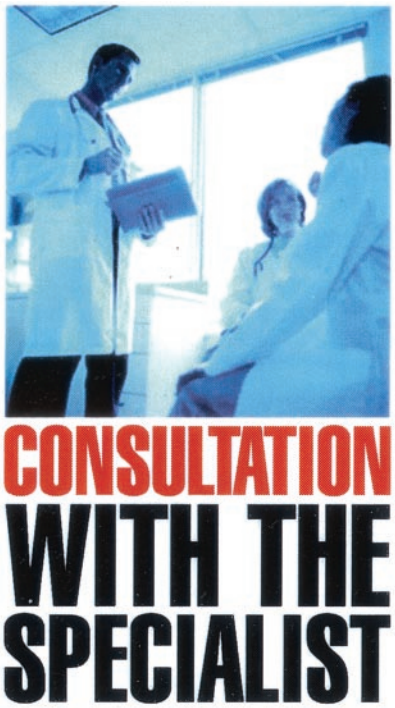

Respiratory Failure in Children

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Objectives

After completing this article, readers should be able to:

1. Explain when hypercarbia interferes with metabolism.
2. Discuss why oximetry is not a sensitive indicator of respiratory failure in patients who are receiving supplemental oxygen.
3. List the indicators of chronic respiratory failure.
4. Describe the usual findings on blood gas analysis in patients who have chronic respiratory failure.
5. Identify which patients should be monitored regularly for the development of respiratory failure.

Case

A 16-year-old boy who has Duchenne muscular dystrophy presents to the pediatric clinic with a 2-day history of nasal congestion, intermittent headache, cough, and low-grade fever. The patient's mother reports that her son's cough was worsening last night, his chest was very congested, and he was sleepy this morning and difficult to arouse. The patient was transported to the clinic by a local ambulance company, who administered 2 L of supplemental oxygen by nasal cannula for mild respiratory distress. On physical examination, the patient is sitting in his wheelchair and requires gentle stimulation to remain awake and engage in conversation. His responses to questions are appropriate but slow and not typical for him. The patient has mild tachycardia, nasal flaring, and paradoxic respirations. His cough is weak. On auscultation, there are bilateral rhonchorous breath sounds with diminished air entry in the lower lung fields. The oxyhemoglobin saturation by pulse oximetry is 91% on 2 L of oxygen per nasal cannula. An arterial blood gas shows a pH of 7.18, a $Paco_2$ of 70 mm Hg, a Pao_2 of 60 mm Hg, and an Hco_3 of 34 mEq/L. The patient is transferred to the pediatric intensive care unit, where he is treated with noninvasive positive-pressure ventilation by face mask and assisted mucus clearance techniques. After 2 days, he is weaned to nocturnal noninvasive ventilation. He is discharged from the hospital after 2 weeks.

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Introduction

Managing a child who is in respiratory failure is challenging for primary care pediatricians. Community physicians are likely to encounter such patients only a few times each year, making it difficult to maintain the necessary clinical skills through daily practice. Furthermore, the stakes are high: Failure to recognize and manage respiratory failure appropriately can result in patient death or long-term disability. The management of respiratory failure depends on whether respiratory insufficiency develops acutely or gradually. Acute respiratory failure (ARF) carries the imminent risk of cardiac arrest. Patients who have chronic respiratory failure (CRF) are less likely to deteriorate precipitously, but they can develop devastating complications such as pulmonary hypertension and cor pulmonale and are at risk for ARF with intercurrent illnesses.

The prompt recognition of respiratory failure requires that physicians maintain an index of suspicion when presented with high-risk clinical situations (Table 1) and remain familiar

Table 1. Common Causes of Respiratory Failure in Children

Respiratory Pump Dysfunction (First-degree Pathophysiology: Hypoventilation)
May be associated with signs of decreased respiratory drive
• Decreased Central Nervous System (CNS) Input
— Head injury
— Ingestion of CNS depressant
— Adverse effect of procedural sedation
— Intracranial bleeding
— Apnea of prematurity
• Peripheral Nerve/Neuromuscular Junction
— Spinal cord injury
— Organophosphate/carbamate poisoning
— Guillian-Barré syndrome
— Myasthenia gravis
— Infant botulism
• Muscle Weakness
— Respiratory muscle fatigue due to increased work of breathing
— Myopathies/Muscular dystrophies
Airway/Lung Dysfunction
Usually associated with signs of increased respiratory drive
• Central Airway Obstruction (First-degree Pathophysiology: Hypoventilation)
— Croup
— Foreign body
— Anaphylaxis
— Bacterial tracheitis
— Epiglottitis
— Retropharyngeal abscess
— Bulbar muscle weakness/dysfunction
• Peripheral Airways/Parenchymal Lung Disease (First-degree Pathophysiology: Disordered gas exchange)
— Status asthmaticus
— Bronchiolitis
— Pneumonia
— Acute respiratory distress syndrome
— Pulmonary edema
— Pulmonary contusion
— Cystic fibrosis
— Chronic lung disease (eg, bronchopulmonary dysplasia)

with the signs and symptoms of acute and chronic respiratory insufficiency (Table 2). Once respiratory failure is recognized, treatment involves initially addressing the adequacy of gas exchange, followed by pursuing the underlying causes and complications of respiratory failure. Prevention is an important aspect of care for children who have chronic respiratory conditions; an ounce of prevention invested in minimizing respiratory infections, atelectasis, obesity, and other conditions that contribute to ARF and CRF in susceptible children is worth a pound of cure.

Pathophysiology of Respiratory Insufficiency

Respiratory failure usually is defined as an arterial partial pressure of carbon dioxide ($Paco_2$) of greater than 50 mm Hg, an arterial partial pressure of oxygen (Pao_2) of less than 60 mm Hg, or both. Hypercarbia and hypoxia have different consequences. Increased $Paco_2$ does not interfere particularly with normal metabolism until it reaches extreme levels (>90 mm Hg). Above that level, hypercapnia can lead to catastrophic central nervous system depression and respiratory arrest. For patients whose $Paco_2$ levels are lower, the more dangerous consequence of respiratory failure is acute or chronic hypoxemia. Acute hypoxemia, particularly in the presence of a low cardiac output, is associated with tissue hypoxia and a risk of cardiac arrest. Chronic hypoxemia can lead to polycythemia, pulmonary hypertension, and cor pulmonale.

The physiologic basis of respiratory failure determines the clinical picture. Patients in respiratory failure who have normal respiratory drive are breathless and anxious. In contrast, patients who have decreased central drive are comfortable or even somnolent. The causes of inadequate gas exchange can be categorized as: 1) conditions that affect the respiratory pump, and 2) conditions that interfere with the normal function of the lung and airways (Table 1). The primary physiologic cause of respiratory failure in patients who have problems of the ventilatory pump or critical central or upper airway obstruction is hypoventilation. For patients who have lung disease, the physiologic basis of respiratory failure can include hypoventilation, but more commonly it involves a mismatching of blood and gas within the lung or shunting of blood around ventilated alveoli. Hypoventilation, ventilation/perfusion mismatch, and shunt have different characteristics and treatments.

Hypoventilation

Breathing an inadequate volume of air to maintain a $Paco_2$ lower than

Table 2. Signs and Symptoms Pertinent to Respiratory Failure

Increased Respiratory Drive

- Increased rate/depth of breathing
- Anxiety
- Breathlessness/dyspnea
- Retractions
- Accessory muscle use:
  - Sternocleidomastoid
  - Intercostal
  - Alar nasae (nasal flaring)

Decreased Respiratory Drive

- Decreased rate/depth of breathing
- Lethargy
- Confusion
- Snoring

Respiratory Muscle Fatigue

- Paradoxic "see-saw" respirations
- Grunting
- Irregular/uncoordinated breathing

Hypoxemia

- Cyanosis
- Digital clubbing
- Increased pulmonary closure sound
- Failure to thrive

Evidence of Lung Disease

- Wheezing/rhonchi/rales
- Retractions
  - Suprasternal
  - Intercostal
  - Subcostal

Evidence of Respiratory Muscle Weakness

- Chest wall paradox
- Shallow breathing
- Ineffective cough

Hypercapnia

- Throbbing morning headaches
- Disrupted sleep
- Decreased level of consciousness

Evidence of Loss of Airway Protective Reflexes

- Absent gag and cough reflexes
- Gurgling respirations

Evidence of Critical Upper Airway Obstruction

- Stridor
- Drooling
- Muffled/absent breath sounds

50 mm Hg can result from a decreased drive to breathe (narcotic overdose), an inability to breathe (muscle weakness), or lung or airway disease that increases the work of breathing beyond the ability of the patient to sustain normal gas exchange (exhaustion). Ventilation is determined by the respiratory rate and the size of each breath; thus, a low respiratory rate and shallow breathing are both signs of hypoventilation. The $Paco_2$ increases in proportion to a decrease in ventilation. If a child's $Paco_2$ normally is 40 mm Hg, reducing ventilation by 50% will increase the $Paco_2$ to about 80 mm Hg. With hypoventilation, the Pao_2 falls approximately the same amount as the $Paco_2$ increases. The relationship between oxygenation and hypoventilation is complicated by the shape of the hemoglobin dissociation curve and the effect of oxygen therapy. Because of the dissociation curve, a patient who exhibits alarming CO_2 retention might have a near-normal oxygen saturation. For example, the child whose $Paco_2$ has increased from 40 to 80 mm Hg, a dangerous level of hypoventilation, might have a Pao_2 that has decreased from 100 to 60 mm Hg and, therefore, maintain an oxygen saturation of 90%. For this reason, oximetry is not a sensitive indicator of the adequacy of ventilation. This is particularly true when a patient is receiving oxygen; a patient who has a $Paco_2$ of 80 mm Hg could have a normal Pao_2 of 100 mm Hg if given just 26% oxygen to breathe. Even minimal oxygen therapy can maintain normal oxygenation in patients who are seriously hypoventilating, as illustrated in the case study of the patient who has muscular dystrophy and maintains an oxygen saturation of 91% despite hypoventilating to a $Paco_2$ of 70 mm Hg.

Lung/Airway Disease

Lung dysfunction leads to respiratory failure when a patient who has lung disease is unable to support normal gas exchange by increasing ventilation. For a variety of reasons, children in the first 2 years after birth are less able to compensate for lung/airway dysfunction and, therefore, are more prone to develop respiratory failure compared with adults and older children. Diseases of the lung or airways affect gas exchange most often by disrupting the normal matching of ventilation and perfusion or by causing a shunt. A child who has either of these problems usually can maintain a normal $Paco_2$ as lung disease worsens simply by breathing more. Carbon dioxide retention develops in lung disease only when the patient no longer can sustain the necessary increase in breathing, usually because of muscle fatigue. Although breathing more usually is adequate to maintain a normal $Paco_2$, it has little effect on oxygenation. Therefore, hypoxemia is the hallmark of lung disease. Patients who have hypoxemia due to ventilation/perfusion mismatch can be discriminated from those who have a shunt by administering oxygen; oxygen therapy typically corrects hypoxemia in patients who have ventilation/perfusion mismatch and has little effect in patients who have shunts.

Arterial Blood Gases and Oximetry in Respiratory Failure

Arterial blood gas tensions and acid/base status are the definitive indicators of gas exchange. In most situations, capillary gases closely approximate arterial values. If capillary or arterial sampling is difficult, the pH and Pco_2 on a venous sample can be useful; for patients who have good perfusion, venous Pco_2 typically is 5 to 10 mm Hg higher and the pH is minimally lower than arterial values. The $Paco_2$ is the best measure of the adequacy of breathing. This value also can be estimated by measuring end tidal Pco_2 if a patient has normal lung function. In ARF, the serum bicarbonate concentration rises slightly and the arterial pH falls. If the $Paco_2$ remains elevated or rises slowly, the kidneys conserve bicarbonate, the serum bicarbonate concentration increases, and the arterial pH remains near or returns toward normal. Renal "compensation" begins within 1 day of the onset of respiratory failure. Venous bicarbonate values do not always reflect arterial bicarbonate precisely. Nevertheless, for patients who do not have a cause for a metabolic acidosis, a stable and near-normal serum bicarbonate value is reassuring. Definitive assessment of respiratory status, however, depends on measuring arterial blood gases. Typical values in respiratory failure are presented in Table 3. The boy in the case study illustrates the condition of ARF superimposed on CRF. His progressive muscle weakness caused CRF, with resultant CO_2 retention and renal compensation documented by the elevated Hco_3 measurement on the arterial blood gases. However, his $Paco_2$ is higher than his normal baseline, as indicated by the low pH, and is consistent with an acute worsening of his respiratory function due to the intercurrent respiratory infection.

Table 3. Arterial Blood Gas Determinations

	pH	Pco_2 (mm Hg)	Po_2 (mm Hg)	Hco_3 (mEq/L)
Normal	7.40	40	100	24
ARF	7.24	60	50	24
CRF	7.35	60	50	34
A/CRF	7.28	70	50	34

ARF=acute respiratory failure; CRF=chronic respiratory failure; A/CRF=acute on chronic respiratory failure

Oximetry provides an invaluable and usually accurate measurement of oxygenation. It is, however, important to recognize its technical limitations (Table 4) and that patients in respiratory failure can have normal or near-normal oxygen saturations, particularly when receiving oxygen. When a child is breathing room air, an oxygen saturation of more than 95% is good evidence of adequate breathing.

ARF

ARF is the most common cause of cardiac arrest in children. The importance of early recognition and appropriate management of ARF is underscored by the dismal outcome of children who progress to cardiac arrest.

Recognition

When presented with a child who has a decreased level of consciousness, slow/shallow breathing, or increased respiratory drive, the possibility of ARF should be considered and the adequacy of breathing systematically evaluated by history, physical examination, and measurements of gas exchange. A brief history should identify any underlying diseases and the extent and rate of change in mental status and breathing. An efficient approach to the examination is to begin

Table 4. Technical Limitations of Oximetry

Condition	Limitation
Dark skin pigment Decreased perfusion Anemia Bright external light Motion	Causes inadequate signal
Venous pulsations – Severe right heart failure – Tricuspid regurgitation – Tourniquet or blood pressure cuff above site	Results in low reading
Abnormal hemoglobin concentration – Methemoglobin	Unreliable reading (tends to read 80% to 85% saturation regardless of actual saturation)
– SS hemoglobin	Saturation accurate, but hemoglobin dissociation curve shifted to right
– Carboxyhemoglobin	Spuriously high saturation readings

Table 5. Rapid Cardiopulmonary Assessment

Evaluation of General Appearance

- General color
- Mental status, responsiveness
- Activity, movement, muscle tone
- Age-appropriate response to environmental stimuli

Airway, Breathing, Circulation

- Airway
  - Clear
  - Maintainable with positioning and suctioning
  - Not maintainable without invasive intervention
- Breathing
  - Respiratory rate
  - Effort and mechanics
  - Air entry/depth of respirations (tidal volume)
  - Skin color
- Circulation
  - Heart rate (tachycardia, bradycardia)
  - Pulses (volume, discrepancy between central and distal)
  - Capillary refill
  - Extremity temperature
  - Mental status

with the "30-second cardiopulmonary assessment" outlined in Table 5. If any of the components of this assessment supports the possibility of ARF, oxygen therapy should be started and bag-mask ventilation considered. Oximetry should be monitored and blood gas measurement strongly considered. As soon as ARF is recognized, steps to transfer the patient to an emergency facility or intensive care unit should be initiated.

Treatment

The first priorities in treating ARF are to assure adequate gas exchange and circulation (the ABCs of cardiopulmonary resuscitation). Oxygen should be delivered to maintain the arterial oxygen saturation above 95%. Although oxygen administration carries the risk of decreasing ventilatory drive in some patients who have chronic hypoventilation, this is not a contraindication to oxygen therapy while a patient is being observed closely and ventilation can be supported if necessary. If ventilation is or appears to be inadequate, breathing should be initiated with a bag-mask system with added oxygen. Tracheal intubation may be indicated in some patients (Table 6). Ventilation can be maintained with a bag-mask system in most patients, and intubation should be delayed until personnel skilled in this procedure are available. The patient should be transferred rapidly to a referral facility by a transport team that is experienced in the management of the pediatric airway.

CRF

In pediatrics, CRF is seen most commonly in patients who have respiratory muscle weakness (muscular dystrophy, anterior horn cell disease) or severe chronic lung diseases (bronchopulmonary dysplasia, end-stage cystic fibrosis) (Table 1). Because CRF usually has an insidious onset, a major challenge for the physician is recognizing that breathing is becoming inadequate. This is accomplished best by careful monitoring of children at risk for CRF at regular intervals. Occasionally, the cause of CRF can be identified and treated, thus re-establishing respiratory sufficiency without an ongoing need for support. This is not the case for most children who have CRF, making the second major challenge of managing CRF to design a regimen of ventilatory support and monitoring that is safe and provides the maximal quality of life for the child and family.

Diagnosis

Most children who present in CRF do not have dyspnea. By definition, CRF develops gradually, allowing the kidneys sufficient time to increase the serum bicarbonate concentration and, thereby, maintain the arterial

Table 6. Indications for Tracheal Intubation

- Cardiopulmonary failure/ cardiopulmonary arrest
- Severe respiratory distress/ respiratory muscle fatigue
- Loss of cough or gag
- Need for prolonged support due to apnea or hypoventilation
- Interhospital transport of a patient who has the potential for respiratory failure

pH at normal or near-normal levels. Because respiratory drive is determined by pH rather than by $Paco_2$, dyspnea does not increase. For patients who have end-stage cystic fibrosis, muscular dystrophy, severe bronchopulmonary dysplasia, or similar conditions (Table 1), assessment of the adequacy of ventilation should be a part of each visit (Table 2). Occasionally, children who have diseases that have an acute or subacute onset and compromise ventilation (myasthenia gravis, diaphragmatic weakness) present initially with signs and symptoms of CRF, but this is unusual. Of the signs and symptoms of CRF that pediatricians should recognize, morning headaches, altered mental status, increased respiratory symptoms, cardiomegaly, and decreased baseline oxygenation are particularly useful. CRF often presents first during sleep, when respiratory drive is normally less vigorous; parents and physicians should be aware that disordered sleep, daytime hypersomnolence, and morning headaches can be early signs of CRF.

Borderline respiratory compensation may be identified when a child who has an underlying disease develops an intercurrent illness. An asthma exacerbation, pneumonia, atelectasis, and influenza all can compromise breathing in the short term and precipitate respiratory failure in previously compensated children. Fever can be an important cause of decompensation and should be treated aggressively because the metabolic rate, CO_2 production, and O_2 consumption increase 10% to 15% for every degree centigrade elevation in body temperature.

Measurements

The diagnosis of CRF is confirmed by blood gas analysis (Table 3). Regular objective measurements are useful to monitor the respiratory status of patients who have underlying conditions that place them at risk for CRF. Many physicians monitor serum bicarbonate levels at regular intervals in patients who have chronic diseases that compromise breathing because CRF usually is associated with a gradual increase over baseline. Oximetry readings that are below baseline may indicate lung disease, but they also should raise the possibility of CRF. Polysomnography or, if formal studies are not easily available, continuous nighttime oximetry can detect early decompensation during sleep in patients who have disordered sleep or muscular dystrophy when gas exchange is normal during wakefulness. Polycythemia and right ventricular hypertrophy on electrocardiography or echocardiography are evidence of prolonged hypoxemia. A number of bedside or office measurements of pulmonary function, such as maximal pressures and vital capacity, can help identify a level of muscle weakness that often is associated with CRF (Table 7).

Table 7. Pulmonary Function Testing in Chronic Respiratory Failure

- Pulse oximetry
- Blood gas
- Forced vital capacity
- Negative inspiratory pressure
- Maximal expiratory pressure
- Peak cough flow
- Expiratory flow rates

Treatment

As in children who have ARF, the first priority in treating CRF is to determine whether ventilatory support is indicated. Unless a child who has a chronic underlying disease has had a sudden deterioration (as indicated by an uncompensated respiratory acidosis), mechanical ventilation usually is not necessary immediately. It often is possible to restore adequate ventilation in such patients by treating the underlying disease or by improving the patient's pulmonary function. For example, in patients who have muscular dystrophy, decreased cough, inability to breathe deeply, and immobility all may contribute to poor airway clearance and atelectasis that can result in CRF. Aggressive chest physical therapy, frequent repositioning, and use of airway clearance assist devices such as the in-exsufflator can effect a dramatic improvement in overall respiratory status. In many patients at risk of respiratory failure, a component of airway inflammation/asthma that would be inconsequential for most children can make the difference between CRF and clinical stability. In these cases, a brief corticosteroid burst and chronic anti-inflammatory therapy often are helpful. Aggressively treating fever is always important.

OXYGEN THERAPY. Because CRF frequently is associated with mild hypoxemia, even in the absence of lung disease, and because many children who have conditions that lead to CRF also have lung problems such as

airway disease and atelectasis, many children who have compensated or uncompensated CRF have intermittent or continuous hypoxemia. In most patients, modest concentrations of added oxygen can maintain saturations greater than 92%, thereby preventing or reversing the pathologic consequences of chronic hypoxemia. It is important to remember, however, that oxygen therapy can be dangerous for a few patients. In some patients who have CRF, the increased drive to breathe in response to acidosis (increased $Paco_2$) is abnormally blunted, and the usually weaker response to hypoxia is the primary stimulus to respiration. For these patients, correcting hypoxia by administering oxygen can blunt the respiratory drive, leading to a disastrous decrease in ventilation and severe hypercarbia, at which point the level of consciousness falls and breathing ceases. This complication is unlikely to occur, however, in patients who do not have an elevated $Paco_2$ and in whom the oxygen saturation is kept between 90% and 95%.

Respiratory depression should be considered in any patient whose level of consciousness decreases after oxygen therapy is initiated. Unfortunately, many patients have been denied the benefit of oxygen therapy because of their physician's concern about the possibility of respiratory depression.

VENTILATORY SUPPORT. There has been a move toward noninvasive ventilation in children who have CRF. At one time, the major option for providing respiratory support for children who had CRF was tracheostomy and ventilation with one of several mechanical ventilators approved for home use. Tracheostomy still is necessary in many patients who require continuous support or who have poor airway protective reflexes. In an increasing number of patients, however, biphasic positive-pressure ventilation delivered through a face mask or through the nose is adequate. This is particularly attractive for patients who require support only at night, but it can be used for longer periods as long as skin integrity is maintained and the child is able to be off of support for periods of hours each day. Other modalities that can be useful to augment ventilation are a "rain jacket" negative-pressure ventilator that fits around the thorax and a rocking bed. Temporary noninvasive ventilation is useful for supporting patients who normally are compensated but are at risk of decompensating during an intercurrent illness.

Prognosis

The prognosis for children who have CRF depends on the underlying illness. Once CRF is recognized, most children improve dramatically with treatment of the underlying disease and, when necessary, supplemental oxygen or ventilatory support. At one time, many children who had advanced cystic fibrosis, muscular dystrophy, or bronchopulmonary dysplasia died shortly after the onset of chronic hypoxemia because of progressive cor pulmonale. With appropriate monitoring and treatment of CRF, most children who have such conditions now live longer and have a greatly improved quality of life.

Suggested Reading

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PIR Quiz

Quiz also available online at www.pedsinreview.org.

5. A 6-year-old girl is hospitalized with Guillain-Barré syndrome. Physical examination reveals an axillary temperature of 97.9°F (36.6°C), a respiratory rate of 24 breaths/min, a heart rate of 110 beats/min, and a blood pressure of 116/70 mm Hg. She is alert and interactive. Pulse oximetry shows an oxygen saturation of 100%. She is receiving 25% oxygen through a Venturi mask. She has considerable weakness of the upper and lower extremities and a weak cough. Capillary blood gas measurements show a pH of 7.38, Pco_2 of 43 mm Hg, and Po_2 of 98 mm Hg. Chest radiography is normal. Continuous electrocardiographic monitoring shows no abnormality. Which of the following is likely to be the *earliest* indicator of respiratory failure?

   A. Decrease in oxygen saturation below 95%.
   B. Hypertension.
   C. Increasing chest wall retractions.
   D. Increasing end-tidal CO_2.
   E. Peaked p waves on electrocardiography.

6. A 2-year-old boy who is known to be positive for human immunodeficiency virus is admitted with rapid respirations and grunting. Physical examination shows a respiratory rate of 70 breaths/min with intercostal retractions, a heart rate of 160 beats/min, and an axillary temperature of 99.3°F (37.4°C). Auscultation reveals bronchial breathing and occasional scattered rales. Chest radiography shows diffuse infiltrates throughout lung the fields. Of the following, the *most* likely abnormality that would be detected on arterial blood gas examination is:

   A. Decreased pH.
   B. Decreased Po_2.
   C. Increased Pco_2.
   D. Increased base excess.
   E. Increased bicarbonate concentration.

7. In which of the following conditions does pulse oximetry provide the *most* accurate reflection of arterial hemoglobin-oxygen saturation (Sao_2)?

   A. Bronchiolitis.
   B. Carbon monoxide poisoning.
   C. Congestive heart failure.
   D. Hypovolemia.
   E. Methemoglobinemia.

8. A 16-year-old boy who has cystic fibrosis is admitted for increased difficulty breathing and a cough. Physical examination reveals a respiratory rate of 26 breaths/min and a heart rate of 100 beats/min. The chest is barrel-shaped, and there are intercostal and suprasternal retractions with prolonged expiration. Auscultation reveals expiratory wheezing and rales throughout the lung fields. Chest radiography shows marked hyperinflation and diffuse bilateral infiltrates. He is started on 70% oxygen via a nonrebreathing face mask with a reservoir. His pulse oximetry shows an oxygen saturation of 95%. Of the following, the *most* likely findings on arterial blood gas determination would be:

	pH	Pco_2 (mm Hg)	Po_2 (mm Hg)	Hco_3 (mEq/L)
A.	7.25	70	75	29
B.	7.40	40	50	24
C.	7.25	30	75	16
D.	7.55	30	50	26
E.	7.55	50	75	44

Consultation with the Specialist: Respiratory Failure in Children

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