1. The process of informed consent involves that the patient or the patient’s surrogate:
   a. Is/are competent.
   b. Have comprehensible information about the medical situation and treatment.
   c. Have information about alternative treatments and their consequences, and have an understanding of what they have learned.
   d. All of the above.
   e. Only A and B.

2. Children who can make their own decisions and give consents for medical treatment separate from their parents include:
   a. Children who are pregnant or are already parents.
   b. Children who graduated from high school.
   c. Children who have joined the armed forces.
   d. Children who live separately and independent from their parents.
   e. All of the above.

3. Appropriate statements pertaining to the mature minor doctrine include all of the following except:
   a. A minor (child) should have the opportunity to accept or decline life-sustaining treatment, such as mechanical ventilation or dialysis.
   b. The child may refuse a blood transfusion that might otherwise be essential for appropriate medical care, if this is because of long-standing, well-thought beliefs, such as those held by adolescents who are Jehovah’s witnesses.
   c. The law does recognize that some children have legitimate independent claims regarding their medical care that may differ from the expressed wishes of their parents.
   d. This legal entitlement means that the proposed decision maker is actually competent.
   e. Physicians must assess the decision-making capacity of patients or their surrogates.

4. In order to give an informed consent, the patient or legal guardian must possess a decision-making capacity. This capacity has several features and elements that include all of the following except:
   a. The patient or surrogate does not need to have the ability to manipulate the information provided to them.
   b. Capacity includes the ability to deliberate about alternative options.
   c. Capacity to make medical decisions involves specific determinations for each significant decision.
   d. Capacity involves the ability to understand and communicate about the medical situation.
   e. Capacity involves the ability to make a choice among alternatives.

5. You are involved in the treatment of a critically ill child with sepsis and multiple organ dysfunction syndrome at a university children’s hospital. In this situation, all of the following actions and statements are true except:
   a. Parental religious beliefs should not prevent this child from receiving a clearly beneficial therapy.
   b. The best interest of the child should remain the guiding principle in most cases where there is any dispute with the parents.
   c. Treatment can go forward with permission from only one parent.
   d. When parents refuse involvement of trainees in the care of their child, the best course of action is to remind them that this is a teaching institution and proceed with the care with your trainees.
   e. Children may receive treatment by court approval over and against parental wishes, when the therapy constitutes the standard of care.

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6. In order for a patient to succeed in a claim for damages, he/she must prove:
   a. That the physician failed to meet the standard of care.
   b. That the physician’s error led to legally recognized injuries.
   c. That the physician’s error approximately caused the patient to suffer legally recognizable damage.
   d. All of the above.
   e. A only.

7. Regarding the doctor–patient relationship involved in a malpractice suit, which of the following statements is true?
   a. It is illegal to alter patient’s medical records at a later time even when a reason for an addition is indicated.
   b. It can be hard to deny charges that inappropriate care was provided when the medical record has little or no information.
   c. When a physician treats a patient with a chronic medical problem, he/she is liable for the entire problem even after one encounter.
   d. Most jurisdictions state that the physician is responsible for the patient’s noncompliance.
   e. All of the above.

8. In the United States, a 14-year-old who is healthy without any significant past medical history is most likely to die from:
   a. Suicide. d. Accident.
   b. Homicide. e. Brain tumor.
   c. Leukemia.

9. “Baby Doe” regulations include that health care providers cannot withhold medically beneficial treatment from a child on the basis of a handicap. An exceptions includes:
   a. If the infant is imminently dying.
   b. The treatment would be inhumane.
   c. The infant is permanently comatose.
   d. All of the above.
   e. A and B only.

10. You are called to the pediatric intensive care unit (PICU) as soon as possible because the parents of a 6-month-old child with Down’s syndrome and atri-ventricular canal defect in congestive heart failure are very angry at the staff, and are expressing dissatisfaction with the care provided to their child. Upon your arrival, you notice the father is indeed very angry and is asking to transfer his child to another institution. All of the following would be appropriate responses except:
    a. Listening to their concerns is one of the most effective interventions in dealing with this family.
    b. A team meeting with this family should be promptly convened.
    c. You should remind the parents that you and the staff were up all night taking care of this child, and at this point, everybody on the team is somewhat tired and exhausted.
    d. Accept the emotional outburst of the father calmly.
    e. Assure the parents that their child is being appropriately cared for and comforted.

11. Measures that can be taken to prevent hostility among parents, such as the ones in Question 10, include:
    a. Orientation to policies of the PICU as soon as possible after the admission of the child.
    b. Introduction of the staff soon after admission.
    c. If there is evidence that the parents are showing signs of dissatisfaction with the care, a team meeting with the family should be promptly convened.
    d. Family education to alleviate any knowledge deficit.
    e. All of the above.

12. A 12-month-old baby of a single mother, who was apparently being watched by the mother’s boyfriend, was admitted to your PICU for persistent seizures. Physical examination was significant for the presence of multiple retinal hemorrhages, and the computed tomography scan revealed intracerebral hemorrhage and a subdural hematoma. Correct statements pertaining to this case include all of the following except:
    a. A complete copy of the medical record is extremely helpful during the initial investigation.
    b. If you are asked to testify in court regarding this case, a monitory compensation is expected.
    c. Accidental injury, other than a car accident, rarely causes intracranial injury in infants.
    d. Remind your staff that the parents of this child should be treated in the same professional and supportive manner that is employed with the parents of any other critically injured child.
    e. In discussing this case with one of your residents who will be testifying in court, it is crucial to remember that most physicians are unprepared by training and experience to go to court as expert witnesses.
CHAPTER 1: RESPIRATORY SYSTEM

1. E The Murphy eye side hole does not provide protection against obstruction of the endotracheal tube. The incidence of tube obstruction is approximately 5% in the pediatric population, and approximately 80% of tube obstructions occur in endotracheal tubes that are 3.5 mm in diameter or smaller. The channel on the straight blade is the visual pathway for the person performing the intubation. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 59–64.)

2. A Subglottic stenosis occurs in 2–6% of pediatric patients following tracheal intubation. (Parkin JL, et al. Ann Otolaryngology, 1976; 85:673.)

3. E All are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 65–76)

4–5. D, E Postextubation croup occurs in approximately 5% of intubated children and usually resolves in 24 hours. It is more common in patients with frequent coughing episodes and in patients who move more frequently while intubated. It has been shown to be more prevalent in children 1–4 years of age, particularly in association with any type of surgery in the head/neck area. (Kemper, et al. Crit Care Med, 1991; 19:352.)

6–8. E, E, E The mortality rate for tracheostomy is 1–3%. The mortality rate and complications are highest in infants. Following tracheostomy, there appears to be an increase in airway secretions for 24–48 hours during which time the patient will need frequent suctioning. The patient will also need to be evaluated for possible air leak, such as subcutaneous emphysema or pneumomediastinum, and monitoring for postoperative bleeding. (Zeifouni A, et al. J Otolaryngology, 1993; 22:431-434; Crysdale, WS. Ann Otorhinolaryngology, 1988; 97:493.)

9. C The tracheostomy tubes, in fact, may measure 0.5 mm larger than the previously used endotracheal tube, because the site of insertion is below the cricoid cartilage. The initial change of the tracheostomy tube must be done with the surgeon in attendance as a precaution against complications. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 72–73.)

10. A This is a rare complication of prolonged tracheostomy, and it is most likely a result of erosion of the innominate artery. Under these circumstances, a cuffed tracheostomy tube should be passed beyond the site of bleeding and immediately inflated. (Crysdale WS. Ann Otorhinolaryngology, 1988; 97:493–499.)

11. B An anterior (and not a posterior) tracheal flap at the operation site for tracheostomy is one of the etiologies of obstruction following decannulation. Other etiologies include: fusion of vocal cords, granuloma, and temporary adductor failure. (Carter P, et al. Ann Otorhinolaryngology, 1983; 92:398–401; Sasaki CT, et al. Ann J Dis Child, 1978; 132:266–269.)

12. D Tracheostomy tubes are not plugged prior to decannulation, as this may increase the airway resistance significantly, and a tracheostomy stoma is usually left to heal on its own. Plastic tracheostomy tubes have been associated with less evidence of stricture and subsequent tracheal stenosis. Tracheostomy tubes are placed below the cricoid cartilage. (Sasaki CT. Am J Dis Child, 1978; 132:266–269.)
13. T, T The same principles applied for tracheal intubation in a patient with closed head injury should be applied here. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 65–68.)

14. D Contraindications to nasotracheal intubation include bleeding diatheses and suspicion of basilar skull fracture. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 65–68.)

15. C In a patient with closed head injury, one should avoid ketamine because it increases intracranial pressure, possibly through a cholinergic mechanism. In a setting of hypotension and shock, thiopental, particularly in the usual dose of 2–4 mg, should be avoided because it may potentiate hypotension, which might be detrimental to the patient. Vecuronium seems to cause minimal hemodynamic disturbances, and therefore, in combination with lidocaine and low-dose (1–2 mg/kg) thiopental would be the most appropriate combination in this patient. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 63–70.)

16. A In a patient with hypovolemia or shock, ketamine seems to be the most appropriate choice because it is a cardiovascular system stimulant, along with vecuronium, which is associated with minimal hemodynamic disturbances, and therefore, in combination with lidocaine and low-dose (1–2 mg/kg) thiopental would be the most appropriate combination. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 63–70.)

17. D With turbulent airflow, the resistance to airflow is proportionate to density. A helium–O2 (HeliOx) mixture has a lower density than an O2–nitrogen mixture. This leads to a reduced resistance to airflow. Use of an oxyhood is not recommended because helium tends to separate as a layer at the top of the oxyhood. It usually is given through a tight-fitting face mask. The ventilator transducer is calibrated with an air–O2 mixture, and therefore, with a HeliOx mixture, the tidal volume may not be accurate unless it is measured directly. (Kemper KJ. Crit Care Med, 1991; 19:356; Ellean C. J Pediatrics, 1993; 122:132–135.)

18. E The theory is that increased negative interstitial pressure is a contributing factor to the development of pulmonary edema in association with upper airway obstruction. To further review theories that explain the development of pulmonary edema in children with croup and epiglottitis, see the following references. (Travis KW, et al. Pediatrics 1977; 59:695; Lichtenstein S. Fed Proc 1975; 34:436.)

19,20. E, E Children who develop hyaline membrane disease or have pulmonary hypoplasia owing to a wide variety of reasons including diaphragmatic hernia, children with tracheoesophageal fistula, and those who develop early neonatal infections resulting from, but not limited to, group B streptococcal infection, ureaplasma, respiratory syncytial virus, or cytomegalovirus, seem to be at a higher risk of developing bronchopulmonary dysplasia. Other risk factors include male sex, white race, and a birth-weight of less than 750 g. (Kennedy KA. Semin Perinatol, 1993; 17:247.)

21. D Negative, rather than positive, pleural pressure has a tendency to promote formation of pulmonary edema. All other factors in the question tend to promote pulmonary edema. (Robin ED. N Engl J Med, 1973; 288:239.)

22. E Refer to answers 19–20.

23–24. E, D Air within the connective tissue sheath leads to compression of the surrounding peripheral airway with subsequent increased airway resistance and hyperinflation. Impaired lymphatic drainage promotes pulmonary edema. Once extra-alveolar air develops, it may dissect into the subcutaneous space and mediastinum. Further extension into the pericardium and peritoneum may occur. The primary event appears to be epithelial necrosis. (Watts, JL. Pediatrics 1977; 60:273; Hansen TN. Clin Perinatol 1984; 11:653.)

25. D Infants with bronchopulmonary dysplasia (BPD) have been shown to have a blunted arousal response to hypoxia. Increased chest wall compliance places these infants at a mechanical disadvantage, particularly during periods of decreased or low intercostal muscle activity, such as during rapid eye movement during sleep. The peripheral chemoreceptors are intact in these babies. Prolonged ventilatory support may lead to disuse atrophy of respiratory muscles. (Gray M. Pediatrics 1988; 82:59; Knosely AS. J Pediatr 1988; 113:1074.)

26. E Normally, the blood flow through the right coronary artery occurs during both diastole and systole,
as opposed to the blood flow through the left coronary artery, which occurs primarily during diastole. In infants with bronchopulmonary dysplasia, with the development of pulmonary hypertension and particularly with progressive pulmonary hypertension, the blood flow through the right coronary artery becomes limited to diastole as right ventricular pressure and volume increase. (Berman W. Pediatrics, 1982; 70:708.)

27. A Infants with BPD have been shown to develop a significant reduction in pulmonary vascular resistance in response to low flow oxygen therapy. Acute, recurrent hypoxia precipitated by a variety of factors such as handling, feeding, or infection may precipitate pulmonary hypertension or pulmonary hypertensive crises with sudden death. (Long, LA. Pediatrics, 1980; 65:203. Grag M. Pediatrics, 1988; 81:635.)

28–30. C, C, C Improved mucociliary clearance is a recognized effect of β₂-agonists. Methylxanthines increase chemoreceptor sensitivity to carbon dioxide and induce hyperthermia rather than hypothermia. (Santa-Cruz R. Am Review of Resp Dis 1974; 109:458; Aranda JV. Clin Perinatol, 1979; 6:87.)

31–32. D, D Diuretics cause decreased transvascular efflux of fluid in the lung, and have been associated with improved survival in patients with BPD. Recognized side effects of furosemide include chloride depletion, metabolic alkalosis, renal calcification, and ototoxicity. Some of these factors have been implicated in poor growth and poor outcome in infants with BPD. (Perlman JM. Pediatrics, 1986; 77:212; Hurnagle KG. Pediatrics, 1982; 70:360.)

33–35. E, D, D Respiratory acidosis, hyperinflation, disuse atrophy from prolonged mechanical ventilation, and tracheal intubation have been associated with decreased respiratory muscle capacity. Advantages of tracheostomy include a stable airway with more freedom of mobility and oral stimulation. Tracheostomy decreases anatomic dead space, and therefore is unlikely to lead to elevation of carbon dioxide. It also decreases work of breathing partly through the same mechanism. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 107–108.)

36. D Use of pulmonary vasodilators would lead to ventilation–perfusion mismatch which is likely to increase the dead space. Allowing the patient’s spontaneous respiratory rate to have a higher contribution to the total ventilatory support while on mechanical ventilation will decrease dead space, as does tracheostomy. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 183–186.)

37. B Self-explanatory. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 97–98.)

38. A Diffusion defect as the only cause of gas exchange abnormalities is extremely rare. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 97–98.)

39. D Diaphragmatic hernia, if not detected and corrected before 16 weeks of gestation, will lead to irreversible changes in the lung; in this case, the left lung, which is expected to remain hypoplastic. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 105–106.)

40. E Canals of Lambert do not develop until approximately 6 years of age. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 105–106.)

41. A, B The intra-alveolar Pores of Kohn do not develop until after 2 years of age. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 105–106.)

42. E Dead space ventilation = alveolar ventilation × (alveolar CO₂ – exhaled CO₂) ÷ alveolar CO₂. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 107–108.)

43. C Dead space ventilation = (arterial CO₂ – exhaled CO₂) ÷ arterial CO₂ × alveolar ventilation. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 107–108.)

44. B The normal ratio of dead space ventilation to alveolar ventilation is 0.3 or less. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 107–108.)

45–47. A, B, C Expiratory braking refers to the increase in airway resistance in the upper airway during exhalation, which leads to an increase in end expiratory
l lung volume. This would lead to an increase in functional residual capacity (FRC). It is decreased during active sleep because it is arousal-dependent. Specific compliance and specific conductance are the same for adults and children. (Kosch PC, Stark AR. J Appl Physiol, 1984; 57:1126–1133.)

48. B Time constant = resistance \times compliance. Whenever one of the components of the time constant (i.e., either the resistance or the compliance) increases, the movement of air from one lung unit to another would be prolonged, leading to an increase in the time constant. Therefore, applying these principles in the diagram, because the resistance in the airway leading to unit A is increased, and the compliance of unit C is also increased, these two units will contain less volume of gas when inflation is interrupted prematurely. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 104–106.)

49. B Diaphragmatic hernia adversely affects the pulmonary vasculature and lead to pulmonary hypoplasia if it is not corrected before 16 weeks of intrauterine life. (Please see Answer 40.)

50. C Regional or localized hypoxic pulmonary vasoconstriction does not increase pulmonary vascular resistance significantly. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 106–112.)

51. E All statements are examples of a shunt. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 110–112.)

52. D The alveolar air exchange equation makes all of the above assumptions. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 110,111.)

53. D Cardiac output equals oxygen consumption divided by arteriovenous oxygen content difference, and therefore, if oxygen consumption increases for a constant cardiac output, the mixed venous oxygen content must decrease. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 108,109.)

54. D This is the major mechanism (i.e., low perfusion/ventilation [V/Q] segments) in adults. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 111,112.)

55. E Transfer factor decreases with age. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 112,113.)

56. D Hemoglobin (Hb)-F is more easily oxidizable compared to Hb-A. (Martin H, et al. Nature, 1963; 200:898–900.)

57. D Neonates and young infants are more susceptible to the development of methemoglobinemia because (1) the iron in Hb-F is oxidized more readily; and (2) the young infant is relatively deficient in the enzyme, met-Hb reductase. When the levels of met-Hb exceed 30–40%, cyanosis and symptoms of decreased O₂ transport are noted. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 114–116.)

58. D Resting oxygen consumption in a 1-week-old infant is three times that of an adult based on the body weight per kilogram. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 118.)

59. C O₂ consumption (VO₂) = Cardiac Output

\[ \text{CaO₂ – CVO₂} \]

Where \( \text{CaO₂} \) = arterial O₂ content.
\( \text{CVO₂} \) = mixed venous O₂ content.
\( \text{O}_2 \) delivery (DO₂) = Q \times \text{CaO₂}
\( Q = \text{Cardiac Output} \)
\( \text{CaO₂} = \text{Hb (grams\%) \times 1.34 \times O₂ saturation} + \text{PaO₂ \times 0.003} \)

Therefore alterations in cardiac output or peripheral circulatory disturbances (that alter blood flow at the capillary level) will affect O₂ consumption. \( P_{50} \) affects the unloading of O₂ from Hb. The higher the \( P_{50} \), the more the unloading of O₂ to tissue. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 118.)

60. B Peripheral chemoreceptors respond to a falling oxygen saturation in a linear fashion by increasing the inspired minute ventilation. There is an exponential increase in minute ventilation as PaO₂ falls, particularly at PaO₂ less than 60 torr. (Berger AJ, et al. N Engl J Med, 1977; 297:194–198.)

61. D Laryngeal and bronchial receptors respond to increasing CO₂ in a linear fashion. (Berger AJ, et al. N Engl J Med, 1977; 297:194–198.)
62. D The carbon dioxide response curve which relates alveolar CO₂ to alveolar ventilation is shifted to the left in the neonate. (Rigatto H. Apnea. Pediatr Clin North Am, 1982; 29:1105.)

63. D Preterm infants have a characteristic breathing pattern referred to as periodic breathing (i.e., pauses in respirations lasting 5–10 seconds). Owing to the higher O₂ demand, newborn infants compensate by having a higher minute ventilation and a shift in the CO₂ response curve to the left. The carotid bodies are present in preterm infants. (Rigatto H. Pediatr Clin North Am, 1982; 29:1105.)

64. D Total respiratory system compliance equals lung compliance plus chest wall compliance. With age, there is a progressive reduction in chest wall compliance which accounts for a reduction in the total respiratory system compliance. (Sharp JT, et al. J Appl Physiology, 1970; 29:775–780.)

65–67. D, D, C Closing capacity (CC) is the lung volume below the FRC where alveoli in dependent lung regions have a tendency to collapse.

In infants, CC is often equal to or greater than FRC, and therefore, tidal breathing often takes place in the range of CC. This phenomenon is a result of the very low elastic recoil of the chest, and it increases the risk of atelectasis. (Smith CA. The Physiology of the Newborn Infant. Springfield, IL, 1976; pp. 206–207.)

68–69. D, D This may result in ischemia of respiratory muscles at a high respiratory rate. Low levels of sarcoplasmic reticulum in the fetal diaphragmatic muscle have been observed. (Maxwell LC, et al. J Appl Physiol, 1983; 54:551.)

70. B Babies who were born prematurely continue to be at high risk of apnea postoperatively (following general anesthesia) and therefore, should be monitored for 24–48 hours after anesthesia. Aminophylline will increase breathing without significantly altering the CO₂ and pH around the respiratory center. It appears to increase the sensitivity of the respiratory center to carbon dioxide. Patients with adenotonsillar hypertrophy who undergo surgical resection may be admitted to the pediatric intensive care unit (ICU) because of airway obstruction from postoperative edema or sometimes owing to decreased ventilatory drive after anesthesia. The increased opioid activity found in the spinal fluid in these patients may be a contributing factor to decreased ventilatory drive noted perioperatively. (Kurth CD, et al. Anesthesiol, 1987; 66:483; Gislason T, et al. Chest 1989; 96:250; Lavaher S. Thorax, 1989; 44:121.)

71. E Work of breathing is increased because of chest wall distortion secondary to instability of the chest wall. (Robotham JL. Crit Care Med, 1979; 7:563.)

72. D The more compliant chest wall of the young child contributes to the clinical manifestation of diaphragmatic paralysis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 123, 247.)

73. C The upper airway contributes, to a much higher degree, to total respiratory resistance in children than in adults. This may mask the physiologically more important airway resistance. (Cook LD, et al. J Clin Invest, 1957; 36:440)

74. B, A The relationship between alveolar ventilation and both PaO₂ and PaCO₂ are nonlinear, as depicted on the graph. (Benumof J. In: Miller RD. Anesthesia, Churchill, Livingstone, NY 1981; pp. 699.)

75. B West Zone I occurs when ventilation is wasted. Alveolar pressure remains constant, whereas pulmonary artery pressure tends to increase from apex to base in the erect posture. Hyperinflation, pulmonary embolus, and shock all lead to a decrease in pulmonary blood flow, with consequent wasting of ventilation. (Benumof J. Anesthesia, Churchill, Livingstone, NY 1981; pp. 699.)

76. C The so-called West Zone 4 of the lung develops when there is interstitial edema, and under those circumstances, there will be less transduction of fluid across the capillary membrane. (Benumof J. Anesthesia, Livingstone, NY 1981; pp. 699.)

77. A Pressure = Flow × Resistance (i.e., mean pulmonary pressure = CO × pulmonary vascular resistance). (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 138.)

78. A, B Regional hypoxic pulmonary vasoconstriction does not result in significant elevation of pulmonary artery pressure, and it seems to be a protective
mechanism for the host. (Fishman AP. Civ Res, 1976; 38:221.)

79,80. B, E Compliance of the chest wall is described in option D. Bronchiolitis primarily affects the airway and, unless associated with significant pneumonia, it does not increase the elastic recoil of the lungs. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 138,139.)

81. B Dynamic compliance is smaller than static compliance because dynamic compliance is equal to the change in volume divided by peak inspiratory pressure minus positive end expiratory pressure (PEEP), as opposed to static compliance, which equals volume divided by plateau pressure minus PEEP. Because peak inspiratory pressure is greater than the plateau pressure, the dynamic compliance would be smaller than the static compliance. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 138,139.)

82. B All other conditions are associated with decreased compliance. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 138,139.)

83. A In fact, airway resistance accounts for more than 80% of nonelastic resistance. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 140–142.)

84. B Time constant is the product of compliance (C) and resistance (R). Mathematically, 63% of lung inflation or deflation occurs with one time constant. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 105,106.)

85. B Hyperinflation leads to increased physiological dead space. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 1992; pp. 142,143.)

86, 87. E, B Pulmonary edema is more likely to lead to a decrease in functional residual capacity rather than an increase in closing capacity. Elimination of secretions and use of effective bronchodilators are useful strategies to improve closing capacity.

88. A Because blood flow falls more dramatically than ventilation from the base of the lung toward the apex of the lung, the ventilation perfusion ratio increases exponentially as one moves up the lung. (West JB. Ventilation/Blood Flow and Gas Exchange, 3rd Edition; Oxford, Blackwell Scientific, 1977; p. 30.)

89, 90. B, D A significant portion of the tidal volume dissipates when the compliance of the ventilatory circuit is high. Patient’s compliance and resistance also affects the actual delivered tidal volume. The exhalation valve is usually kept close to the airway opening in order to minimize the circuit volume. (Rogers MC. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 147–150.)

91–93. A, B, D In clinical medicine, carbon monoxide (CO) poisoning is probably the most common application of hyperbaric O2 therapy. The half-life of CO is actually decreased to 23 minutes at 3.0 atmospheric pressure, as opposed to 180 minutes with 100% oxygen at the normal atmospheric pressure. Sixty to 90 minutes of hyperbaric oxygen at 2 to 2.5 atmospheric pressure seems to be safe, without significant central nervous system (CNS) toxicity, although other side effects mentioned in the question are possible. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 156,157.)

94. E With turbulent airflow, the resistance to airflow is proportionate to density (as opposed to viscosity with laminar flow). Because helium is not as dense as nitrogen, it has a beneficial role in patients with upper airway obstruction, such as croup. More recently, the HeliOx mixture has also been shown to improve gas exchange in patients with acute asthma with or without ventilatory support. HeliOx mixture minimizes work of breathing by altering the resistance to airflow. HeliOx mixture is most beneficial at 80:20 or 70:30 ratios. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; p. 157.)

95. A The flow through the ventilator circuit is set at 8 L/minute. Therefore, \(8000 \div 60 \times 0.5 = 66 \div 6 = 11\) mL/kg.

96. C Most of gas exchange takes place during exhalation. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 156–159.)

97–99. D, A, A The inspiratory pressure relief valve is housed in the expiratory limb of the circuit in these ventilators. Actually, in order to minimize work of breathing, the inspiratory gas flow in the continuous
flow circuit should meet the patient’s inspiratory flow rate demand. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 156–159.)

100. A In the pressure support mode of ventilation, the length of the cycle, as well as depth and flow characteristics, are determined by the patient. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 156–159.)

101, 102. D, E All three options are methods of providing an inverse ratio ventilation, which has been used successfully to improve oxygenation and ventilation at a reduced peak inspiratory pressure. During inverse ratio ventilation, the tidal volume is a function of multiple factors, some of which are enumerated in the question. (Tharralt RS, et al. *Chest*, 1988; 94:755.)

103. C The decrease in oxygen delivery associated with elevation of PEEP is usually responsive to adequate fluid resuscitation and inotropic support, unless one is using extremely high levels of PEEP. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 297–300.)

104. D Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 184.

105. E All of these factors are operative when it comes to the adverse hemodynamic effects of PEEP. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 186.)

106. E Unless they progress to a tension pneumothorax or a tension pneumoperitoneum, none of the manifestations of barotrauma mentioned are usually clinically significant (i.e., do not require immediate intervention). (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 186–188.)

107. B During tension pneumothorax, the intrapleural pressure is consistently higher than the atmospheric pressure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 307–309.)

108. B Veno-venous extracorporeal life support (ECLS) usually requires a higher rate of flow because of the recirculation of the previously oxygenated blood. This is true when the pulmonary bed is totally non-functional. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 317,318.)

109. B, A, A Veno-venous ECLS maintains pulmonary blood flow with oxygenated blood, but it does not assist the systemic circulation. On the other hand, veno-arterial ECLS does assist the systemic circulation and it also tends to decrease the pulmonary artery pressure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 317,318.)

110. C The oxygen saturation that is obtained using pulse oximetry is called a functional saturation, and the pulse oximetry obtains the ratio of oxy-Hb divided by the total Hb. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 333–336.)

111. C Methylene blue absorbs light maximally at 668 nm. The pulse oximeter interprets this extra absorbance as reduced Hb, and therefore a lower oxygen saturation is obtained. The oxygen saturation obtained by the pulse oximetry could drop dramatically within 30 seconds of an intravenous administration of methylene blue, and it remains reduced for approximately 2 minutes. (Scheller M. *Anesthesiology*, 1986; 65:550.)

112. A Carboxy-Hb and met-Hb produce these findings. (Barker SJ. *Anesthesiology*, 1987; 66:677.)

113. D With an increase in met-Hb concentration, the saturation on the pulse oximeter decreases and plateaus at approximately 85%. Met-Hb absorbs light significantly at both 660 nm and 940 nm wavelengths, thereby confusing the pulse oximeter photo detector into believing that both oxy-Hb and reduced Hb are increased. This results in increases in both the denominator and numerator. As this happens, the microprocessor-driven algorithm of the red absorbance and infrared absorbance approaches unity and this gives rise to a saturation of approximately 85% on the calibration curve. Hyperbilirubinemia does not interfere with reading of the pulse oximetry. (Barker SJ, et al. *Anesthesiology*, 1988; 68:279.)

114, 115. D, E In the presence of normal oxygen extraction and utilization by the tissue, an increase in oxygen delivery will not result in decreased mixed-venous oxygen saturation. Increased oxygen consumption leads to a decrease in mixed venous oxygen
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saturation, and not vice versa. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 210,211.)

116. D This capnogram reveals irregularity in the pattern of the exhalation of the CO2 which most likely reflects irregularity in the pattern of breathing of this patient. Adding 20 cmH2O of pressure support will decrease the work of breathing by overcoming the work that is necessary to open the demand valve mechanism that is operating in this ventilator. It will also help to overcome some of the resistance of the endotracheal tube. (Carlon G, et al. Crit Care Med, 1988; 16:550.)

117. E Hypoventilation is likely to lead to a gradual increase in the level of end tidal CO2. All other clinical conditions indicated in the question lead to a sudden decline in end tidal CO2 levels. (Carlon G, et al. Crit Care Med, 1988; 16:550.)

118. A The presence of a gas bubble in a syringe will usually affect the PaO2. The effect on the PaO2 will depend on the amount of oxygen that is inspired by the patient. In patients on room air, this will lead to a false elevation of PaO2 (atmospheric PO2 is usually higher than alveolar PO2). On the other hand, in patients who are receiving a high fraction of inspired oxygen and have normal lungs, the presence of an air bubble in a syringe may spuriously lower the PaO2. Excess heparin does lead to a drop in PaCO2, but usually there are no changes in the pH level because it is neutralized by the acidity of heparin. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 353–359.)

119. E Reticulocytes and band forms are highly metabolic immature cells that are most likely to lead to a change in the blood gas results. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 355–359.)

120. D Aspirin, especially with overdose, is likely to lead to high anion gap metabolic acidosis (HAGMA). All other drugs do not. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 361.)

121. D Other abnormalities of the CNS, esophagus, and cardiovascular system have been reported in association with choanal atresia. Therefore, evaluation for possible other anomalies should be done in patients with posterior choanal atresia. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 231–233.)

122. D Nasal obstruction is usually seen when the mass is located at the base of the brain. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 231–233.)

123. E All are features of angiofibroma. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 231–233.)

124. A, A, B, B Vocal cords in infants are concave, the anterior attachment to the trachea is lower, and the glottis is located higher in the neck compared with an adult. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 231–233.)

125. C At 4–6 months of age, the epiglottis loses contact with the soft palate and assumes a more erect posture, allowing oral (mouth) breathing. The lateral diameter of the newborn glottis is only about 4–5 mm, and at birth the trachea is approximately 5–7 cm in length. The glottis assumes the adult location at C6 by about 12 years of age. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 231–233.)

126. A (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 231–232.)

127. B \[\pi R^2 = 16 \pi\] when the diameter is 8 mm which gives rise to a radius of 4 mm. With a uniform 1 mm reduction in the size of the airway, this will decrease the diameter from 8 to 6 mm, and decrease the radius from 4 to 3 mm. Now \[\pi R^2 = 9 \pi, \ 9 \div 16 = 54\%\], which means that the diameter of the airway has been decreased by 44%. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 231–233.)

128. E All of these are measures that may be needed to intervene with laryngospasm. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 233–234.)

129. B Congenital anomalies are the most common cause of chronic stridor in children less than 2 years of age. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 235–238.)
130. C Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 235–238

131. C Laryngotracheomalacia is characterized by normal voice. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 236.)

132. C, C Both laryngomalacia and airway or subglottic hemangioma usually present with symptoms before 6 months of age. In both cases, the treatment is conservative because in most cases, the problem resolves spontaneously by the end of the 2nd birthday. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 235–238.)

133. E All of these conditions pose difficult airway management. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 235–238.)

134. D All of the choices are complications that may be noted in the postoperative period following repair of cleft lip and cleft palate. Occasionally, bronchospasm is also seen. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 235–238.)

135. C Both these conditions are characterized by macroglossia with a short neck, which combine to produce a difficult airway. Both of these conditions belong to the mucopolysaccharidoses. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 241.)

136. E Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 241.

137. A The typical age for this condition is younger than 3 years. It is important to obtain inspiratory radiographs to evaluate the thickness of the retropharyngeal soft tissue. Measurement of this soft tissue is important in the diagnosis of the retropharyngeal abscess. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 242.)

138. B In fact, frequent tracheal suctioning is necessary in these patients to prevent airway obstruction because the infection/inflammation induces an increase in airway secretions. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 242–245.)

139. D These are the indications for extubation in a patient with a viral croup. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 244–246.)

140. D The amount of subcutaneous emphysema of the neck area does not correlate with the severity of airway injury. Nasotracheal intubation should be avoided in patients with midfacial fractures, and also in patients suspected of having a fracture of the base of the skull. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 245–248.)

141. A Seventy to eighty percent of subglottic stenosis occurs following endotracheal intubation. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 245–248.)

142. E The accepted duration of time for intubation to prevent subglottic stenosis is unknown. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 245–248.)

143. D A HeliOx mixture in various combinations has been shown to be effective in the management of postextubation stridor and burn victims with significant stridor. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 245.)

144. D Most of these airway papillomas resolve by the teenage years. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p 252.)

145. A These patients respond to relatively low concentrations of inspired oxygen. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 258–260.)

146. D High antidiuretic hormone (ADH) levels in association with elevation of renin has been reported in patients with bronchiolitis. (Gozal D, et al. Pediatr Res, 1990; 27:204–209.)

147. C High ADH in association with high aldosterone levels has been reported in patients with respiratory syncytial virus bronchiolitis. Because of this combination of hormonal abnormalities, there is a decrease in urine output associated with a normal urine sodium concentration. (Gozal D, et al. Pediatr Res, 1990; 27:204–209.)
Maximum mid-expiratory flow rate is one of the flow volume parameters that demonstrates the most severe decrease during an attack of asthma. This is also the parameter that is the last to improve following treatment for acute asthma. Patients with asthma, particularly those that are in status asthmaticus, have an increased residual volume. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 264–270.)

Transmural pressure = intraluminal pressure – extraluminal pressure. With higher negative inspiratory pressure, as seen with status asthmaticus, there is an increase in afterload during inspiration with a subsequent decrease in left ventricular output, which is followed by a sharp increase in left ventricular output during subsequent expiration. This leads to the phenomenon of pulsus paradoxus (PP). A decrease in PP may indicate an improvement in the patient’s condition (i.e., a smaller fall in pleural), but it may also indicate the patient’s fatigue and worsening clinical condition. Another factor that contributes to PP is ventricular interdependence, which can be exaggerated by the pulmonary hypertension, as it may be seen with severe status asthmaticus. The hypoxia that is seen during status asthmaticus results from V/Q mismatch, excessive O\textsubscript{2} requirement secondary to increased metabolic demand, and a degree of interstitial edema. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 268–270.)

The degree of hypoxemia does not correlate with the degree of airway obstruction as assessed by the reduction in forced expiratory volume in 1 second (FEV\textsubscript{1}). (McFaden ER, et al. *N Engl J Med*, 1968; 278:1029.)

As the FEV\textsubscript{1} drops below 20% predicted, PCO\textsubscript{2} rises, hypoxemia occurs, and pulsus paradoxus is present in almost all of these patients. (McFaden ER, et al. *N Engl J Med*, 1968; 278:1029.)

0.3 liters $\rightarrow$ 1 PSI

$\chi$ liters $\rightarrow$ 1100 PSI

therefore:

$\chi = \frac{1100 \times 0.3}{1} = \frac{330 \text{ L}}{4 \text{ L/minute}} = 82.5 \text{ minutes}$

The lungs emerge as a bud from the pharynx at day 26 following conception. This bud elongates, separates from the esophagus, and continues to divide to form the main bronchi. Extensive subdivision in the pseudoglandular stage leads to formation of the conducting airway, the most peripheral of which are the terminal bronchioles, which give rise to respiratory bronchioles and alveolar ducts during the canalicular stage. During this later stage, the acinus is formed. An acinus is the gas exchange unit associated with a single-terminal bronchiole, and will eventually contain three orders of respiratory bronchioles: alveolar ducts, alveolar sacs, and alveoli.

The Saccular stage was formerly thought to be the last stage of lung development prior to birth. However, because alveoli form before birth, the termination of this period is now arbitrarily set at 35–36 weeks’ gesta-
At the beginning of this phase (28 weeks’ gestation) the terminal structures are called saccules. They are cylindrical structures with a smooth wall. They become subdivided by ridges called secondary crests. Further subdivision between crests results in small spaces termed subsaccules. Exactly when these subsaccules can be termed alveoli is a matter of debate. The range of timing is between 29–36 weeks’ gestation. Most of postnatal formation of alveoli occurs over the first 1–1.5 years of life. Pores of Kohn are not established until several years after birth. (Langston C, Kida K, Reed M, et al. Am Rev Resp Dis, 1984; 129:607.)

160. B \[\text{PaO}_2 = \frac{\text{PiO}_2 - \text{PaCO}_2}{\text{RQ}}\]
\[= \frac{(747 - 47) \times 0.4 - 40}{0.8}\]
\[= 280 - 50 = 230\]
Alveolar arterial O2 gradient = PAO2 – PaO2
\[= 230 - 100 = 130\]

(Kandra TG, Rosenthal M. Int Anesthesiol, 1993; 31:119–121.)

161. B Please see Jodka PG, Heard SO. Int Anesthesiol, 2000; 35:1–10.

162. E Bronchopleural fistulae (BPF) can result from blunt trauma, barotraumas, or inflammatory diseases of the lung. Patients with BPF can present acutely because of pulmonary flooding or tension pneumothorax, or subacutely with an insidious clinical course. A persistent air leak without evidence of technical problem in the pleural drainage apparatus also indicates a BPF. Several techniques can be employed using bronchoscopy to localize the proximal endobronchial site of the fistulous tract. Occasionally, air bubbles can be seen emanating from the segmental bronchus. Washing the suspected segment with saline and coughing may accentuate the bubbling. Techniques for obliteration of the fistula bronchoscopically have also been described. (McManigle JE, et al. Chest, 1990; 97:1235–1238.)

163. C After a delay of 2–8 minutes, intramuscular ketamine (4–8 mg/kg) produces anesthesia for 20–40 minutes. More than 90–92% of ketamine is absorbed after an intramuscular injection. (White PF, Way WL, Trevor AJ. Anesthesiology, 1982; 56:119.)

164. D The cellular proliferative phase, after alveolar injury, is characterized by type II cellular hyperplasia, which appears to be a reparative process. These cuboidal cells may virtually cover the entire alveolar surface. They will later transform into the thin, type I alveolar epithelial cells. (Royall JA, Matalon S. In: Fuhrman BP, Zimmerman JJ. Pediatric Critical Care, Mosby Yearbook 1992, pp. 445–456.)

165. A Tachypnea is the earliest sign of respiratory muscle fatigue. As a compensation for the decrease in efficient tidal volume, the respiratory rate increases in an attempt to maintain minute ventilation. (Nunn JF. Applied Respiratory Physiology, 3rd Edition. Boston: Butterworth, 1987, p. 109.)

166. C CO2 binds with deoxy-Hb to form carbamino-Hb, which is one of the forms in which CO2 is transported in the blood. However, only 10% of CO2 in blood is transported in this form. Myoglobin approaches full saturation at a PO2 of 15–30 mmHg, which is the level pertaining to voluntary muscle. The bulk of its oxygen may be released only at very low O2 tension. 2,3-Diphosphoglycerate decreases the affinity of O2 for Hb, and thus, facilitates release of O2 to tissues and so does carbon dioxide. This latter phenomenon is also known as the Bohr effect. O2 binds to one of the six coordination bonds of the iron atom. Hydrogen binds to the imidazole ring of histidine on the globin chains of the Hb molecule. (Nunn JF. Applied Respiratory Physiology, 4th Edition; pp. 273–275; Guyton AC. Textbook of Medical Physiology, 8th Edition; pp. 440–442.)

167. E Increasing PEEP will diminish left-to-right shunting by increasing the pulmonary vascular resistance. All other measures stated in the question would increase left-to-right shunt flow. (Meliones JN, et al. Respiratory Support in Infants & Children; p. 352.)

168. D Linoleic acid is the primary precursor of arachidonic acid. (Abman S, Stenmark K. Am J Physiology, 1992; 262: L214.)

169. C \[\text{O}_2 \text{ delivery (DO}_2\text{)} = \frac{\text{Cardiac output (CO) } \times \text{Arterial O}_2 \text{ Content (CaO}_2\text{)}}{\text{Hb (gm%)} \times 1.34 \times \text{O}_2 \text{ Sat} + \text{PaO}_2 \times 0.003}\]

In this case, increasing the Hb from 9 to 14 gm% will increase O2 delivery the most. (Fahey JT, Lister G. In:
170. **A** J receptors act to stimulate breathing.

171. **C** Pulmonary capillary endothelial damage is one of the earliest changes in ARDS. Capillary congestion occurs with intraluminal aggregation of platelets, fibrin, and neutrophils. Pulmonary capillary endothelial cells undergo swelling and focal necrosis with destruction of mitochondria, endoplasmic reticulum, and ribosomes during the first few hours of ARDS. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 297, 298.)

172. **A** The Mapleson D breathing circuit (shown in figure below) can be described as a T-piece with an expiratory limb. The fresh gas inlet is located near the patient, and the expiratory pressure release valve is near the reservoir bag. The pressure release valve opens as pressure increases during expiration and a portion of the expired gas along with fresh gas is released into the atmosphere. During the next inspiration, the patient receives a combination of fresh gas and the exhaled gas. The content of this inspired gas is determined by:

1. Rate of fresh gas flow: A fresh gas flow more than two times the minute ventilation prevents rebreathing.
2. Patient’s tidal volume: the amount of rebreathing increases as the tidal volume increases.
3. Duration of expiration: a short expiratory pause provides inadequate time to flush the alveolar gas (occurs with faster respiratory rate); this allows rebreathing.

![Mapleson D breathing circuit diagram](Barash PG, Cullen BF, Stoelting RK. *Clinical Anesthesia*, 2nd Edition; pp. 654.)

173. **C** When peak airway pressure is allowed to increase to a level beyond that which is necessary to maximally distend the lungs, barotrauma and lung injury result. Because regional differences in lung resistance and compliance often coexist, maintaining a constant tidal volume may overdistend areas of the lung that are aerated if the remainder of the lung is collapsed. Similarly, maintaining a constant inspiratory flow pattern when regional differences in lung units exist will selectively increase distention of lung units with lower resistance. (Haake R, et al. *Chest*, 1987; 1:608.)

174. **D** Pulmonary conditions associated with decreased compliance, such as pulmonary fibrosis and ARDS or increased airway resistance such as bronchial asthma and chronic obstructive pulmonary disease (COPD), have the potential for being homogenous. This homogeneity can result in regional overdistention during positive pressure ventilation. Hyperinflation secondary to airway narrowing or collapse, such as seen with auto-PEEP, increases end-expiratory lung volume, but does not result in lung expansion of the hyperinflated lung units until airway pressure exceeds the level of auto-PEEP. Although the work of breathing during spontaneous breathing is increased by auto-PEEP, end-inspiratory lung volumes do not increase. (Bone RC, Stober G. Med Clin Noth Am, 1983; 67:599.)

175. **D** Changes in intrathoracic pressure correlate highly with changes in lung volume. Changes in intrathoracic pressure are independent of lung compliance. An increase in respiratory rate with lung conditions associated with increased expiratory airway resistance will result in dynamic hyperinflation, because there is inadequate time for exhalation. Examples are COPD, asthma, and other causes of intrathoracic airway obstruction. Thus, overdistention is possible with a fixed tidal breath or tidal volume. Because regional lung compliance, even in healthy individuals, is different under all conditions, uniform expansion of all lung units by positive pressure ventilation at any setting probably never occurs. (Marini JJ. In: Pinsky MR, Dhainaut JFA, Ed. *Pathophysiologic Foundations of Critical Care*, 1993; pp. 453–471.)

176. **B** Please see Marini JJ. In: Pinsky MR, Dhainaut JFA, eds. *Pathophysiologic Foundations of Critical Care*, 1993; pp. 453–471.

177. **A** Nitric oxide is synthesized from the amino acid arginine by the action of the enzyme nitric
oxide synthetase. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 36, 78, 111, 206.)

178. **D** Systemic-to-pulmonary shunt is often created in neonates and infants with an underlying cardiac defect in order to improve pulmonary blood flow and oxygenation. Examples are the (modified) Blalock-Taussig shunt that connects the subclavian artery to the pulmonary artery using a synthetic material, and the aortic to pulmonary window, which usually connects the ascending aorta to the pulmonary artery. Conditions that lead to a reduction in pulmonary artery pressure and pulmonary vascular resistance would increase the flow across the shunt with an increase in left-to-right shunt. Examples include: alkalosis, vasodilators such as hydralazine and nitroprusside, an increase in the concentration of inspired oxygen, and selective pulmonary vasodilators, such as nitric oxide. Interventions that lead to an increase in pulmonary vascular resistance, such as increasing PEEP, would lead to a reduction in pulmonary blood flow and a reduction in the left-to-right shunt. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 460.)

179. **B** Tachypnea in this infant would be the earliest evidence of inspiratory muscle fatigue. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 319–332.)

180. **C** Nitric dioxide is the toxic by-product. The rate of formation of this toxic product is dependent on the duration of contact between oxygen and nitric oxide. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 36, 78, 111, 206.)

181. **E** Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 157, 245–246.

182. **C** Histological features of infants with BPD include squamous metaplasia of the airway epithelium (large and small airways), increased peribronchial smooth muscle with fibrosis, submucosal edema, and inflammation with hypertrophy of submucosal glands. In the parenchyma, there are areas of fibrosis with atelectasis alternating with areas of hyperinflation, which, on gross examination of the lungs, has a cobblestone appearance. In more long-standing cases, there is diminution in alveolarization and surface area. The decrease in the number of alveoli probably reflects the onset of the insult with subsequent failure of the ability to regenerate new alveoli. This is associated with an increased number of small pulmonary arteries, which may contribute to pulmonary hypertension. The pulmonary arterial tree shows proliferation of the intima, smooth muscle hypertrophy, distal extension of smooth muscles, and adventitial thickening. (Abman SH, Groothius JR. Pediatr Clin North Am, 1994; 41, pp. 277–291.)

183. **C** Upper airway obstruction usually does not lead to an alveolar–arterial oxygen gradient. On rare occasions when upper airway obstruction is complicated by postobstruction pulmonary edema, this is possible. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 231–296.)

184. **E** The lungs have a tendency to collapse, while the chest wall has a tendency to move outward. Thus the elastic forces of the lung and the chest wall are in opposite directions. These two opposing forces are linked by the pleural surfaces and the net pressure is the intrapleural pressure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 145.)

185. **B** Bronchogenic cyst accounts for 5% of mediastinal masses. It is found in five major locations: right paratracheal region (20%); carinal region (51%); hilar region (9%); paraesophageal (14%); and pericardial/retrosternal. (Taussig LM. *Pediatric Respiratory Medicine*, 1999; pp. 1118.)

186. **E** Hysteresis refers to the failure of a system to follow identical paths of response during application and during withdrawal of a force. In the lungs, this is due mainly to surface properties and alveolar recruitment–derecruitment. In the chest wall, this is because of muscles and ligaments, both of which exhibit hysteresis. (Taussig LM. *Pediatric Respiratory Medicine*, 1999; pp. 100–101.)

187. **A** Increasing the length of muscle fibers (to a limited extent) would increase the force of contraction and thus the efficiency of the diaphragm. The diaphragm is most efficient at the lung volume that corresponds to the FRC, and thus increasing the end-expiratory lung volume above this does not improve the efficiency of the diaphragm. Increasing the radius of
curvature increases the efficiency of the diaphragm. The diaphragm of an infant has less radius of curvature than that of an adult, and is less efficient. (Fuhrman BP, Zimmerman JJ. Pediatric Critical Care, 2nd Edition; pp. 407.)

188. A Bronchoalveolar lavage in ARDS is characterized by predominance of polymorphonuclear leukocytes (PMNs), of 10 greater than 85%. (Reynolds HY. Am Rev Resp Dis, 1987; 135:250–263.)

189. B Massive hemoptysis is relatively uncommon in cystic fibrosis patients. It occurs in 10% of adolescents and adult patients with cystic fibrosis. Massive hemoptysis usually occurs from the bronchial circulation resulting from the higher systemic pressure compared with the pulmonary circulation. Often an untreated exacerbation of the disease is a triggering factor, but sometimes there is no clear cause. If hemoptysis persists, bronchial artery embolization is warranted; this requires bronchial arteriography. (Sweeney N, Fellows K. Chest, 1990; 97:1322–1326.)

190. B In infants, the continuous muscle tone of the thorax is important to maintain FRC, because the chest wall is very compliant and lacks the rigidity necessary to oppose the elastic recoil of the lung, which tends to lower FRC. With age, as chest wall compliance decreases and the chest wall becomes more rigid and capable to oppose the elastic recoil of the lungs, FRC increases. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 112–128.)

191. B A HeliOx mixture is less dense than a nitrogen–O2 (air) mixture. With turbulent flow (seen with upper airway obstruction, such as subglottic stenosis), resistance to air flow is proportional to density. A HeliOx mixture is useful in reducing the resistance to flow and work of breathing. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 275–276.)

192. B Refer to the answer for Question 182.

193. D $\text{PAO}_2 = (\text{BP} – \text{Vapor Pressure}) \times \text{FiO}_2 – \text{PaCO}_2 / \text{RQ}$

Because PaCO$_2$ and RQ are assumed to remain constant, they will remain the same under both situations: $(760 – 47) \times 0.27 = 192.51$

In order to keep the PaO2 the same, and therefore compensate for the same degree of alveolar–arterial oxygen gradient as the atmospheric pressure decreases, the alveolar oxygen tension must remain the same (i.e., 192.51). Therefore,

$\text{FiO}_2 = 192.51 / 632–47 = 0.3290$

(Reynolds HY. Am Rev Resp Dis, 1987; 135:250–263.)

194. B The function of this protein is to promote formation of a surfactant layer. It is, therefore, essential for effective reduction of the surface tension induced by surfactant. (Fuhrman BP, Zimmerman JJ. Pediatric Critical Care, 2nd Edition; pp. 382,383.)

195. E Type I alveolar cells are less in number than type II alveolar cells (which synthesize surfactant), but they cover a much larger area of the lung. Their primary function is to reduce the barrier to gas exchange. (Fuhrman BP, Zimmerman JJ. Pediatric Critical Care, 2nd Edition; pp. 445,446.)

196. C Forced vital capacity (FVC) is easily measured during spirometry. Data obtained from a specific patient can be compared with those from subjects who have the same height, weight, and age. FVC is highly reproducible and has a narrow range of normal values. It is affected in both obstructive and restrictive lung diseases. FVC may decline in the supine position by up to 20% in normal subjects and up to 38% in patients with underlying neuromuscular diseases. (Civeta JM, et al. Critical Care, 2nd Edition; pp. 565,566.)

197. A Work = Force × Distance. When it comes to the respiratory system, work is defined as the pressure that is generated by the respiratory muscle to move a particular volume of gas. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 2nd Edition; pp. 129,130.)

198. B Alveolar O2 tension = (Barometric Pressure – Vapor Pressure) × FiO$_2$ – PaCO$_2$ / RQ

$(760 – 47) \times 0.21 – 85 / 0.8$

$713 \times 0.21 – 106.25 = 43.38$

$\text{PAO}_2 – \text{PaO}_2 = 10$
Therefore: \( \text{PaO}_2 = 43 - 10 = 33 \text{ mmHg} \)

(Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 90.)

199, 200. E, A The half-life of a drug is function of clearance (CL) and volume of distribution (Vd) according to the following formula:

\[
\text{Half-life} = \frac{0.693 \times \text{Vd}}{\text{CL}}
\]

Thus half-life is affected not only by elimination, but also by Vd. For instance, during ECLS, most of the increase in the half-life is owing to an increase in the Vd, rather than a change in drug clearance. A drug’s half-life can also be used to determine the time it takes for the drug to reach a steady-state concentration, a state in which the amount of drug administered equals the amount cleared by the body.

- After 3 half-lives 87% of steady-state concentration is achieved.
- After 4 half-lives 93% of steady-state concentration is achieved.
- After 5 half-lives 97% of steady-state concentration is achieved.

(Fuhrman BP, Zimmerman JJ. *Pediatric Critical Care*, 2nd Edition; p. 1281; Behrman BE, et al. *Nelson Textbook of Pediatrics*, 15th Edition; p. 294.)

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**CHAPTER 2: CARDIOVASCULAR SYSTEM**

1. A The Mueller maneuver (inspiration against closed glottis) increases afterload similar to phenylephrine. The Valsalva maneuver has the opposite effect. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 369–380.)

2. E The ventricular afterload is best approximated by ventricular wall stress, or the degree of stretching of the ventricular muscle. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 369–380.)

3. D All three mechanisms described are operative in the process of ventricular interdependence. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 369–380.)

4. A In the failing heart, or congestive cardiac failure, the effect of changes in intrathoracic pressure on afterload is predominant. Afterload is best approximated by ventricular wall stress. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 390,391.)

5, 6. B, E The right ventricle does not receive more highly oxygenated blood than the left ventricle because of the phenomenon of “streaming,” wherein the blood that is returning from the umbilical vein, through the inferior vena cava, is directed to the left atrium owing to the presence of a flap in the inferior vena cava. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 17–23.)

7. B After birth, because of expansion of the lungs and separation of the placenta, there is a reduction in pulmonary vascular resistance with the first breath and an increase in systemic vascular resistance resulting from loss of the low resistance placenta. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 397–411.)

8. C Arterioles contribute the most to the total peripheral resistance. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 409–413.)

9. A The major portion of oxygen consumption by the heart is directed toward the myocardial wall tension. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 420–422.)

10, 11. D, D Myocardial wall tension is directly proportional to intraventricular pressure and also directly proportional to the intraventricular volume. However, myocardial wall tension is inversely proportional to the myocardial wall thickness. Therefore, in a situation where the wall of the myocardium is thin, there is an increase in myocardial wall tension, and this is likely to lead to increased myocardial oxygen consumption because the majority of oxygen consumed by the heart is utilized by myocardial wall tension. A heart that is dilated (which means that there is increased intraventricular volume associated with a large preload) in the presence of a thin left ventricular wall is a heart that would be considered least efficient. (Rogers MC, et
12. B The diagram represents the following: area A is referred to as systolic time index, and area B is referred to as diastolic time index. Because an increase of heart rate increases myocardial oxygen consumption, tachycardia would adversely affect both of these variables, as does hypotension. Area B represents a time where the myocardium receives its blood and oxygen supply. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 420–421.)

13–15. E, E, D Ischemic heart disease in infants and children should be sought whenever there are risk factors such as those mentioned in Question 13. In the presence of these risk factors, ischemic heart disease in infants is not uncommon. Some of the clinical situations are enumerated in Question 15. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 422–424.)

16. A Eighty percent of infants with anomalous left coronary artery, if untreated, will die before their first birthday. There is frequently a history of screaming with feeding. History of cyanosis at birth is not a recognized feature. This condition may mimic endocardial fibroelastosis or myocarditis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 424,425.)

17. A Kawasaki syndrome is a leading cause of ischemic heart disease in children. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 426–428.)

18. E Coronary artery involvement and cardiac abnormality are more commonly seen in children with Kawasaki syndrome, who are male, less than 1 year of age, have had a fever for longer than 2 weeks, and who have an erythrocyte sedimentation rate of more than 100 mm/hour. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 426–428.)

19. E These are recognized cardiac abnormalities in a setting of trauma. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 431.)

20. B With high permeability pulmonary edema, the ratio of extravascular lung water to total lung weight increases, and the blood-free dry weight of the lung is increased because of the presence of protein in the extravascular fluid. The total dry weight of the lung is also increased. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 432–435.)

21. E $K_f$ is the filtration coefficient. $\sigma$ is the reflection coefficient. When $\sigma$ is equal to 1, there is complete restriction to passage of protein across the capillary membrane. On the other hand, when $\sigma$ is equal to 0, there is no restriction to passage of protein across the capillary membrane. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 432–435.)

22. D Administration of bleomycin and cyclophosphamide, as well as radiation therapy, are known to potentiate the cardiotoxicity of anthracycline. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 436–438.)

23. C Cardiotoxicity because of doxorubicin is indeed dose-dependent, and is usually seen at doses higher than 450 mg/M². (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 436–438.)

24. D The combination of doxorubicin and cyclophosphamide leads to cardiomyopathy with subsequent cardiac failure, which can present with pulmonary edema. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 436–438.)

25. D The lungs are the most frequently affected organs with heroin overdose. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 440–441.)

26. E All of the pulmonary vascular physiological changes listed are true. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 441–454.)

27. C It will take at least 24 hours for the levels of 2,3-diphosphoglycerate to increase in response to hypoxia. All of the other physiological responses to hypoxia are true. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 445–448.)

28. C There does not seem to be a strong correlation between the level of PaO₂ and incidence of Tet spells. All other options are true. (Rogers MC, et al.
29. D Cyanosis in the face of a normal PaO₂ occurs with smoke inhalation, which is particularly associated with CO poisoning. An overdose on shoe dye leads to met-hemoglobinemia. Both these clinical conditions are characterized by a normal arterial oxygen tension, but a decreased measured oxygen saturation. Patients with a very high hematocrit also may present with cyanosis, which is usually a peripheral cyanosis in the presence of a normal PaO₂. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 456–458.)

30. D All of the three conditions mentioned benefit from a right-to-left shunt in preserving the cardiac output. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 101–112, 755–763, 804–805.)

31. D The first priority in these patients is to obtain an echocardiogram in order to rule out any residual abnormalities that might be contributing to the abnormal cardiac output and oxygenation. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 618–620.)

32. A Owing to pulmonary vascular endothelial dysfunction after cardiopulmonary bypass, oxygen is often not a very strong pulmonary vasodilator. With a pH greater than 7.45, it appears that the pulmonary vascular resistance decreases independent of the arterial carbon dioxide pressure (PaCO₂). The other options are true. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 618–620.)

33. C These findings suggest that there is increased pulmonary blood flow, which is likely to lead to pulmonary congestion, and also a diastolic overload on the right ventricle. Use of hyperventilation and tolazoline will lead to further pulmonary congestion and may lead to deterioration of the patient’s overall condition. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 863–868.)

34–36. B, D, D Most thoracic duct injuries occur following an extrapericardial procedure, usually a palliative procedure such as a systemic-to-pulmonary shunt. Prior to enteral feeding, the pleural fluid may be serosanguinous. It turns into a milky color following enteral feeding. Malnutrition because of loss of protein and fat is a recognized complication, which must be managed appropriately. All are indications for surgical ligation of the thoracic duct for persistent choloorthorax. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 481,482.)

37. B, A, A The purpose of the modified Fontan procedure is to eliminate the obligatory diastolic overload on the single ventricle and also to improve oxygenation. Following the Norwood procedure, a systemic-to-pulmonary shunt is created, and any situation that increases pulmonary vascular resistance leads to a decreased pulmonary blood flow, with subsequent hypoxemia. On the other hand, an increase in pulmonary vascular resistance in a patient with a modified Fontan procedure will lead to cardiogenic shock. This is owing to the fact that blood flow from the right side of the heart to the lungs is gravity-dependent because of absence of a contractile right heart. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 868–874.)

38. C The creation of a fenestration between the upper chambers of the heart will allow shunting of the blood from the right side to the left side of the heart in a setting of increased pulmonary vascular resistance, which in turn will maintain cardiac output. It has also been shown to decrease incidence of pleural effusion and mortality. The fenestration can be closed in a cardiac catheterization laboratory at a later date. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 881–883.)

39, 40. D, C To allow reconditioning of the left ventricle pulmonary artery binding and a Blalock-Taussing shunt are important. Focal myocardial ischemia may occur and this may affect the left ventricular function, either focally or globally. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 825–836.)

41. A Refer to answer for Question 34.

42. D Myocardial ischemia can occur with resultant ventricular dysfunction (refer to answer for Question 40).
43. It is usually done on the same side as the arch or the side in which the arch descends. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; p. 746.)

44. Ketamine is a myocardial depressant in a denervated heart. It is likely to depress the myocardial function in this setting. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1563–1564.)

45. A stiff myocardium with poor myocardial compliance is a recognized problem in the postoperative period following repair of Tetralogy of Fallot. Adequate volume expansion with subsequent decrease in afterload, as a result of a decrease in vasoconstriction, is likely to improve myocardial perfusion and myocardial compliance. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 856–857.)

46. Left ventricular stroke work index reflects contractility. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 482–486.)

47. Compensatory mechanisms are least efficient with shock that is cardiogenic in origin. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 577–589; Perkin RM, Levin DL. J Pediatr, 1982; 101:163.)

48. In a setting of myocardial dysfunction, the effects of positive pressure ventilation on afterload predominate over the effect on preload. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 390,391.)

49. An increased negative intrathoracic pressure associated with upper airway obstruction would increase the left ventricular afterload. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 390,391.)

50. Mueller maneuver is inspiration against the partially closed glottis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 369–380.)

51. It may increase the shunt. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; p. 202.)

52. Thiocyanate is removed by dialysis. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; p. 202.)

53. Nitroglycerin tends to decrease central venous pressure and pulmonary artery occlusion pressure without significantly lowering blood pressure. Therefore, it is the preferred drug in patients with marginal blood pressure. Sodium nitroprusside, on the other hand, is the preferred drug for patients who have a preserved blood pressure. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby, 1995; pp. 202–205.)

54–57. In the United States, trauma is the leading cause of death in children beyond infancy. Shock is the major contributor to mortality in these cases. Cases of hypovolemic shock can be successfully treated with crystalloid solutions when sufficient volumes are administered. It has been shown that replacement of up to 50% of the total blood volume of the patient with crystalloids is not associated with significant expansion of the interstitial space. Fluid administration equivalent to 200% of blood volume will result in edema fluid accumulation, particularly if administered rapidly. Hetastarch is available as 6% solution in 0.9 saline. Therapeutically it is equivalent to albumin but the cost is much less. The administration should not exceed 10–20 mL/kg/day because of the concern about derangement in hemostasis. Carcillo et al. (JAMA 1991) found that fluid resuscitation rapidly in excess of 40 mL/kg in the first hour was associated with improved survival in children with septic shock. The risk of pulmonary edema was not increased. Plasma catecholamines are significantly elevated in shock states and impaired cellular metabolism occurs early with septic shock. (Carcillo JA, Davis AL, Zoritsky A. JAMA 1991; 266:1242; Martex AJ, et al. Arch Surg, 1970; 101:421; Hauser CJ, et al. Hosp Physician, 1980; 16:38.)

58. Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 585, 590,591.

59–62. Fat embolism is a recognized complication of orthopedic procedures and fractures. It may also occur in sickle cell disease during painful crises. The treatment is supportive. Microscopic urine analysis may reveal fat globules. Removal of these emboli are not technically possible. Air embolism can occur in all of the clinical settings referred to in the
question. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 224, 225.)

63–65. C, C, D This is a patient with acute hemorrhagic pancreatitis. Potential complications are hypocalcemia, hyperglycemia, ARDS, and shock. Appropriate interventions would include volume resuscitation, management of the hypocalcemia, and appropriate management of the respiratory dysfunction. Surgical exploration is not indicated at this time. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1175–1178.)

66–69. C, B, D, E In a patient with a pure hypovolemia and in the absence of any other complications, such as infection or myocardial dysfunction, a careful repeated physical examination and monitoring of the peripheral perfusion and urine output is usually adequate for fluid management. However, if the patient’s condition becomes complicated, then central venous catheter insertion for monitoring should be a consideration. Hypokalemia and hypocalcemia may develop following vigorous correction of metabolic acidosis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 588–597.)

70. C The newborn myocardium is indeed less compliant, and this leads to increased myocardial wall stress with increased myocardial oxygen consumption. (Nichols DG, et al. Critical Heart Disease in Infants and Children, Mosby, 1995; pp. 18–26.)

71. D Nerve fibers representing baroceptors located in the atrial and ventricular wall, primarily in the distribution of left coronary artery, mediate this reflux. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 513.)

72, 73. D, C Self-explanatory.

74, 75. B, C Dysrhythmias are more often seen during catheter insertion into the right ventricle and include premature ventricular contractions and ventricular tachycardia.

During measurement of cardiac output by the thermal dilution technique, cardiac output is inversely proportional to the area under the curve. Prolonging the upstroke/downstroke of the curve leads to false elevation of the area under the curve, which would lead to false overestimation of the cardiac output. (Nichols DG, et al. Critical Heart Disease in Infants and Children, Mosby, 1995; pp. 481–488.)

76–79. B, A, B, B Point B is end diastolic volume. B–C is an isovolemic contraction; aortic valve opens at Point C. C–D is the period of systole; a fluid bolus increases end diastolic volume (B–B1). Increased contractility leads to a lower end systolic volume (A–A1). Increased afterload is associated with higher systolic pressure (C2–D2) but smaller stroke volume. (Nichols DG, et al. Critical Heart Disease in Infants and Children, Mosby, 1995; pp. 25–32.)

1–3. A, E, E With central hypoventilation syndrome, apnea usually occurs during quiet sleep, however, it can happen during rapid eye movement sleep (REM). Included in questions 2 and 3 are some of the reorganized causes of central hypoventilation syndrome. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 235–238.)

4. C, D Both achondroplasia and Arnold-Chiari malformation give rise to a mixed type of apnea, and neither typically causes central apnea. (Canfield P, et al. Clin Pediatr, 1982; 21:684; Pauli RM, et al. J Pediatr, 1984; 104:342.)

5. B The absence of any chest wall movement is highly suggestive of a central apnea. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 235–238.)

6. A There will be some abnormalities in the respiratory function owing to the loss of abdominal muscle activity and loss of their participation in the respiratory effort. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 239–241.)

7. E The hypotension is typically associated with bradycardia and may be very difficult to manage.
8. C Somatosensory evoked potentials detect brain wave activities in response to peripheral nerve stimulation, and therefore, it evaluates the entire neural track from the cortex down to the peripheral nerve. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 693,694; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; p. 604.)

9. A Spinal cord injury without any significant radiographical abnormalities is commonly seen in the pediatric population. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 239–241.)

10–15. A, D, E, E, E Various portals of entry of the organism into the body are recognized. In 20% of cases, a portal of entry is not found based on history and physical examination, and therefore, absence of the portal of entry is not very rare. Also of note, is that cultures may not reveal the causative organism. Generalized seizures are not a recognized feature of tetanus; however, the so-called “respiratory convulsions” can develop and require immediate attention to opening and maintaining the airway, which often includes endotracheal intubation. The mortality resulting from tetanus is most commonly secondary to respiratory abnormalities. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 241–244.)

16. C, C, B, B Autonomic dysfunction is a recognized feature of both poliomyelitis and Guillain-Barre syndrome, and the mortality in both results from respiratory dysfunction. Poliomyelitis generally presents with asymmetric scattered weakness, as opposed to the symmetric weakness that is noted with Guillain-Barré syndrome, and the clinical progression is usually rapid with poliomyelitis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 242–246; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; p. 638.)

17. E All of the features mentioned are correct, and this is a case of poliomyelitis, which is very rare in the United States; however, it is still seen in developing countries and can certainly be imported into the United States. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 242–244.)

18. C Syndrome of inappropriate antidiuretic hormone secretion (SIADH) occurs with both respiratory syncytial virus infection and Guillain-Barré syndrome. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 132, 244–246; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; p. 638.)

19. C Residual volume is not clinically useful in this setting. The two most commonly used parameters for monitoring of patients with neuromuscular disease who might require tracheal intubation or liberation from mechanical ventilation, are FVC and maximum or negative inspiratory force. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 245–247; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 1341–1345.)

20. D In patients with neuromuscular disease, depolarizing muscle relaxants, such as succinyl choline, should be avoided because of the possibility of cardiac dysrhythmias, and sedatives should be used very cautiously. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 239–247. Fuhrman BP; *Pediatric Critical Care*, 2nd Edition; pp. 360,361.)

21, 22. A, A Pneumonia is a common complication in patients with Guillain-Barré syndrome. Among the options, sinus tachycardia is the most common abnormality in these patients. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 244–246; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; p. 638.)

23, 24. D, B Diaphragmatic paralysis secondary to a phrenic nerve injury most commonly follows a palliative repair of a congenital cardiac defect such as a Blalock-Taussig shunt. In infants and children, this entity is much more likely to lead to gas exchange abnormalities and could be analogous to a flail chest in an adult. This arises because of the highly compliant chest wall and poor ability of the intercostal muscle to stabilize the chest wall. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 247–249; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 360,361.)

25–29. D, B, C, E, D There are various subtypes of myasthenia gravis. Juvenile myasthenia gravis is usually seen in teenage years. Onset of symptoms often
follows a viral respiratory infection and cranial nerves, particularly extraocular movements are predominantly involved. Other autoimmune diseases, such as systemic lupus erythematosus or thyroid disorders, may be associated.

*Congenital myasthenia gravis* has an onset a few days after birth with poor feeding and respiratory difficulty/failure. Family history is often present in a sibling, but history of myasthenia in the mother during pregnancy is absent.

*Neonatal myasthenia gravis* is uniformly born to mothers with myasthenia gravis; one out of five is transient in nature (as the autoantibodies resolve), and responds well to anticholinesterase medications.

*Familial infantile myasthenia gravis* is usually not born to mothers with myasthenia gravis even though there is often history of myasthenia gravis in a sibling. These patients develop marked respiratory depression and require tracheal intubation. The subsequent clinical course is characterized by episodes of muscle weakness in the first 2 years of life, which may progress to respiratory failure. Episodes do respond to anticholinesterase therapy.

Following general anesthesia with tracheal intubation, patients with myasthenia gravis may develop stridor with or without respiratory distress secondary to the following factors: glottic/subglottic edema owing to traumatic intubation, laryngeal muscle weakness, or vocal cord paralysis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 249–251.)

30. A Succinylcholine in general should be avoided in patients with neuromuscular disease. Peripheral muscle weakness does not seem to correlate well with respiratory muscle weakness. Plasmapheresis has been shown to decrease the duration of endotracheal intubation and mechanical ventilatory support postoperatively in patients with myasthenia gravis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 249–251.)

31. D The level of consciousness is typically preserved in patients with infantile botulism. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 251–253; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 639,640.)

32. C Recovery of the diaphragm seems to occur prior to recovery of peripheral muscles. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 251–253.)

33. D All of the statements regarding evoked potentials are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 690–696; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 604, 682.)

34. B In severe head injury, the vasoresponsivity to changes in blood pressure is lost earlier than that in response to CO₂. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 649–652.)

35. D In meningitis, the autoregulation to cerebral blood flow seems to be intact. (Ashwal S, et al. J Pediatr, 1990; 117:523–530.)

36. A In a patient with head injury and coma, absence of cortical waves bilaterally with the somatosensory evoked potentials is associated with poor outcome. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 694–695. Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 604, 682.)

37. D Cerebral blood volume is an important determinant of intracranial pressure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 648–650.)

38. D All of the statements are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 648–650.)

39. A The characteristics/pattern of cerebral ischemia is: ischemia, reactive hyperemia, followed by delayed hypoperfusion. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 701; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 671–687.)

40. E Layers CA₁ and CA₃ of the hippocampus are one of the most vulnerable areas of the brain to ischemia; others include the cerebellum and layers 3, 5, and 6 of the cerebral cortex. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 701,702; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 671–687.)
41, 42. C / D, E The corneal reflex tests cranial nerves 5 and 7. Midbrain lesions induce a midsize minimally reactive pupil, whereas Pontine lesions induce a pinpoint pupil. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 739–744.)

43. D In experimentally induced status epilepticus, which is divided into phase I and phase II, it has been shown that phase I is characterized by hypertension, lactic acidosis, hyperglycemia, and hyper- or normokalemia, whereas phase II is characterized by hypoglycemia, hyperkalemia, hyperthermia, and respiratory compromise. (Lothman E. *Neurology* 1990; 40:13.)

44. C With highly lipid soluble drugs, free brain concentration does correlate with free serum concentration of the drug. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 765–768.)

45. D Diazepam is one of the most lipid-soluble of anticonvulsants, and therefore, it has a very large Vd because of its high lipid solubility. The Vd of diazepam is at least five times that of lorazepam, and diazepam has significant metabolites, which tend to accumulate and contribute to the prolonged or delayed effects. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 766,767; Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 629,630.)

46. B Phenobarbital does have a low lipid solubility, and this accounts for the very slow onset of action. The pharmacokinetics of phenytoin is nonlinear, and this accounts for a significant increase in toxicity at increasingly higher doses. Infants do have a higher elimination capacity for anticonvulsants compared with older children and adults. Lorazepam, when used repeatedly over a period of 48 hours for status epilepticus, may become progressively less effective owing to development of tolerance. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 767–769.)

47. A Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 810,811.

48. D Cytotoxic edema involves primarily the cells, and therefore, is seen primarily in the gray matter. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 647,648.)

49. D Glutamic acid is normally found in very small concentrations in the brain interstitial fluid. When it is released from the cell in high concentrations, it is very cytotoxic to glial cells, and also contributes to increased intracranial pressure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 705,706.)

50. B Initial concentration of a drug is equal to the dose administered divided by its Vd. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 766–768.)

51. C The majority of blood flow to the brain is committed to the gray matter, which contains the cells. Arterial oxygen tension does have a significant influence on the cerebral blood flow. Water constitutes about 65% of total brain content. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 648–650.)

52. C, D, B, A, E, F, G Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 737–744.

53. E All have been recognized and associated with head injury. Other abnormalities include ST segmented T-wave changes on the electrocardiogram (EKG). (Rogers MC, et al. *Crit Care Med*, 1980; 8:213,214.)

54. C Some of the initial compensatory mechanisms in response to increased intracranial volume are because of displacement of the spinal fluid from the intracranial to intraspinal space. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 646–660.)

55. E, D, C, B, A, E Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 817–834.

56. A Homocystinuria is the metabolic abnormality that is most likely to be associated with the development of stroke. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 868–869.)

57. C This patient apparently developed communicating hydrocephalus most likely as a result of blockage of the arachnoid villi within the dural sinuses, the site of drainage for of cerebrospinal fluid back to the
venous circulation. (Fuhrman BP. Pediatric Critical Care, 2nd Edition; p. 658.)

58. E Cerebrospinal fluid rhinorrhea is seen in approximately 7% of basilar skull fractures, and in the vast majority of cases it resolves within a period of a few weeks. Ecchymosis in the periorbital area is referred to as raccoon’s eye. Corticosteroids have not been shown to be definitely beneficial in a setting of closed head injury. Cerebrospinal fluid rhinorrhea is uncommon in children less than 10 years of age because of underdevelopment of sinuses. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 816,817.)

59. A A significantly depressed skull fracture requires surgical intervention. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 855.)

60. E All of the statements are true regarding cerebral circulation. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 859–872.)

61. A, B, C Please see Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 859–872.

62. A Please see Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 859–872.

63. T, F, T, F, T, T Please see Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 859–872.

64. D Please see Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 859–872.

65. T, T, T Please see Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 859–872.

CHAPTER 4: INFECTIOUS DISEASES

1. E In pediatric and newborn services, lower respiratory infections are the most common type of nosocomial infection followed by bacteremia, urinary tract, cutaneous, and surgical wound infection. Staphylococcus aureus predominates as the most common cause of lower respiratory infections in newborns, not Klebsiella. Klebsiella is the most common organism isolated from pediatric lower respiratory tract nosocomial infections. Other common lower respiratory pathogens include Pseudomonas aeruginosa, Coagulase-negative staph, and Escherichia coli. E. coli is the most common cause of pediatric, nosocomial, urinary tract infections. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 976, 997, table 30.2.)

2. A Pneumococcus and Branhamella are the most common organisms causing sinusitis in the general pediatric population younger than 10 years of age. In intensive care patients with a nasotracheal tube in place, a variety of Gram-negative organisms, including Pseudomonas aeruginosa, Klebsiella, Proteus, E. coli, Enterobacter, and Serratia are found. Often, these infections are polymicrobial. Direct aspiration and culture of the material should direct therapy. Ocular infections are often caused by P. aeruginosa and may progress if left untreated. Infection from environmental contaminants also occurs. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 987,988.)

3. E Gram-negative organisms, not anerobes, are the dominant organisms that colonize the trachea in patients who are intubated. Colonization is increased in those patients receiving cimetidine or antacids. Respiratory equipment, including nebulizers, medications, and hand ventilators may also become contaminated and contribute to respiratory infections. Uncuffed endotracheal tubes contribute to the aspiration of oral secretions. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 989.)

4. C Local inflammation does not correlate with the duration of arterial catheter insertion and is not predictive of catheter tip colonization. All other responses are true. Catheters placed by surgical cutdown have twice the incidence of infection and nine-fold increase in septicemia. Disposable transducers used for 4 days had no higher risk of infection than those used for 2 days. At 8 days, the prevalence of contamination was significantly higher for the transducers (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 981,982.)
5. E Approximately 30% of total parenteral infection infections are caused by fungi with Candida albicans, Candida sp., and Torulopsis being primarily responsible. All of the other statements are true. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 984.)

6. T, T, T, F, T In spite of the inherently invasive nature of extracorporeal membrane oxygenation, few reports of infectious complications have arisen. Approximately 5% of the cannulas placed for extracorporeal life support became infected. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 986,987.)

7. D Cleansing with providone-iodine or the use of antibiotic impregnated catheters have not been shown to significantly lower the incidence of urinary tract infections in the ICU. Gram-positive isolates predominate in urinary tract infections in both sexes. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 991,992.)

8. B The prophylactic use of antibiotics does not significantly decrease the incidence of infection associated with intracranial pressure monitoring devices, and therefore, use of prophylactic antibiotics in this setting is not indicated. Increasing the frequency of breaks into the system, such as obtaining samples or flushing the catheter with saline, does increase the risk of infection. However, placement of these catheters either in the ICU or the operating room has not been shown to make a substantial difference in terms of the rate of infection. The presence of blood within the ventricular system does increase the risk of infection. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 993,994.)

9. D Ampicillin and an aminoglycoside alone will not be adequate coverage for intraabdominal infection. It is necessary to cover for anerobic bacteria as well. Therefore, a combination of ampicillin, gentamycin, and clindamycin is one approach the child with abdominal sepsis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1012,1013.)

10. A, B, B, B Early-onset neonatal group B streptococcal infections are usually seen within the first week of birth. Early-onset disease is primarily a disease of premature infants less than 35 weeks gestation and weighing less than 2500 g at birth. Late-onset infection can be delayed up to 3 months after birth. There is a poor correlation between the late-onset group B streptococcal infection and maternal colonization, 95% of the isolates are type III, and there is a higher association with meningitis, as opposed to association with pneumonia that is seen with early-onset group B streptococcal infection. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1016,1017.)

11. T, F, T, T, F, T, T, T The initial antibiotic therapy of the sick neonate generally consists of ampicillin and an aminoglycoside. Whereas the combination of ampicillin and gentamicin is synergistic against group B streptococcal infection, the addition of chloramphenicol to ampicillin is of no additional benefit. The immaturity of the immunological system of the newborn predisposes this group of patients to susceptibility to group B streptococcal infection. It is the deficiency in complement, antibodies, and plasma components that is thought to be responsible for the short-term outcome improvement in simple and double volume exchange transfusions. Listeria monocytogenes generally affects extremes of age and pregnant women, and it has a bimodal presentation similar to group B streptococcal infection (i.e., early-onset and late-onset). (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1019,1020.)

12. A, B, B, B Late-onset L. monocytogenes infection is usually seen in healthy, full-term infants who are born to mothers who are asymptomatic at the time. The vast majority of infections are from type 4B, and there is a higher association with meningitis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1020–1022.)

13. D The majority of infections in which a source is identified are related to maternal genital infections. The incubation period for neonatal herpes is usually longer than 7 days. The likelihood of the neonate contracting the disease is correlated with a prolonged rupture of membranes (>6 hours) in a mother with active genital infection. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1023,1024.)

14. T, F, F, T, F T Herpetic meningoencephalitis occurs in approximately 50% of neonatal diagnoses. Mothers with genital lesions need not be isolated from their babies, in contrast to mothers with oral or perioral lesions who should be preferably isolated from their newborn babies. The prognosis for babies with dissemi-
nated infection is approximately 90%. Herpes simplex virus 2 has an increased rate of pneumonitis and disseminated intravascular coagulation, which may relate to its poorer outcome when compared with herpes simplex virus 1. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1023,1024.)

15. E *Neisseria meningitidis* is usually endemic and is commonly carried in the nasopharynx of the healthy population. The infection is more commonly in males. Influenza A and B are associated with an increased susceptibility to infection. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1025–1027.)

16. D Several attempts at classifying meningococcal disease severity and prognosis have occurred. The characteristics that are associated with a worsened outcome represent failure of the child’s organ systems to adequately compensate for the disease. A low leukocyte count in the periphery or in the cerebrospinal fluid (CSF) may represent a failure of the host’s neutrophils to mount an adequate response. Similarly, the presence of shock, petechiae, and thrombocytopenia are unfavorable. The elevation of the sedimentation rate is, in part, owing to elevation of the acute phase reactants, which includes fibrinogen, and this will take at least 24 hours. A sedimentation rate of 100 mm/hour (as stated in the question) would suggest that the infection has been going on for more than several hours, and it would constitute a good prognostic feature. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1026,1027, tables 31.13, 31.14, 31.15, 31.16, and 31.17.)

17. D The cardiovascular collapse and instability associated with meningococcal infection was originally thought to be resulting primarily from adrenal dysfunction. However, large doses of exogenous corticosteroids were not always effective in reversing the shock state, and therefore, the more recent prevailing theory is that the cardiovascular collapse is actually secondary to endotoxemia, with its effect in inducing multiple organ dysfunction syndrome. Fulminant meningococcemia has an estimated mortality rate of 85%. Petechiae are frequently present in this disease and are related to a failure of the hematopoietic system and disseminated intravascular coagulation. Corticosteroids are a promising intervention that have not been demonstrated to universally reverse the shock state. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1026–1029.)

18. D Myocarditis, which is believed to be a form of vasculitis, generally develops 4–7 days after onset of infection, and pneumonia can be very severe and require mechanical ventilatory support. The recommendation of using Rifampin prophylaxis for household and day-care contacts is universally agreed on. Corticosteroids are a promising intervention that have not been demonstrated to universally reverse the shock state. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1029–1032.)

19. E Petechiae and ecchymosis may be noted with any of the infections mentioned, although they are typically associated with *Neisseria* infection. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1032–1034.)

20. E *H. influenzae* infection may mimic meningococcemia. Adrenal hemorrhage has been noted in 55% of the fatal cases of *H. influenzae* sepsis. Intractable hypotension and cardiac dysfunction usually lead to death in affected patients. Rifampin prophylaxis should be initiated immediately after diagnosis of the *H. influenzae* type B infection, in household contacts. It should be incorporated into the therapeutic antibiotic regimen of the index case in the last few days of therapy, and should not be delayed until one month after completion of antibiotic therapy. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1032–1034.)

21. B A history of freshwater lake swimming is an important etiological risk factor for *N. meningitidis*. Otitis media is often seen in association with *H. influenzae* meningitis. Meningitis in the vast majority of cases does not actually involve the parenchyma. It is limited to the three layers of the meninges. The Virchow-Robin spaces are a continuous extension of the subarachnoid space, which will allow the bacteria to gain access into the subarachnoid space, and maybe to the most superficial surface of the brain. Meningitis, when severe, is often associated with cerebral edema. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1040–1060.)

22. D Even in the absence of an index case within the day care setting, children who attend day care centers are at higher risk of developing meningitis. Convulsions occurring within the first 24–72 hours of meningitis may represent febrile seizures, and therefore have a better prognosis. Convulsions that develop
beyond this period carry a less favorable prognosis. Limitation of ocular movement may be owing to abnormalities in the 3rd cranial nerves, and does not always indicate increased intracranial pressure. When papilledema is noted on the first day of admission of meningitis, other etiologies should be sought, particularly an intracranial mass lesion, such as a brain abscess. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1047–1060.)

23. D Limitation of ocular movements may result from irritation of cranial nerves III, IV, or VI. Convulsions do occur in at least 30% of meningitis cases. Those convulsions that are limited to the first 24–72 hours carry a better prognosis. See response to question 22. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1047–1049.)

24. E Bacterial culture of the CSF is considered the gold standard. The presence of any neutrophils in the CSF in the newborn period should be treated with a high degree of suspicion. This may be one of the early manifestations of meningitis. However, in newborn infants polymorphonuclear leukocytes may comprise up to 60% of the total CSF white cell population and still be considered normal. The opening pressure in the neonate is between 90 and 110 mm H2O, whereas in the older child and adult it may be as high as 180 mm H2O. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1049–1051, table 32.4.)

25. T, F, F Spinal fluid remains clear with up to 500 white blood cells/mm³. Erythrocytes raise the CSF protein concentration by about 15 mg/dL for every 1000 red blood cells/mm³. A CSF lactate level of more than 14 mg% is considered abnormal. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1049–1047.)

26. D Children who are diagnosed and admitted to the hospital for meningitis have commonly received some form of antibiotic, usually oral, prior to presentation. This form of antibiotic usually is not sufficient to treat meningitis, and therefore, it does not improve the outcome in these patients. Several hours after the administration of an appropriate antibiotic, it is certainly possible to inhibit bacterial growth in the spinal fluid. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1051,1052.)

27. A Tuberculous meningitis, which is usually a basal form of meningitis, is more likely to present with focal neurological signs and papilledema, particularly cranial nerve palsies such as cranial nerves VII, VIII and IX. Cryptococcal meningitis may present only with behavioral changes, or it may present with symptoms of a space-occupying lesion. The opening pressure in neonates may be as high as 110 mm H2O. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1052–1060; American Academy of Pediatrics. In: Pickering LK, ed. 2000 *Red Book: Report of the Committee on Infectious Diseases*. 25th Ed. Elk Grove Village, IL.)

28. E By day 5 of treatment with antibiotics, 85% of children with *H. influenzae* meningitis will be afebrile. SIADH has been noted in more than 50% of patients with meningitis. Under these circumstances, restriction of fluid and close monitoring of fluids and electrolytes are a necessary part of the management of these patients. Subdural effusions, which are a recognized complication of meningitis, generally resolve spontaneously and do not require surgical intervention in the vast majority of cases. Nosocomial infection is a common cause of recurrent treatment after initial treatment for meningitis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1052–1054.)

29. T, T, F, T The causes of fever that persists beyond the 10th day in the setting of meningitis are subdural effusions, drug fever, arthritis, brain abscess, and nosocomial infection. Thirty to fifty percent of fevers are idiopathic. Persistence of a positive CSF culture would be a poor prognostic feature in patients with bacterial meningitis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1053–1060.)

30. E The frequency of shunt infections varies between 2 and 30% and is influenced by a variety of factors. Children suspected of having a shunt infection or meningitis should receive coverage with antibiotics for Gram-positive organisms including the *Staphylococcus* species, as well as Gram-negative organisms. Staphylococcal species are the most common. Initial therapy should include vancomycin because the frequency of methicillin-resistant staphylococci is high. Respiratory isolation of the patient for the initial 24 hours of antimicrobial therapy is an important epidemiological consideration. The data regarding the use of dexamethasone in meningitis are controversial. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1061,1062.)
31. T, T, F, F, T The CSF cytology in tuberculous meningitis mimics the lymphocyte predominance found in viral meningitis. CSF glucose is classically reduced and the protein level is elevated. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1050–1061.)

32. E Aseptic meningitis is an inflammatory process of the meninges that results from a number of different etiologies. An elevated protein, a pleocytosis, and the absence of organisms on Gram stain and culture characterize it. The etiologies associated with this diagnosis are rather large and include viral, bacterial, and fungal causes. Admission of the patient to the hospital depends on the certainty of the diagnosis. To the extent that the patient is stable and the likelihood of a partially treated bacterial etiology is ruled out, outpatient management may be acceptable. (Mandell ML, et al. *Principles and Practice of Infectious Diseases*, 3rd Edition; pp. 1367–1379.)

33. E Enteroviral infections are higher in lower socioeconomic groups, have a 3- to 5-day incubation, and are typically seen in the latter part of the summer. The meningitis associated with these infections usually has a benign course. (American Academy of Pediatrics. In: Pickering LK, ed. *2000 Red Book: Report of the Committee on Infectious Diseases*, 25th Ed. Elk Grove Village, IL.)

34. T, F Please see Mandell ML, et al. *Principles and Practice of Infectious Diseases*, 3rd Edition; pp. 1367–1379.

35. E With HSV infection, particularly with meningoencephalitis, the electroencephalogram displays abnormalities typically in the frontal and temporal lobe area of the brain. All of the other responses are true (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1064–1066.)

36. D Arboviruses are arthropod-born viruses that are a common cause of encephalitis. These infections are usually seen in late summer and spring, and they are transmitted by arthropods. St. Louis encephalitis is the most common arbovirus infection in the United States, and is generally a mild disease. The highest mortality usually occurs with Eastern equine encephalitis. California encephalitis is usually a mild disease. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1062–1064.)

37. B, A, C, D The St. Louis encephalitis virus is distributed throughout most of the US and causes major epidemics that peak later than other arboviruses. Most infections are asymptomatic, and less than 1% have overt neurological disease. Western equine encephalitis is the usual cause of arbovirus encephalitis and California encephalitis viruses occur in the central and eastern United States, and cause diseases with a fulminant and mild course, respectively. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1063–1065.)

38. B Interestingly, in spite of the name, Rocky Mountain Spotted Fever occurs primarily in the eastern United States, including the Ohio Valley area. The disease is a tick-born illness. See Question 47. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1106–1109.)

39. D Over the past 30 years, cyanotic congenital heart disease has replaced suppurative otitis media or mastoiditis and suppurrative sinusitis as the most common predisposing factor for brain abscess. This is true for the industrial nation, but even in developing nations, it is the most likely predisposing factor. Overall, a predisposing factor can be determined in approximately 85% of all patients with brain abscess, and therefore, a meticulous evaluation for a predisposing factor is warranted in these patients. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1071–1073.)

40. D Brain abscesses formed by hematogenous seeding tend to develop at the junction of gray and white matter and usually in the distribution of the middle cerebral artery; hence, the predominant location in the temporal and parietal lobes. Beyond the neonatal period, meningitis is a rare form of brain abscess. Seizures, when they occur, are more typically generalized. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1071–1074.)

41. D Normal brain parenchyma is highly resistant to invasion by microorganisms and therefore, abscess formation seems to occur only in areas of the brain with focal ischemia, necrosis, or marginal perfusion. Poor vascular supply in the white matter or at the junction of the gray and white matter makes these areas the most likely to be affected by brain abscess. With the exception of the neonatal period, abscess infrequently complicates a course of bacterial meningitis. In the neonatal period, *Citrobacter diversus* and *Proteus mirabilis* are the most common etiological agents that usually cause meningitis.
and are subsequently complicated by brain abscess. When seizures develop in association with brain abscess, they are most commonly a generalized seizure. In up to 30% of brain abscess cases, the microbiology is polymicrobial, which could be a combination of aerobic and anaerobic organisms. Suppurative complications of otitis media or sinusitis are becoming less and less common as an etiological agent or predisposing factors for brain abscess. Because of poor penetration into the abscess cavity, aminoglycosides are not effective for treatment of brain abscess. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1073–1075.)

42. D Unlike the epidural space, the subdural space is not limited by attachment of the dura to the skull sutures, allowing extension and the spread of the subdural empyema over a wide area of the cerebral hemispheres. The potential subdural space is restricted at the base of the brain, and therefore involvement of the base of the brain is rare with subdural abscesses. In infants, subdural empyema generally complicates acute meningitis, and therefore is caused by the organisms commonly implicated in causing meningitis. Because the incidence of *H. influenza* type B as a cause of meningitis in infants has decreased dramatically in the United States, this organism is becoming less and less an etiological agent for subdural empyema. The magnetic resonance imaging (MRI) is the diagnostic imaging procedure of choice for subdural empyema. Advantages of MRI over the computed tomography (CT) scan include the lack of bone artifact, the ability to detect the smaller extracranial fluid collection, and improved ability to differentiate extracranial collection of fluid from other differential diagnoses such as cerebritis, cerebral edema, and venous thrombosis. MRI can also detect the density difference from elevated protein concentration, and therefore distinguish a subdural abscess from other sterile collections, such as subdural effusions. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1071–1077.)

43. C, B, A In the child and young adult, the most common organism causing localized parameningeal infections, such as a subdural empyema, are the various aerobic streptococci, such as *S. pneumoniae*, staphylococci of either the epidermidis or the aureus species. α-Hemolytic streptococci are the most frequently isolated organisms from brain abscesses in patients with cyanotic congenital heart disease. *S. aureus* is the usual organism causing spinal epidural abscess and accounts for 80% of cases. See response to Question 51. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1072–1079.)

44. D Proposed and simplified diagnostic criteria for toxic shock syndrome in children include: fever equal to or greater than 39°C, lymphopenia, rash, shock, diarrhea and vomiting, and irritability. The Centers for Disease Control and Prevention has not adopted these simplified criteria, however. Toxic shock syndrome can also be caused by streptococci, and is one example of severe group A streptococcal disease. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1103–1106.)

45. D Staphylococcal toxic shock syndrome is caused by a coagulase positive staphylococcus that liberates an exoprotein known as TSST-1. The host does not form neutralizing antibodies to the toxin for at least 2 years after infection. This, in addition to the noninvasiveness of the organism, may help to explain the recurrent nature of disease, especially in menstrual cases. Menstrual cases are seen exclusively in the white Caucasian population. Patients who present with elevated serum creatinine, particularly when the serum creatinine is greater than 3 mg/dL tend to have a prolonged hospital course. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1103–1106.)

46. B See response to Question 47 (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1106–1109.)

47. B Rocky Mountain Spotted Fever is caused by *Rickettsia rickettsii*. In the eastern regions of the United States, *Dermacentor variabilis* is the most common tick involved, whereas in the western region, the *Dermacentor andersoni* is the most common tick involved. The disease is usually prevalent in the summer months, and the highest incidence of disease among children age 5–9 years. More than half of all cases appear in persons younger than 19 years of age. The incubation period is 2–14 days, with an average of 7 days. Man is only incidentally involved when bitten by an adult tick. The initial presentation consists of headaches, malaise, and myalgias. The rash generally appears within 2–4 days after the fever, and has been noted in nearly all children with the disease. The eruptions begin as discrete macules, first observed on the ankles and feet, and shortly thereafter on the wrists and hands. Regardless of the pro-
progression of the rash, the rash is almost always most pronounced over the extremities and almost always involves the palms of the hands and the soles of the feet. Over a period of several days, the rash becomes petechial and purpuric. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1106–1109.)

48. E Legionnaire’s disease was first recognized in 1976 after an outbreak of pneumonia in Philadelphia. The organism, *L. pneumophila*, accounts for only about 15% of pneumonia in adults, but it causes acute pulmonary disease, mostly among adult males. The disease has also been noted in infants and children and the prevalence of elevated titers in children is quite high in some communities. The presenting complaints are usually fever, nonproductive cough, encephalopathy, and seizures; cerebellar signs may be markedly severe in these patients. The lung disease is usually lobar in nature; hepatic and renal abnormalities are often also noted. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1109–1110.)

49. D Super antigens are antigens that are derived from either bacteria or viruses that interact with the major histocompatibility class II proteins and activate T-cells by binding to the variable region of the β chain of the T-cell receptor. Stimulation of the T-cell receptors leads to polyclonal T-cell activation, which results in release of massive amounts of tumor necrosis factor-α and interleukin-6. These cytokines are most likely the elements responsible for the shock and multiorgan dysfunction seen in these diseases. Super antigens differ from conventional antigens mainly in the manner in which they are processed and presented to the T-cell receptors. The polyclonal activation generally results in a reduction of the number of circulating CD4+ lymphocytes; however, this reduction is usually reversible and transient. Super antigens are potentially involved in all the three disorders mentioned in the question. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1103.)

50. B, A Some differences have been noted between children and adults with toxic shock syndrome. Whereas only a small percentage of adults have had a prominent prodromal illness, nearly all children have between 1 and 6 days of symptoms preceding the illness. These symptoms include fever, mucosal hyperemia, erythroderma, vomiting, diarrhea, dizziness, and myalgias. The vast majority of adults admitted to the hospital have hypotension at presentation. This finding is not prominent in children at the time of admission, although it may develop later during the hospitalization. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1103–1106.)

51. D, A, B, C Brain abscesses are the most frequently encountered form of localized intracranial infection in children. Death usually occurs with rupture of the abscess and spread of the infection into the ventricular system or herniation secondary to mass effect. *Citrobacter* and *Proteus* are the most common etiological agents in the newborn period. In patients with congenital heart disease, α-hemolytic streptococci are common. Patients who have traumatic injuries are affected by *S. aureus*. Immunocompromised patients are at risk for Nocardia brain abscesses. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1071–1076.)

CHAPTER 5: HEMATOLOGY AND ONCOLOGY

1. A Trauma is the leading cause of death in the 1- to 15-year-old population. Neoplastic disease is second. The other responses are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1433–1434.)

2. D Infection is higher in patients with central lines than those without. Recent retrospective data suggest that there are no differences in infection rates between subcutaneously implanted versus externalized catheters. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1438.)

3. E Fifty-five to seventy percent of febrile episodes in oncology patients are of infectious origin. Blood cultures are positive in less than 50% of cases of serious disseminated fungal infections. *C. albicans* and *Aspergillus* species are the most common fungal organisms. Neutropenia is closely correlated with morbidity and mortality. Pneumocystis is unlikely in this clinical scenario; however, pneumocystis is responsible for up to 50% of nonbacterial pneumonitis in oncology patients. Chest radiographs demonstrate bilateral infiltrates radiating from the hilum. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1438–1445; table 42.6.)
4. D The half-life of transfused platelets is 7 days; with significant alloimmunization, it can be hours. All of the other statements are correct. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1442, 1443.)

5. D, E, C, B, F, A Chemotherapy may promote the development of a coagulopathy associated with an increased risk of hemorrhage or thrombosis. Actinomycin D and other antibiotics decrease the vitamin K-dependent clotting factors. Anthracycline increases fibrinolysis. l-Asparaginase may cause hypofibrinogenemia. Methotrexate can cause an antithrombin III deficiency. Vincristine may cause chronic hepatic dysfunction. Glucocorticoids increase the levels of factors II, VII, VIII, and X. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1442–1457.)

6. A Primary pulmonary parenchymal involvement with leukemia is very rare. Parenchymal involvement is occasionally seen with histiocytosis X and metastatic disease (e.g., osteogenic and Ewing sarcoma, and Wilms tumor). All other statements are correct. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1445–1447, table 42.8.)

7. T, T, F, T, T, F A variety of chemotherapeutics, as well as radiotherapy, can cause cardiomyopathy. Effects appear to be dose related. Radiotherapy may cause pericarditis with a chronic effusion. Histologically, interstitial fibrosis with vascular narrowing is seen. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1447–1448.) The onset of *Pneumocystis carinii* infection typically occurs 3–6 months after bone marrow transplantation. Bacterial or fungal infection can occur within the first 2 weeks. Cytomegalovirus (CMV) infection occurs 6–12 weeks after bone marrow transplantation. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1454, 1455.)

8. B, D, A, C The child with neoplastic disease may acquire a variety of neurological deficits related to the neoplasm, the therapy, or a combination of both. Methotrexate is associated with aseptic meningitis, arachnoiditis, demyelination, somnolence, and chronic leukoencephalopathy. Cisplatin may cause ototoxicity, cerebral edema, and seizures. Vincristine is associated with SIADH. 5-FU may cause acute cerebellar ataxia. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1449–1452; table 42.17.)

9. B, D, A Chemotherapy is commonly associated with renal injury. High-dose methotrexate is associated with renal tubular injury. Cisplatin may cause tubular necrosis; cyclophosphamide and ifosfamide are both associated with hemorrhagic cystitis. l-Asparaginase is not associated with renal injury. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1452, 1453; table 42.18.)

10. D CMV infection occurs most commonly 6–12 weeks after bone marrow transplantation. All of the other statements are correct. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1454, 1455.)

11. A, D, B, C, E Chemotherapeutic agents form the mainstay of treatment for childhood neoplasms. All agents act by disrupting some aspect of normal cell growth or division. Antimetabolites interact with various cell enzymes (e.g., methotrexate inhibits the activity of dihydrofolate reductase). Vincristine, a vinca alkaloid, inhibits microtubule function within the cell. This prevents the formation of the spindle apparatus during metaphase, thus inhibiting cell division. The anthracycline (daunorubicin and doxorubicin) and actinomycin D inhibit the synthesis of DNA in tumor cells. Alkylating agents like cyclophosphamide cause breaks in the DNA strands. The glucocorticoids are directly lymphocytotoxic to lymphoid leukemia and lymphoma cells. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1456–1458; tables 42.23, 42.24, 42.26, and 42.27.)

12. E Von Willebrand’s disease is the most common inherited bleeding disorder. One percent of the population has detectable abnormalities in the von Willebrand’s disease protein. All of the statements are correct. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1414–1416; figure 41.2.)

13. T, F, T, T, T, F The preferred source for factors II, VII, X, and antithrombin III is fresh frozen plasma (FFP). Vitamin K is not a stored vitamin. (Rogers, MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1415, 1416.)
14. **D** Antithrombin III, protein C, and protein S are the main components of the antithrombotic system. Thrombomodulin and heparin cofactor II, among others, are also included as endogenous anticoagulants. Protein B is not included among these components. (Rogers, MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1421; figure 41.3.)

15. **E** When the liver itself is diseased, abnormal coagulation results. The liver synthesizes fibrinogen, prothrombin, protein C, protein S, antithrombin III, plasminogen, and factors V, VII, IX, X, XI, and XII. All of the above statements are correct. (Rogers, MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1408, 1409, 1415, 1416.)

16. **B** ε-Aminocaproic acid prevents the breakdown of the fibrin clot by complexing with plasmin to prevent its fibrinolytic activity. Protamine is used to reverse the effects of heparin. Vitamin K deficiency generally occurs within 2–3 days following cardiopulmonary bypass. D-dimers are rarely elevated. (Rogers, MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1405, 1406; table 41.11; Chang, AC, et al. Pediatric Cardiac Intensive Care; pp. 397–399.)

17. **D** Massive transfusion is defined as the replacement of at least one blood volume; estimated as 75 mL/kg for children less than 1 year of age and burn victims, and 70 mL/kg for all others. All of the other statements are true. (Rogers, MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1403–1408.)

18. **C** A variety of metabolic abnormalities can be induced by massive transfusion. 2,3-Diphosphoglycerate is decreased in transfused red cells, which increases red cell affinity for oxygen. Thus, oxygen unloading to tissues may be impaired. All other statements are correct. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1421, 1422; Gilman, AG, et al. Goodman and Gilman’s The Pharmacological Basis of Therapeutics, 8th Edition; pp. 1316.)

19. **E** Antithrombin III primarily inhibits the vitamin K dependent procoagulant factors (II, VII, IX, and X). Deficiency will lead to recurrent thrombosis. Heparin induced antiplatelet antibodies occur in approximately 5% of patients receiving heparin therapy. Most cases are mild with platelet counts higher than 100,000/μL. All of the other statements are correct. (Rogers, MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1406, 1407.)

20. **D** Two forms of acute, heparin-induced thrombocytopenia occur. The mild form occurs in approximately 5% of patients, 4–15 days after initiation of full-dose heparin therapy (platelet counts higher than 100,000/μL). Severe thrombocytopenia occurs less frequently. The more severe form is associated with thrombotic complications. All of the other statements are correct. (Gilman, AG, et al. Goodman and Gilman’s The Pharmacological Basis of Therapeutics, 8th Edition; pp. 1316.)

21. **E** Protein C activation is controlled by several different mechanisms, including by thrombomodulin. Protein C activation and thrombin generation are tightly coupled. Acquired and hereditary deficiencies are the primary cause of thrombophilia. (Nathan, DG, et al. Nathan and Oski’s Hematology of Infancy and Childhood, 5th Edition; pp. 1545–1547.)

22. **E** In contrast to heparin, which acts as a cofactor with antithrombin III to prevent coagulation, the plasminogen activators, urokinase, streptokinase, and tissue plasminogen activator increase fibrinolysis, thereby lysing the clot. Plasminogen is cleaved into plasmin by these activators. Plasmin lyases clot directly. All of the other statements are correct. (Rogers, MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1415, 1416, 1423, 1424.)

23. **E** Both heparin and plasminogen activators (streptokinase, tissue plasminogen activator) may be used to treat arterial thrombosis. The partial thromboplastin time should be kept 1.5–2.0 times normal during heparin therapy. Note: younger neonates may be resistant to thrombolytic therapy, possibly because of lower levels of plasminogen. (Rogers, MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1415, 1416, 1423, 1424.)

24. **E** Disorders in children that are treated with chronic anticoagulants include cardiac disorders (prosthetic valves, Blalock-Taussig shunts, endovascular shunts), some cerebrovascular events, and Kawasaki’s disease. All of the above statements are correct.
25. C, D, A Aspirin is a potent and irreversible inhibitor of cyclooxygenase and thromboxane A2. Sulfinpyrazone, like aspirin, is also a nonsteroidal anti-inflammatory agent that reversibly inhibits cyclooxygenase. (Gilman, AG, et al. *Goodman and Gilman's The Pharmacological Basis of Therapeutics*, 8th Edition; pp. 652, 1524.)

26. D Thromboprophylaxis for prosthetic heart valves has reduced the occurrence of thromboembolic events from approximately 6% to less than 2%. The other statements are true. (Michelson AD, et al. *Chest* 1998; 114(5 Suppl): 748S–769S.)

CHAPTER 6: RENAL SYSTEM

1. A Kidneys are able to maintain renal blood flow over a wide range of systemic blood pressures by autoregulation of intrarenal vascular resistance. Therefore, hypotension with renal hypoperfusion may or may not produce ischemic renal injury. However, these autoregulatory mechanisms are not well developed in neonates. Neonates have high renin levels, which in turn, are associated with decreased glomerular filtration rate (GFR) and reduced outer cortical blood flow. The cortical glomeruli are immature and so are their corresponding tubules. This pattern of high renin and reduced outer cortical blood flow makes neonates more vulnerable to renal dysfunction as a result of hypotension of systemic pressures only slightly below the normal range. In animal studies, newborn animals have decreased production of atrial natriuretic peptide in response to saline challenge. All these factors combined make the incidence of acute renal failure in neonates, after cardiac surgery, higher than in older infants and children. (Nichols DG, et al. *Critical Heart Disease in Infants and Children*, Mosby 1995; pp. 125, 562.)

2. C Furosemide causes vasodilation of the cortical vasculature by direct action and through release of prostaglandins. Furosemide maintains renal blood flow and tubular blood flow when cardiac output is compromised. Mannitol is also a vasodilator of the cortical vasculature that increases renal blood flow either directly by drawing fluid from extravascular to intravascular space, thus increasing total plasma volume, or by increasing prostaglandin production. Increased plasma volume alone does not fully explain the effects of mannitol, because volume expansion with saline improves renal blood flow without improving GFR. The improvement in GFR seen with mannitol is associated with a decrease in afferent and efferent arteriolar resistance, which is probably mediated by prostaglandins. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1192–1194, 1202.)

3. D Clinical studies comparing prophylactic administration of mannitol (or furosemide) with maintenance of adequate intravascular volume during cardiopulmonary bypass failed to reduce the incidence of postoperative renal dysfunction. However, there are experimental studies that have shown some beneficial effects of mannitol. Mannitol has been shown to be effective in preventing deterioration of renal function before administration of Amphotericin B and Cis-Platinum. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1194,1195; Nichols DG. *Critical Heart Disease in Infants and Children*, Mosby 1995; pp. 129,130; Olivero JJ, et al. Br Med J, 1975; 1:550; Hayes D, et al. Cancer, 1977; 39:1372.)

4. C Etiologies of postoperative oliguria in this patient include: (1) intra-operative blood loss; (2) third space volume loss; (3) bilateral ureteral obstruction; (4) cardiac failure; and (5) increased intra-abdominal pressure. In this patient, the latter is important to recognize (because it appears that intravascular volume has been expanded and cardiac output is normal) because prompt surgery to relieve increased intra-abdominal pressure is associated with rapid diuresis. The development of this problem is best avoided by direct measurement of intra-abdominal pressure either via the esophageal route or per gastrostomy. Data indicate that the abdominal wall should not be closed if pressure exceeds 20 mmHg. In this case, it is best to employ a silo with delayed closure to allow time for the compliance of the abdominal wall to increase. (Yaster M, et al. *Anesthesiology*, 1986; 65:A449.)

5. E Children have a lower mortality compared with adults. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1198–1201.)
6. A An increased P–R interval is seen before changes in P-wave because the A–V node is much more sensitive to hyperkalemia than the S–A node. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 1201.)

7–8. E, D All of the strategies mentioned are appropriate for oliguria in a setting of suspected renal insufficiency. With the onset of acute renal failure, hyponatremia is more commonly seen owing to the dilutional effect of intake of fluid orally, which is mostly hypotonic. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 1202. Nichols DG. *Critical Heart Disease in Infants and Children*, Mosby 1995; pp. 128–138.)

9–12. B, A, D, C In the absence of significant symptoms, hypocalcemia does not need to be aggressively treated. Aggressive treatment with calcium in the presence of hyperphosphatemia, and particularly when the product of calcium and phosphorus exceeds 60, increases the risk of calcium deposition in various tissues within the body. Acidosis raises the level of ionized calcium and thus mitigates against the occurrence of symptomatic hypocalcemia. Caution must be exercised in correcting acidosis abruptly, as a rapid decline in the level of ionized calcium may precipitate tetany. Dysequilibrium syndrome is not seen with peritoneal dialysis, as the process is very slow, as compared with hemodialysis, which is done over a few hours. Such a high dose of vitamin C is unnecessary in patients with renal failure. Patients with hemolytic uremic syndrome (HUS) seem to have a better outcome with early institution of dialysis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1201–1205; Kaplan BS, et al. Can Med Assoc J, 1981; 124:429.)

13. A Severe hypertension with hypertensive encephalopathy is a recognized feature of rapidly progressive glomerulonephritis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; p. 1214.)

14. A The initial concentration of a drug equals the dose administered divided by Vd: C = D ÷ Vd (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 766–768.)

15. D Saline diuresis is the most appropriate treatment for hypercalcemia. (Bilezikian J. N Engl J Med, 1992; 326:1196.)

16. A, A, A, C, D, A Please see Williams GH. N Engl J Med, 1988; 319:1517.

17,18. D, D High levels of urea act as an osmotic diuretic in the postoperative period. High normal intravascular volume is precisely what is desirable in the postoperative period in order to avoid the risk of thrombosis in the graft. Preoperative transfusion (with consequent hypervolemia) would increase the risk of congestive cardiac failure in the postoperative period. For cadaveric kidney transplantation, there is a positive correlation between the number of transfusions and the graft survival; the survival seems to be optimal with a transfusion from five or more different donors. With living related donors, it is unclear whether transfusion has any beneficial effects on the survival of the graft. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1237–1240.)

19. C, A Increased platelet consumption is a feature of both HUS and disseminated intravascular coagulopathy. However, deficiency of prostaglandin I₂ activity is associated only with HUS. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1231–1235.)

20. C Atracurium undergoes spontaneous degradation referred to as Hofmann degradation; however, some authorities believe that ester hydrolysis is the major pathway for degradation of atracurium. (Fuhrman BP. *Pediatric Critical Care*, 2nd Edition; pp. 1346,1347.)

21. E Please see Bennet WM. Clin Pharmacokinetics, 1988; 5:326.

22. B, C, D, A Furosemide acts at the loop of Henle, chlorothiazide at the distal tubule, spironolactone at the cortical collecting duct, and mannitol is freely filtered by the glomerulus.

23. D, A Water intoxication is characterized by absence of clinical signs of dehydration, hyponatremia, and a low urinary sodium. In SIADH, the urine osmolality continues to be high in spite of low serum sodium and osmolality. Congenital adrenal hyperplasia is associated with hyperkalemia and acidosis. Three percent salt given at an initial dose of 4 mL/kg will increase serum sodium by approximately 3–4 mEq/L and will abort the seizure. (Rogers MC. *Textbook of Pediatric Intensive Care*, 2nd Edition; Williams & Wilkins, pp. 1249–1250.)
CHAPTER 7: ENDOCRINE SYSTEM

1. D An increased anion gap (AG) is usually present with greater prerenal azotemia, and is not directly related to hyperglycemia. The shift of extracellular phosphate into the intracellular space does not occur until diabetic ketoacidosis is reversed by the administration of insulin and fluids. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1263–1264; Adrogue H, Wilson H, Boyd A. N Engl J Med 1982; 307:1603.)

2. A In most children with a diagnosis of diabetes mellitus who develop diabetic ketoacidosis, the precipitating event is an omission of insulin, whether inadvertent or deliberate. The other causes are also possible, but not as likely. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1261.)

3. A Hyperosmolality has also been associated with electroencephalogram (EEG) changes during diabetic ketoacidosis. The mortality in children with cerebral edema from diabetic ketoacidosis can approach 80%. An increased risk is present for children less than 5 years of age who have a new diagnosis of diabetes mellitus complicated by a prolonged untreated case of diabetic ketoacidosis. Aggressive rehydration, especially with hypotonic fluids, may acutely decrease an already hyperosmolar state in the child precipitating a picture of cerebral edema. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1266–1267; Krane EJ, Walman JK, Walsdorf JI. N Engl J Med 1985; 316:857.)

4. E Intravenous antibiotics should be administered pending lumbar puncture. Progressive deterioration of mental status in this patient would be an indication to obtain a cranial CT scan to evaluate for cerebral edema. As stated in the rationale in Question 1 of this section, children under the age of 5 newly diagnosed with diabetes mellitus and who also have a complicated course of diabetic ketoacidosis, have an increased risk of cerebral edema. Airway and primitive reflexes should be monitored with the possibility of early intubation, if any question of those is compromised. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1267–1269.)

5. C It is not uncommon to have hyperglycemia in association with a head-injured child. Most likely, as a result of an increase in catecholamines and corticosteroids, there is an increase in blood sugar. Hyperglycemia has already been shown to be associated with the degree of severity in brain injury. Some data suggests that ischemic brain injury may be worse in those patients who have hyperglycemia in their recovery phase as opposed to those patients who had normoglycemia. Any coagulopathy that may exacerbate an ischemic picture also may worsen the severity of brain injury. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1271,1272. Pulsinelli W, Levy D, Sigsbee B, et al. Am J Med 1983; 74:540.)

6. E The first step in the treatment of hypoglycemia in a child is initiation of dextrose bolus followed by an infusion of 10% dextrose. All of these conditions are characterized either by the inability to release glycogen from the liver or depletion of glycogen from the liver, and therefore, glucagon is unlikely to be effective. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1273,1274; Kogut M, Gluck L, Kone T, Dodge P, eds. Current Problems in Pediatrics, Chicago: Yearbook, 1974, p. 3.)

7. E The stimulus for the mechanisms which elevate blood glucose in the setting of hypoglycemia is primarily CNS hypoglycemia. The body’s measures which help to remedy hypoglycemia are primarily the release of epinephrine and glucagon with their effects being additive. The neonate requires a considerable amount of glucose, especially in the perioperative period. There is a significant decline in glycogen storage within the liver within the first 3 postnatal hours. If hypoglycemia is resistant to medical therapy, a laparotomy may be indicated to determine the presence of a tumor or subtotal pancreatectomy. Ketonic hypoglycemia is the most common form in children. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1272–1275; Mozam F, Rodgers B, Talbert J et al. Arch Surg 1982; 117:1151.)

8. B Maximum effects of arginine vasopressin result in an osmolality of 1400 mOsmol/L with a urine output of 0.5 mL/kg/hour. It is important to initiate DDAVP treatment as soon as diabetes insipidus is made as the diagnosis, to prevent large surges in fluid loss. The goal is to double the urine osmolality in comparison to the plasma, and obtain a urine output of 2
mL/kg/hour. Death can occur within 1–5 days after the presentation of diabetes insipidus with cerebral insult. Low urine osmolality (<300 mOsmol/L) and serum osmolality higher than 295 mOsmol/L is consistent with a diagnosis of diabetes insipidus without the presence of any osmotic diuretics. In the absence of ADH hormone, the urine flow will continue to increase in the range of 15–20 mL/kg/hour with a significant increase in serum osmolality. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1275–1280.)

9. A, D, B, C Fluid overload leads to polyuria accompanied by plasma hypo-osmolality. With osmotic diuresis, the urine osmolality remains close to that of plasma. In the absence of osmotic diuretics, when the plasma osmolality is more than 295 mOsmol/L, while the urine osmolality remains 300 mOsmol/L or less, the diagnosis of diabetes insipitus is very likely. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1275–1280.)

10. A, B, C Gastroenteritis has minimal urine sodium losses. It maintains a relatively high urine/plasma osmolality. There is a significant degree of urine sodium losses in HUS. With a high FE_{Na} (>3%), a urine osmolality of approximately 300 mOsmol/L and a 1:1 urine/plasma osmolality, SIADH has the highest amount of urine sodium losses with a very high urine osmolality and 2:1 urine/plasma osmolality. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1276–1278.)

11. B With the lack of ketonuria, a negative toxicology screen and significant hepatomegaly on exam, the diagnosis is most likely the result of a storage disease. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1272–1275.)

12. A Blood should be collected anaerobically because CO₂ loss can alter the pH, and therefore, affect the binding of albumin. RBCs, if not removed, may cause acidosis as a result of lactate production. Some anticoagulants may attach to calcium and cause misinterpretation of calcium levels. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1281.)

13. C Lack of weightbearing is more severe in children as a result of hypercalcemia and immobility. Levels of total serum calcium more than 15 mg% may be life-threatening. Digitalis toxicity increases in the setting of hypercalcemia. Mithramycin should be avoided if surgery is anticipated because of the possibility of severe marrow suppression as a result of its administration, and therefore, complicating thrombocytopenia which could lead to significant bleeding. The product of the concentration of calcium and phosphorus should be kept below 60 when treating hypercalcemia. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1282–1285.)

14. D For any patients admitted to the ICU with hypocalcemia, treatment should be initiated unless the hypocalcemia is borderline and without symptoms. Hypocalcemia, which is resistant to the administration of repeated doses of intravenous calcium chloride, may be an indication of hypomagnesemia, hypoparathyroidism, or vitamin D insufficiency. Magnesium sulfate should not be used because the possibility exists for a complex to be formed between magnesium sulfate and calcium. A rapid magnesium infusion leads to a peak adrenal excretion. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1283–1285.)

15. C The daily requirement is approximately 0.3–0.4 mEq/kg/day intravenously. With a decrease in glomerular filtration rate, magnesium replacement may precipitate hypermagnesemia. Intravenous magnesium should not be given as a rapid bolus (as discussed previously). Magnesium chloride is preferable to magnesium sulfate because the sulphate can bind calcium. Rapid magnesium infusion leads to a poor clinical response because peak magnesium levels are associated with peak renal excretion. Aminoglycosides have been known to cause hypomagnesemia. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1285–1288; Chernow B, Smith J, Rainey T, et al. Crit Care Med 1982; 10:193.)

16. E Propranolol, labetalol, lidocaine, nitroglycerine, morphine, and verapamil are not dependent on hepatic blood flow. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1200–1205.)

17. B Dexamethasone has the least sodium retaining properties, and therefore, it is very appropriate for clinical situations where relative hypovolemia is desired. Prednisone and methylprednisolone are intermediate in terms of their salt retaining properties. Synthetic steroids are less avidly bound to protein and they undergo slower hepatic degradation, which makes them very effective in clinical practice. (Rogers MC, et al.
18. **A, A, C** Pyloric stenosis usually induces metabolic alkalosis rather than metabolic acidosis, and CAH is associated with hyponatremia in the presence of hyperkalemia. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1252–1254.)

19. **D** Ketoconazole, Bactrim®, and Etomidate are all known to cause adrenal suppression. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1252–1254.)

20. **D** With long-term steroid use, morning administration will minimize hypothalamic–pituitary–adrenal axis suppression. Also, with prolonged use of the administration of steroids, it is best to administer the dose in the morning, because this will coincide with peak diurnal variation in the endogenous steroid levels. The 30-minute adrenocorticotropic hormone (ACTH) administration test is a reliable test for adrenal suppression. Prolonged use of 12 mg/m²/day of cortisol does not cause clinical significant hypothalamic–pituitary–adrenal axis suppression. Methylprednisolone does not interfere with the common radioimmune assay method of cortisol administration. Dexamethasone administration will not interfere with subsequent measurement of cortisol, and therefore, it is used in the so-called dexamethasone suppression test. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1251–1256.)

### CHAPTER 8: NUTRITION AND GASTROINTESTINAL SYSTEM

1. **E** The fat requirement in infants is 4 g/kg/day. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1142–1145.)

2. **D** The ebb phase and the flow phase are characteristic features of hypermetabolism and not features of a starvation syndrome. The ebb phase is similar to a shock stage during which the metabolic rate is slow. The flow phase is characterized by increased metabolism. Normally with aerobic glycolysis, the end product is pyruvate. Subsequently the end products enter the tricarboxylic acid cycle (kreb cycle) for production of the high-energy adenosine triphosphate. With substantial ketonemia, this process is inhibited, and therefore, utilization of glucose is impaired. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1145–1148.)

3. **B** During hypermetabolism, which is characterized by an initial ebb phase followed by a flow phase, there is usually an associated hyperglycemia owing to decreased sensitivity to the effect of insulin, although the level of insulin may actually be higher than usual. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1145–1148.)

4. **B** After several days of starvation, the serum glucose and insulin levels gradually decrease but eventually a plateau is reached. Levels of ketones, however, continue to rise along with an increase in the level of glucagon. With continuation of starvation, nitrogen excretion falls. There is an adaptation of the brain to use the rising level of available ketones. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1145–1148.)

5. **B, C, A** These are the respiratory quotients for the various fuels. The respiratory quotient is highest for carbohydrate. Therefore, with patients who have a problem with elimination of carbon dioxide, the administration of carbohydrate should be lowered in order to minimize carbon dioxide production. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1150,1151.)

6. **E** Preventive measures that are used for stress ulceration in the ICU include feeding, which by itself, acts as a protective barrier for the gastric mucosa, or the administration of H₂ blockers, which may be administered by continuous infusion. These would include ranitidine or famotidine. Administration of antacids has been shown to be as effective as H₂ blockers. Alternatives include administration of sucralfate, which has been shown to be comparable to H₂ blockers. Enteral feedings seem to stimulate release of the hormone gastrin. Administration of gastrin itself is not one of the measures that is clinically used in an ICU as a preventative measure against stress ulceration. (Rogers MC, et
al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1167,1168.)

7. D Administration of an elemental diet has been associated with an increased release of the hormone gastrin, which seems to be trophic for the gastric mucosa. (Choctaw W, et al. Arch Surg, 1980; 115:1073.)

8, 9. D, A With gastric lavage and hemodynamic support, usually most patients with gastritis and bleeding will respond. H2 blockers have not been shown to stop gastric bleeding faster than lavage. Endoscopy should be performed to identify the site of bleeding, which if found, endoscopic therapy with electrical or laser cautery may be indicated and helpful. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1167,1168.)

10. D Vasopressin, an antidiuretic hormone, appears to be released during laparotomy and contributes to the decreased motility of the small bowel. Other contributing factors are hypokalemia, particularly with potassium levels of less than 2.5 mEq/L. The colon is the portion of the bowel most dependent on neural control to achieve motility. This is the portion of the bowel that is most sensitive to anesthesia-induced inhibition of motility, and the last to recover. The role that handling or direct manipulation of the gut plays in the development of ileus is not very clear. (Livingston E, Passaro E. Dig Dis Sci, 1990; 35:121.)

11. E Ogilvie's syndrome, which is a localized ileus of the bowel leading to pseudo-obstruction, is associated with inflammatory conditions in the intra-abdominal or para-abdominal regions. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1168,1169.)

12. D Postoperative intussusception that is usually ileoileal rather than ileocecal (which is seen in late infancy) is a problem that can be overlooked in the postoperative patient, particularly in patients who are receiving analgesia for postoperative care along with nasogastric suctioning to decompress the bowel. However, this is important to recognize to avoid morbidity and mortality. (Ein H, Ferguson J. J Pediatr Surg, 1971; 6:16.)

13. E Please see the answer to Question 10.

14. D In the setting of postoperative ileus, if the cecum is very dilated, particularly if the diameter is greater than 12 cm, there is a very high risk of perforation even in the absence of mechanical obstruction. The only effective treatment for postoperative ileus is nasointestinal intubation to decompress the bowel and supportive measures. Neostigmine has not been shown to be a safe therapeutic intervention, and is associated with significant side effects. (Adams J. Arch Surg, 1974; 109:513; Livingston E. Dig Dis Sci, 1990; 35:121.)

15. D Because of the counter-current mechanism, the oxygen delivery is least to the tip of the villi. Impaired blood flow to the bowel leads to dilation of the bowel which leads to overgrowth of bacteria, and this can lead to malabsorption, including fat malabsorption. (Perman PA. In: Hokelman RA. *Principles of Pediatrics*, New York, McGraw Hill, 1978; p. 808.)

16. C Clostridium difficile is an important infection to recognize in the ICU, particularly where broad-spectrum antibiotics have been utilized. It presents with diarrhea, which can be bloody in nature and associated with significant volume loss. When this is diagnosed, usually by obtaining a toxin assay, oral vancomycin or intravenous metronidazole are usually effective. Stool culture for corona virus and rotavirus are important for epidemiological studies, but will not contribute to a patient’s therapeutic intervention, nor do small bowel radiography or colonoscopy. (Viscidi RP. Pediatrics, 1981; 67:381.)

17. C Acute pancreatitis is a medical condition characterized by inflammation of the pancreas with subsequent release of the enzymes amylase and lipase. The degree of serum amylase does not seem to be proportional to the severity of acute pancreatitis. Serum lipase levels seem to be elevated for a longer period of time than serum amylase. Pancreatic trypsinogen serum levels seem to rise early in the course of pancreatitis and remain elevated for up to 5 days. In a clinical situation where amylase and lipase are normal and there is a high suspicion of pancreatitis, one could look at the level of trypsinogen. Some of the bad prognostic signs of acute pancreatitis include hyperglycemia, leukocytosis, hypocalcemia, and azotemia. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1175–1178.)

18. D Reye’s syndrome which has practically vanished and is very infrequently seen today, is characterized
by alteration of mental status that can progress to coma in association with derangement of the liver enzymes and alteration in the coagulation profile. However, an increased level of bilirubin or jaundice is not a recognized feature of this condition. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1178–1180.)

19. C Patients who develop fulminant hepatic failure as a result of hepatitis B virus infection (when compared with patients who do not progress to hepatic failure) tend to have earlier appearance of antibodies against hepatitis B surface antigen. Also, they have earlier appearance of antibodies against hepatitis B, E-antigen, and more rapid clearance of the hepatitis B surface antigen. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1179–1181.)

20. D Intravenous fat emulsions may not be tolerated well in patients with significant hepatic disease because it may not be metabolized by these patients. Accumulation of fatty acids intrahepatically may further compromise the hepatic function. Furthermore, non-esterified fatty acids may compete with tryptophan for binding to albumin. This may increase the risk of encephalopathy. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1179–1181.)

21–23. D, C, E The initial intervention for an upper gastrointestinal (GI) hemorrhage is gastric lavage and supportive measures which would include correction of any coagulopathy and use of volume expanders, either crystalloids or colloids. In patients who are hemodynamically unstable owing to upper GI hemorrhage, adequate volume expansion is crucial and this should not be withheld, even in patients who have evidence of edema. If the patient does not respond to initial intervention, an endoscopy should be performed, and if any localized area of bleeding is identified, this can be treated through endoscopy with electrical or laser cautery, or with the application of topical coagulants as indicated. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1178–1187.)

24. D Patent foramen ovale is not a recognized cause or a contributing factor to hypoxia in patients with hepatic failure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1182.)

25. D Ibuprofen, a nonsteroidal anti-inflammatory medication, can reduce renal plasma flow as well as glomerular filtration rate. This would result in water retention, dilutional hyponatremia, and ascites, which might be resistant to diuretic therapy. It appears that prostaglandins are important in renal vasodilation and ibuprofen may compromise this physiologically important parameter that maintains renal blood flow in patients in hepatic failure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1183.)

26. B Hepatorenal syndrome is characterized by low urine sodium owing to the hyperaldosteronism. The associated high antidiuretic hormone levels lead to urine osmolality, which is generally greater than the serum osmolality. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1183.)

27. C Hepatorenal syndrome can develop in a setting of isovolemia. However, preventive measures that have been shown to be helpful for this clinical condition include avoiding large volume paracentesis in order to avoid intravascular volume depletion, as well as use of potent diuretics, which can also lead to intravascular volume depletion. Use of dopamine has not been shown to be effective for this clinical condition. In the early stages when this condition is suspected, expansion of intravascular volume with salt-poor albumin to raise the central venous pressure to the upper limits of normal is a helpful preventive measure. Other preventive measures include avoidance of prostaglandin antagonists, such as ibuprofen. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1183.)

28. E Arterial ammonia is preferred to venous ammonia; however, there is no positive correlation between the grade of encephalopathy and the height of the ammonia. Not all patients with hepatic encephalopathy (HE) have elevated ammonia levels. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1183.)

29. E Measures to decrease protein intake, as well as elimination of colonic bacteria by use of oral lactulose, oral antibiotics, such as neomycin, have been shown to be effective for HE. Use of hypertonic glucose to provide calories is also an important measure in the management of these patients. (Butterworth RF. Dig Dis Sci, 1992; 37: 321–327.)

30, 31. D, E In a patient with HE, there is inappropriate pathological cerebrovascular tone along with
altered permeability of the blood–brain barrier that contributes to their symptomatology. In these patients, intracranial pressure monitoring along with hyperventilation to lower the PCO2 will facilitate management. Steroids have not been shown to decrease mortality in these settings. These patients should be considered for hepatic transplantation and evaluated for this procedure in the initial stages of ICU admission because it has been shown that if the patient progresses to decorticate posturing and becomes ventilator dependent, it usually too late to initiate liver transplantation. (Zaki AEO, et al. Experientia, 1983; 39:988; Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1193–1195.)

32. B, A, D, C, E Pulmonary edema is rarely associated with administration of OKT3. Therefore, patients who are receiving OKT3, usually in the post-operative period, are monitored in the intensive care setting. Their fluids and electrolytes are adjusted very carefully to prevent pulmonary edema. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1202–1204.)

33. A Please see Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1197–1199.

34. B This form of hemolytic anemia is usually self-limited and resolves spontaneously within 2–4 weeks. During this period, a serial reticulocyte count is often helpful in monitoring the progression or regression of this hematological problem. Haptoglobin may not be useful in this setting because the level of haptoglobin may be decreased owing to underlying liver disease. (Ramsey G, et al. N Engl J Med, 1984; 311:1167.)

35. A Liver disease is usually not homogenous, and therefore, drug metabolism is affected to a variable degree depending on the type of medication. It seems that the process of glucuronidation is more resistant to abnormalities in function than the process of oxidation, and therefore, in treating a patient with liver disease, preference should be given to drugs that are metabolized through this pathway. For drugs that undergo significant hepatic biotransformation clearance of these drugs tends to be proportional to the degree of liver blood flow. (Bass NM, Williams RL. Clin Pharmacokinetics, 1988; 6:396.)

36. C Branched chain amino acids have been shown to be of some use in chronic liver disease, however, they do not resolve HE on a consistent basis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1184–1186.)

37. A, D, C, B These are the half lives of various proteins, which can be used to evaluate the nutritional status of patients. Albumin has the longest half-life of 20–21 days. On the other hand, pre-albumin has a half-life of 2 days, and transferrin has a half-life of 8 days. Retinol-binding protein has a very short half-life of only 10 hours, and therefore, can be evaluated in patients who are suspected of having a recent onset of their nutritional deficiency. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 1149.)

38. C Medium-chain triglycerides (C6 to C12) inhibit gastric emptying less than long-chain fatty acids, and are absorbed from the GI tract faster than long-chain fatty acids. Consequently, they convert into energy more rapidly than the long-chain fatty acids, or long-chain triglycerides. Medium-chain triglycerides are absorbed directly into the systemic circulation through the portal venous system, instead of being absorbed through the lymphatic lacteals and subsequently into the thoracic duct. (Fuhrman BP. Pediatric Critical Care, 2nd Edition; p. 907.)

39. E The presence of reducing substances in stool suggests carbohydrate malabsorption. Disaccharidases, which are located on the brush border may be diminished following acute injury and contribute to malabsorption of carbohydrates. Protein hydrolysate formulas, such as alimentum, nutramigen, and pregestimil are predigested for ease of nutrient absorption and are suitable to critically ill infants. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1152,1153.)

40. B Stress ulcers are a recognized complication in critically ill children and are usually located high in the fundus of the stomach. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition, 1996; pp. 1165–1167; Menguy R, Master YF. Gastroenterology, 1974; 66:1172.)

41. C Ranitidine (Zantac®), Famotidine, and proton pump inhibitors decrease gastric concentration. Sucralfate does not affect gastric pH or its concentration. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1165–1168; Fuhman
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42. A Iced saline lavage offers no advantages over room temperature saline lavage. A significant reduction in core body temperature is a potential complication of iced saline gastric lavage in young children. (Furhman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 919–932; Levin D, et al. Essentials of Pediatric Intensive Care, 1990; pp. 565–572.)

43. C The majority of patients who die because of fulminant hepatic failure are found to have cerebral edema. Many of these patients have evidence of transtentorial herniation. Infection and sepsis are common but usually do not cause death. Gastrointestinal hemorrhage is also common, and is usually related to gastritis or ulceration. (Ware AJ. Gastroenterology, 1971; 61:877; Canalese J. Gastroenterology, 1982; 23:625.)

44. B Local complications of pancreatitis include pancreatic necrosis, pancreatic abscess, and pseudocyst formation. ARDS may occur with pancreatitis. Renal dysfunction is seen frequently in the setting of acute pancreatitis, and is related to hypoperfusion, hypotension, and volume loss. Specific renal injury, such as glomerulonephritis, has not been noted with acute pancreatitis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition, pp. 583,584. Frey CF, Bradley EL. Surg Gynecol Obs, 1988; 167: 282.)

45. D Toxic megacolon is usually a complication of ulcerative colitis but is rarely involved in patients with pseudomembranous enterocolitis, Crohn’s disease or ischemic colitis. Factors involved in precipitation of toxic megacolon include barium enema, opiates, anti-cholinergics, anti-diarrheal agents, and electrolyte derangements. (Dorudi S, Berry AR, Kettlewell MG. Br J Surg, 1992; 79:99–103; Ulshen M. Nelson’s Textbook of Pediatrics, 15th Edition, pp. 1080–1087.)

46. D This patient has typhlitis, which is a necrotizing colitis involving the cecum. This is common among patients with immune deficiency. Typhlitis is a life-threatening condition that causes severe abdominal pain, GI bleeding, and fever. Medical management includes discontinuing oral intake, aggressive fluid management followed by total parenteral nutrition, antibiotics, and FFP to maintain adequate coagulation status. Colonoscopy would be contraindicated because of risk of perforation. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1174,1175; Katz JA, Wagner ML. Cancer, 1990, 65: 1041–1047.)

CHAPTER 9: IMMUNOLOGY

1. E The developmental pattern of immunoglobulins (Igs) is as follows: IgG transfer across the placenta occurs as early as 8 weeks gestational age. Its level is directly proportional to gestational age, but is still less than 50% of term levels at 28 weeks gestation. The IgG levels fall during the first four months of extraterine life reaching adult levels by 4–6 years of age. By the 10th week of gestation, the fetus is capable of producing IgM and may make large quantities in the presence of a congenital infection. IgA is not measurable until late in gestational life and is very limited in the infant, failing to reach adult values until puberty. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 916.)

2. C The immunological function of the neonate undergoes maturation in both the cellular and humoral components with the child’s development. The neonate’s T-cells are unable to produce certain cytokines, which affects the interaction between T-cells and B-cells. In addition, there is a greater reactivity of T-suppressor cells relative to T-helper cells compared with those of the normal adult. Premature and full-term infants are deficient in all of the measurable products of complement activation. The newborn’s phagocytes exhibit diminished motility, adherence, and chemotaxis. Bacterial killing by polymorphonuclear leukocytes, which depends on the generation of oxygen-derived free radicals, is intact in healthy-term and most premature newborns. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 916,917.)

3. A, B C3a is an anaphylatoxin that induces smooth muscle contraction, histamine release from basophils and mast cells, and increased vascular permeability. The C5b–C9 components are referred to as
the membrane attack complex, which leads to cell lysis. 
(Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 917: Table 28.2.)

4. **T, F, T, T, T, F** A variety of drugs and diseases can affect immune function. For example, N2O decreases both T-cell responses to mitogen, and B-cell proliferation and activity. Halothane decreases phagocytosis, bacterial killing, and chemotaxis and has a depressant effect on reticuloendothelial phagocytic activity. The administration of thiopeptals and other barbiturate agents at anesthetic levels for as little as 30 minutes can produce granulocytopenia. Longer exposures to pentobarbital have resulted in an 80% decrement in the circulating granulocyte count. The major adverse effect on immunity produced by narcotics, such as morphine sulfate, is depression of leukocyte chemotaxis. A surgical wound dramatically increases the circulating neutrophil count. This is related to certain humoral effects of trauma, most notably, a strong, acute catecholamine release that is one component of the body's nonspecific response to stress. Blood levels of B-lymphocytes and T-lymphocytes decrease in response to surgical stress. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 918-919, table 28.3.)

5. **B** Secretory IgA appears later than serum IgA (already limited in the infant; see Question 1). Diseases whose defense depends primarily upon secretory IgA, such as some of the viral respiratory agents (e.g., respiratory syncytial virus) and infectious diarrheas, remain prevalent throughout infancy. The infant is at risk for encapsulated organisms and cannot localize infections well. IgM production by the non-infected newborn does not reach adult levels until 1–2 years of life. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 916,917.)

6. **B** A number of immune function alterations have been documented following major trauma. Many of these have also been implicated in the post-trauma sepsis syndrome. Of those listed, only the primary response to immunization does not increase. Prostaglandin E2, interleukin-6, tumor necrosis factor-α, and transforming growth factor-β all increase. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 919, table 28.4.)

7. **A** Although the majority of infections in the post-surgical patient are nosocomial, the most likely offending organisms are aerobic Gram-negative bacteria (*Escherichia coli*, *Proteus*, *Pseudomonas*, *Klebsiella*, *Enterobacter*, and *Acinetobacter*). Controversy exists over the use of prophylactic antibiotics; however, prophylactic antibiotics are most beneficial in injuries involving the large and the small bowel, and in soft tissue crush and extremity avulsion injuries. In the setting of GI contamination, Gram-negative and aerobic organisms are particularly likely. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 919,920, table 28.5.)

8. **E** In the management of burn victims, feeding by the enteral route is preferable to parenteral nutrition, because food in the gut can decrease the rate of organism and toxin translocation across the GI tract. Burns lead to a reduction in Ig levels, chemotaxis, and T-helper lymphocytes. Colonization of the burn wound in 5–7 days postinjury is predominantly with Gram-positive bacteria. Arginine influences postburn recovery by stimulating wound-healing, potentially through its roles in the formation of nitric oxide, by enhancing growth hormone secretion from the pituitary gland and by directly modulating immune function. However, high concentrations of dietary lipids, especially of the ω-6 series, may contribute to the development of postburn sepsis by augmenting the plasma concentration of prostaglandin E2 and prostacyclin. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 921.)

9. **B, C, A** As mentioned earlier, in the early postburn period, Gram-positive organism infections predominate. The most likely organism is *Staphylococcus aureus*, which has an insidious course, and is associated with a low mortality. Of the Gram-negative organisms that later colonize the wound, *Pseudomonas aeruginosa* and *E. coli* are the most prevalent. *Pseudomonas* infection can be particularly dangerous because it has a propensity to further devitalize intact tissue, and may convert a partial thickness burn to a full thickness one. *Candida albicans* and other *Candida* species can cause some of the most severe infections, and are associated with the highest mortality. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 921–922, tables 28.7 and 28.8.)

10. **C** The second most common cause of death in the pediatric trauma victim who survives the initial postinjury period is infection. In addition, infection is
the major cause of death among burn victims who survive initial fluid resuscitation. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 919, 921.)

11. **T, F, T** Kwashiorkor (protein deficiency) and Marasmus (protein and calorie deficiency) have more significant effects on cell-mediated immunity. Children with kwashiorkor have very small thymus glands, with relative atrophy of lymph nodes and spleen. Qualitatively, this is expressed as an increased incidence of infections with viral (especially measles and disseminated herpes), fungal (*Candida*), and opportunistic organisms (*Pneumocystis carinii*). In kwashiorkor, the thymus is typically small. The B-cell system is relatively spared in children with protein calorie malnutrition. Seroconversion in response to immunization with diphtheria and tetanus toxoids, pneumococcal polysaccharide, and polio vaccines is normal even in malnourished children. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 921–924, tables 28.9 and 28.10.)

12. **A** Although steroids do not diminish serum antibody concentrations, they decrease the circulating pool of T-lymphocytes by sequestering these cells in extravascular sites. Steroids also reduce the production of IL-1 and IL-2 and adversely affect macrophage maturation. This results in diminished antigen processing and presentation to lymphocytes for antibody production. Monocytic killing of bacteria and fungi is inhibited by the administration of steroids. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 923.)

13. **A, B** Cyclosporine asserts its major effects by decreasing both IL-2 production, and γ-interferon. The major complications of cyclosporine include renal failure and systemic hypertension. Azathioprine inhibits purine synthesis and decreases RNA and DNA synthesis. The major complications of azathioprine include nausea, vomiting, diarrhea, and decreases in both white blood cell and platelet counts. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 924, table 28.11.)

14. **D** Asplenia in children can result from a variety of conditions ranging from trauma to sickle cell disease. This makes the ability to perform randomized clinical studies of asplenia difficult. Congenital asplenia is most often associated with cardiac abnormalities (heterotaxy). Absence of the spleen, whether anatomic or functional, predisposes the young child to potentially fatal sepsis from encapsulated bacterial species. The most prevalent offender is the pneumococcus in 50% of cases. *Haemophilus influenzae* type B, meningococcus, and group A streptococci account for 25%. There is general agreement that immunization of asplenic patients with pneumococcal vaccine should be performed. Children who are less than 2 years of age have a poor response to pure polysaccharide vaccines, so immunization at a young age is not feasible. Although penicillin prophylaxis has become routine, others have suggested that prompt administration of antibiotics with any febrile illness will more reliably reduce the percentage of fatal episodes. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 926–927.)

15. **C** Such a communication is known as a dural fistula and may be suspected by the finding of rhinorrhea or otorrhea following closed head trauma. In adults, posttraumatic meningitis has been reported in up to 25% of those with basilar skull fractures. Organisms most frequently implicated are *Streptococcus pneumoniae* (50–90%), *Haemophilus influenzae* type b (9%), and other streptococcal species (10%), with other organisms, such as *Neisseria meningitides* (5%), *Staphylococcus aureus* (5%), *S. epidermidis* (2%), and enteric Gram-negative organisms (4%). (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 920.)

16. **D, C, E, B, A** Wiskott-Aldrich Syndrome is an X-linked recessive disorder manifested by eczema, thrombocytopenia, and recurrent infections with encapsulated bacteria. The disorder is related to an inability to produce antibody to polysaccharide capsule. Serum immunoglobulins show a decrease in IgM, with an increase in IgA and IgE. Ataxia-Telangiectasia is an autosomal recessive disorder involving 11q22-23. The disorder results from a defect in DNA recombination. Breakpoints involve genes that encode for T-cell receptors. Associated conditions include IgA deficiency and lymphosarcoma. Chronic granulomatous disease involves a defect in any one of the four components of the enzyme NADPH oxidase, essential for bacterial killing in the neutrophil. 65% are X-linked, and the remainder are autosomal recessive. Organisms that are catalase-positive (*S. aureus*) can produce chronic infection by preventing phagocytes from using microbial generated hydrogen peroxide. Chediak-Higashi Syn-
drome involves defective chemotaxis, phagocytosis, and natural-killer (NK) activity because of elevated levels of cyclic adenosine monophosphate. Abnormal giant granules formed by the fusion of lysosomes are seen in cells that contain lysosomes. The clinical characteristics include recurrent pyogenic infections, albinism, photophobia, and nystagmus. Schwachman-Diamond Syndrome is a disorder that involves deficiency of the exocrine function of the pancreas and neutropenia secondary to bone marrow failure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 932–936.)

17. **E** A recurrent infection with only one source is often related to an anatomical defect rather than immunodeficiency. All others listed are characteristics that should make the intensivist suspicious of an underlying immunodeficiency. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 936)

18. **C** Childhood sexual abuse and undetermined risk factors account for less than 1% of pediatric AIDS cases. Perinatal transmission from infected mother to infant is the most common means by which children acquire HIV infection. HIV antibody screening of all donated blood products, as well as donor self-exclusion programs, were initiated in the early 1980s has lead to a finite risk of transmission from infected blood products. Enzyme-linked immunosorbent assay (ELISA) is the primary screening test for HIV infection because of its very high degree of sensitivity, reproducibility, and low cost. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 946–949.)

19. **C** Although adults initially infected with HIV undergo an acute influenza like illness accompanied by a rapid fall in CD4 counts and a rise in viral antigenemia, children rarely demonstrate such clinical symptoms or viremia. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 946,947.)

20. **T, T, F, T, F, F** Among perinatally infected children, the earliest clinical manifestations include lymphadenopathy, hepatosplenomegaly, hypergammaglobulinemia, and skin disease including candidal dermatitis or seborrhea. ELISA is the primary screening test for HIV infection because of its very high degree of sensitivity, reproducibility, and low cost. The ELISA detects antibodies to HIV usually within 6–12 weeks of the primary infection. Western blot is the most widely used confirmatory test for HIV. The Western blot detects viral protein antigens. In infants younger than 18 months of age, serum tests for IgG antibody to HIV do not differentiate between infant and passively acquired maternal antibody. Polymerase chain reaction permits amplification of HIV viral DNA. This process is as sensitive and specific as viral culture. Hypergammaglobulinemia, not hypogammaglobulinemia is an early clinical manifestation of HIV infection. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 946–951.)

21. **E** *P. carinii* pneumonia (PCP) becomes attached to the type I alveolar cells of the lung. Most normal children have serological evidence of latent infection with *P. carinii*. Therefore, this infection in older children and adults is presumably the result of reactivation of the organism. Patients with PCP typically have the tetrad of nonproductive cough, fever, dyspnea, and tachypnea. Bronchoalveolar lavage is the most widely used method for obtaining lung fluid to diagnose PCP. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 953,954.)

22. **A, B, B** Patient intolerance to trimethoprim–sulfamethoxazole may result in cutaneous reactions ranging from a mild rash to TEN or Steven’s-Johnson syndrome. Pentamidine has a wide variety of side effects including pancreatitis, hypoglycemia, hyperglycemia, neutropenia, thrombocytopenia, and azotemia. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 954,955.)

23. **C** Aerosolized pentamidine, in adults has been shown to be a less effective regimen than intravenous pentamidine for PCP. All of the other statements are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 955.)

24. **B** Measles is highly contagious and may have devastating effects on the lung. Measles itself may be associated with pneumonia. In addition, other viruses and bacteria may secondarily infect the lung. Herpes simplex virus, adenovirus, *S. aureus*, and Gram-negative bacilli are among the most frequent etiologies. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 957.)

25. **D** Children with biopsy proven minimal change disease or focal glomerulosclerosis have been
treated with prednisone, but no clear responsiveness has been demonstrated. Although 30–55% of infected children will develop renal disease at some point in their illness, children tend not to follow the clinical pattern of adult patients including a rapid loss of renal function and death. Children will often manifest hypernatremia and histological evidence of focal segmental glomerulosclerosis. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 961,962.)

26. F, T, T, T, T, T, T In series that include asymptomatic, mildly symptomatic, and children with advanced neurological disease, a 19.6% prevalence rate of HIV encephalopathy is reported. Although rare, primary CNS lymphomas are the most common intracranial mass lesions that develop in HIV-infected children. In a multicenter study sponsored by the National Institute of Child Health and Human Development, intravenous Ig-treated children with CD4 counts higher than 200/mm³ had a significant decrease in both documented and suspected bacterial infections, as well as days in hospital when compared with controls. The most important mycobacterial infections in HIV-infected children are those caused by Mycobacterium tuberculosis, and the Mycobacterium avium-intracellulare complex. A significant and troublesome infection among HIV-infected children is candida esophagitis. Treatment options include ketoconazole orally, fluconazole orally, or amphotericin B intravenously. The risk of acquiring HIV infection from needle stick exposure is approximately 0.5%. Epidemiological evidence suggests that blood is the single most infectious medium for HIV in the medical setting. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 958–967.)

27. E Of the total lymphocyte population, 55–75% are T-cells. B-cells are activated by antigen to secrete antibody. IgG and IgM are the only Ig classes that are capable of activating the classical complement pathway. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 931,932.)

28. E DiGeorge Syndrome includes clinical features of thymic aplasia, parathyroid aplasia, and conotruncal cardiac defects. The deficiency of cell mediated immunity in DiGeorge is a result of the thymic aplasia, and a relative absence of T-cells not B-cells. All of the other responses are true. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 931,932.)

CHAPTER 10: METABOLIC DISORDERS

1. B, C, A, D Slight elevation of ammonia is noted with organic acidemias; however, this is usually minimal compared with the significant hyperammonemia that is noted with urea cycle defects. Maple syrup urine disease (a disorder of branched chain amino: leucine, isoleucine, and valine) is characterized by metabolic acidosis and ketosis and significant hypoglycemia. The level of lactic acid will be significantly elevated with congenital lactic acidosis. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 820–825.)

2. E Urea cycle is present and is active only within the liver. In the brain there is no urea cycle that can detoxify ammonia. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1299–1302.)

3. C In a newborn who develops significant seizures within the first 24 hours after birth, prior to having consumed a significant amount of protein in the diet, and who does not have significant metabolic acidosis or elevation of ammonia levels, one has to think about nonketotic hyperglycinemia. Ornithine transcarbamylase deficiency is an X-linked disorder of ureacyte and is usually associated with significant elevation of blood ammonia levels. Methylmalic acidemia, propionic acidemia, and isovolemic acidemia are organic acid disorders and are usually characterized by HAGMA. Maple syrup urine disease is discussed in Question 1, and is a disorder of branched chain amino acids caused by defective branched chain ketoacid dehydrogenase. Management of glucose and pH is most important in these patients. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1299–1302.)

4. D All other responses are appropriate. The accepted hypotheses include:

   a. The synergistic neurotoxin hypothesis which states that HE results from the synergistic
accumulation of toxins, including ammonia, mercaptans, and short-chain fatty acids.

b. The false neurotransmitter hypothesis states the octopamine acts as a false neurotransmitter and is taken up and released by neurones that normally store norepinephrine and dopamine.

c. The neural inhibitory hypothesis implicates γ-aminobutyric acid (GABA) in the pathogenesis of HE.

(Jones, EA. Hepatology, 1984; 4:1235.)

5. A Hypoglycemia produces selective necrosis of the superficial cortical layers sparing the non-neuronal elements (unless hypoglycemia is profound and prolonged). Infarction is usually absent even after severe hypoglycemia. In Reye’s syndrome, nonspecific cytotoxic cerebral edema has been seen with swelling of astrocyte foot processes. The hallmark of HE is proliferation and enlargement of the so-called Alzheimer-type astrocyte, which is basically a protoplasmic astrocyte. Long-standing heparin encephalopathy has been shown to be associated with degeneration changes in layers 5 and 6 of the cerebral cortex. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition, pp. 792–794.)

6. B, A, C, D, E These are some of the co-factors that have been shown to be helpful in the various metabolic disorders. (Batshaw M. Enzyme, 1987; 38:242; Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 820–825.)

7. C With adrenocortical insufficiency, hypotension is associated with low levels of stress hormones. Thus, hypoglycemia is more of a possibility than hyperglycemia. (DiAeage AM, Levine LS. Nelson’s Textbook of Pediatrics, 15th Edition; pp. 1613-1617; Kaplan SA. Clinical Pediatric Endocrinology, 1990; pp. 181–234; Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 351–363.)

8. C Diabetic ketoacidosis in the pediatric patient is a potentially life-threatening event. Ketosis and hyperglycemia result from an imbalance of glucagon and insulin levels with an increase in catecholamines, growth hormones, and glucocorticoids. An increase in somatostatin is not associated with diabetic ketoacidosis, but it down regulates the production and release of glucagon and growth hormones. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1261–1270; Kaplan SA. Clinical Pediatric Endocrinology, 1990; pp. 127–164.)

9. B, A, D, C Diabetic ketoacidosis or any acute metabolic acidosis state will decrease PCO₂ by 1–1.5 mmHg for each millimole change in bicarbonate concentration. In severe scoliosis, there is retraction of the chest wall causing chronic alveolar hypoventilation, with the development of chronic respiratory acidosis. A bicarbonate rise of 4 mmol will occur with a rise in 10 mmHg of PCO₂. Here both pH and bicarbonate concentrations are higher than expected for the level of PCO₂ elevation suggesting a mixed acid base disorder-metabolic acidosis superimposed or chronic respiratory acidosis. Botulism will cause rapid onset of respiratory failure causing pure respiratory acidosis. In acute respiratory acidosis, the pH will fall by approximately 0.08 U for each 10 mmHg of PCO₂. Plasma bicarbonate will increase 1 mmol/L for each increase of 10 mmHg in PCO₂. Salicylate intoxication causes acute metabolic acidosis. It also stimulates the respiratory center causing coincident respiratory alkalosis. A decrease in PCO₂ is out of proportion to the fall in plasma bicarbonate, which is suggestive of mixed acid–base disorder-metabolic acidosis and respiratory alkalosis. (Kaplan SA. Clinical Pediatric Endocrinology, 1990; pp. 181–234; Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 351–363.)

10. D The rapid ACTH stimulation test is only a screening test that should be verified by a more definitive test when the patient’s condition stabilizes. An abnormal response may be due to either primary or secondary adrenocortical insufficiency. A false-negative result has been reported in patients with early ACTH deficiency. (Kaplan SA. Clinical Pediatric Endocrinology, 1990; pp. 181–234; Furhman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 826–843.)

11. C Nearly all critically-ill patients have decreased serum levels of T3 and 50% have a decrease in the level of T4 concentration with normal or low thyroid-stimulating hormone (TSH). The reduction in T3 levels results from a decrease in deiodinase activity that occurs in critical illness. This is reflected in the increase in serum level of T3 that occurs during critical illness. This enzyme is responsible for the degradation reverse of T3, explaining the increase in serum levels of reverse T3 that occurs in critical illness. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1290–1297; Wilson D, et al. J Ped, 1982; 101:113.)
CHAPTER 11: PAIN MANAGEMENT

1. D Neuroanatomical pathways are present at birth; they do not develop at 4 months of age. All of the other statements are correct. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1548–1550.)

2. D The μ receptor agonists are the most commonly used opioids for analgesia. The μ₁ receptor is the subtype that provides supraspinal analgesia. The μ₂ receptor produces respiratory depression, inhibition of GI motility, and spinal analgesia. Furthermore, the μ₂ receptors cause bradycardia and sedation. Newborns may be sensitive to an age-related receptor phenomenon that leads to opiate-induced respiratory depression. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1552–1554.)

3. D, B, A, D, A The μ₂ receptor inhibits GI motility. Methadone, morphine, fentanyl, and meperidine are agonists for the μ receptor. The κ receptor inhibits antidiuretic hormone release. The δ receptor produces analgesia, respiratory depression, euphoria, and physical dependence. The psychotomimetic effects that are observed with some opiates including dysphoria and hallucinations are associated with the σ receptor. Phencyclidine is an agonist for the σ receptor. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1552–1554; tables 46.2 and 46.3.)

4. B, C, A Neonatal seizures can occur with the use of morphine at commonly prescribed doses. Meperidine is also associated with seizure activity. This, however, can occur in any age group and is related to the buildup of the toxic metabolite normeperidine. The serotonin syndrome occurs following the use of serotomimetic agents of which meperidine is included. When used alone or in combination with monoamine oxidase inhibitors a symptom complex characterized by myoclonus, rigidity, hyperreflexia, shivering, confusion, agitation, restlessness, coma, autonomic instability, low-grade fever, nausea, diarrhea, diaphoresis, flushing, and rarely, rhabdomyolysis and death can occur. The development of chest wall rigidity is a side effect associated with the rapid administration of fentanyl. This effect can be treated with the administration of either a neuromuscular blocker or naloxone. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1554–1559; Bodner RA, et al. Neurology 1995; 45:219–223.)

5. E All of the statements regarding the μ receptor agonist drugs are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1552–1560.)

6. B Fentanyl is approximately 100 times more potent than morphine and is largely devoid of hypnotic or sedative activity. Sufentanil is approximately 10 times more potent than fentanyl. Alfentanil is approximately 10 times less potent than fentanyl. The α₁ acid glycoprotein is an acute phase protein that inhibits platelet aggregation and phagocytosis, and may help to regulate collagen fiber formation in healing. Fentanyl is tightly bound to α₁ glycoprotein. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1557–1558; Fuhrman BP, *Pediatric Critical Care*, 2nd Edition; p. 807; Wilson AS, et al. Anesth Analg 1997; 84: 315–318.)

7. B Meperidine is 10 times less potent than morphine. It is unique among the opioids in its ability to stop shivering from amphotericin, blood products, anesthetics, and hypothermia. The metabolite normeperidine causes CNS excitation and seizures. See response to Question 4 from this section regarding the interaction of meperidine and monoamine oxidase inhibitors. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1558,1559; Bodner RA, et al. Neurology 1995; 45:219–223.)

8. D Methadone has a half-life of approximately 19 hours. Clonidine is useful as an adjunct to treat opioid withdrawal. The bioavailability of methadone from the GI tract is excellent. It is estimated at 80–90%. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1559.)

9. D Hydromorphone has a rapid onset of action with a 4- to 6-hour duration. It is 6–7 times more potent than morphine and 10 times more lipid soluble. Its half-life is 3–4 hours. Hydromorphone is far less sedating than morphine and associated with fewer systemic side effects. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1559.)

10. D Lipid solubility affects the transition of opioid analgesics from the aqueous side of the CSF to
the lipid phase of the underlying neuraxis where the receptors are located. Hydrophilic agents, such as morphine, have a greater latency and duration of action than the more lipid soluble agent fentanyl. However, the lipid soluble agents produce more segmental analgesia associated with less rostral spread than the less lipid soluble agonists. Spinal opiates produce analgesia without altering autonomic or neuromuscular function. In addition, both light touch and proprioception are preserved. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1561,1562.)

11. D Naloxone is a nonselective, opioid antagonist that works in small doses to alleviate the respiratory depression associated with opioids without affecting the analgesic properties. It is rapidly metabolized in the liver. Naloxone has no effect on the mental status of patients who have not received opioids. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1562,1563.)

12. B Vertical or horizontal nystagmus heralds the onset of the loss of consciousness after ketamine administration. Ketamine is an excitatory hallucinogen that produces a coupled increase in both cerebral metabolism and blood flow. This has the effect of raising intracranial pressure. Ketamine also increases mean arterial blood pressure, heart rate, and cardiac output through an increase in plasma catecholamine levels. If a patient is catecholamine deficient, profound hypotension or death may occur. Nightmares occur as a side effect in about 10% of adult patients and 5% of pediatric patients. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1563,1564.)

13. T, F, T, T, T, T Ketamine is a ventilatory depressant that reduces the ventilatory response to carbon dioxide. Laryngeal reflexes remain intact but this does not preclude the potential for aspiration. Ketamine increases pulmonary compliance by direct action on bronchial smooth muscle and indirectly by increasing plasma catecholamine levels. The drug is highly lipid soluble. Its redistribution rather than biotransformation or elimination is responsible for its short half-life. Reduction in liver blood flow leads to a prolongation of the serum half-life of ketamine. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1563,1564.)

14. E The local anesthetics are of two types, amides and esters, which are both weak bases that block nerve conduction at the sodium channel when they are in their ionized form. In order to gain access to the channel, the drug must cross the membrane. It does this in its ionized form. The minimum concentration of a local anesthetic is the concentration necessary to block nerve impulse conduction. Unmyelinated nerve fibers carry nociceptive information and have a lower minimum concentration than heavily myelinated fibers. Less local anesthetic is necessary to block the transmission of pain than is necessary to produce muscle paralysis. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1565–1567.)

15. 4, 2, 3, 5, 1 The absorption of local anesthetic is dependent on the site of the block. The order of absorption from highest to lowest is intercostal, intratracheal, > caudal/epidural > brachial plexus > distal peripheral > subcutaneous. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1566.)

16. E GABA is the major inhibitory neurotransmitter in the brain. Glycine is an inhibitory neurotransmitter in the spinal cord and brainstem. The GABA receptor has two α and two β subunits. Binding of benzodiazepines to the α subunits of the GABA receptor facilitates binding to the β-receptors and promotes membrane hyperpolarization and resistance to neuronal excitation. The benzodiazepines can blunt or abolish the respiratory responses to hypercarbia and hypoxia. They produce hypoventilation by reducing tidal volume. The benzodiazepines produce minimal cardiac affects. However, they do reduce preload and afterload. They increase, rather than decrease coronary sinus blood flow and myocardial oxygen consumption. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1573–1574.)

17. T, F, T Midazolam is four times more potent than diazepam and can be painlessly administered. When used for continuous sedation, usually for more than a week, dependence and withdrawal may occur when the drug is stopped. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1574–1576.)

18. B, A Recovery from the effects of thiopental and methohexital are a result of drug redistribution, and not biotransformation or elimination. Pentobarbital is used to induce sleep. It acts within 10–15 minutes of an intravenous or intramuscular injection and lasts...
approximately 2–6 hours. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1576.)

19. **T** The incidence of cardiac arrest associated with anesthesia is 1:600 for infants, 1:700 for children, and 1:2500 for adults, respectively. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1577.)

20. **E, C, A, B, D** The preoperative assessment of surgical risk or postoperative complications has been classified according to the American Society of Anesthesiologists. The classification ranges from class 1 to class 5. As the patient’s physiological dysfunction worsens, a higher class is attained. This higher class has an increased risk of anesthetic-related complications and death. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1577; table 46.12.)

21. **E** All of the agents listed stimulate histamine release. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1577.)

22. **T, F, T** The inhalational agents mentioned are commonly used in pediatric anesthesia. The rate of delivery of the agent to the alveolus is the function of minute ventilation and inspired concentration, but the rate of removal is dependent on cardiac output and the solubility of the agent. Nitrous oxide is relatively insoluble and can achieve steady state within 5–10 minutes. Halothane requires 15–20 minutes to achieve steady state. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1579–1583.)

23. **E** Hyperkalemia is associated with the administration of succinylcholine, not hypokalemia. All of the other statements are true regarding succinylcholine. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1584–1585.)

24. **E** All of the statements regarding nondepolarizing neuromuscular blockers are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1584–1587.)

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CHAPTER 12: PHARMACOLOGY/TOXICOLOGY

1. **D** In this case, the lean body-weight (the 50th percentile of weight for age) is the appropriate weight to use. (Rowland M, Tozer TN. *Clinical Pharmacokinetics: Concept and Applications*. Lea & Febiger, Philadelphia; pp. 100–110.)

2. **C** When drug accumulation is expected, which means that the ratio of dosing interval/half-life is less than three, and when there is a need to establish a therapeutic level rapidly, then a loading dose is necessary. Loading dose = Concentration × Volume of distribution. (Kearns GL. Clin Pharmakokinet, 1989; 17[Suppl 1]:29.)

3. **C** First one has to calculate the creatinine clearance, which is = \( L \times K \) / serum creatinine, where \( L \) is length of the child, \( K \) is a constant and it equals .45 for this age. Therefore, the creatinine clearance for this patient = 75 × 0.45 / 1.2. Assuming that the normal creatinine clearance is 100 mL/minute/1.73 M², the renal index would = 28.1 mL/minute, which is equal to 0.28 for this patient. Plugging all these numbers into the equation provided would result in 4.5 mg/kg/day as the appropriate dose. \( K = 0.45 \) for infants; 0.55 for 1–3 years; and for adolescents, 0.7 for boys and 0.55 for girls. (Bennett WM. Clin Pharmakokinet, 1988; 52:326.)

4. **E** Patients at risk of adrenal hypofunction who are admitted to the PICU (for nontrivial illness) require additional doses of corticosteroid coverage. The physiological dose is 12.5 mg/M² body surface area (BSA)/day of hydrocortisone. Patients with a febrile illness presumed to be secondary to a nontrivial infection, deserve doubling of the maintenance dose. Patients with a major trauma, major surgery, or generalized sepsis deserve 3–4 times the maintenance dose. When time allows, high-dose corticosteroids must be initiated 1–2 days prior to surgery, and weaned over a period of 5–7 days. Because the risk of undertreatment is higher than overtreatment in patients with a serious illness or trauma, it is reasonable for a clinician to administer 100–200 mg/M² BSA/day of hydrocortisone to these patients. Gastric acidity partially inactivates oral steroids and, therefore, higher doses are often necessary. (Migeon C. In: Collaly R, et al. *Recent Progress in Pediatric Endocrinology*, New York, Raven Press, 1981; pp. 465–522.)
5. A Adrenergic receptors comprise four subtypes: \( \alpha_1, \alpha_2, \beta_1, \) and \( \beta_2. \) Each of these subtypes and the family keeps growing. \( \alpha \) Receptors are typical postsynaptic receptors, mediating smooth muscle contraction in both the vascular tree (causing intense vasoconstriction) and the genitourinary system. \( \alpha_2 \) Receptors include presynaptic and nonsynaptic sites (such as on platelets). \( \alpha_2 \) Receptors tend to inhibit release of norepinephrine from sympathetic nerve terminals resulting in relaxation of vascular and GI tract (GIT) smooth muscles. Phenoxybenzamine, or \( \alpha_1 \) blocking agent, is the most selective \( \alpha_1 \) blocking agent, and is used for preoperative management of patients with pheochromocytoma. Prazosin is a potent but less selective \( \alpha \) blocker, and its blockade of \( \alpha_2 \) receptors (presynaptic receptors) cause uninhibited release of norepinephrine, thus countering the \( \alpha_1 \) receptor blockade. Phentolamine is likewise not a selective \( \alpha_1 \) blocker. Atenolol is a selective \( \beta_1 \) blocker. (Bravo E. N Eng J Med, 1984; 311:1298. Hoffman B. N Eng J Med, 1980; 302:1390.)

6. E Cocaine is absorbed from respiratory, GIT, and genitourinary mucosa. It is metabolized in the liver by esterases. It is metabolized by plasma pseudocholinesterase and nonenzymatic hydrolysis. The two major cocaine metabolites in urine are benzoylecgonine and ecgonine methyl ester. Most urine drug screening tests detect benzoylecgonine. There is a greater potential for toxicity in patients with pseudocholinesterase deficiency because cocaine will be less metabolized. Drug abusers ingest an organophosphate in an attempt to prolong the effects of cocaine, which also increases the risk of cocaine toxicity. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 855–856.)

7. D Deferoxamine does interfere with subsequent laboratory determination of iron level, and under these circumstances, the most accurate method of measuring serum iron is using the atomic absorption spectrophotometric method. Interestingly, deferoxamine actually potentiates the activity of Yersinia enterocolitis. Children usually require 24 hours or less of deferoxamine therapy. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 532–534.)

8–9. E, E Numerous substitutions of the phenylethylamine structure are possible resulting in different amphetamine-like compounds. These are referred to as amphetamines, although a more precise name, phenylethylamines, exists. The diagnosis of amphetamine overdose depends on a high degree of suspicion along with clinical judgment. Diagnosis by history alone is rarely helpful. There is no reliable blood analysis test and the quantitative urine test is not particularly useful for acute settings. One of the major differentiating features between cocaine and amphetamines is the duration of action, which lasts for about 2 hours in the case of cocaine. The half-life of amphetamines on the other hand ranges from 8 to 30 hours. Amphetamines enhance the release of, and block the reuptake of, catecholamines, resulting in excess stimulation of both \( \alpha \) and \( \beta \) receptors. At higher doses, they can cause release of serotonin. The clinical manifestations are that of cardiovascular and CNS excitation. Do not neglect to obtain a rectal temperature in these patients. Hyperthermia, if not recognized and treated aggressively, may be rapidly fatal in association with delirium. These patients are often very agitated and require sedation because agitation against restraints may exacerbate the associated rhabdomyolysis. Benzodiazepines are the drug of choice because neuroleptic agents lower seizure threshold, alter temperature regulation, and may induce dystonia. Death is often from hyperthermia, dysrhythmias, or intracerebral hemorrhage. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 863–869.)

10–16. B, A, C, C, D, D, E Any child who has ingested more than 20 mg/kg body weight of elemental iron and who has not vomited spontaneously (and is awake) may be given syrup of ipecac and brought to the emergency department. If GI symptoms develop within 6 hours in these children or children who have ingested less than 20 mg/kg body weight of elemental iron and have a level of less than 500 mg/dL of serum iron, they may be discharged home because it is unlikely that children who present within 1 hour of ingestion, and who have not vomited, may benefit from ipecac (if not already given at home), as adult-strength pills are too large to be removed by lavage. Lavage may be performed if chewable forms are ingested or if pill fragments are seen in the vomitus or on the abdominal radiograph.

The properties of iron that promote its toxicity include: (1) first order or concentration dependent absorption that is seen even in the overdose setting, (2) absorbed iron cannot be rapidly excreted. Patients with massive overdose by history or clinical manifestations should be presumed to have taken a significant
ingestion prior to determination of serum iron levels. The most valuable time to assess serum iron is 4–6 hours after ingestion. At this time, tablet breakdown is almost complete, but iron has not been completely distributed to tissues.

Because administration of deferoxamine (DFO) interferes with the standard calorimetric method of iron measurement, the laboratory must be informed of this fact. In this case, atomic absorption method is an accurate method and overcomes the false-negative results associated with the former test. DFO is a specific iron-binding agent that binds free inorganic iron to form ferric oxide (which is reddish in color) that is excreted in urine. It is given intravenously because GIT absorption is poor. The efficacy of DFO is not explainable entirely on the basis of the amount of iron excreted. Therefore, it is possible that toxicity is prevented by making iron less available for cellular binding where toxicity occurs. Hb, cytochrome, and other protein-bound iron are not chelated. Activated charcoal is ineffective in the setting of iron poisoning, as are any of the lavage solutions that could theoretically bind the iron in the stomach.

(Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 523–530.)

17–23. A/E/A, A, B, D, A, E, C The major metabolic pathway for elimination of salicylates when therapeutic doses are used is conversion to salicyluric acid and salicylphenolic glucuronide. However, this metabolic pathway follows the Michaelis-Menten kinetics, which is a saturable form of kinetics. Therefore, in the setting of overdose, this metabolic pathway becomes completely saturated and an alternative pathway has to be available for metabolism of salicylate. This alternative pathway is option E (salicylate excretion unchanged in urine), and therefore, this route of elimination becomes of paramount importance during salicylate intoxication. Because two of the major pathways become saturated, the half-life increases from 2 to 4 hours at therapeutic doses to as long as 20 hours. Also, protein binding decreases from 90% at therapeutic levels to less than 75% at toxic levels, and Vd increases from 0.2 to 0.3 L/kg. A nomogram is of limited value and was developed to be used only 6 hours or more after a single ingestion of nonenteric coated aspirin when blood pH is known to be 7.4. Repeat testing of serum salicylate levels is mandatory every 2–4 hours after ingestion. In children, the respiratory alkalosis is transient and usually occurs with metabolic acidosis.

Respiratory acidosis with salicylate toxicity warrants an evaluation for another toxin or for pulmonary dysfunction, such as pulmonary edema, which is a rare complication of salicylate overdose. Alteration in mental status in the presence of metabolic derangements make pure acetaminophen overdose suspect, and elevation of temperature directly resulting from salicylate toxicity is an indication of severe toxicity, and often is a preterminal condition in the adult population.

Aspirin was the leading cause of child poisoning in the past; however, the incidence of poisoning resulting from aspirin has been declining over the last several years. Because acidemia tends to affect the protein binding of salicylate, hyperventilation to maintain some degree of alkalemia is clinically important in salicylate poisoning. Because salicylates are a weak acid, salicylates are ionized and less mobile in an alkaline environment, whereas with acidemia, more salicylate leaves the blood and enters the cerebral spinal fluid.

In the setting of hypokalemia, it is often difficult to achieve alkalinization because under these circumstances, there is a limitation on excretion of hydrogen ion into the tubular lumen, and one has to correct the hypokalemia in order to be able to achieve alkalinization of the urine.

(Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 501–510.)

24–32. D, A, D, C, E,E, C, D, E Acetaminophen (N-acetyl-P-aminophenol [APAP]) is rapidly absorbed from GIT and peak plasma levels almost always occur within 4 hours of ingestion. The drug is metabolized in the liver by: (1) sulfation; (2) glucuronidation, (3) p-450 oxidase system, producing the intermediate metabolite (NAPQI) thought to be responsible for the toxicity; NAPQI is normally detoxified by conjugation with reduced glutathione and excreted in urine as mercapturic acid or cysteine conjugates.

A small fraction of acetaminophen is excreted in urine unchanged. This and the product of sulfation and glucuronidation are nontoxic. In the setting of overdose, when more than 70% of glutathione is depleted, NAPQI binds covalently to hepatocytes, inducing hepatic necrosis, which is usually centrilobular with periportal sparing. Children seem to be more resistant to the toxicity of acetaminophen, presumably because of the higher activity of the sulfation pathway. One exceptional group is children on anti-convulsants, such as phenobarbital, which accelerates the p-450 mixed-function oxidase system with production of higher lev-
els of NAPQI, which is the main metabolite responsible for toxicity. These children are a higher risk and must be treated at a lower level of serum acetaminophen.

Because APAP is so rapidly absorbed through the GIT, gastric emptying is of benefit only in the first 2 hours after ingestion. Because APAP is effectively absorbed to activated charcoal and also because binding of N-acetylcysteine (NAC) to charcoal is probably clinically insignificant, most physicians would use activated charcoal with NAC, with possible repeating of the loading dose of NAC. NAC is taken up by the hepatocytes and acts as a precursor for glutathione and sulfate, replenishing reduced glutathione. When given more than 24 hours after ingestion of APAP, NAC acts as an antioxidant. NAC is administered when APAP is in the toxic range based on the Rumack and Mathew nomogram. NAC is also indicated when (1) initial AST and prothrombin time are elevated, suggesting significant ingestion, (2) when there is a history of prior or present vomiting with ingestion of more than 140 mg/kg body weight, or (3) when there is a history of a large APAP ingestion at an unknown time.

The clinical manifestations of APAP toxicity is divided into four phases: Phase I is characterized by nausea, vomiting, and malaise; phase II is characterized by hepatic dysfunction; phase III is characterized by sequelae of significant hepatic dysfunction with jaundice and coagulopathy; and phase IV occurs if phase III is not reversible.

In younger children with significant toxicity, hypotension, hypothermia, and apnea may be noted. Liver enzymes (alanine aminotransferase and aspartate aminotransferase), bilirubin and prothrombin time and partial thromboplastin time should be measured every 24 hours for 4 days while therapy proceeds. It is important to recognize that APAP measured by the calorimetric method is unreliable in the presence of high salicylates, bilirubin levels, or renal failure. In these circumstances, high-pressure liquid chromatography and enzyme immunoassay may be employed.

(Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 487–495.)

33, 34. E, C An acutely poisoned patient with a very high level of theophylline may be awake, alert, and merely tachycardic. If this patient does not exhibit tachycardia, the diagnosis of theophylline overdose is suspect or concurrent ingestion should be excluded.

The cardiac toxicity is owing to massive catecholamine (release of epinephrine and norepinephrine) stimulation of the myocardium and is aggravated by hypokalemia, hypercalcemia, and hypophosphatemia. β-Adrenergic stimulation is responsible for the electrolyte abnormalities, acid–base disturbances, and vasodilation. Metabolic acidosis, hypokalemia, and hyperglycemia are recognized features. The hypokalemia is from a transcellular shift (into the skeletal muscles).

The cardiovascular toxicity (dysrhythmias and hypotension) is worsened by hypoxia and co-administration of medications with β-adrenergic or anticholinergic activity. Anti-emetics with anticholinergic activity may worsen dysrhythmias, and if a pressor is used to elevate blood pressure, a pure a-adrenergic agent is preferred.

Massive theophylline toxicity can be effectively treated by hemoperfusion, and therefore, strong consideration should be given to initiating transfer of this patient to a facility with these capabilities, while the patient is still stable. At this same time, multiple dose-activated charcoal, intravenous β-blockers, and other supportive measures should be continued. The indications for initiation of hemoperfusion include a theophylline level greater than 90 mg/mL at any time; a theophylline level of more than 70 mg/mL 4 hours after ingestion of a sustained release tablet; and a theophylline level of more than 40 mg/mL with seizures, hypotension, or dysrhythmias. The author has treated a 2-year-old child with a theophylline level of 120 mg/mL without hemoperfusion.

(Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 567–574.)

35. C Among the opioids in this question, morphine-6-β-glucuronide is the most potent. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; p. 776.)

36. B Normeperidine is a metabolic product of meperidine, and it causes CNS excitation and seizures when it accumulates. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 777–778.)

37. A, B, C The dose of sodium nitrite needs adjustment for Hb concentration, whereas sodium thiosulfate needs adjustment for body weight. The efficacy of both these medications is increased by the coadministration of high concentrations of oxygen. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 1195, 1228, 1229.)
38. E All of the above combinations can lead to cyanide production. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 1215–1222.)

39–41. D, C, D An upper GI endoscopy is warranted to evaluate for formations of concretions and bezoars. Hypokalemia is more likely to develop because of the β-adrenergic agonist type effect of theophylline. Repeated doses of activated charcoal should be continued throughout the hemoperfusion procedure in order to minimize further absorption of theophylline into the circulatory system. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 568–574.)

42. C Intraventricular conduction defects are most commonly associated with propoxyphene overdose. Meperidine has a tendency to cause seizures, morphine causes respiratory insufficiency, and heroine has been associated with pulmonary abnormalities, such as ARDS. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 175, 198, 777.)

43. D Hypotension is multifactored in origin. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 175, 198, 777.)

44,45. A, E Propoxyphene is associated with heart block and intraventricular conduction defect abnormalities. Often, much higher doses of naloxone may be needed to reverse the toxicity resulting from propoxyphene. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; p. 198.)

46, 47. B, C Activated charcoal may be helpful in body packers. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 175, 198, 777.)

48. E 3-Methylfentanyl is an extremely potent opioid and may require higher doses of naloxone to reverse its toxicity. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; p. 30.)

49–52. B, A, C, C/D/D/A/A/B With methanol intoxication, the onset of toxic symptoms or the development of metabolic acidosis is often delayed for 24 hours, with a range of 1–72 hours from the time of ingestion. Methanol is converted to formaldehyde and then formic acid (FA). The latter is responsible for the toxicity of methanol particularly with late recognition, with subsequent build-up of FA. Two factors that correlate best with poor outcome are: (1) delay of appearance of toxic symptoms for longer than 10 hours, and (2) elevated levels of FA. Clinically, the most characteristic clinical findings are symptoms of blurred vision (the sign of dilated pupils with sluggish response to light) and hyperemia of the optic disc. These features correlate best with metabolic acidosis.

Oxalaturia and elevated levels of glycolic acid are features of ethylene glycol (EG) poisoning. HAGMA and hyperventilation are features of both. The degree of AG or EG poisoning is the largest seen in any metabolic acidosis. However, the onset of high AG metabolic acidosis may be delayed, and therefore, if the clinical suspicion is high, ethanol therapy should be initiated promptly. Because ethanol has a greater affinity for alcohol dehydrogenase than either methanol or EG, when ethanol is administered in sufficient concentration (100–150 mg/dL), it competitively inhibits formation of toxic metabolites, allowing the primary alcohol to be eliminated in urine unchanged. An optimal blood ethanol level, 100–150 mg/dL, should be attained, either orally (using a 15–20% concentration) or intravenously using a 10% concentration. Ethanol should be continued during hemodialysis at a higher dose because ethanol itself is dialyzable.

Alkalinization with NaHCO₃ is also helpful because renal clearance of glycolic acid is enhanced and the amount of undissociated FA is decreased at a higher pH, thereby limiting access to the CNS.

Additional therapeutic measures EG ingestion may include 100 mg of thiamine intravenously or 50 mg of pyridoxine intravenously every 6 hours until acidosis is resolved and EG level is zero. Pyridoxine in the presence of magnesium may shunt the metabolism of EG metabolites from glycolic acid to the harmless glycine, and thiamine may reduce production of oxalic acid.

For methanol intoxication, 50–75 mg of folic acid every 4 hours for 24 hours has been suggested. Folic acid may enhance the elimination of FA.

(Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 827–836.)

53, 54. C, C The typical initial dose of naloxone in an adolescent is approximately 2 mg intravenously. If the first dose of naloxone fails to reverse symptoms, then 2–4 mg intravenously should be given up to a total dose of 10–20 mg. In a setting where there is no ventilatory insufficiency, it is not essential to initiate high-dose naloxone. Once the patient responds, two-
thirds of the dose that reversed the respiratory depression needs to be used on an hourly basis until the patient recovers. (Goldfrank LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 26, 27, 100, 422, 770–772, 784, 785.)

55–59. D, E, D, C, E Cyclic antidepressants induce their toxic effects by:

a. Inhibition of re-uptake of neurotransmitters such as norepinephrine and Dopamine.
b. Membrane depressant effect by slowing sodium influx into myocardial cells during Phase 0 of depolarization.
c. α-Adrenergic blockade.
d. Inhibition of central sympathetic reflexes.
e. Anticholinergic and antihistamine effects.

Many of the signs noted during toxicity are caused by central and peripheral anticholinergic effects, which include agitation, confusion, hallucinations, coma, seizures (central), tachycardia, hypertension, hyperthermia, dry skin, and urinary retention (peripheral).

Cyclic antidepressants are divided into first-generation (or tricyclic) antidepressants and second-generation (or cyclic) antidepressants. These drugs have a more specific mechanism of action but their toxicity profile remains the same. Patients with antidepressant overdose often develop wide, complex dysrhythmias, hypotension, and seizures within minutes of ingestion. If a life-threatening event is going to occur, it will occur within the first 6 hours of hospitalization (most often within 2 hours of admission to the emergency department). After initial stabilization, a 12-lead EKG should be obtained and the patient placed on cardiac monitor. The finding of a small S-wave in leads I and AVL and a small R-wave in AVR along with a prolonged QT and sinus tachycardia are highly specific and sensitive for cyclic antidepressants (CAs). However, absence of these EKG changes does not exclude a cyclic antidepressant overdose (CAO). The duration of QRS has been shown to be prognostic of seizures and dysrhythmias; QRS greater than 100 msec, 30% risk of seizures; QRS more than 160 ms, 50% risk of dysrhythmias. Blood should be sent for electrolytes, glucose, and if ingestion was intentional, an acetaminophen level. It is not clinically useful or cost-effective to obtain a plasma cyclic antidepressant level because there is no good correlation between levels and symptomatology. However, with levels exceeding 1000 mg/mL, dysrhythmias and seizures are usually seen.

CAs have a membrane depressant effect on the myocardium by slowing sodium influx into the myocardium during phase 0 of depolarization. This leads to intraventricular conduction defects, dysrhythmias, decreased cardiac output, hypotension, and decreased coronary perfusion. The effects of CAs on sodium channels can be attenuated by increasing the blood pH to 7.50–7.55, either by hyperventilation or Na+ HCO3. At this pH, it appears that CA uncoupled from sodium channels, whereas hypotension and acidosis enhance their binding. (Lidocaine may also be effective in treating ventricular dysrhythmias.) Therefore, aggressive treatment of hypotension and metabolic acidosis is essential. If hypotension does not respond to fluid resuscitation, then depending on the underlying etiology, inotropic support or vasopressors may be used. Norepinephrine will increase the vascular tone, whereas dobutamine will increase the contractility without increasing the vascular resistance dramatically. Dopamine should be in this setting because of its arrhythmogenic potential.

Seizures that develop in the setting of CAO are usually brief and respond to lorazepam. For persistent seizures, phenobarbital is recommended. Phenytoin is not recommended because of the potential for dysrhythmias. Other drugs that must be avoided include: class IA and IC antiarrhythmias (membrane stabilizers); propanolol and verapamil (myocardial depressants); flumazenil (inhibits the chloride channel of α-adrenergic and β-adrenergic receptors similar to CA). Because of the rapid deterioration of mental status in patients with CAO, ipecac should not be used. Multiple dose charcoal does enhance elimination of CA and physostigmine has not been shown to be safe and/or effective in this setting.

(Goldfrank, LR. Goldfrank’s Toxicologic Emergencies, 6th Edition; pp. 726–731.)

60. A, B, C, D These are the drugs/toxins that should be in the differential diagnosis of HAGMA:

a. Paraldehyde ingestion can be diagnosed by its distinctive pungent odor. Other findings include: gastritis, mental status changes with possible coma, tubular acidosis, azotemia, oliguria, and proteinuria.

b. Toluene abuse by inhalation takes two forms:

i. Huffers inhale from a toluene-soaked cloth.

ii. Baggers inhale from a plastic bag containing toluene placed over the head.
They may present with HAGMA or renal tubular acidosis. Other symptoms are GI disturbances, musculoskeletal weakness, or neuropsychiatric disorders.

c. Isopropyl alcohol ingestion is characterized by hyperosmolality and ketonemia (with ketonuria) but without significant metabolic acidosis.

d. Iboniazide overdose is associated with seizures. Seizing patients should receive 1 g of pyridoxine for every gram of iboniazide ingested at a rate of 1 g every 2–3 minutes. If the seizures stop, the remainder may be given more slowly in D5W. A maximum dose of 5 g may be administered.

(Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 627,628.)

61. D Tachycardia may be a common feature. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 1105–1109.)

62. C Although there are authorities who believe that pralidoxime must be given within 24 hours of exposure to organophosphates, there are also reports that pralidoxime is still effective when administered beyond 24 hours after exposure. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 1117,1118.)

63. B Clinical manifestations of organophosphate poisoning are not seen until a significant portion of the cholinesterase is inhibited, and the end point for atropinization is inhibition or significant reduction in upper airway and tracheal secretions. Tachycardia is not a contraindication to atropine in this setting. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 1117,1118.)

64, 65. D, B During hemoperfusion, compounds are cleared from blood as they come in contact with an adsorbent (surface) material contained in a cartridge, within an extracorporeal circuit. The adsorbent material could be: (1) Charcoal-best for polar compounds, such as salicylates, or (2) Amberlite XAD-4-best for lipid soluble compounds, such as theophylline, phenobarbital, CAs, meprobamate, and digoxin.

Extraction of many of these compounds is almost complete and the clearance often equals the blood flow through the circuit. Many of the pharmacokinetic factors that limit the applicability of diagnosis are not significant during hemoperfusion. Thus, molecular weight, degree of protein, binding in the plasma, and water solubility are not limiting factors during hemoperfusion because of the high adsorbent area that comes in contact with the blood. The Vd remains important however. Drugs with a large Vd may be completely extracted from the blood as they pass through the adsorbent, but if only a small amount is present in the plasma compartment, only a small total amount may be removed from the body. The most frequent complications are hypotension and thrombocytopenia. Other complications are hypoglycemia, hypocalcemia and hypothermia.

(Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1302–1304.)

66. E Repeated dose activated charcoal is effective for all of these medications except for iron. Activated charcoal is ineffective in a setting of iron poisoning. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 66–69.)

67. A, A, A Carbamates do not penetrate the CNS, and their effects are usually reversible and transient. Only atropine is usually needed in a setting of overdose. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1304–1307.)

68. D PKₐ is an important concept in pharmacology particularly in the setting of overdose. For example, with salicylates, which have a PKₐ of 3.1 at a pH of 3.0, the ratio of ionized to non-ionized is 1:1. However, if the pH is increased to 7.4, the ratio of ionized to non-ionized increases to 2500:1, and this will dramatically help with elimination of the drug through the kidneys. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 503–505.)

69. D Because K⁺ is exchanged with H⁺ in the renal tubules, hypokalemia with total body K⁺ deficit will hinder urinary alkalinization. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 503–510.)

70. E For a compound to be dialyzed efficiently, it must be poorly protein bound (<90%) and highly water soluble, have a small Vd so that the majority of the drug is in the plasma, and have a small molecular-
weight; compounds with a molecular-weight higher than 500 are progressively less dialyzable. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1302–1303.)

71. **E** See answer to Questions 64 and 65.

72. **A, B, B, B** Organophosphates bind irreversibly to acetylcholinesterase (the enzyme that normally hydrolyses acetylcholine). As a result, acetylcholine accumulates at the synaptic site with subsequent continuous stimulation of the neuromuscular junction. Clinical manifestations are fasciculations, weakness, and paralysis. Myoclonus is associated with the other three groups of drugs. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1304–1306.)

73. **E** The pulse oximetry detects the ratio of oxy-Hb to the total Hb and is incapable of measuring the other different types of Hb, such as carboxy-Hb or met-Hb. Therefore, the saturation that is obtained may be erroneous. Under these circumstances, one has to measure the oxygen saturation using the co-oximeter. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 122,123, 321, 461.)

74. **D** Drugs recognized to form gastric concentrations in the setting of overdose include barbiturates, salicylates, ferrous sulfate, and slow-release theophylline preparations. In these case circumstances, attempts should be made to eliminate these concentrations from the stomach including use of endoscopy because they contribute to the toxicity of these drugs. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1326–1339.)

75. **E** Activated charcoal should be administered in almost all cases of poisoning after emesis and lavage are accomplished. Exceptions are in cases of ingestion of (1) Corrosives—whether alkaline or acids, as charcoal does not absorb either one effectively and the dark charcoal may interfere with endoscopic examination; (2) Anticholinergics—overdose with ileus is an obvious situation when repeated dose-activated charcoal should not be used; (3) Enteric-coated preparations are not well-adsorbed by activated charcoal. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 2nd Edition; pp. 1326–1339.)

76. **C** Hypovolemia is the most likely cause of hypotension in a patient with significant intoxication, although other etiologies must be kept in mind and should be appropriately evaluated and treated. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 726–731.)

77. **C** Alkalosis seems to minimize binding of CAs to sodium channels in the myocardium with resultant suppression of dysrhythmias. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 726–731.)

78. **E** All of the above drugs induce a state of sympathetic stimulation and therefore are likely to be associated with hypertension. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 1–100.)

79. **A, B, C, D, E** Detection of a distinctive odor may be a clue to diagnosis of specific poisoning. This question addresses some clinical examples. (Goldfrank LR. *Goldfrank’s Toxicologic Emergencies*, 6th Edition; pp. 1–100.)

**CHAPTER 13: TRAUMATOLOGY**

1. **D** Generally, following hemorrhage in humans, a rise in osmolality is directly related to the glucose concentration in the plasma, not the result of an influx of sodium. All of the other statements are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1467–1470.)

2. **C** Catecholamines produce hyperglycemia, hyperlipidemia, increased oxygen consumption, and hyperkalemia, and α-stimulation reduces insulin and glucagon secretion. The overall effect of catecholamines on the islet cells it to not only increase glucagon, but also decrease insulin secretion. Cortisol decreases the peripheral utilization of glucose but the increase in plasma cortisol is designed to produce an increase in osmolality in response to hemorrhage. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1470.)

3. **T, T, T, F, F, T, T** Angiotensin II is a powerful vasoconstrictor. Aldosterone works on the ascending
loop of Henle and in the collecting ducts of the kidney to increase sodium and water absorption. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1470–1472.)

4. C An attempted open-operative cricothyroidotomy may cause irreversible damage to the larynx. All of the other statements are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1474–1476.)

5. B, A, C Hydroxyethyl starch, albumin, and lactated ringers are commonly used fluid replacement solutions. Their physical properties differ and may affect selection. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1481,1482, table 43.5.)

6. C Almost all plasma coagulation factors are stable in banked blood, with the exception of factor V and VIII. With massive transfusion, defined as greater than 2 blood volumes in a child, hemostatic defects may occur as a result of dilution or a decrease in the platelet and circulating protein coagulation factors. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1482,1483.)

7. C In Weil’s 5-2 or 7-3 rule, the 5-2 applies to the central venous pressure (CVP), and the 7-3 rule applies to the pulmonary capillary wedge pressure (PCWP). Volume boluses are administered and the pressure response is measured. When the CVP is less than 8 or the PCWP is less than 12, 10–20 mL/kg of isotonic solution is infused over 10–15 minutes. If the CVP increases by more than 5 or the PCWP increases by more than 7, the infusion is stopped. Immediate fasciotomy is indicated when a pressure greater than 60 cm H$_2$O is present. Cardiac tamponade presents with paradoxical pulse and hypotension. A pulmonary hematoma takes only a few days to resolve. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1485–1490.)

8. F, T, T, T, F, T, F, T, T The rupture is more likely on the left because of the presence of the liver on the right acting as a cushion to the diaphragm. Pulmonary compliance decreases with adult respiratory distress syndrome. An intravenous pyelogram is indicated for gross hematuria with clinical evidence of renal injury and unstable clinical course of blood loss and a possible renal artery injury. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1491–1495.)

9. B Loss of consciousness of 3 minutes or more is an indication for a skull film. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1495, table 43.7.)

10. E There is a decrease in the perfusion, which results in the initial ischemic insult to the spinal cord following trauma. All of the other statements are true (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1496.)

11. T, T, T Please see Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 65, 1498, and table 2.10.)

12. B Child victims of abuse are usually younger than 2 years of age. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1498, table 43.8.)

13. E Hypertension is a commonly described phenomenon associated with thermal injury. The increase in plasma renin activity and aldosterone increases intravascular volume and raises blood pressure. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1522.)

14. E Pulmonary dysfunction after thermal injury may be secondary to inhalational injury, aspiration, shock, sepsis, congestive heart failure, or trauma. The presence of inhalational injury increases mortality by 20%, whereas pneumonia increases the risk of mortality by 40% in burn patients. In the resuscitation phase of burn injury, lung injury results from hypoxia and subsequent reoxygenation, CO and cyanide toxicity, airway edema, chest wall, and pulmonary compliance problems. Hypoproteinemia may contribute to edema formation in the postresuscitative phase. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1522.)

15. T, T, F, F, T, T, T, T, T, T, T

16. C, D, A, D C, C, A, B Renal blood flow decreases immediately after injury. Later, GFR increases coinciding with the onset of the postburn...
hypermetabolic state. Hepatic dysfunction is commonly encountered in thermal injury, and can generally be found in more than 50% of patients. Thrombocytopenia appears first, then is followed by thrombocytosis several days later. Significant increases in fibrinogen, Factors V and VIII occur. RBC mass decreases. Hypoxia occurring in the first 48 hours was the most common cause of encephalopathy and was related to smoke and CO inhalation sustained in enclosed fires. Acalculous cholecystitis is of two types in the burn patient. The first involves bacterial seeding in septic patients and the second arises in patients with dehydration, ileus, or pancreatitis in whom the gallbladder is distended with sterile fluid. Burn-injured patients are immunocompromised. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1522–1525.)

First-degree burns are superficial burns isolated to the epithelial cells and characterized by erythema and mild blistering. Second-degree burns involve a tissue depth into the dermis. A superficial partial-thickness burn is moist, red, and tender. It becomes pale, but dermal papillae can be visualized through the eschar within a few days. Third-degree burns extend through all layers of the skin and invade the hypodermic fat. Fourth-degree burns involve deep injury to bone, joint or muscle. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1525–1526.)

17. B With the Rule of 9’s, the front and back are each assigned 18% of BSA; each arm is assigned 9%; each leg is assigned 18%. Therefore, a burn that involves 9% (arm), plus 18% (leg), plus 18% (back), equals 45% total BSA burn. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1526.)

18. B, C, A A minor burn involves less than 5% of the BSA and no significant involvement of the hands, feet, face, or perineum. A moderate sized burn involves between 5 and 15% of the body surface area. Alternatively, any full-thickness component also qualifies. Involvement of the hands, feet, face, perineum, or the presence of a complicating factor, such as chemical or electrical injury, also constitutes a moderate burn. A severe burn is characterized by more than 15% total BSA burn or the presence of smoke inhalation or CO poisoning. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1526, figure 45.2.)

19. B House fires account for 84% of burn associated fatalities, the cause of which is most frequently smoke inhalation rather than tissue damage from flames. Chemical burns should be flushed with water for 20–30 minutes, not alcohol. Tetanus prophylaxis must be addressed in all burn patients. Scald burns are the most common type of pediatric burn and the home is the most common location. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1526,1527, table 45.1.)

20. E The criteria for transfer to a burn center include significant underlying disease, associated inhalation injury, 10% BSA or more of partial or third-degree burns in children younger than 10 years of age or more than 20% BSA in children older than 10 years of age, third-degree burns more than 5% in any age group, electrical and chemical burns, and burns associated with major trauma. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1527,1528.)

21. T, F, T, T, T Systemic blood pressure is usually maintained after thermal injury despite hypovolemia, thereby making blood pressure an insensitive measure of volume status. Generally, children with less than 5% of their BSA burned do not require intravenous fluid therapy. Children with a burn exceeding 15% BSA will require intravenous resuscitation. If the burn size exceeds 30% BSA, placement of a central venous catheter is recommended. Muscle relaxants and sedation are contraindicated in the child who has signs of upper airway obstruction up until the airway is secured. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1528.)

22. B, A, E, B, B Mafenide is an excellent antibacterial. It inhibits carbonic anhydrase and may lead to acidosis. It can be painful, but penetrates the eschar rapidly. It is applied twice daily. Silver sulfadiazine is a broad antibacterial agent that is painless. It penetrates fairly well through the eschar. It is contraindicated in pregnancy and has unknown absorptive properties in the fetus. Bacitracin is limited in its antibacterial action, has poor eschar penetration, but is easy to apply and cosmetically acceptable. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1530, table 45.2.)

23. D The Parkland formula recommends lactated Ringer’s solution in the first 24 hours postburn in
the amount of 4 mL/kg/% BSA burn. One half of this volume is given in the first 8 hours postburn and the remainder given over the remaining 16 hours. The resuscitation should be adjusted to maintain a urine output of 0.5–1.0 mL/kg/hour. On the second postburn day, maintenance fluid of a glucose-containing hypotonic fluid may begin. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1529.)

**24. T, T, F, F, T** Resistance to silver sulfadiazine is common for *Enterobacter cloacae*, *S. aureus*, and occasionally *P. aeruginosa*. All three of these organisms are usually sensitive to Mafenide. Silver nitrate can induce methemoglobinemia. Ideally surgical excision and closure of the wound should take place as soon as the child is stable enough for anesthesia. More than 105 organisms per gram of tissue constitute burn wound sepsis. Early surgical closure decreases significant blood loss. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1530–1531, table 45.2.)

**25. E** An adverse effect on the immune function may occur if lipid content is more than 15% of total diet kcals particularly if it is high in the ω-6 fatty acids. Enteral feeds prevent hypermetabolism and catabolism in contrast to parenteral feeds. Positive nitrogen balance may be achieved earlier with the institution of enteral nutrition within the first 4 hours. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1531–1532.)

**26. E** Thermal injury from smoke inhalation is usually limited to the supra-glottic airway. Inhalation injury accounts for more than 50% of the mortality associated with major burns. Carbon monoxide poisoning accounts for approximately 50% of the poisonings in the United States per year. The largest source of CO is generated from the incomplete combustion of carbon-containing compounds. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1534–1536.)

**27. D** The oxy-Hb dissociation curve is shifted to the left in CO poisoning, thereby enhancing oxygen affinity for Hb and impeding oxygen delivery from blood to tissue. The toxic effects of CO result from its direct action on the cytochrome-oxidase system and not solely on the reduced oxygen carrying capacity of the blood. If a significant amount of time has passed since the exposure of CO poisoning, an abnormal level may not be discovered. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1534–1536, figure 45.4.)

**28. D** The heart rate and coronary blood flow increase in response to CO. Pulmonary edema occurs in about 10–30% of cases, however, the mechanism for pulmonary edema remains speculative. Cerebral blood flow and edema also increase. The cherry-red skin color is not commonly seen clinically. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1534–1536, table 45.4.)

**29. T, F, F, T, T, T, T, T** Muscle necrosis leads to myoglobinuria and subsequent acute renal failure. Salivary amylase is responsible for development of hyperamylasemia. A mild acidosis actually shifts the oxy-Hb dissociation curve to the right, increasing release of oxygen to the tissues and so should not be treated. The half-life of CO is 5–6 hours in room air, 1.5 hours in 100% FiO2, and less than 30 minutes in 100% FiO2 in 2.5 atmospheres. Hyperbaric oxygen treatment should be instituted when a patient has a CO Hb of more than 25%, signs and symptoms of CO poisoning, and a hyperbaric oxygen facility available. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1534–1538, table 45.5, 45.6, and 45.7.)

**30. C, A, B** CO concentrations affect the presenting symptoms. A CO Hb concentration of more than 0.195 is rapidly fatal, a CO Hb of 0.022 is associated with disturbed judgement, and a concentration of 0.007 is associated with shortness of breath with vigorous exercise. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1537, table 45.5.)

**31. E** Cyanide poisoning from smoke commonly occurs and acts synergistically with CO toxicity. Smoke injury decreases ciliary function. Patients with pulmonary injury may be asymptomatic with a normal chest radiograph on presentation. Arterial blood gases may also be normal for the first 12–24 hours. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1539.)

**32. D** At low voltages, alternating current is more dangerous than direct current because of its ability to freeze the extremity to the electrical source. Joule’s law states that power equals amperage squared...
times resistance \((P = I^2R)\). Surface burns result from the
ignition of clothing or from the heat of the current travel-
ing close to the skin. Arc burns are produced by a cur-
rent that travels external to the body, as an electric arc
forms between two objects of opposite charges. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 1540.)

33. F, T, T, F, F, F, T Water content and a thinner stratum corneum decrease skin resistance in children. The conducting system of the heart is particularly vul-
erable and ventricular fibrillation can occur with a
current of 100 mA passing through the chest. Transient arrhythmias are present in 30% of patients. Tetanic spasms of respiratory muscles occur at 30 mA. Neuro-
logical findings are common. Loss of consciousness, spinal cord lesions, deafness, seizures, and changes in mood commonly occur after electrical injury. Nearly two-thirds of people struck by lightning live. (Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; p. 1541.)

34. A \(\text{CO}_2\) autoregulation is better maintained
than blood pressure autoregulation.

35. D When using uncrossmatched blood, it is
best to obtain at least an ABO and Rh type and partial
crossmatch. This is sometimes referred to as an incom-
plete or partial crossmatch. The immediate phase cross-
match eliminates serious hemolytic reactions because
of errors in the ABO typing. It will fail to detect only a
few unexpected antibodies outside of the ABO system,
most of which are clinically insignificant. If time does
not permit even a preliminary screen, ABO and Rh
type-specific, uncrossmatched blood is still preferable
(and more abundant). Of patients never exposed to
blood, fewer than 1 in 1000 will have an unexpected
antibody detected in the immediate phase crossmatch.
(Rogers MC, et al. Textbook of Pediatric Intensive Care, 3rd Edition; pp. 1482,1483.)

36. E FFP provides the equivalent clotting fac-
tors of a single unit of fresh whole blood. The adminis-
tration of FFP should be considered when 200% of the
calculated circulating blood volume has been replaced
with crystalloids and red cell concentrates. A precipi-
tous fall in platelet count may not be tolerated, as well
as a slow decline in thrombocytopenic patients. Platelet
administration begins when 100–150% of the calcu-
lated circulating blood volume has been replaced with
 crystalloid and red cell concentrates. The dilutional
coagulopathy is rapidly corrected once perfusion is
restored, but may be exacerbated by the development
or persistence of hypotension. (Rogers MC, et al. Text-
book of Pediatric Intensive Care, 3rd Edition; p. 1483.)

37. C Bleeding and edema within an intact fasci-
cal compartment can lead to the development of
increased pressure, muscle ischemia, and death.
Whereas pulses may be intact distally with a compart-
ment syndrome, one constant finding is severe pain
even with passive motion. Muscle compartment pres-
sures can be evaluated during the secondary survey of
the trauma patient using an 18-gage needle and water
manometer. Compartment pressures of 40 cm \(H_2O\)
should cause concern, whereas pressures greater than
60 cm \(H_2O\) require fasciotomy. (Rogers MC, et al. Text-
book of Pediatric Intensive Care, 3rd Edition; p. 1486.)

38. C In addition to measuring urine output, the
bladder catheter facilitates the diagnosis of urinary tract
injury and rhabdomyolysis. An oral gastric tube should
be placed in all patients with abdominal trauma. This
procedure removes air from the stomach and improves
ventilation, empties liquid and particulate matter,
decreases the likelihood of aspiration, and provides
diagnostic information concerning the presence of
blood in the upper GI tract. If a pelvic fracture is sus-
pected or seen on a radiograph, a rectal examination
should be performed to evaluate the possibility of bone
fragment injury to pelvic structures. Pain on passive
range of motion is a constant finding in compartment
syndrome. See response to question 37. (Rogers MC, et
al. Textbook of Pediatric Intensive Care, 3rd Edition;
pp. 1485–1487.)

39. D Almost all deaths from thoracic injury in
children occur after the victim reaches the resuscitation
center, and most children can be treated successfully
with prompt diagnosis and aggressive early manage-
ment. Penetrating injuries to the chest are unusual in
children and usually result from fractured ribs rather
than from external missiles. The mediastinum of the
child is more mobile and this contributes to a low inci-
dence of major vessel and airway injury. However, seri-
ous intrathoracic injury may be present in the absence
of obvious chest wall injury. (Rogers MC, et al.
Textbook of Pediatric Intensive Care, 3rd Edition;
pp. 1487,1488.)

40. A Cardiac arrest from blunt chest trauma is
nearly always associated with multiple system injuries,
and results from hypovolemia either from external or internal blood loss. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1487,1488.)

41. **D** Flail chest injuries are rarely seen in children because high-velocity direct-chest trauma is uncommon. Additionally, rib fractures are less common in children than adults because children have very pliable ribs that are resistant to fracture. Contusions and/or penetrating injury of the lung parenchyma are frequently involved. The initial therapy should include humidified oxygen and a limitation of crystalloid resuscitation, if the remainder of the injuries permit, so that there will be a decrease in extravasation of fluid into the injured pulmonary parenchyma and a limitation of the secondary acute pulmonary edema. Definitive treatment of the flail chest takes place in the PICU by controlled ventilation and PEEP. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1488,1489.)

42. **E** The least common occult and potentially serious injury to the chest of a child with multiple trauma is esophageal rupture. From most to least common, the injuries are pulmonary contusion, pulmonary laceration, pulmonary hematoma, tracheobronchial tear, myocardial contusion, diaphragmatic rupture, partial aortic or great vessel disruption, and esophageal perforation. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 1489.)

43. **C** In the setting of pulmonary contusion, overhydration should be avoided because fluid will sequester in the damaged lung tissue and complicate the clinical condition. Radiographical evidence of a pulmonary contusion includes early consolidation of the lung parenchyma, which may be focal in nature, with resolution over 2–6 days. Empyema, or abscess formation, may occur after pulmonary contusion secondary to the extravasation of fluid and blood into the alveolar and interstitial spaces. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 1489,1490.)

44. **E** Drowning is the third most common cause of death by unintentional injury among persons of all ages in the United States, and the second leading cause of injury deaths in children younger than 15 years old. Males account for 78% of all deaths from drowning. Approximately 50% of the drowning deaths occur in the summer. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 875–877.)

45. **E** The majority of drowning accidents occur in the southern and western United States; Saturday is the most common day of the week for drowning accidents; private pools are the most common sites for submersion accidents involving children; and drowning rates are highest among the African-American population. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 875–877.)

46. **T, F, T, T** Drowning is death from asphyxia caused by submersion in water. Death usually occurs at the time of submersion or within 24 hours. Most human drowning victims aspirate less than 3–4 mL/kg of fluid. Fresh water causes surfactant to denature and become nonfunctional. Seawater either dilutes surfactant concentrations or washes the surfactant out of the alveolus entirely. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 875–881.)

47. **E** The pathophysiology of submersion injury can include the processes of asphyxia, fluid overload, pulmonary injury, and hypothermia with the diving reflex. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 878–883.)

48. **D** Therapeutic hypothermia has not been shown to improve outcome. A body temperature of less than 32°C causes the cessation of shivering. Resuscitation of drowning victims should continue until the core temperature is at least 32°C. Pupillary dilatation occurs at a core temperature of less than 30°C. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 882–889.)

49. **T, F, T, T, T, F, F, T** Chest radiographs do not correlate with clinical outcome. Steroids have not been shown to be useful in improving the outcome for ischemic or anoxic insults. Intracranial pressure monitoring has not been shown to improve outcome in submersion injury. PEEP is often useful in treating the pulmonary dysfunction that is associated with a near-drowning episode, which is unresponsive to supplemental oxygen. The drowning victim will often swallow a large amount of water, which may induce emesis and subsequent aspiration. Consciousness is then lost. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 878–889.)
50. E The EEG may not be reliable in very young and particularly premature infants, because there are reports of return of neuronal function and EEG activity after the demonstration of electrocerebral silence. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 896–899.)

51. D No corroborative testing is required in the case described. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 895–900).

52. A Stereotyped movement of the extremities and extensor posturing can be seen in patients who are clearly brain dead; these have been termed the Lazarus sign. Spinal and deep tendon reflexes are found on physical examination in at least 50% of brain dead patients. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; p. 902.)

53. T, T, T, T All of these statements are true. (Rogers MC, et al. *Textbook of Pediatric Intensive Care*, 3rd Edition; pp. 895–902.)

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**CHAPTER 14: STATISTICS**

1. D Type I or $\alpha$-error is more likely to occur when you have too many variables rather than too few subgroups. (Fuhrman BP, et al. *Pediatric Critical Care*, 2nd Edition; pp. 172–174.)

2. D The power of a study is equal to $1 - \beta$-error, which was set in this study at 0.2, and therefore the power of this study would be 0.8 or 80%. The P-value of this study was set at 0.05, and there is only a 20% chance that the authors actually missed an improvement owing to the new drug. There is an 80% chance that an improvement from the new drug was not missed. (Fuhrman BP, et al. *Pediatric Critical Care*, 2nd Edition; pp. 172–174.)

3. A Groups are not a factor for nominal data. (Fuhrman BP, et al. *Pediatric Critical Care*, 2nd Edition; pp. 169,170.)

4. A In evaluating how strong the correlation is, it is more dependent on how tightly all the points are scattered around the slope line. Correlation does not make judgments as to how one variable affects or predicts another. A regression coefficient permits prediction. (Hermansen M. *Biostatistics: Some Basic Concepts*; p. 56.)

5. E Relative risk (RR) refers to the risk of having complications in the presence of a risk factor compared to the absence of the factor. The range of RR is from 0 to infinity. A RR of 1.0 indicates that there is no difference in risk. Calculation of RR is explained in the answer to Question 6. (Hermansen M. *Biostatistics: Some Basic Concepts*; pp. 164–166.)

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6. B

| Infection | Present | Absent | Total |
|-----------|---------|--------|-------|
| Nurse Present | A = 75 | B = 25 | 100   |
| Absent      | C = 125| D = 775| 900   |
| Total       | 200    | 800    | 1000  |

Relative risk $= \frac{\text{Risk of infection when risk factor present}}{\text{Risk of infection when risk factor absent}}$

$= \frac{A / A + B}{C / C + D}$

$= \frac{75/100}{125/900}$

$= 5.4$

(Hermansen M. *Biostatistics: Some Basic Concepts*; pp. 164–166.)

7. C Standard error of the mean (SEM) is a descriptive statistic, and SEM helps determine the range in which the population mean exists. SEM equals standard deviation divided by the square root of the number of variables, and therefore, SEM is always smaller than standard deviation.

$$SEM = \frac{SD}{\sqrt{n}}$$

(Hermansen M. *Biostatistics: Some Basic Concepts*; pp. 38–41.)

8. A, B, C, E, D Self-explanatory. (Hermansen M. *Biostatistics: Some Basic Concepts*; pp. 38–41.)
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9, 10. B, A

\[
CL = \frac{\text{Dose at Steady State (D}_{SS})}{\text{Concentration at Steady State (C}_{SS})}
= K_{el} \times Vd
\]

\[
K_{el} = \frac{0.693}{\text{Half-life}}
\]

\[
C_{SS} = \frac{20 \times 100}{0.693 \times 1.1}
= 2.6 \text{ mg/mL}
\]

It takes four times the half-life to reach a steady state concentration.

\[
= 4 \times 100
= 6.7 \text{ hours}
\]

(Kearns GL. Clin Pharmacol, 1988; 7:198.)

11. C Parametric methods of statistical analysis use distribution assumptions (i.e., normal distribution) of data, and the distribution is described by mean and standard deviation. Nonparametric methods are also called distribution-free. These methods are based on analysis of ranks rather than actual data, and therefore, they are sometimes called rank methods. Skewed data are commonly analyzed by nonparametric methods. Methods using ranks are especially suitable for data, which are scores rather than measurements. Examples include apgar scores and stages of disease. (Altman DG. Practical Statistics For Medical Research; pp. 171–173.)

**CHAPTER 15: ETHICS**

1. D All of the above are involved in the process of informed consent. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 10,11.)

2. E These children are referred to as emancipated minors. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 10–12.)

3. D It does not assume that the patient or the surrogate is competent. This must be assessed by the physician, as indicated in answer E. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 9–14.)

4. A It does include the ability to manipulate the information and deliberate about alternatives. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 9–14.)

5. D The best course of action is direct inquiry into their fears and guilt, which is likely to provide the best resolution for all parties. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 9–14.)

6. D In order to succeed in a claim for damage, it must meet all the above criteria. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 18–22.)

7. B As long as the reason and the date are stated, adding to medical records is not illegal. Jurisdictions vary on the essence of compliance. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 20–22.)

8. D Accidents and adverse effects are the most common causes of death in children 13–15 years of age. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; p. 4.)

9. D “Baby Doe” regulations (introduced in 1982 by the Federal government) prohibit withholding or withdrawing of beneficial medical treatment from any infant on the basis of handicap or prognosis for quality of life. The three exceptions are: (1) infant is permanently comatose; (2) treatment is inhumane; and (3) infant is immediately dying. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; p. 34.)

10, 11. C, E It is more helpful in dealing with these families to have a caring attitude rather than a defensive attitude. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 38–41.)

12. A The complete record may be needed if the case goes to trial. It is rarely helpful in the initial investigation because so much of the record pertains to investigation. (Fuhrman BP, et al. Pediatric Critical Care, 2nd Edition; pp. 18–23.)