Major Instructional Objectives for
Fundamentals of Veterinary Clinical Pathology, 2nd edition

This document is provided as an example of instructional objectives for a sophomore course in a veterinary curriculum in which instruction includes lectures, case discussions, and the use of Diagnostic Pathfinder. The referenced pages are in Fundamentals of Veterinary Clinical Pathology, 2nd ed.

Clinical pathology is the study of clinical diseases or disorders. One major goal of this pathology course is to learn how a disease or disorder creates changes that can be detected by clinical laboratory tests. In other words, learn the pathogeneses of abnormal laboratory data — or, learning what laboratory data tell us about an animal's illness or response to a disorder.

These instructional or educational objectives list the major outcomes for a veterinary student in a sophomore clinical pathology course. That is, a veterinary student should be able to accomplish these tasks by the end of the course. The *Major Objectives* are divided (without separate headings) into three major groups within each instructional unit (most units defined by chapters in *Fundamentals of Veterinary Clinical Pathology*, 2nd edition).

1) Those involving the *interpretation of laboratory data* that produces a diagnosis or a group of possible diagnoses (also called *differential diagnoses* or *rule-outs*).
2) Those dealing with *pathogeneses of laboratory data* found in common clinical disorders and conditions (***most objectives of this course fall into this area***).
3) Those dealing with *pre-analytical or analytical events or facts* that limit the value of laboratory data or create erroneous laboratory data.

For the *Interpretation of Laboratory Data* objectives, there are two recurring tasks for the cases.
      Given relevant historical information, physical examination findings, other clinical information, and clinical laboratory data for domestic mammals with common clinical disorders (examples are discussed during class sessions), a student should:

- State appropriate terms to describe abnormalities and use classifications if appropriate (e.g., acute inflammatory leukogram or renal azotemia)
- Propose appropriate pathologic states, physiologic conditions, pathologic syndromes, or specific diseases that might cause the defined abnormalities. The specificity of the proposed disorders should be appropriate for the available information (or the conclusion that can be justified); e.g., the leukogram justifies a conclusion that the animal has an inflammatory disease (but do not know where, why, or the cause), or the data can justify a conclusion of acute bacterial cystitis.

When a student is asked to *interpret the laboratory data*, the student should attempt to complete those two tasks. It is important to recognize that the major challenge for most cases in this course is not arriving at a correct diagnosis. The "paper cases" are used to provide a framework or context for learning the pathogeneses of abnormal laboratory data.

For the *Pathogeneses of Laboratory Data* objectives, the expectations are described in a separate section below. It is difficult to describe the level of knowledge expected for all laboratory data in a few sentences. Basically, the *pathogenesis* goals are either to:

- Learn how a variety of clinical disorders and conditions can produce the same laboratory test result.
- Learn the *hows* and *whys* at a sufficient depth so that laboratory data can provide clues to the variety of clinical disorders and conditions that occur in animals.

For the *Pre-analytical and Analytical Aspects* objectives, a student should be able to explain when the reported laboratory data represent poor sample handling, poor sample quality, unique sample properties, or the limitations of an analytical procedure.

Pathogenesis

Pathogenesis (*patho-* disease; *-genesis* origin, creation, production) is the sequence of events that occur during the development of or the response to a disease. In the context of clinical pathology, *pathogenesis* of laboratory data starts with the initial pathologic event that causes changes in tissues, cells, or body fluids which eventually produce abnormal laboratory data.

One challenge we face is determining the appropriate level of the pathogenesis explanation. For example – explain the pathogenesis of azotemia in chronic renal failure.
- At the organ level, the explanation might be: Chronic renal disease damages the kidneys so that they do not remove urea and creatinine from plasma adequately and thus urea and creatinine accumulate in plasma.

However, there are many chronic renal diseases that damage the kidneys but those animals do not develop azotemia. Thus, the organ explanation may not have adequate depth to understand the variety of renal diseases.
- At the tissue or cellular level, the explanation might be: Chronic renal disease damages enough nephrons so that renal functional capacity is greatly reduced (generally, < 25 % remaining functional nephrons). At this point, the fewer functional glomeruli causes reduced renal blood flow and thus a decreased glomerular filtration rate (GFR). Once the GFR is reduced sufficiently, urea and creatinine accumulate in plasma.

Understanding the azotemia of chronic renal failure at this level leads to a better understanding of the diseases that cause chronic renal failure, other manifestations of chronic renal failure, and the therapeutic and prognostic aspects of chronic renal failure.

The different pathogenesis levels can be divided as follows.
- Organ: changes in an organ that creates the abnormal laboratory data – rarely an adequate explanation in this course
- Cellular: what happens to cells; or how do cells create the abnormality – common level for abnormal cell concentrations or the microscopic features of cells
- Physiologic: what are the cellular or physiologic responses to hormones, to tissue damage, or to cellular or tissue dysfunction – common level for clinical chemistry abnormalities
- Biochemical: what happens in biochemical pathways – occasional level for either clinical chemistry abnormalities or microscopic features of cells
- Molecular: what happens with a molecule's interaction with other molecules – this level of understanding is usually not needed for clinical disorders or conditions

In this course, we will try to provide many examples of the different levels of pathogeneses. Usually, pathogeneses should be explained at least at the cellular level if abnormal laboratory data involve cells (e.g., neutrophilia or spherocytosis). Pathogeneses should typically be explained at least at the physiologic level if the abnormal laboratory data involve abnormal biochemical data (e.g., hyperglycemia or hyponatremia).

Other major pathogenesis concepts should be remembered when describing the processes that result in abnormal laboratory data.
- An abnormal analyte concentration in a body fluid typically represents a disruption of equilibrium. Blood concentrations in health reflect a balance between an analyte entering the blood and the analyte leaving the blood. An abnormal concentration indicates an imbalance in those processes.
- An abnormal analyte concentration in a body fluid was caused by either an initial event (e.g., pathologic, physiologic, or pharmacologic) or a physiologic response to the initial change (e.g., destruction of pancreatic β-cells lead to decreased insulin release which lead to decreased glucose utilization by cells which leads to hyperglycemia). Therefore, to understand pathogeneses, we need to know what the initial event is.

- When considering potential reasons for abnormal blood analyte concentrations, one should think of basic processes that might cause such concentrations.
  - If there is an increased analyte concentration, is it due to increased rate of entering blood (if so, how?) or a decreased rate of leaving blood (if so, how?).
  - If there is a decreased analyte concentration, is it due to decreased rate of entering blood (if so, how?), an increased rate of leaving blood (if so, how?), or destruction within the blood (if so, how?).

There is not one standard method of describing pathogeneses. *Diagnostic Pathfinder* is designed to present pathogeneses in a multilevel sequence. We will try to model this method during the case discussions and with the "Expert Solutions" in *Diagnostic Pathfinder*.

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Major Objectives for Chapter 1: Introductory Concepts

Chapter 1 material is different from all the other chapters, and thus there are different kinds of learning objectives. The amount of time and the expected learning outcomes will probably differ considerably in sophomore-level clinical pathology courses. At a minimum, students should be able to do the following because such knowledge provides a framework for other instructional units.

1. Describe the similarities and differences of blood, plasma, and serum and how each of those fluids are collected or prepared for laboratory analysis. (p. 5–7)
2. Describe the differences between a qualitative, semi-quantitative, and a quantitative assay. (p. 8)
3. Given measured laboratory data, recognize what the reported significant figures tell us about the degree of certainty in a measurement; e.g., what is the difference between one assay that measures 1.2 mg/dL and another assay that measures 1.234 mg/dL (1,234 µg/dL). (p. 9–11)
4. Using information in Table 1.6, be able to convert conventional units to SI units; or SI units to conventional units. (p. 13–15)
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. (p. 16–20)
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. (p. 16–20)
7. Describe how reference intervals are established and their purposes. (p. 16–20)
8. Explain what reference intervals represent and when parametric or non-parametric methods should be used to establish reference limits. (p. 16–20)
9. Explain why reference intervals published in textbooks may not be adequate for evaluating your patient. (p. 16–20)
10. Explain why interpretation of laboratory data may differ if the value one uses for a decision threshold is different from a reference limit. (p. 20, 44)
11. Explain the differences between preanalytical errors, analytical errors, and postanalytical errors; and be able to recognize examples of each. (p. 21)
12. Contrast or 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. (p. 21–25)
13. Identify and classify the appropriate analytical property in clinical situations. (p. 21–25)
14. 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. (p. 26)
15. Contrast or compare a control solution and a standard solution. (p. 22–24)
16. Define coefficient of variation (CV) and explain why a CV of 10% may be very acceptable for one assays but very unacceptable for another assay. (p. 22–23)
17. Contrast random error and systematic error (bias). (p. 21–22)

18. 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. (p. 38)
19. Contrast or 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. (p. 38–39)
20. Contrast and compare diagnostic sensitivity and PV (+). Contrast and compare diagnostic specificity and PV (–).(p. 38–39)
21. 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. (p. 38–39)
22. Explain how the following alter the calculated values for the diagnostic sensitivity, diagnostic specificity, and diagnostic accuracy.
    a. Prevalence of disease (p. 40–42)
    b. Use of a poor "gold standard" to determine the presence or absence of disease (p. 39)
    c. Increasing the decision threshold value; or decreasing the decision threshold value (p. 40)
    d. Using a healthy animal group as the "disease absent" group
23. Recognize or list the diagnostic property that is most important for a screening test. ... for a confirmation test. (p. 38–39)

Major Objectives for Chapter 2: Leukocytes
24. Interpret leukogram data including abnormal leukocyte concentrations and common microscopic findings such as toxic neutrophils and reactive lymphocytes
25. Explain, list, or recognize the physiologic or pathologic processes or mechanisms that cause the following:
    a. Inflammatory neutrophilia with a regenerative left shift (p. 71–74)
    b. Inflammatory neutrophilia without a left shift (or mature neutrophilia) (p. 74–76)
    c. Inflammatory neutrophilia with a degenerative left shift (p. 71)
    d. Steroid neutrophilia with a regenerative left shift (p. 75)
    e. Steroid neutrophilia without left shift (or mature neutrophilia) (p. 75)
    f. Steroid neutrophilia with a right shift (p. 71, 75)
    g. Physiologic (shift) neutrophilia (p. 76)
    h. Inflammatory neutropenia with a left shift (p. 76–79)
    i. Neutrophilia concentration within reference interval concurrent with a left shift (p. 70–79)
    j. Bovine inflammatory neutropenia (p. 77–79)
    k. Bovine inflammatory neutrophilia (p. 90)
    l. Peripheral-destruction neutropenia (p. 79)
    m. Granulocytic-hypoplasia neutropenia (p. 79)
    n. Ineffective-production neutropenia (p. 80)
    o. Inflammatory lymphocytosis (p. 81)
    p. Physiologic (shift) lymphocytosis (p. 81–82)
    q. Neoplastic (leukemic) lymphocytosis (p. 82–83)
    r. Inflammatory lymphopenia (p. 83–85)
    s. Steroid lymphopenia (p. 85)
    t. Depletion lymphopenia (p. 85)
    u. Lymphoid-hypoplasia lymphopenia (p. 85)
    v. Inflammatory monocytosis (p. 85)
    w. Steroid monocytosis (p. 85)
    x. Eosinophilia associated with mast cell degranulation (p. 86)
    y. Eosinophilia concurrent with inflammatory neutrophilia (p. 86)
    z. Eosinophilia not associated with other leukogram changes (p. 86–88)

aa.   Concurrent findings of lymphocytosis and eosinophilia in an obviously stressed dog (p.
                    83 & 87)
26.   Explain, list, or recognize the reasons for:
           a.   Increased WBC concentration when nucleated erythrocytes are in the blood (p. 68–69)
           b.   The calculated concentrations of leukocytes in a leukogram being estimated
                    concentrations (p. 67–68)
           c.   Increased WBC concentration when nucleated erythrocytes are in the blood (p. 68–69)

Major Objectives for Chapter 3: Erythrocytes
27.   Interpret erythrogram data including:
           a.   Calculated Hct, spun Hct, blood Hgb concentration, RBC concentration, MCV, MCHC,
                    MCH, reticulocyte percentage, corrected reticulocyte percentage, and reticulocyte
                    concentration
           b.   Abnormal microscopic features including macrocytosis, microcytosis, hypochromia,
                    agglutination, rubricytosis (appropriate or inappropriate), increased polychromasia,
                    reticulocytosis, basophilic stippling, Heinz bodies, Howell-Jolly bodies, acanthocytosis,
                    codocytosis, crenation, eccentrocytosis, keratocytosis, pyknocytosis, schizocytosis,
                    spherocytosis, concurrent normocytosis & normochromia (mostly in p. 135–151)
           c.   A positive or negative Coombs' test (direct antiglobulin test)
           d.   Hemoglobinemia or icteric plasma
           e.   Those with macrocytic anemias, microcytic anemias, normocytic normochromic anemias,
                    hypochromic anemias, and hyperchromic anemias
           f.   Those with anemia and with or without reticulocytosis
           g.   Results of a saline dispersion test
28.   Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the
         following:
           a.   Anemia of inflammatory disease (p. 161)
           b.   Anemia of chronic renal disease (p. 161–162)
           c.   Anemia of erythroid hypoplasia or aplasia ((p. 162–163)
           d.   Anemia of pure red cell aplasia (p. 163)
           e.   Anemia of immune-mediated nonregenerative anemia (ineffective erythropoiesis) (p.
                    164)
           f.   Anemia of acute blood loss (Fig. 3.8, p. 168)
           g.   Anemia of chronic blood loss that results in an iron deficiency (Fig. 3.9, p. 169)
           h.   Extravascular hemolytic anemia (p. 170)
           i.   Intravascular hemolytic anemia (p. 170)
           j.   Immune-mediated anemias (idiopathic, penicillin-induced, parasitic, neonatal) (p. 176;
                    Fig. 3.12, p. 178)
           k.   Anemias due to erythrocytic parasite infection (*Mycoplasma, Anaplasma, Babesia,
                    Cytauxzoon)* (note: cytauxzoonosis is very different from the others) (p. 181–182, 184,
                    139)
           l.   Heinz body anemias (p. 186–187)
           m.   Eccentrocytic anemias (p. 187–188)
           n.   Post-parturient hemoglobinuria (p. 189–190)
           o.   Fragmentation anemia (p. 191)
           p.   Heparin-induced hemolysis in horses (p. 192–193)
           q.   Anemia associated with erythroid neoplasia (p. 163)
           r.   Erythrocytosis due to dehydration (p. 195)
           s.   Erythrocytosis due to splenic contraction in horses and dogs (p. 195)
           t.   Erythrocytosis due to chronic hypoxia (p. 195)
           u.   Erythrocytosis due to erythroid neoplasia (p. 195)
29.   Explain, list, or recognize the reasons for:
           a.   Falsely low Hct values due to excess EDTA (p. 129–131)

       b.   Falsely increased or decreased MCV values with altered plasma Na$^+$ concentrations (p. 129–131)

       c.   Reasons for falsely increased MCHC or MCH values due to hemoglobinemia, lipemia, markedly icteric plasma, and extreme leukocytosis (p. 157–158)

       d.   Falsely increased MCV values due to cold agglutinins (p. 156)

       e.   False negative Coombs' test results

30.   Interpret iron profile data including serum iron concentration, serum TIBC, serum UIBC, % saturation, serum ferritin concentration, and quantity of stored iron in marrow.

31.   Explain, list, or recognize the physiologic or pathological processes or mechanisms that cause the following:

       a.   Hypoferremia due to blood loss (p. 203–204)

       b.   Hypoferremia due to inflammation (p. 204)

       c.   Hyperferremia due to iron supplementation (p. 203)

       d.   Decreased TIBC due to inflammation (p. 206)

       e.   Hyperferritinemia due to inflammation (p. 208)

       f.   Hypoferritinemia due to blood loss (p. 208)

Major Objectives for Chapter 4: Platelets

32.   Interpret thrombogram data including abnormal platelet concentrations and common microscopic findings such as giant (shift, stress, etc) platelets (p. 234)

33.   Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:

       a.   Primary (idiopathic) immune-mediated thrombocytopenia (p. 238)

       b.   Consumptive thrombocytopenia (p. 240–241)

       c.   Decreased-production thrombocytopenia (p. 235)

       d.   Reactive (non-clonal) thrombocytosis (p. 245)

       e.   Clonal thrombocytosis (p. 244–235)

       f.   Specific thrombocytopenias: chemotherapeutic (p. 235), estrogen-induced (p. 236), immune-mediated (p. 237), DIC (p. 241), infection-related (p. 242)

       g.   Petechial hemorrhages associated with thrombocytopenia (p. 227)

34.   Explain, list, or recognize the reasons for:

       a.   Pseudothrombocytopenia (p. 233)

       b.   Platelet clumps in a blood film (p. 230, 233)

Major Objectives for Chapter 5: Hemostasis

35.   Interpret hemostasis data including:

       a.   (Primary hemostasis) Prolonged buccal mucosal bleeding time (p. 263), vWF ratio or percentage (p. 267–268), platelet concentration (p. 234)

       b.   (Secondary hemostasis): prolonged PTT (p. 281–282), prolonged PT (p. 283), prolonged TT (p. 285), hypofibrinogenemia (p. 286, 396), decreased antithrombin activity (p. 289–290), prolonged "PIVKA" assay (p. 288–289)

       c.   (Fibrinolysis): increased FDP concentration (p. 297–298) , increased D-dimer concentration (p. 300)

36.   Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:

       a.   Thrombocytopenia (see Chapter 4 objectives)

       b.   Thrombopathias (hereditary vs acquired) (p. 265–266)

       c.   Prolonged BMBT in thrombocytopenic states (p. 302–303)

       d.   Prolonged BMBT without a thrombocytopenic states (p. 302)

       e.   Prolonged PTT due to an inherited coagulopathy (p. 281)

       f.   Prolonged PT due to an inherited coagulopathy (p. 283)

       g.   Prolonged PTT but not PT with patient heparinization (p.283)

       h.   Prolonged PTT &/or PT due to hepatic disease (p. 304–305)

i.   Prolonged PTT &/or PT due to vitamin K antagonism (p. 305–306)
j.   Prolonged PTT &/or PT due to cholestasis or intestinal malabsorption (p. 306)
k.   Prolonged PTT &/or PT due to vasculitis, severe tissue necrosis, sepsis, or disseminated neoplasia (p. 306)
l.   Hypofibrinogenemia due to vasculitis, severe tissue necrosis, sepsis, or disseminated neoplasia (p. 309)
m.   Increased FDP &/or D-dimer concentrations due to vasculitis, severe tissue necrosis, sepsis, or disseminated neoplasia (p. 309)
n.   Decreased AT activity due to vasculitis, severe tissue necrosis, sepsis, or disseminated neoplasia (p. 309)
o.   Decreased AT activity due to protein-losing nephropathy (p. 290)
p.   Prolonged PTT &/or prolonged PT &/or decreased AT activity due to heparinization (p. 310)

37.   Explain, list, or recognize the reasons for:
a.   Prolonged PTT &/or PT when an animal has an erythrocytosis (p. 276–277)
b.   Altered hemostasis data when a hemostasis system (primary or secondary) is activated during sample collection (p. 274, 276)
c.   Very little serum collected when a blood sample (collected in a clot tube) is placed in a refrigerator soon after collection. (hint p. 264–265)

Major Objectives for Chapter 6: Bone Marrow and Lymph Node
38.   Interpret bone marrow reports or lymph node reports including:
a.   Hypercellular or hypocellular marrow
b.   Hypercellular marrow with decreased G:E, normal G:E, or increased G:E ratio
c.   Hypocellular marrow with decreased G:E, normal G:E, or increased G:E ratio
d.   Thrombocytopenia with megakaryocytic hyperplasia or hypoplasia
e.   Anemia and reticulocytopenia with an increased G:E or decreased G:E ratio
f.   Anemia and reticulocytosis with an increased G:E or decreased G:E ratio
g.   Neutrophilia with an increased G:E or decreased G:E ratio
h.   Neutropenia with an increased G:E or decreased G:E ratio
i.   Anemia, reticulocytopenia, neutropenia, and thrombocytopenia with a normal G:E ratio
j.   Bone marrow lymphocytosis with typical small lymphocytes or atypical large lymphocytes
k.   Hypocellular marrow and 50 % of nucleated cells are small lymphocytes
l.   Hypercellular marrow and > 20 % of nucleated cells are blastic cells
m.   Lymphadenomegaly with > 75 % small lymphocytes and no other significant finding
n.   Lymphadenomegaly with > 75 % small lymphocytes and an increased plasma cell percentage
o.   Lymphadenomegaly with > 75 % small lymphocytes and an increased neutrophil &/or macrophage percentage
p.   Lymphadenomegaly with > 75 % small lymphocytes and an increased eosinophil percentage
q.   Lymphadenomegaly with < 50 % small lymphocytes and an increased percentage of medium &/or large lymphocytes
r.   Lymphadenomegaly with nearly 100 % small lymphocytes
s.   Being able to state the neoplastic cell lineage with these diagnoses (p. 343):
    i.   Acute myeloid leukemia
    ii.   Acute myelomonocytic leukemia
    iii.   Acute erythroleukemia
    iv.   Acute myeloblastic leukemia
    v.   Chronic myeloid leukemia
    vi.   Primary erythrocytosis
    vii.   Polycythemia vera

viii.   Acute lymphocytic leukemia
ix.   Chronic lymphocytic leukemia
x.   Thrombocythemia

39. Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:
  a. Marrow hyperplasia (p. 337–338)
  b. Megakaryocytic hyperplasia (p. 337)
  c. Erythroid hyperplasia with a regenerative anemia (p. 334)
  d. Erythroid hyperplasia with a nonregenerative anemia (p. 336)
  e. Granulocytic hyperplasia with a neutrophilia (p. 336)
  f. Granulocytic hyperplasia with a neutropenia (p. 336-–337)
  g. Marrow lymphoid hyperplasia (p. 340)
  h. Aplastic anemia (p. 338)
  i. Pure red cell aplasia (p. 339)
  j. Marrow hypoplasia due to estrogen toxicosis (p. 338, 163, 236)
  k. Erythroid hypoplasia due to acute myeloid leukemia (p. 342)
  l. Erythroid hypoplasia due to acute lymphocytic leukemia (p. 342)

40. Explain, list, or recognize the reasons for:
  a. Hypocellular aspirate from an animal with a hypercellular marrow (p. 329, 334)
  b. Mostly lysed cells in a lymph node aspirate (p. 359)
  c. Clotted erythrocytes and platelets in a marrow aspirate (p. 328)

Major Objectives for Chapter 7: Proteins
41. Interpret serum and plasma protein concentrations including:
  a. Hyperproteinemia, hyperalbuminemia, and hyperglobulinemia
  b. Hyperproteinemia due to hyperglobulinemia
  c. Hyperproteinemia, hypoalbuminemia, and hyperglobulinemia
  d. Normoproteinemia with hypoalbuminemia and hyperglobulinemia
  e. Hypoproteinemia, hypoalbuminemia, and hypoglobulinemia
  f. Hypoproteinemia due to hypoalbuminemia
  g. Hyperproteinemia with a polyclonal gammopathy
  h. Hyperproteinemia with a monoclonal gammopathy
  i. Hyperfibrinogenemia
  j. Hypofibrinogenemia

42. Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:
  a. Hyperproteinemia due to dehydration (p. 379)
  b. Hyperproteinemia due to inflammation (p. 380–381)
  c. Hyperproteinemia due to B-lymphocyte neoplasia (p. 381–383)
  d. Hyperproteinemia with a polyclonal gammopathy (p. 381)
  e. Hyperproteinemia with a monoclonal gammopathy (p. 382–383)
  f. Hyperalbuminemia due to dehydration (p. 390)
  g. Hypoalbuminemia due to inflammation (p. 380)
  h. Hypoproteinemia due to blood loss (p. 385)
  i. Hypoproteinemia, hypoalbuminemia, or hypoglobulinemia due to urinary protein loss (p. 385–386)
  j. Hypoproteinemia, hypoalbuminemia, or hypoglobulinemia due to intestinal protein loss (p. 387)
  k. Hypoproteinemia, hypoalbuminemia, or hypoglobulinemia due to cutaneous protein loss (p. 387)
  l. Hypoproteinemia due to pleural or peritoneal exudation (p. 387)
  m. Hypoproteinemia due to acute, severe vasculitis (p. 387)

       n.   Hypoproteinemia, hypoalbuminemia, or hypoglobulinemia due to hepatic insufficiency or failure (p. 388)

       o.   Hypoproteinemia, hypoalbuminemia, or hypoglobulinemia due to exocrine pancreatic insufficiency (p. 388–389)

       p.   Hypoproteinemia, hypoalbuminemia, or hypoglobulinemia due to mucosal disease of the small intestine (p. 388–389)

       q.   Hypoproteinemia, hypoalbuminemia, or hypoglobulinemia due to neoplasia (p. 389)

       r.   Hyperfibrinogenemia due to inflammation (p. 372, 380, 394)

       s.   Hyperfibrinogenemia due to dehydration (p. 394)

       t.   Hypofibrinogenemia due to DIC (p. 394, 309)

43.   Explain, list, or recognize the reasons for:

       a.   Factitious hyperalbuminemia in a BCG assay (pseudohyperalbuminemia (p. 390)

       b.   Factitious hypoglobulinemia secondary to pseudohyperalbuminemia (p. 375)

       c.   Increases in the refractometric plasma [TP] not due to plasma proteins (p. 373)

       d.   Differences between total solids concentration and total protein concentration (p. 373)

Major Objectives for Chapter 8: Urinary System

44.   Interpret serum chemistry and urinalysis data including:

       a.   Serum urea and creatinine concentrations

       b.   Color and clarity of urine

       c. USG_{ref} values

       d.   Urinalysis chemistry data including pH, protein, glucose, ketones, heme, and bilirubin

       e.   Urinalysis microscopic findings including pyuria, hematuria, cylindruria, crystalluria, and bacteriuria

       f.   Urine protein to creatinine ratio

45.   Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:

       a.   Increased serum urea concentration due to dehydration (p. 429–430)

       b.   Increased serum creatinine concentration due to dehydration (p. 429–430)

       c.   Increased serum urea and/or creatinine concentration due to chronic renal disease (p. 430–431)

       d.   Increased serum urea and/or creatinine concentration due to acute renal disease (p. 430–431)

       e.   Increased serum urea and/or creatinine concentration due to urinary tract obstruction (p. 431)

       f.   Increased serum urea and/or creatinine concentration due to uroperitoneum (p. 431)

       g.   Increased serum urea concentration due to marked gastric or intestinal hemorrhage (p. 431–432)

       h.   Decreased serum urea concentration due to hepatic insufficiency (p. 435–436)

       i.   Decreased serum urea concentration due to persistent diuresis (p. 436)

       j. USG_{ref} of 1.025 in a healthy dog (p. 447)

       k. USG_{ref} of 1.005 in a healthy dog (p. 447)

       l. USG_{ref} of 1.050 in a healthy dog (p. 447)

       m. USG_{ref} of 1.007 to 1.013 in polyuric chronic renal failure (p. 450)

       n. USG_{ref} of 1.007 to 1.013 in oliguric chronic renal failure (p. 450)

       o. USG_{ref} of 1.025 and polyuria in an animal with diabetes mellitus (p. 450)

       p. $USG_{ref} < 1.015$ in a case of central diabetes insipidus (p. 450)

       q. $USG_{ref} < 1.015$ in a case of hyperadrenocorticism (p. 450–451)

       r. $USG_{ref} < 1.015$ in an animal with hypercalcemia (p. 451)

       s. $USG_{ref} < 1.015$ in a case of hypoadrenocorticism (p. 451)

       t. $USG_{ref} < 1.015$ in a case of hepatic insufficiency (p. 451)

       u. $USG_{ref} < 1.015$ after fluid therapy (p. 447)

       v.   Alkalinuria due to bacterial infection in urinary tract (p. 457)

w. Proteinuria due to intravascular hemolysis, lymphoma or myeloma, renal amyloidosis, glomerulonephritis, proximal renal tubular disease, urinary tract inflammation, or urinary tract hemorrhage (p. 458–462)

x. Glucosuria due to diabetes mellitus or proximal renal tubular disease (p. 463–464)

y. Ketonuria due to ketosis (p. 464–465)

z. Positive heme reaction due to intravascular hemolysis, rhabdomyolysis, or urinary tract hemorrhage (p. 465–466)

aa. Bilirubinuria due to hemolytic disease or due to cholestatic disease (p. 466–467)

bb. Pyuria due to urinary tract infection, urinary tract neoplasia, or urolithiasis (p. 469–471)

cc. Pyuria due to genital tract inflammation (p. 471)

dd. Hematuria due to urinary tract trauma or inflammation, thrombocytopenia, or coagulopathies (p. 471–472)

ee. Bacteriuria due to pathologic and non-pathologic disorders or conditions (p. 472)

ff. Cylindruria due to renal disease (p. 473)

gg. Ammonium biurate crystalluria in hepatic insufficiency (p. 470, 679)

hh. Struvite crystalluria due to urinary tract infection (p. 470)

ii. Increased $(Prot/Crt)_u$ due to intravascular hemolysis, lymphoma or myeloma, renal amyloidosis, glomerulonephritis, proximal renal tubular disease, urinary tract inflammation, or urinary tract hemorrhage (p. 479–480)

46. Explain, list, or recognize the reasons for:

a. "Proteinuria" (via reagent strips) due to alkalinuria (p. 458)

b. Inaccurate assessment of renal concentrating ability when there is marked glucosuria or proteinuria (p. 447)

c. Alkalinuria due to delayed sample analysis (p. 457)

d. Absence of erythrocytes in urine sediment in some cases of pathologic or iatrogenic hematuria (p. 471)

Major Objectives for Chapter 9: Monovalent Electrolytes and Osmolality

47. Interpret serum and plasma electrolyte concentrations and serum osmolalities including:

a. Hypernatremia and hyponatremia

b. Hyperchloremia and hypochloremia

c. Hyperkalemia and hypokalemia

d. Increased and decreased bicarbonate concentrations

e. Increased and decreased total CO_2 concentrations

f. Increased anion gaps

g. Increase L-lactate concentration

h. Increased β-hydroxybutyrate concentration

i. Hyperosmolality and hypoosmolality

j. Calculated osmolality values

48. Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:

a. Hypernatremia due to H_2O deprivation, diabetes insipidus, or salt poisoning (p. 501–502)

b. Normonatremia in a dehydrated animal that has renal disease or GI disease (p. 503)

c. Normonatremia in an edematous animal that has congestive heart failure (p. 504–505)

d. Hyponatremia due to GI disease, hypoadrenocorticism, diabetes mellitus, sweating (horses), congestive heart failure, or uroperitoneum (p. 505–508)

e. Hyperkalemia due to inorganic acidosis, rhabdomyolysis, renal failure, obstructive uropathy, uroperitoneum, and hypoadrenocorticism (p. 511–516)

f. Hypokalemia due to metabolic alkalosis, anorexia, diabetes mellitus, GI disease, and sweating (horses) (p. 516–519)

g. Decreased Na^+ to K^+ ratio in animals with hypoadrenocorticism, diarrhea, diabetes mellitus, and uroperitoneum (p. 519–520)

h. Hyperchloremia due to H_2O deprivation, diabetes insipidus, salt poisoning, or diarrhea (p. 523)

i. Hypochloremia due to vomiting, displaced abomasum, ketoacidotic diabetes mellitus, anaerobic states that result in lactic acidosis, hypoadrenocorticism, sweating (horses), congestive heart failure, or uroperitoneum (p. 524–525)

j. Increased serum HCO_3^- concentration due to vomiting, displaced abomasum, or a respiratory acidosis (p. 529–530)

k. Decreased serum HCO_3^- concentration due to disorders that cause lactic acidosis or ketoacidosis, renal failure, uroperitoneum, or disorders that cause diarrhea (p. 531–533)

l. Increased anion gap in ketosis, lactic acidosis, renal failure, and antifreeze poisoning (p. 537)

m. Decreased anion gap due to hypoproteinemia (p. 538)

n. Hyperlactatemia due to poor tissue perfusion or strenuous exercise (p. 540–542)

o. Ketonemia due to diabetes mellitus disorders, bovine ketosis, or starvation (p. 544–545)

p. Increased serum osmolality in a dehydrated animal that is hypernatremic (p. 501–502, 552)

q. Increased serum osmolality in a dehydrated animal that is hyponatremic (p. 552)

r. Increased serum osmolality in a hydrated animal that is normonatremic (p. 553)

s. Decreased serum osmolality (p. 552)

49. Explain, list, or recognize the reasons for:
a. Pseudohyponatremia due to lipemia or marked hyperproteinemia (p. 508–509)

b. Pseudohyperkalemia due to hemolysis or thrombocytosis (p. 514)

c. Pseudohyperchloremia due to KBr therapy (p. 522)

d. Pseudohypochloremia due to lipemia (p. 508)

e. Decreased serum HCO_3^- concentration due to poor sample handling (p. 527–528)

f. Hyperlactatemia due to delayed blood sample analysis (p. 540)

g. Differences between serum colloidal osmotic pressure (oncotic pressure) and serum osmolality and the factors in serum that affect both properties (p. 546)

Major Objectives for Chapter 10: Blood Gases, Blood pH, and Strong Ion Difference

50. Interpret blood gas data including:
a. Increases and decreases in plasma pH values

b. Increases and decreases in plasma P_aCO_2 &/or plasma P_vCO_2

c. Increases and decreases in plasma P_aO_2

d. Increases and decreases in plasma HCO_3^- concentrations

e. Increases and decreases in plasma total CO_2 concentrations

51. Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:
a. Increased serum HCO_3^- concentration due to vomiting, displaced abomasum, bovine renal failure, or a disorder that causes respiratory acidosis (p. 529–530, 576)

b. Decreased serum HCO_3^- concentration due to disorders that cause lactic acidosis or ketoacidosis, renal failure, uroperitoneum, disorders that cause diarrhea, or a respiratory alkalosis (p. 531–534, 574)

c. Increased P_aCO_2 (or P_vCO_2) due to respiratory disease or disorders that restrict respiration or as a compensation to alkalemia (p. 575)

d. Decreased P_aCO_2 (or P_vCO_2) as a response to hypoxemia or acidemia (p. 576–577)

e. Acidemia due to disorders that cause lactic acidosis (p. 531, 539), ketoacidosis (p. 531), renal failure (p. 532, 440), or extensive pulmonary disease (p. 575)

f. Alkalemia due to disorders that cause gastric or abomasal loss of HCl, bovine renal failure, or hypoxemia (p. 529, 576)

g. Decreased P_aO_2 due to pulmonary disease (p. 582–583)

h. Increased P_aO_2 during gas anesthesia (p. 581)

      i. P_aO_2 is within reference intervals when anemia is causing hypoxia (p. 583)

      j.   Tissue hypoxia when there is not hypoxemia (p. 583–584)

52.   Explain, list, or recognize the reasons for:

    a.   Decreased serum HCO_3^- concentration due to poor sample handling (p. 527–528)

    b.   Decreased P_aCO_2 (or P_vCO_2) due to sample being exposed to air or when collected with excess heparin (p. 527–528, 573)

    c.   Increased P_aO_2 due to sample being exposed to air or when collected with excess heparin (p. 573)

    d.   Decreased P_aO_2 and decreased pH when there is delayed analysis of a heparinized blood sample (p. 573)

Major Objectives for Chapter 11: Calcium, Phosphorus, Magnesium, and their Regulatory Hormones

53.   Interpret serum or plasma calcium, phosphorus, magnesium, and their regulatory hormone data including:

    a.   Hypercalcemia, hypocalcemia, and alterations in free Ca^{2+} concentrations

    b.   Hyperphosphatemia and hypophosphatemia

    c.   Hypermagnesemia and hypomagnesemia

    d.   Increases in iPTH or PTHrp concentrations, decreases in iPTH concentrations, or iPTH concentrations WRI with a concurrent hypercalcemia

54.   Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause:

    a.   Hypercalcemia in hyperparathyroidism, malignancies, rodenticide toxicosis, equine renal failure, canine hypoadrenocorticism, and canine renal failure (p. 598–602)

    b.   Hypocalcemia in hypoparathyroidism, chronic renal disease (dogs, cats, and cattle), post parturient state or during early lactation (p. 602–607)

    c.   Alterations in free Ca^{2+} concentrations due to acidemia or alkalemia (p. 611–612)

    d.   Hyperphosphatemia due to dehydration, renal failure, uroperitoneum, urinary tract obstruction, hypoparathyroidism, and myopathies (p. 618–619)

    e.   Hypophosphatemia due to anorexia, hyperparathyroidism, hyperinsulinism, and milk fever (p. 619–621)

    f.   Hypermagnesemia due to renal failure (p. 622)

    g.   Hypomagnesemia due to grass tetany or prolonged anorexia (p. 623)

    h.   Increased iPTH concentrations due to parathyroid neoplasia, chronic renal disease, or a diet with a low $Ca^{2+}:PO_4$ ratio (p. 627, 598, 604, 607)

    i.   Increased PTHrp concentrations due to malignancies (p. 628)

55.   Explain, list, or recognize the reasons for:

    a.   Hypocalcemia or hypomagnesemia due to hypoproteinemia and/or hypoalbuminemia (p. 602, 623)

    b.   Hyperphosphatemia due to in vitro hemolysis or delayed blood sample handling (p. 617)

    c.   Pseudohypocalcemia due to collection of blood into an EDTA anticoagulant (p. 596)

    d.   Decreased fCa^{2+} concentration when blood sample collected with excess heparin (p. 611)

    e.   Altered fCa^{2+} concentrations when blood or serum sample is not handled anaerobically (p. 611)

Major Objectives for Chapter 12: Enzymes

56.   Interpret serum enzyme data including increased activities of ALP, ALT, AMS, AST, CK, GGT, GMD, ID, LD, and LPS

57.   Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:

    a.   Increased activities of ALP, ALT, AST, GGT, GMD, ID, and LD due to hepatic, biliary, or hepatobiliary disorders or conditions (p. 642–645, 650–667)

    b.   Increased activities of ALP due to glucocorticoids in dogs and hyperthyroidism in cats (p. 642–645, 657–658)

c. Increased activities of AST, LD, CK, or ALT due to muscular disorders (p. 642–645, 652–654, 662–663)
d. Increased activities of AMS or LPS due to pancreatic disease, dehydration, or renal disease (p. 642–645, 664–667)
e. Increases in PLI concentration due to pancreatic disease (p. 668)

58. Explain, list, or recognize the reasons for:
a. Alterations in AST, LD, or CK activities due to in vitro hemolysis or delayed blood sample handling (p. 652)
b. Different enzyme data when assays are performed at different temperatures or with different substrates (p. 647)

Major Objectives for Chapter 13: Liver function
59. Interpret CBC, serum chemistry, or urinalysis data that suggest or indicate hepatic dysfunction
60. Explain, list, or recognize the physiologic and pathologic processes or mechanisms that cause the following:
a. Hypoproteinemia due to hepatic disorders (p. 678, 388)
b. Ammonium biurate crystalluria due to hepatic disorders (p. 678, 470)
c. Hyposthenuria due to hepatic disorders (p. 678, 451)
d. Hyperbilirubinemia due to in vivo hemolysis, anorexia (horses, cattle), and cholestasis (obstructive or functional) (p. 678, 684–689)
e. Bilirubinuria due to in vivo hemolysis or cholestasis (p. 678. 689, 467)
f. Increased direct bilirubin, indirect bilirubin, unconjugated bilirubin, conjugated bilirubin, or δ-bilirubin concentration in pathologic or physiologic states (p. 683)
g. Hypercholemia (increased bile acid concentration) due to portosystemic shunts, diffuse liver disease, and cholestasis (obstructive or functional) (p. 678, 691–693)
h. Hyperammonemia due to portosystemic shunts, diffuse liver disease, and, in horses, intestinal disease (p. 678, 699–701)
61. Explain, list, or recognize the reasons for:
a. Falsely decreased serum bilirubin concentration after sample is exposed to daylight (p. 683)
b. False ammonium concentrations due to delayed blood sample analysis (p. 699)
c. False ammonium concentrations when sample is handled aerobically (p. 699)

Major Objectives for Chapter 14: Glucose, Ketoamines, and Related Regulatory Hormones
62. Interpret serum (blood, plasma) glucose, ketoamine, and insulin concentrations
63. Explain, list, or recognize the physiologic, pathologic, or pharmacologic processes or mechanisms that cause the following:
a. Hyperglycemia due to excitement, eating a meal, stress, β-cell destruction, feline pancreatic insular amyloidosis, acute pancreatitis, hyperadrenocorticism, equine hyperpituitarism, pheochromocytoma, steroid therapy, intravenous glucose therapy, xylazine & detomidine therapy, and insulin overdose (p. 714–719)
b. Hypoglycemia due to functional β-cell neoplasia, hypoadrenocorticism, hepatic insufficiency, spontaneous bovine ketosis, and insulin overdose (p. 719–721)
c. Increased fructosamine concentration in persistent hyperglycemic states or hypothyroidism (p. 723–725)
d. Decreased fructosamine concentration in persistent hypoglycemic states, hypoproteinemic states, or hyperthyroidism (p. 723–726)
e. Increased glycated hemoglobin percentage in persistent hyperglycemic states (p. 723–725)
f. Decreased glycated hemoglobin percentage in persistent hypoglycemic states and acute hemolytic or blood loss anemic disorders (p. 723–726)
g. Hyperinsulinemia and/or increased IRI:G ratio due to functional β-cell neoplasia or hyperglycemic disorders (p. 728–729)

h.   Hypoinsulinemia and/or increased IRI:G ratio due to β-cell damage or hypoglycemic disorders (p. 728–729)
64.   Explain, list, or recognize the reasons for:
    a.   Pseudohypoglycemia due to delayed removal of serum from clotted blood, marked leukocytosis, or marked erythrocytosis (p. 709)
    b.   Artifactual hypoglycemia due to collection of blood into NaF-oxalate tubes (p. 709–711)
    c.   Differences between blood glucose concentration and serum glucose concentration when there is a marked erythrocytosis (p. 711–712)

Major Objectives for Chapter 15: Exocrine Pancreas and Intestine
65.   Interpret serum AMS & LPS activities, serum TLI, PLI, cobalamin, or folate concentrations, and fecal α_1-PI concentrations
66.   Explain, list, or recognize the physiologic or pathologic processes or mechanisms that cause the following:
    a.   Hyperamylasemia, hyperlipasemia, increased TLI concentration, or increased PLI concentration in acute pancreatitis (p. 741–743)
    b.   Decreased TLI concentration or decreased PLI concentration in chronic pancreatitis or pancreatic acinar cell atrophy (p. 744)
    c.   Increased TLI concentration in azotemic dogs (p. 744)
    d.   Decreased cobalamin or folate concentrations due to pancreatic or intestinal disorders (p. 749–752)
    e.   Increased fecal α_1-PI concentration in dogs and cats with intestinal diseases (p. 753)
    f.   Flat xylose or glucose absorption curves in dogs or horses with intestinal diseases (p. 754–756)
67.   Explain, list, or recognize the reasons for:
    a.   Increased TLI concentrations in a non-fasted dog (p. 744)
    b.   False feline or equine TLI (or PLI) data if a canine assay is used (p. 743, 745)
    c.   False folate concentration if there is in vitro hemolysis (p. 751)
    d.   False cobalamin concentration if the sample is exposed to daylight (p. 749)

Major Objectives for Chapter 16: Lipids
68.   Interpret serum triglyceride and cholesterol concentrations
69.   Explain, list, or recognize the physiologic or pathologic processes or mechanisms that cause the following:
    a.   Hypercholesterolemia due to protein-losing nephropathy, hypothyroidism, cholestasis, diabetes mellitus, and eating a meal (p. 773–777)
    b.   Hypocholesterolemia due to hepatic insufficiency (p. 770)
    c.   Hypertriglyceridemia due to eating a meal, equine hyperlipemias, acute pancreatitis, and diabetic disorders (p. 777–778)
70.   Explain, list, or recognize the reasons for:
    a.   Increased triglyceride or cholesterol concentrations in a non-fasted dog or cat (p. 768, 771)

Major Objectives for Chapter 17: Thyroid Function
71.   Interpret serum thyroxine, free thyroxine, TSH, and TgAA concentrations
72.   Explain, list, or recognize the physiologic, pathologic, or pharmacologic processes or mechanisms that cause the following:
    a.   Hyperthyroxemia due to thyroid neoplasia or administration of TSH (p. 789–790)
    b.   Absence of hyperthyroxemia in feline hyperthyroidism due to thyroid adenoma (p. 790)
    c.   Hypothyroxemia or decreased free $[T_4]$ due to lymphocytic thyroiditis (or other causes of thyroid gland damage), non-thyroidal disease, and some drug treatments (p. 790–792)
    d.   Increased TSH concentrations due to lymphocytic thyroiditis (or other causes of thyroid gland damage) (p. 794)

e.   Increased TgAA concentration due to lymphocytic thyroiditis (p. 794–795)
f.   Failure to suppress [T_4] with T_3 treatments in a cat (p. 796–797)

73.   Explain, list, or recognize the reasons for:
        a.   Positive interference by thyroxine autoantibodies on measurement of thyroxine concentration (p. 786–787)

Major Objectives for Chapter 18: Adrenocortical Function

74.   Interpret serum or plasma cortisol, ACTH, and aldosterone concentrations and urine cortisol:creatinine ratios

75.   Explain, list, or recognize the physiologic, pathologic, or pharmacologic processes or mechanisms that cause the following:
        a.   Normocortisolemia in a dog with hyperadrenocorticism (p. 808)
        b.   Increased urinary cortisol to creatinine ratio due to hyperadrenocorticism or non-adrenal disease (p. 812–813)
        c.   Increased ACTH concentration in hypoadrenocorticism (p. 813–814)
        d.   Increased ACTH concentration in hyperadrenocorticism (p. 813–814)
        e.   Decreased ACTH concentration in hyperadrenocorticism (p. 813–814)
        f.   Inadequate cortisol suppression in LDDST in PDH, FAN, or non-adrenal disease (p. 816–817)
        g.   Inadequate cortisol suppression in HDDST in PDH, FAN, or non-adrenal disease (p. 816–818)
        h.   Escape from suppression in LDDST or HDDST (p. 816–818)
        i.   Adequate cortisol suppression in PDH or non-adrenal disease (p. 816–818)
        j.   Exaggerated cortisol response to ACTH in PDH, FAN, or non-adrenal disease (p. 820)
        g.   Poor cortisol response to ACTH in FAN (p. 820)
        h.   Normal cortisol response to ACTH in PDH, FAN, or non-adrenal disease (p. 819–820)
        i.   Aldosterone concentrations within reference intervals, decreased, or increased in adrenal disorders (p. 824–826)

76.   Explain, list, or recognize the reasons for:
        a.   Falsely low ACTH concentrations if sample is not handled properly (p. 807–808)

Major Objectives for Chapter 19: Cavitary Effusions

77.   Interpret results of the analysis of cavitary effusions
        a.   Routine analysis: color, clarity, TP_{ref}, TNCC, and results of microscopic examination
        b.   Special analyses: concentrations of cholesterol, triglyceride, urea, creatinine, Na^+, Cl^-, K^+, lactate, glucose, and bilirubin

78.   Explain, list, or recognize the physiologic or pathologic processes or mechanisms that cause the following:
        a.   Protein-poor transudates in cirrhosis and protein-losing nephropathies (p. 841–843)
        b.   Protein-rich transudates in heart failure (p. 843)
        c.   Exudates due to infections (p. 843–844)
        d.   Exudates due to non-infectious disorders (p. 843–844)
        e.   Hemorrhagic effusions (p. 844–845)
        f.   Lymphorrhagic effusions (p. 845–846)
        g.   Chylous effusions (p. 846)
        h.   Effusion of uroperitoneum (p. 846–847)
        i.   Effusion of bile peritonitis (p. 843–844)
        j.   Effusions associated with neoplasms (p. 843–844)

79.   Explain, list, or recognize the reasons for:
        a.   Falsely increased TP concentration in chylous effusion (p. 849)
        b.   Falsely increased L-lactate concentrations due to delayed analysis of an effusion (p. 859)
        c.   Falsely decreased glucose concentrations due to delayed analysis of an effusion (p. 860)