroid hyperplasia
  - e. Granulocytic hyperplasia and neutrophilia
  - f. Granulocytic hyperplasia and neutropenia
  - g. Granulocytic hypoplasia and neutropenia
  - h. Megakaryocytic hyperplasia and thrombocytosis
  - i. Megakaryocytic hyperplasia and thrombocytopenia
  - j. Megakaryocytic hypoplasia and thrombocytopenia
111. Define the following: effective erythropoiesis, ineffective erythropoiesis, effective granulopoiesis, ineffective granulopoiesis, pure red cell aplasia, aplastic anemia, agranulocytosis, myelofibrosis, myelitis, myelophthisis, leukemia, myeloproliferative disease, lymphoproliferative disease, acute leukemia, chronic leukemia, myelodysplastic syndrome, polycythemia vera, primary erythrocytosis, thrombocythemia.
112. List and recognize the structural changes in cells that represent dysmyelopoiesis, dyserythropoiesis, or dysthrombopoiesis.
113. List three laboratory methods that can be used to differentiate and identify neoplastic hemic cells.
114. Classify the changes in a marrow if given an animal's Hct, RC, neutrophil concentration, platelet concentration, marrow cellularity, G:E ratio, and characterization of the megakaryocyte pool.
115. Define or match terms with definitions of the following: hyperplastic lymph node, reactive lymph node, lymphadenitis, lymphoid neoplasia, lymphoma, metastatic neoplasia.
116. State the types of lymphadenopathies that are differentiated by cytologic biopsies. For each type, name the major abnormal cell population found AND why the cells are in the enlarged lymph node.
117. If given a detailed description of cells aspirated from an enlarged lymph node, classify the lymphadenopathy.
118. Explain how the results of lymph node and marrow biopsies may help clarify the cause of a lymphocytosis.

Chapter 7: Proteins

119. Given pertinent historical or physical findings and serum or plasma protein concentrations,
  - a. List and classify abnormalities using appropriate terms.

- b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
120. Describe the common analytical methods for serum or plasma proteins (including units of measurement) and explain the purposes or potential values of the analytical procedures. Specifically,
- a. List two methods of determining serum total protein concentration and recognize the major analytic principles of the methods.
  - b. State the common assay that is used to measure serum albumin concentration and recognize its major analytic principle.
  - c. State the common method that is used to determine serum globulin concentration.
  - d. List the two methods of measuring plasma fibrinogen concentration.
121. List and recognize the substances in plasma that can give falsely increased  $[TP_{ref}]$ .
122. List the 2 major processes that produce hyperproteinemia. For each, list the major pathologic states that produce the hyperproteinemia and explain the pathogenesis of the hyperproteinemia.
123. Compare and contrast polyclonal and monoclonal gammopathies including the diseases that produce them and the types of proteins found.
124. Define and describe Bence Jones proteins.
125. List the 4 major processes that produce hypoproteinemia. For each, list the major pathologic states that produce the hypoproteinemia and explain the pathogenesis of the hypoproteinemia.
126. List the 2 processes that produce a true hyperalbuminemia. State a cause of pseudohyperalbuminemia and briefly describe the reason for its occurrence.
127. List the 3 major processes that produce hypoalbuminemia. For each, list the major pathologic states that produce the hypoalbuminemia and explain the pathogenesis of the dysproteinemia.
128. List the 2 processes, and their associated pathologic states, that produce hyperfibrinogenemia.
129. List the 2 processes, and their associated pathologic states, that produce hypofibrinogenemia.
130. List and recognize the categories or types of disorders that cause these findings.
- a. Hyperproteinemia due to a monoclonal gammopathy in either the  $\beta_2$  or  $\gamma$  region
  - b. Hyperproteinemia due to a polyclonal gammopathy
  - c. Hyperproteinemia due to panhyperproteinemia
  - d. Hyperproteinemia due to increased  $\alpha_2$  globulins
  - e. Hyperproteinemia with increased  $\alpha_2$ -globulins, a polyclonal gammopathy and concurrent hypoalbuminemia
  - f. Dysproteinemia with a normal [total protein], hypoalbuminemia, and a monoclonal gammopathy
  - g. Hyperproteinemia with hypoalbuminemia and hyperglobulinemia
  - h. Hyperproteinemia with hyperalbuminemia and hyperglobulinemia
  - i. Dysproteinemia with a normal [total protein], hypoalbuminemia, and hyperglobulinemia
  - j. Hypoproteinemia, hypoalbuminemia, hypoglobulinemia, and a regenerative anemia

- k. Hypoproteinemia, hypoalbuminemia, hypoglobulinemia, and a microcytic hypochromic anemia
  - l. Hypoproteinemia, hypoalbuminemia, and proteinuria
  - m. Hypoproteinemia, hypoalbuminemia, and a small liver
  - n. Hypoproteinemia, hypoalbuminemia, and hypoglobulinemia in a clinically healthy 3-month-old calf
  - o. Hypoproteinemia and hypoalbuminemia in a dog with weight loss and chronic small bowel diarrhea
  - p. Hypoproteinemia and hypoalbuminemia in a cat with a neoplasm
131. Define the following and give one or two examples of each.
- a. Acute phase proteins
  - b. Positive acute phase proteins
  - c. Negative acute phase proteins
  - d. Delayed response proteins
  - e. M-proteins
132. Explain the differences between a selective and a non-selective hypoproteinemia; give examples of each in your explanations. Explain why an animal with a concurrent hypoalbuminemia and hypoglobulinemia may not have a non-selective hypoproteinemia or panhypoproteinemia.
133. Explain why an animal may develop hypoalbuminemia in the following conditions: inflammatory disease, hepatic insufficiency, malabsorption or maldigestion, cachectic states, malnutrition and starvation, marked hyperglobulinemia, external or internal hemorrhage, immune-complex glomerulonephritis or renal amyloidosis, small intestinal disorders, and thermal burn. Of these disorders, state those in which you expect to find concurrent hypoproteinemia.
134. Extra credit material
- a. PP:F or (TP:Fib)<sub>p</sub> ratios (p. 270-271)
  - b. IgG (272-275)
  - c. SPE interpretations (Plate 5; between Chapters 3 & 4)

#### Chapter 8: Urinary system

135. If given serum concentrations of urea or creatinine (increased or decreased), urinalysis results, and pertinent patient information,
- a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
136. Explain the pathogeneses of the following in an animal with chronic renal insufficiency or renal failure.
- a. Azotemia
  - b. USG<sub>ref</sub> near 1.010
  - c. Polyuria
  - d. Oliguria
137. Explain the pathogeneses of the following in an animal in acute renal failure.
- a. Azotemia
  - b. Oliguria

138. List the 2 major processes that produce azotemia. For the one that relates to the urinary system, explain how the 3 types produce azotemia. For each, list major pathologic states that produce azotemia.
139. List the criteria that are used to differentiate pre-renal, renal, and post-renal azotemia; appropriately classify azotemias using these criteria.
140. List the major processes that produce decreased serum urea (or UN) concentrations. List disorders that produce low serum urea concentrations via those processes.
141. Compare and contrast the pathophysiologic processes that may result in concurrent increases in serum urea and creatinine concentrations or increases in one but not the other.
142. Explain what a decreased creatinine clearance tells us about an animal. Explain why it does or does not help differentiate pre-renal, renal, or post-renal disorders.
143. Explain why concentrations or activities of the following serum analytes may become abnormal if a decreased GFR is caused by acute or chronic renal diseases (note major species differences): UN, creatinine, inorganic phosphorus, total calcium, potassium, hydrogen, amylase, lipase.
144. If given an animal's hydration status and its  $USG_{ref}$  value, state if the  $USG_{ref}$  is an expected physiologic response or a pathologic state and explain the basis of your conclusion.
145. Explain why  $USG_{ref}$  is typically an excellent method of estimating urine solute concentration. List the situations when it is not.
146. Explain the clinical significance of the following urine abnormalities (i.e., what do the data tell you about the animal?):
  - a. Appearance: cloudy, red, orange, brown, black
  - b. Chemical features: aciduria, alkalinuria, proteinuria, glucosuria, bilirubinuria, ketonuria, positive heme reaction
  - c. Sediment findings: pyuria, hematuria, cylindruria, bacteriuria, crystalluria (phosphate or struvite, calcium oxalate dihydrate, calcium oxalate monohydrate, calcium carbonate, ammonium biurate, urate, bilirubin).

For each, explain the possible pathogenesis(es) of the abnormality and state disorders (diseases, pathophysiologic states) which could cause the abnormality.
147. Explain the conditions and name the animal in which the detection of protein and bilirubin in urine are not considered abnormal findings.
148. For each of the semi-quantitative chemical procedures on a UA reagent strip, state the substance(s) that the assay is designed to detect.
149. Recognize which of the following proteins (or protein groups) are detected by the urine protein reagent-strip assay (Ames type): albumin,  $\alpha$ -globulins,  $\beta$ -globulin,  $\gamma$ -globulins, kappa or lambda chains, hemoglobin. State why the reagent-strip assay does not accurately measure the concentrations of all urine proteins.
150. Explain the possible advantage(s) of the sulfosalicylic acid procedure over the reagent pad method.
151. State the pathologic states which might give an increased urinary protein:creatinine ratio and explain the pathogenesis of the proteinuria for each state.
152. Explain why a urine protein concentration of 50 mg/dL may be strong evidence of a proteinuria in one dog, but in another case be considered an expected finding in a healthy dog.
153. List the 4 types of proteinuria. Given appropriate data, differentiate them.

154. List the 2 types of glucosuria. Given appropriate data, differentiate them.
155. List the 3 types of positive heme reaction. Given appropriate data, differentiate them.
156. List the 2 basic pathophysiologic processes that produce bilirubinuria. Given appropriate data, differentiate them.
157. Using urine and serum colors, an animal's Hct, and results of a urine sediment examination, differentiate hematuria, hemoglobinuria, and myoglobinuria.
158. Explain the pathogenesises of polyuria in the following disorders: renal insufficiency or renal failure, diabetes insipidus, renal diabetes insipidus, diabetes mellitus, hyperadrenocorticism, hypoadrenocorticism, hypercalcemia, hypokalemia, canine pyometra, hepatic insufficiency or failure, psychogenic polydipsia.
159. Explain why animals in renal failure (acute or chronic) or with hypoadrenocorticism can be either polyuric or oliguric.
160. Explain how it is possible that the following can occur in animals with renal disease.
  - a. Decreased creatinine clearance and serum [Crt] WRI
  - b. Glomerular proteinuria without azotemia
  - c. Azotemia without glomerular proteinuria
161. Extra credit material
  - a. UN:Crt ratio (p. 296)
  - b. USG by reagent strip (p. 304)
  - c. Quantitative urinalysis (p. 326-332 except the (Prot:Crt)<sub>u</sub> ratio)
  - d. H<sub>2</sub>O deprivation and antidiuretic hormone (p. 332-334)

#### Chapter 9: Monovalent electrolytes and osmolality

162. If given serum electrolyte concentrations, osmolality, and other relevant data (CBC, serum chemistry results, urinalysis results, and patient information),
  - a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
163. State the specific analytes that are measured to determine serum concentrations of sodium, potassium, chloride, and total carbon dioxide content. Explain the relationship between serum bicarbonate concentration and total carbon dioxide content.
164. Using the ratio between total body Na<sup>+</sup> and total body H<sub>2</sub>O (tbNa<sup>+</sup>:tbH<sub>2</sub>O ratio), explain the pathophysiologic events which cause hypernatremia, normonatremia, or hyponatremia in: a) a dehydrated animal; b) an animal with normal hydration; c) an edematous animal.
165. List the two basic processes that produce hypernatremia.
166. Explain or recognize the pathogenesis of the hypernatremia that may be found in the following conditions.
  - a. Frozen water source
  - b. Prolonged fever
  - c. Central diabetes insipidus
  - d. Ruminant acidosis (grain overload)
  - e. Salt poisoning
167. List and recognize the two basic processes that produce a pathologic normonatremia.
168. Explain or recognize the pathogenesis of the normonatremia that may be found in the following conditions.

- a. Vomiting or diarrhea
  - b. Renal disease
  - c. Osmotic diuresis
  - d. Furosemide or thiazide diuretics
  - e. Congestive heart failure
169. Compare the edematous states that occur with hepatic cirrhosis and nephrotic syndrome; what do the disorders have in common regarding regulation of  $\text{Na}^+$  and  $\text{H}_2\text{O}$  balance?
170. List and recognize the five basic processes that produce hyponatremia.
171. List and recognize the conditions that may produce a pseudohyponatremia (or pseudohypochloremia) and which analytical methods are or are not affected by such conditions.
172. Explain and recognize the pathogenesis of the hyponatremia that may be found in the following conditions.
- a. Vomiting or diarrhea
  - b. Hypoadrenocorticism
  - c. Ketonuria
  - d. Sweating in horses
  - e. Congestive heart failure
  - f. Hyperglycemia (with or without ketonuria)
  - g. Uroperitoneum
173. List and recognize the two basic processes that produce hyperkalemia.
174. Explain and recognize the pathogenesis of the hyperkalemia that may be found in the following conditions.
- a. Inorganic metabolic acidosis
  - b. Rhabdomyolysis
  - c. Renal insufficiency or failure
  - d. Urinary tract obstruction
  - e. Hypoadrenocorticism
175. Explain why *in vitro* hemolysis may cause hyperkalemia in some animals, but not in others.
176. Explain why  $[\text{K}^+]$  is typically greater in serum than in plasma. In what situation does this process result in a pseudohyperkalemia?
177. List and recognize the two basic processes that produce hypokalemia.
178. Explain and recognize the pathogenesis of the hypokalemia that may be found in the following conditions.
- a. Metabolic alkalosis
  - b. Anorexia
  - c. Osmotic diuresis
  - d. Ketonuria
  - e. Conditions that cause hypochloremic metabolic alkalosis
  - f. Diarrhea
  - g. Choke or dysphagia
179. Explain and recognize the reason for a decreased serum  $\text{Na}^+:\text{K}^+$  ratio in the following conditions.
- a. Hypoadrenocorticism
  - b. Renal failure
  - c. Uroperitoneum
  - d. Diabetes mellitus

- e. Repeated drainage of chylous thoracic effusions
180. List or recognize the four basic processes or pathologic states that produce hyperchloremia.
181. Explain and recognize the pathogenesis of the hyperchloremia that may be found in the following conditions.
- a. Frozen water source
  - b. Prolonged fever
  - c. Central diabetes insipidus
  - d. Ruminant acidosis (grain overload)
  - e. Salt poisoning
  - f. Bovine esophageal obstruction that causes a metabolic acidosis
182. Explain why the presence of a normochloremia and concurrent metabolic acidosis suggests or indicates an increased anion gap.
183. List and recognize the four basic processes or pathologic states that produce hypochloremia.
184. Explain and recognize the pathogenesis of the hypochloremia that may be found in the following conditions.
- a. Vomiting or diarrhea that produces hyponatremia
  - b. Hypoadrenocorticism
  - c. Persistent vomiting in monogastric mammals or displaced abomasum in ruminants
  - d. Ketoacidosis or lactic acidosis
  - e. Sweating in horses
  - f. Congestive heart failure
  - g. Hyperglycemia (with or without ketonuria)
  - h. Uroperitoneum
185. List and recognize the four major processes that produce an increased  $[\text{HCO}_3^-]$  or  $[\text{tCO}_2]$ .
186. Explain and recognize the pathogenesis of an increased  $[\text{HCO}_3^-]$  that may be found in the following conditions.
- a. Gastric or abomasal loss of  $\text{H}^+$
  - b. Loop or thiazide diuretics
  - c. Hypokalemia
  - d. Contraction alkalosis
187. List and recognize the four major processes that produce a decreased  $[\text{HCO}_3^-]$  or  $[\text{tCO}_2]$ .
188. Explain and recognize the pathogenesis of a decreased  $[\text{HCO}_3^-]$  that may be found in the following conditions.
- a. Lactic acidosis
  - b. Ketoacidosis
  - c. Renal failure
  - d. Uroperitoneum
  - e. Diarrhea
189. Explain the relationships between the following.
- a. Total body  $\text{Na}^+$  content and total body  $\text{H}_2\text{O}$  content
  - b. Serum  $[\text{K}^+]$  and acid-base status
  - c. Serum  $[\text{K}^+]$  and total body  $\text{K}^+$
  - d. Serum  $[\text{Cl}^-]$  and serum  $[\text{Na}^+]$ :
  - e. Serum  $[\text{Cl}^-]$  and serum  $[\text{HCO}_3^-]$
  - f. Serum  $[\text{Na}^+]$  and serum  $[\text{K}^+]$

190. If given necessary measured values:
  - a. Calculate an anion gap.
  - b. Explain the clinical significance of a normal or increased anion gap values (i.e., what does the anion gap value tell you about the animal?).
  - c. List or recognize common disorders that might cause an increased or decreased anion gap.
191. State the serum analyte that is the major contributor to the anion gap in a healthy mammal. State what would happen to the anion gap if the concentration of that analyte decreased.
192. Explain and recognize why animals with the following disorders have an increased anion gap.
  - a. Metabolic acidosis and concurrent hypochloremia
  - b. Metabolic acidosis and concurrent normochloremia
193. Define and recognize the definitions of osmolality, osmole, solute, solvent, and osmo. gap.
194. Briefly explain how a freezing-point osmometer measures a sample's osmolality.
195. State the relative contribution of electrolytes ( $\text{Na}^+$ ,  $\text{K}^+$ ,  $\text{Cl}^-$ ,  $\text{HCO}_3^-$ ) to total osmolality in serum of healthy animals compared to the contributions of urea, glucose, and proteins.
196. List the endogenous solutes which can cause an increased serum osmolality (e.g., > 10 mosm/kg above reference interval)
197. List the endogenous solutes which can cause a decreased serum osmolality (e.g., < 10 mosm/kg below reference interval)
198. Explain the difference between serum osmolality and effective serum osmolality. Explain how effective serum osmolality can be estimated from measured values of osmolality and certain solute concentrations.
199. If given necessary measured values,
  - a. Calculate an osmo. gap.
  - b. Explain the clinical significance of either normal or increased osmo. gap values (i.e., what does the osmo. gap value tell you about the animal?).
  - c. List common disorders that might cause an increased osmo. gap.
200. Compare and contrast the interpretations of a measured osmolality versus a calculated osmolality. When do increased or decreased values for both mean the same thing? When don't they? When is a calculated osmolality useful?
201. Explain why a dehydrated animal might have:
  - a. An increased serum osmolality
  - b. A serum osmolality WRI
  - c. A decreased serum osmolality
202. If given serum electrolyte concentrations and other relevant data (CBC, serum chemistry results, urinalysis results, and patient information), recognize data which are consistent with these disorders or conditions.
  - a. Hypoadrenocorticism
  - b. Dehydration (hypertonic, isotonic, or hypotonic)
  - c. Gastric loss of  $\text{H}^+$  that produces a hypochloremic metabolic alkalosis
  - d. Intestinal disease that produces a metabolic acidosis
  - e. Ketoacidosis
  - f. Lactic acidosis
  - g. Renal failure
  - h. Cirrhosis

- i. Nephrotic syndrome
- j. Diabetes mellitus
- k. Uroperitoneum
- l. Renal tubular acidosis (proximal or distal)

Chapter 10: Blood gases, blood pH, and strong ion difference

203. If given an animal's values for serum  $[\text{Na}^+]$ , serum  $[\text{K}^+]$ , serum  $[\text{Cl}^-]$ , serum  $[\text{HCO}_3^-]$  (or  $\text{tCO}_2$  content), blood gas & pH, serum osmolality, and other supportive information or laboratory data,
- a. List and classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
204. Explain the abnormalities in  $\text{PCO}_2$ ,  $\text{PO}_2$ , and pH blood values that will occur if a sample is exposed to air or excess heparin or if there is delayed sample analysis.
205. Of the following analytes, recognize which are measured by blood gas and pH instruments; which are calculated from the measured results: pH,  $\text{PO}_2$ ,  $\text{PCO}_2$ ,  $[\text{tCO}_2]$ ,  $[\text{HCO}_3^-]$ , BE,  $\text{SO}_2$ .
206. Explain the pathogenesis of the:
- a. Acidemia that occurs in metabolic or respiratory acidoses
  - b. Alkalemia that occurs in metabolic or respiratory alkaloses
207. List the major processes of the non-respiratory system that produce:
- a. A decreased blood pH
  - b. An increased blood pH
- For each, list and recognize the major conditions or pathologic states that produce the changes. (Note: see electrolyte chapter)
208. List the major processes of the respiratory system that produce an increased blood  $\text{PCO}_2$  or decreased blood  $\text{PCO}_2$ . For each, list or recognize the major conditions or pathologic states that produce the changes.
209. List the major processes of the respiratory system that produce a decreased blood  $\text{PO}_2$ . For each, list or recognize the major conditions or pathologic states that produce the changes.
210. Recognize or explain why anemia may cause hypoxia but not hypoxemia.
211. Recognize or explain how the  $\text{SO}_2$  determination by a blood gas machine and a  $\text{SpO}_2$  from a pulse oximeter are different. Recognize when and explain why the  $\text{SpO}_2$  may remain near reference intervals but the true oxygen saturation is severely decreased.
212. If given an animal's values for serum  $[\text{Na}^+]$ , serum  $[\text{K}^+]$ , serum  $[\text{Cl}^-]$ , serum  $[\text{HCO}_3^-]$  (or  $\text{tCO}_2$  content), blood gas & pH, serum osmolality and other supportive information or laboratory data,
- a. Recognize data which are consistent with hypoadrenocorticism, salt-losing (Na-losing) nephropathy, dehydration (hypertonic, isotonic, or hypotonic), diarrheas, edematous states, diabetes mellitus with or without ketonuria, respiratory acidoses and alkaloses, metabolic acidoses and alkaloses, ketoacidosis, lactic acidosis, ethylene glycol toxicosis, upper GI obstruction in ruminants, renal insufficiency or failure.
  - b. For these pathologic states, explain the pathogenesis of the abnormal laboratory data.

213. Extra credit material
  - a. Temperature correction of blood gas results (p. 388)
  - b. Pulse oximetry (p. 388-389)
  - c. SID (p. 393-398)

Chapter 11: Calcium, phosphorus, magnesium, and their regulatory hormones

214. If given serum concentrations of calcium and phosphorus and other supporting information and pertinent patient information:
  - a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
215. If routine laboratory assays are used, state what is represented by measured serum values for calcium, phosphorus (or phosphate), magnesium, PTH, PTHrp, and vitamin D.
216. Explain why serum protein (esp. albumin) concentrations should be known when you are interpreting a serum calcium concentration.
217. List the three major processes that produce hypercalcemia.
218. Explain and recognize the pathogenesis of hypercalcemia that may be found in the following conditions.
  - a. Primary hyperparathyroidism
  - b. HHM
  - c. Rodenticide toxicosis
  - d. Systemic fungal infections
  - e. Equine renal failure
  - f. Canine hypoadrenocorticism
219. List the five major processes that produce hypocalcemia.
220. Explain and recognize the pathogenesis of hypocalcemia that may be found in the following conditions.
  - a. Hypoalbuminemia
  - b. Primary hypoparathyroidism
  - c. Chronic renal disease in dogs, cats, and cattle
  - d. Pregnancy, parturient, or lactational hypocalcemia
  - e. Hyperphosphatemia
221. Explain and recognize the potential consequences of the following on serum, plasma, or blood [ $\text{fCa}^{2+}$ ].
  - a. Aerobic handling of serum, plasma, or blood sample
  - b. Delayed processing or handling of blood sample
  - c. Excess heparin in blood sample or its subsequent plasma sample
  - d. Collecting sample in an EDTA tube
222. Explain why and recognize that a [ $\text{fCa}^{2+}$ ] may be useful in the following conditions.
  - a. Concurrent hypocalcemia and hypoalbuminemia
  - b. Concurrent hypercalcemia and renal failure or multiple myeloma
  - c. Horses after an endurance or cross-country race
223. List the three major processes that produce hyperphosphatemia.
224. Explain or recognize the pathogenesis of a hyperphosphatemia that may be found in the following conditions:
  - a. Azotemic animals

- b. Hypoparathyroidism
  - c. Acromegaly
  - d. Myopathies or extensive tissue necrosis
  - e. *In vitro* hemolysis
225. List the four major processes that produce hypophosphatemia.
226. Explain and recognize the pathogenesis of hypophosphatemia that may be found in the following conditions:
- a. Hyperparathyroidism
  - b. Anorexia
  - c. Hyperinsulinism
  - d. Milk fever
227. List the three major processes that produce hypermagnesemia.
228. Explain and recognize the pathogenesis of hypermagnesemia that may be found in the following conditions:
- a. Renal failure
  - b. Milk fever
  - c. *In vitro* hemolysis
229. List the three major processes that produce hypomagnesemia.
230. Explain and recognize the pathogenesis of hypomagnesemia that may be found in the following conditions:
- a. Hypoproteinemia
  - b. Prolonged anorexia
  - c. Grass tetany
  - d. Osmotic diuresis
  - e. Ketonuria
  - f. Metabolic alkalosis
231. Explain and recognize the pathogenesis of increased [PTH] that may be found in the following conditions:
- a. Primary hyperparathyroidism
  - b. Chronic renal disease
  - c. Diet with low  $\text{Ca}^{2+}:\text{PO}_4$  ratio
232. Explain and recognize the pathogenesis of decreased [PTH] that may be found in the following conditions:
- a. Hypoparathyroidism
  - b. Hypervitaminosis D
  - c. HHM
233. If given appropriate values for serum  $[\text{tCa}^{2+}]$ ,  $[\text{Pi}]$ ,  $[\text{Mg}^{2+}]$  and other supportive information or laboratory data, recognize data which are consistent with primary hyperparathyroidism, hypoparathyroidism, humoral hypercalcemia of malignancy, systemic fungal infections, hypoalbuminemia, renal insufficiency or failure (in different species), eclampsia or milk fever, growing animals, *in vitro* hemolysis, prolonged anorexia in carnivores, hypervitaminosis D, and hypoadrenocorticism.
234. Extra credit material
- a. Vitamin D section (p. 426-427)
  - b. Calcitonin section (p. 427-428)

Chapter 12: Enzymes

235. If given appropriate values for serum enzyme activities (e.g., ALT, AST, LD, ID, ALP, GGT, CK, AMS, LPS) and other supportive information or laboratory data,
  - a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
236. List and recognize the major cellular sources of the common serum enzymes.
237. State and recognize the major mechanisms that lead to increased activities of each of the common serum enzymes.
238. Explain the three basic pathophysiologic mechanisms that lead to increased serum enzyme activity.
239. Explain and recognize how a serum enzyme's half-life influences our interpretation of laboratory data from one serum sample.
240. Explain why a serum ALT value that is 10xURL may be due to reversible or irreversible hepatocyte damage.
241. Explain how the analytical methods for serum enzymes are different from most of the clinical chemistry assays we have discussed to date (hint: what is measured?).
242. Recognize and state how different assay conditions (pH, substrate, temperature) affect enzymatic assay results.
243. Explain why it is typically best that serum enzyme activity be measured in a fresh and not stored sample.
244. Explain why a serum ALP activity that is 2xURL may actually be a 10-fold increase for that animal.
245. For each of the following serum enzymes, state the major tissue sources of the enzyme and the major mechanisms or processes that lead to increased serum enzyme activity.
  - a. ALT in dogs and cats
  - b. AST in domestic animals
  - c. LD in domestic animals
  - d. ID in domestic animals
  - e. ALP in dogs and cats
  - f. GGT in domestic animals
  - g. CK in domestic animals
  - h. AMS & LPS in dogs
246. Compare and contrast the processes in dogs that produce increased serum ALP activity in cholestasis, after steroid treatments, and after phenobarbital treatments.
247. Explain and recognize the diagnostic differences between total serum lipase activity and pancreatic lipase immunoreactivity (not in FVCP).
248. Extra credit
  - a. GGT activity in urine (p 451-2)
  - b. Other serum enzymes (p. 456)

### Chapter 13: Liver function

249. If given appropriate values for assays that indicate decreased hepatic function (e.g., serum [bilirubin], [bile acid], plasma [ammonium]) and other supportive information or laboratory data,
  - a. List or classify abnormalities using appropriate terms.

- b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
250. Explain the similarities and differences between hepatocellular disease, biliary disease, and hepatic insufficiency. Identify laboratory data that indicate or suggest the presence of each.
251. Explain the pathogenesis of the following in animals with liver disease (in your explanations, describe how liver disease causes the abnormality).
- a. CBC results: anemia, codocytosis, acanthocytosis, microcytosis
  - b. Chemistry results
    - 1) hypercholesterolemia, hyperglycemia, increased hepatic enzymes (ALT, AST, etc), lipemia
    - 2) decreased serum [UN], hypoglycemia, hypoproteinemia, hypoalbuminemia, hypocholesterolemia
  - c. Urinalysis results: lower than expected USG<sub>ref</sub>, bilirubinuria, ammonium biurate crystalluria, urate crystalluria
  - d. Coagulation assays: prolonged PTT, prolonged PT
252. List and recognize the appropriate methods of handling and processing samples for the analysis of bilirubin, bile acid, and ammonium concentrations.
253. State the form(s) of bilirubin which is/are represented by the following terms: total bilirubin, unconjugated bilirubin, conjugated bilirubin, direct bilirubin, indirect bilirubin, and delta bilirubin.
254. List the five major processes that produce hyperbilirubinemia.
255. Explain or recognize the pathogenesis of hyperbilirubinemia that may be found in the following conditions.
- a. Hemolytic disorders, especially extravascular hemolysis
  - b. Fasting or anorexia in horses
  - c. Obstructive cholestasis
  - d. Functional cholestasis
256. Compare and contrast obstructive cholestasis and functional cholestasis. Which laboratory test results may be increased when they are present? Why?
257. List the two major processes that produce an increased fasting [BA].
258. Explain and recognize the pathogenesis of increased [BA] that may be found with the following.
- a. Diffuse hepatocellular disease such as cirrhosis
  - b. Portosystemic shunts
  - c. Obstructive cholestasis
  - d. Functional cholestasis
  - e. Postprandial sample
259. Explain why a bile acid challenge test may be a better assessment of hepatic function than a fasting [BA].
260. List the three major processes that produce hyperammonemia.
261. Explain and recognize the pathogenesis of hyperammonemia that may be found with the following.
- a. Diffuse hepatocellular disease
  - b. Portosystemic shunts
  - c. Postprandial sample

- d. Urea toxicosis in cattle
- 262. Explain why an ammonium tolerance test has better diagnostic sensitivity for hepatic insufficiency than a fasting ammonium concentration.
- 263. Explain why the following are not recommended.
  - a. Serum bile acid concentration in an icteric animal that is not anemic or has a very mild anemia.
  - b. Plasma ammonium concentration for a sample that must be shipped to a reference laboratory.
- 264. By using results from the analysis of blood, serum, or urine, explain how we can differentiate hyperbilirubinemias due to hemolysis from those due to cholestasis (or anorexia in horses).
- 265. Interpret the following information.
  - a. For dogs
    - 1) Serum ALP activity of 10xURL and serum bilirubin WRI
    - 2) Serum ALP activity of 10xURL and hyperbilirubinemia
    - 3) Serum ALP activity WRI and hyperbilirubinemia
  - b. For cats
    - 1) Serum ALP activity of 3xURL and serum bilirubin WRI
    - 2) Serum ALP activity of 3xURL and hyperbilirubinemia
    - 3) Serum ALP activity WRI and hyperbilirubinemia
  - c. For horses
    - 1) Serum GGT activity of 3xURL and a serum bilirubin WRI
    - 2) Serum GGT activity of 3xURL and a hyperbilirubinemia
    - 3) Serum GGT activity WRI and hyperbilirubinemia
- 266. Extra credit
  - a. Icterus index (p 475)
  - b. Dye excretion tests (p. 484)

#### Chapter 14: Glucose and related regulatory hormones

- 267. If given appropriate measured values for [glucose] and [IRI] and other supportive information or laboratory data,
  - a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
- 268. Explain how poor sample handling can cause erroneous glucose values.
- 269. Explain and recognize why a blood glucose concentration does not equal a plasma/serum glucose concentration.
- 270. List the three major processes that produce hyperglycemia.
- 271. Explain and recognize the pathogenesis of a hyperglycemia that may be found with the following conditions or situations.
  - a. Blood sample collected soon after animal ate
  - b. Excitement or fright
  - c. Glucocorticoid-associated 'stress'
  - d.  $\beta$ -cell destruction in dogs
  - e. Pancreatic amyloidosis in cats

- f. Acute pancreatitis
  - g. Hyperadrenocorticism
  - h. Equine hyperpituitarism
  - i. Bovine BVD infection
  - j. Pharmacologic: intravenous glucose, glucocorticoids, ketamine, xylazine or detomidine, excess injected insulin, progestins including megestrol acetate
272. List the five major pathologic processes that produce hypoglycemia.
273. Explain and recognize the pathogenesis of hypoglycemia that may be found in the following conditions.
- a. Pancreatic  $\beta$ -cell neoplasia
  - b. Hypoadrenocorticism
  - c. Hepatic insufficiency
  - d. Lactational hypoglycemia (bovine ketosis)
274. Explain and recognize:
- a. Why an animal's serum [glucose] should be known when you are interpreting an [IRI].
  - b. Why published reference intervals for [IRI] or IRI:G ratios may not be satisfactory to interpret [IRI] or IRI:G ratios in your patient.
275. Extra credit
- a. Immunoreactive glucagon section (p. 502-503)
  - b. Amended insulin:glucose ratio (p. 501-2)

#### Chapter 15: Exocrine pancreas and intestine

276. If given appropriate values for serum TLI, cobalamin, folate, or xylose concentrations,
- a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
277. List and recognize the appropriate methods of handling and processing samples for the analysis of TLI, folate, and cobalamin concentrations.
278. List the two major processes that produce increased serum TLI concentration.
279. Explain and recognize the pathogenesis of increased [TLI] that may be found in the following conditions.
- a. Pancreatic disease
  - b. Physiologic processes
  - c. Renal disease
  - d. Dehydration
280. List the process that produces a decreased serum TLI concentration.
281. Explain and recognize the pathogenesis of decreased [TLI] that may be found in pancreatic disease.
282. Explain and recognize the diagnostic differences between a [TLI] concentration and pancreatic lipase immunoreactivity (not in FVCP).
283. List the two major processes that produce decreased serum cobalamin concentration.
284. Explain and recognize the pathogenesis of decreased serum cobalamin concentration that may be found in the following conditions.
- a. EPI

- b. Intestinal bacterial overgrowth
  - c. Inherited defect in giant schnauzers and border collies
285. List the two major processes that produce increased serum folate concentration.
286. Explain and recognize the pathogenesis of increased serum folate concentration that may be found in the following conditions.
- a. EPI
  - b. Intestinal bacterial overgrowth
  - c. Parenteral supplementation
287. List the major process that produces decreased serum folate concentration.
288. Explain and recognize the pathogenesis of decreased serum folate concentration that may be found in the following conditions.
- a. Intestinal disease
  - b. Packer fan
289. Explain how cobalamin deficiency may produce a clinical disorder that appears to be due to a folate deficiency but the serum folate concentration is WRI.
290. Explain the reasons (*processes*) that produce a flat xylose absorption curve.
291. If given appropriate values for serum TLI, cobalamin, folate, or xylose concentrations, and other supportive information or laboratory data, recognize data that are consistent with:
- a. EPI
  - b. Proximal small intestinal mucosal disease
  - c. Distal small intestinal mucosal disease
  - d. Small intestine bacterial overgrowth
  - e. Acute pancreatitis
  - f. Decreased GFR
  - g. Parenteral administration of cobalamin or folate
292. Extra credit: other methods of evaluating digestive or absorptive functions (p. 517-8)

#### Chapter 16: Lipids

293. If given serum concentrations of cholesterol or triglyceride and pertinent patient information,
- a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
294. List the two major processes that produce hypercholesterolemia.
295. List the two major processes that produce hypertriglyceridemia.
296. Explain or recognize the pathogenesis of hypercholesterolemia or hypertriglyceridemia that may be found with the following.
- a. Postprandial sample
  - b. Acute pancreatitis
  - c. Nephrotic syndrome or protein-losing nephropathy
  - d. Hypothyroidism
  - e. Lipoprotein lipase deficiency
  - f. Cholestasis
  - g. Diabetes mellitus
  - h. Hyperadrenocorticism

- i. Primary versus secondary hyperlipidemia
  - j. Multiple conditions of equine hyperlipidemia: anorexia, pregnancy and lactation
297. List the major process that produces hypocholesterolemia.
298. Explain and recognize the pathogenesis of hypocholesterolemia that may be found in an animal with a portosystemic shunt.
299. Explain the relationships (if any) between the following:
- a. Presence or absence of plasma turbidity versus serum triglyceride concentration
  - b. Presence or absence of plasma turbidity versus serum cholesterol concentration
300. Explain why triglyceride concentrations in chylous effusions are much greater than serum triglyceride concentrations. Explain why a cholesterol:triglyceride ratio is much less in chylous effusions than in non-chylous effusions.

### Chapter 17: Thyroid function

301. If given appropriate canine values for serum  $tT_4$ ,  $fT_4$ , TSH, TgAA concentrations, and pertinent patient information,
- a. List or classify abnormalities using appropriate terms.
  - b. Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - c. Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
302. When considering the analysis of serum for thyroxine concentrations,
- a. Explain why a RIA procedure produced to measure human thyroxine concentrations is not acceptable for canine serum.
  - b. Explain why a RIA procedure may generate a  $[tT_4]$  that is much greater than the true concentration.
303. Explain why an assay designed to measure human  $[TSH]$  will not provide valid results for domestic mammal samples.
304. List the two major processes that produce hyperthyroxemia.
305. Explain and recognize the pathogenesis of a hyperthyroxemia or increased  $[fT_4]$  that may be found in thyroid neoplasia (benign and malignant).
306. List the major process that produces hypothyroxemia.
307. Explain and recognize the pathogenesis of hypothyroxemia or decreased  $[fT_4]$  that may be found with the following.
- a. Primary hypothyroidism (lymphocytic thyroiditis)
  - b. Secondary hypothyroidism
  - c. Non-thyroidal disease or administration of certain drugs
308. Explain or recognize the pathogenesis of an increased  $[TSH]$  that may be found in the following conditions.
- a. Hypothyroidism
  - b. Euthyroid lymphocytic thyroiditis
309. Explain why a  $[TSH]$  in a dog with secondary hypothyroidism is WRI.
310. Sick euthyroidism is a common state in which non-thyroidal disease results in hypothyroxemia and there may be multiple mechanisms that produce the hypothyroxemia. Predict if  $tT_4$ ,  $fT_4$ , or TSH concentrations will change if the following pathologic process is occurring.
- a. Decreased protein binding of  $T_4$
  - b. Inhibition of TSH secretion
  - c. Inhibition of  $T_4$  production

311. If given appropriate canine values for serum  $tT_4$ ,  $fT_4$ , TSH, and TgAA concentrations, recognize data that are consistent with:
- Primary hypothyroidism due to thyroiditis
  - Primary hypothyroidism (idiopathic)
  - Secondary hypothyroidism
  - Sick euthyroidism
  - Thyroiditis without thyroid gland dysfunction

312. If given appropriate feline values for serum  $tT_4$ ,  $fT_4$ , or a  $T_3$ -suppression test and basic clinical information, recognize data that are consistent with:

- Hyperthyroidism
- Euthyroidism
- Hypothyroidism

313. Explain the results of the following  $T_3$ -suppression tests.

	Analyte	Units	Cat 1	Cat 2	Cat 3	Ref. Int.
Pre- $T_3$ sample	$tT_4$	nmol/L	35	30	25	15 - 40
Post- $T_3$ sample	$tT_4$	nmol/L	5	26	24	< 20
Pre- $T_3$ sample	$tT_3$	nmol/L	1.0	2.0	1.8	0.3 - 2.3
Post- $T_3$ sample	$tT_3$	nmol/L	2.5	1.8	4.0	> 2.0

- Cat 1:
- Cat 2:
- Cat 3:

#### Chapter 18: Adrenocortical function

314. If given values for serum cortisol concentrations from LDDST, HDDST, or ACTH stimulation test and basic clinical information,
- List or classify abnormalities using appropriate terms.
  - Propose appropriate ideas or conclusions (i.e., diseases, syndromes, or pathologic states) that might cause the defined abnormalities.
  - Based on your conclusions or ideas, explain the pathogenesis of each defined abnormality if the abnormality could be caused by the disorder.
315. List the major process that produces hypercortisolemia.
316. Explain and recognize the pathogenesis of hypercortisolemia that may be found in the following conditions.
- PDH
  - FAN
  - 'Stress'
317. List the major process that produces hypocortisolemia.
318. Explain and recognize the pathogenesis of hypocortisolemia that may be found in the following conditions.
- Primary hypoadrenocorticism
  - Secondary hypoadrenocorticism
  - Iatrogenic hyperadrenocorticism
  - Iatrogenic hypoadrenocorticism
319. Based on your understanding of physiologic and pathologic conditions involving the adrenal cortices, explain why the  $(Cort:Cr)_u$  has a:
- High diagnostic sensitivity for primary hyperadrenocorticism
  - Low diagnostic specificity for primary hyperadrenocorticism

320. Explain and recognize the pathogenesis of increased or decreased [ACTH] in the following conditions:
  - a. PDH
  - b. FAN
  - c. Primary hypoadrenocorticism
  - d. Secondary hypoadrenocorticism
  - e. Iatrogenic hyperadrenocorticism
  - f. Iatrogenic hypoadrenocorticism
321. Explain and recognize the pathogenesis of increased or decreased aldosterone concentration in the following conditions:
  - a. Primary hypoadrenocorticism
  - b. Iatrogenic hyperadrenocorticism
  - c. Renal failure
  - d. Heart failure
  - e. Diabetes mellitus
322. If given values for serum cortisol concentrations from LDDST, HDDST, or ACTH stimulation test and basic clinical information, recognize data that are consistent with:
  - a. PDH
  - b. FAN
  - c. 'Stress'-induced changes
  - d. Primary hypoadrenocorticism
  - e. Secondary hypoadrenocorticism
  - f. Iatrogenic hyperadrenocorticism
  - g. Iatrogenic hypoadrenocorticism
323. Interpret the following findings for a dog that has clinical signs suggestive of hyperadrenocorticism:
  - a. Increased  $(\text{Cort/Crt})_u$  ratio
  - b.  $(\text{Cort/Crt})_u$  ratio WRI
324. Explain the physiologic reasons that dexamethasone suppression tests in cats and horses have different schedules from those of dogs.

## **Material not in *Fundamentals of Veterinary Clinical Pathology, 2002***

### **Pleural and peritoneal fluid analysis**

325. If given the results of a pleural or peritoneal fluid analysis and other pertinent clinical information, use appropriate terms to classify the effusion as a pure transudate, exudate, septic exudate, non-septic exudate, heart failure effusion, hemorrhagic effusion, chylous effusion, or neoplastic effusion.
326. Describe each component of a routine pleural or peritoneal fluid analysis (including units of measurement) and the purposes or potential value of each analytical procedure.
327. Based on your understanding of inflammatory reactions, explain the difference between peritoneal or pleural effusions that contains many neutrophils versus ones that contains many macrophages.
328. Explain and recognize an advantage and a disadvantage of collecting pleural or peritoneal fluid into an EDTA-containing tube.
329. Explain the pathogeneses (how or why) of each of the following and propose and recognize disorders (diseases, pathologic states) which might cause them: pure

- transudate, exudate, septic exudate, non-septic exudate, heart failure effusion, hemorrhagic effusion, chylous effusion, or neoplastic effusion.
330. Explain why pleural or peritoneal fluid should be submitted to attempt to culture microorganisms if:
- Organisms are seen in a microscopic examination
  - Organisms are not seen in a microscopic examination
331. Compare and contrast the morphologic features of degenerate and nondegenerate neutrophils and their diagnostic significance.
332. Explain why the microscopic evaluation of pleural and peritoneal fluid is an important component of the analysis of effusions; give examples to justify your answer.

### **Synovia analysis**

333. If given the results of a synovia analysis and other pertinent clinical information, use appropriate terms to classify the synovia as a neutrophilic (suppurative, purulent) or mononuclear (nonsuppurative, nonpurulent) inflammatory arthropathy.
334. Describe each component of routine synovia analysis (including units of measurement) and the purposes or potential value of each analytical procedure.
335. Explain why or how the viscosity of synovia interferes with the analysis of synovia and what can be done to synovia to reduce its viscosity.
336. Explain and recognize what a LE cell contains and what a ragocyte contains.
337. Based on your understanding of inflammatory reactions, explain the difference between synovia that contains many neutrophils versus synovia that contain many macrophages.
338. Explain and recognize an advantage and a disadvantage of collecting synovia into an EDTA-containing tube.
339. Explain the pathogenesis (how or why) of each of the following and propose and recognize disorders (diseases, pathologic states) which might cause the following:
- Neutrophilic (suppurative, purulent) inflammatory arthropathy
  - Mononuclear (nonsuppurative, nonpurulent) arthropathy
  - Red synovia
  - Dark yellow to orange synovia
340. Explain why synovia should be submitted to attempt to culture of microorganisms if:
- Organisms are seen in a microscopic examination
  - Organisms are not seen in a microscopic examination

### **CSF analysis**

341. If given the results of a CSF analysis and other pertinent clinical information, use appropriate terms to classify the CSF as an inflammatory or neoplastic pleocytosis.
342. Describe each component of routine CSF analysis (including units of measurement) and the purposes or potential value of each analytical procedure.
343. Explain why quick analysis of CSF is needed to obtain accurate results for some CSF tests.
344. Explain why CSF analysis may be difficult in many veterinary practices.
345. Explain and recognize an advantage and a disadvantage of collecting CSF into an EDTA-containing tube.
346. Explain and recognize why contamination of the CSF with blood can greatly alter the composition of the collected sample.
347. Explain and recognize information that can help us differentiate pathologic hematorrhachis from iatrogenic hemorrhage due to the collection procedure.

348. Explain the pathogenesises (how or why) of each of the following and propose and recognize disorders (diseases, pathologic states) which might cause the following:
- Neutrophilic (suppurative, purulent) pleocytosis
  - Mononuclear (nonsuppurative, non-purulent) pleocytosis
  - CSF with many erythrocytes
  - Dark yellow to orange CSF
  - CSF containing increased total protein concentration
  - CSF containing lipophages
349. Define the following: bilirrhachia, xanthochromia, hypoglycorrachia, pleocytosis
350. Recognize data that represent “albuminocytologic dissociation” (or “protein-cytologic dissociation”) and recognize or explain what it suggests.
351. Explain why CSF should be submitted to attempt to culture microorganisms if:
- Organisms are seen in a microscopic examination
  - Organisms are not seen in a microscopic examination

### **Cytologic Examination of Tissues**

352. In the context of assessing lesions, explain the:
- Advantages of cytologic compared to histologic examinations
  - Disadvantages of cytologic compared to histologic examinations
353. Explain the goals of:
- Biopsy collection methods
  - Biopsy cytopreparatory techniques
  - Cytologic examination of tissues
354. State the types of cells, cell populations, or material that would suggest that a cutaneous or subcutaneous lesion is due to:
- An inflammatory process
  - A neoplastic process
355. State the 3 major groups of cutaneous or subcutaneous neoplasms of dogs; for each, give examples of cytologic features that are used to differentiate them.
356. Define the following terms as they are used to describe cell populations and explain why malignant cells may have each feature.
- Anisokaryosis
  - Increased nuclear:cytoplasmic (N:C) ratio
  - Anisocytosis
  - Poikilokaryosis
  - Pleomorphism
  - Monomorphism
  - Anaplasia
357. Name (list) 6 tumors of canine skin (epidermis, dermis, or subcutaneous) that may be considered discrete or round cell tumors. For each, state or recognize the unique features which allow us to identify the cells of the neoplasms.
358. Explain why the cellular criteria of malignancy are not typically applied as rigorously to canine discrete cell neoplasms (melanoma an exception) as they are to epithelial and non-epithelial (mesenchymal) neoplasia.
359. Explain or list the staining features of the following.
- On Wright-stained slides
    - Bacteria other than *Mycobacterium* sp.
    - Mycobacterium* sp.

- b. On Gram-stained slides
  - 1) Purple (purplish-blue) organisms
  - 2) Pink organisms
  - 3) Pink debris
- c. Based on the above, what stain would best allow for detection of all bacteria?
- 360. Recognized or describe microscopic findings that suggest bacteria are:
  - a. Pathogens
  - b. Nonpathogens (flora, contaminants)
- 361. List the major microscopic features of cells that are the best for differentiating benign cells from malignant cells.
- 362. If given color photocopies of microscopic fields, identify the following cells types or lesions:
  - a. Mast cell neoplasm
  - b. Melanoma
  - c. TVT
  - d. Histiocytoma
  - e. Epithelial cells (well-differentiated)
  - f. Spindle cells (well-differentiated)
  - g. Malignant cells (poorly differentiated)
  - h. Adipocytes (well-differentiated)