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Respiratory diseases are a common problem encountered by physicians who treat pediatric patients.[1] *Respiratory disorders account for one out of five pediatric hospital admissions and about 10% of pediatric emergency department (ED) visits.*[2] *Respiratory disorders also are a major cause of pediatric mortality.*[1,3] *About one-half of all deaths in patients younger than 1 year of age and more than one-third of the deaths in children younger than 15 years of age are due to pulmonary diseases.*[2]

With winter respiratory viruses flourishing, this is an opportune time to review the recognition and management of pediatric patients with respiratory failure.

— The Editor

The Pediatric Patient with Acute Respiratory Failure: Clinical Diagnosis and Pathophysiology

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Introduction

Respiratory failure is the inability of the respiratory system to provide adequate oxygen to meet the body's metabolic requirements and/or to excrete the carbon dioxide produced by the body.

There are many disorders, both within the respiratory tract and outside the pulmonary system, that can cause respiratory failure.[4] *(See Table 1.)* Exchange of gases occurs in the pulmonary capillary bed. Failure of the respiratory system to deliver adequate oxygen to the tissues causes hypoxia, and failure to excrete carbon dioxide results in hypercapnia.[5]

Respiratory System: Anatomy

The respiratory system can be conceptualized as a pump consisting of the nervous system (central nervous system [CNS], spinal nerves, peripheral nerves, and the neuromuscular junction); the effector components (the respiratory muscles and chest wall); the conducting airways; and the air exchange system in the alveoli. The pump drives the system, and the respiratory muscles and chest wall do the work of breathing. The conducting airways are the conduit for movement of air from the atmosphere to the alveoli.[6] The alveoli are where actual diffusion of gases occurs. The nervous system regulates respiration. Failure of the respiratory pump leads to hypercapnia, while alveolar disorders primarily cause hypoxemia.[6]

Respiratory Physiology: Process of Oxygenation

Multiple steps are needed to provide sufficient oxygen for the cells to maintain aerobic metabolism and to remove carbon dioxide, which is important in maintaining the body's pH via the bicarbonate buffer system.

The first step in the process is ventilation, which is the movement of gases between the environment and the lungs,[7] followed by intrapulmonary gas exchange.[8] Diffusion of oxygen and carbon dioxide across the pulmonary capillary membrane occurs, allowing mixed venous blood to release carbon dioxide to the

lungs and then pick up oxygen. This is followed by gas transport, in which adequate amounts of oxygenated blood are transported to the tissues and cells for aerobic metabolism, and carbon dioxide is transported to the lungs for excretion.[9] Finally, tissue/cellular gas exchange occurs in which oxygen is taken up and used by the tissues, and carbon dioxide is released. These processes are controlled by CNS regulation of respiration in the brainstem.

Thus, respiratory failure may involve a failure of oxygenation, a failure of ventilation, or both.[10] Furthermore, failure of oxygenation can result from non-pulmonary factors, including: cardiogenic shock, severe anemia, abnormal hemoglobins (such as methemoglobinemia), an abnormal oxygen carrying capacity (as with carbon monoxide poisoning), or a failure of cellular oxygen uptake (e.g., cyanide poisoning or septic shock).[10]

Classification

Respiratory failure is classified according to the pCO_2 level. Type I failure, also known as normocapnic or non-ventilatory failure, is indicated by hypoxemia (low pO_2) with a normal or low pCO_2. An elevated pCO_2 is the hallmark of Type II failure, also known as ventilatory or hypercapnic failure. A variable degree of hypoxemia also is present with this type.[6]

Type I (normocapnic, non-ventilatory) respiratory failure is commonly due to ventilation/perfusion (V/Q) abnormalities. Other causes of Type I respiratory failure include: impaired diffusion across the alveolar-capillary membrane (as occurs with pulmonary fibrosis) and shunting.[11] *(See Table 2.)*

Type II (hypercapnic) respiratory failure generally is the result of alveolar hypoventilation, increased dead space ventilation, or increased CO_2 production. Factors that impair the central ventilatory drive in the brainstem, restrict ventilation, or increase CO_2 production can cause hypercapnic (Type II) respiratory failure.[11] *(See Table 3.)*

Case No. 1

A 16-year-old male arrives in the ED. No other history is available since the friends who brought him to the ED left.

The vital signs are:

- Temperature (T) = 96°F;
- Pulse (P) = 90 beats/min;
- Respiratory rate (R) = 6 breaths/min;
- Blood pressure (BP) =120/80 mmHg; and
- Pulse oxygen saturation is 76% on room air.

Arterial blood gas (ABG) is: pH = 7.13; pO_2 = 52; pCO_2 = 81; HCO_3 = 26; and oxygen saturation = 75% on room air.

He is unresponsive to painful stimuli, and his pupils are pinpoint. His respirations are shallow. The lungs are clear, and the heart rate is regular. There are needle tract marks on his arms but no signs of trauma. He has a depressed level of consciousness but no focal neurologic findings.

This patient has hypercapnia and hypoxia due to alveolar hypoventilation, resulting in Type II hypercapnic respiratory failure secondary to a control abnormality with a decreased respiratory drive. Of the physiologic events in respiration, diffusion, transport, and the tissue/cellular uptake of oxygen are normal, but ventilation is impaired. His hypoxia and hypercapnia are due to a non-pulmonary mechanism with a narcotic drug (e.g., heroin) overdose depressing his respiratory drive.

He has some signs of respiratory distress, including: abnormal respirations with a decreased tidal volume evidenced by shallow respirations (decreased depth), decreased rate (bradypnea, R = 6 breaths/min), and pattern (apneic at times). He does not have signs of air hunger and increased respiratory effort. He meets the criteria for acute respiratory failure (ARF). *(See Table 4.)* He is given supplemental oxygen, and his ventilations are assisted while other therapy, including naloxone, is given. After observation in the ED, the overdose wears off, and he becomes progressively more alert.

Causes of Respiratory Failure

The etiology of respiratory failure is more diverse than just lung or airway disorders. Respiratory failure can occur from an abnormality in any component of the respiratory system from the CNS to the pulmonary capillary bed where gas exchange occurs,[14] and to the tissues and cells where cellular uptake and utilization of oxygen occur. Nervous system disorders (from the respiratory control center in the medulla and pons via the spinal

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Table 1. Causes of Respiratory Failure by Clinical Pathway or Organ System

NEUROLOGIC DISORDERS
- Central nervous system (control abnormalities with a decreased respiratory drive)
- Spinal nerve pathways
- Peripheral nerves
- Neuromuscular junction

RESPIRATORY MUSCLE DISORDERS
- Primary muscle disorders (decreased ability of muscles)
- Respiratory muscle fatigue (resulting from increased work of breathing)

PULMONARY DISORDERS
- Airway disorders (interference with ventilation), airway obstruction
- Alveolar (parenchymal) diseases

CHEST WALL/PLEURA ABNORMALITIES
- Chest wall disorders (decreased chest wall compliance): flail chest
- Ruptured pleural space (limited movement of lungs): pneumothorax, pleural effusion

CARDIOVASCULAR DISORDERS
- Shock (interferes with oxygen/carbon dioxide transports by inadequate blood flow)

HEMATOLOGIC DISORDERS

(Interferes with oxygen transport by red blood cells)
- Severe anemia
- Abnormal hemoglobins

METABOLIC DISORDERS
- Severe metabolic acidosis
- Increased metabolic rate: burns, fever, infection, sepsis
- Abnormal respiratory quotient
- Abnormal cellular oxygen uptake: cyanide poisoning

nerve pathways to the peripheral nerves and the neuromuscular junction), chest wall/pleura disorders, airway disease (e.g., obstruction), pulmonary diseases, and even cellular uptake/utilization disorders all can lead to respiratory failure.

CNS disorders that lead to respiratory failure are control abnormalities that cause Type II (hypercapnic) respiratory failure and usually present without signs and symptoms of respiratory distress (such as dyspnea, retractions, or tachypnea).[15] Common causes of respiratory failure from CNS disorders include: drug overdoses; anesthesia; sedation; seizures; and CNS infections, injuries, and malformations (i.e., meningitis, encephalitis, brain abscesses, congenital malformations, encephalopathy, ischemia, infarcts, tumors, and trauma). These disorders all exert their effect by depressing the respiratory center in the brainstem.

Disorders of the upper motor neurons or spinal nerves also can cause Type II hypercapnic respiratory failure.[16] This may occur with several disorders, including: cervical spinal cord trauma, demyelinating diseases, myelitis (poliomyelitis, transverse myelitis), and Werdnig-Hoffman syndrome. Diseases of the peripheral nerves (i.e., Guillain-Barré syndrome, post-thoracotomy phrenic nerve damage, or a peripheral neuropathy) also can lead to hypercapnic respiratory failure.

Diseases affecting the neuromuscular junction may cause hypercapnic respiratory failure. These diseases include: myasthenia gravis, botulism, tetanus, organophosphate poisoning, and neuromuscular blocking drugs/anesthetics (i.e., pancuronium and succinylcholine). Fatigue of the respiratory muscles also can lead to ARF.[17,18]

Chest wall/pleura disorders lead to respiratory failure by decreasing chest wall compliance, as with flail chest, severe kyphoscoliosis, congenital or genetic deformities of the chest (e.g., severe dwarfism), or by disruption of the pleural space (e.g., pneumothorax or pleural effusion).[19] These disorders cause respiratory failure by mechanical abnormalities and decreased alveolar ventilation, which result in hypercapnia.

Airway obstruction causes increased airway resistance.[3] Airway obstruction can be in the upper airway (e.g., above the vocal cords) or in the lower airway from the larynx distally. The upper airway includes the nose, paranasal sinuses, and the pharynx. The lower airway includes the larynx, bronchi, bronchioles, and the alveoli. Obstruction in children is commonly due to foreign bodies, or infection, and infrequently due to congenital abnormalities such as a laryngeal web or tracheomalacia. Causes of upper airway obstruction include:
- Foreign bodies;
- Infections (epiglottitis, retropharyngeal abscess, or croup);
- Edema (as with anaphylaxis or laryngoedema);
- Congenital defects (web, stenosis, tracheomalacia, etc.);
- Tumors;
- Adenotonsilar hypertrophy; and
- Subglottic stenosis.

Lower airway obstructions include similar etiologies as with upper airway obstruction (e.g. foreign body, edema, congenital defects, and infections), but also reactive airway disease (asthma and bronchiolitis) secondary to bronchospasm.

In children, respiratory failure most often is due to diseases of the lungs.[15] Pulmonary diseases include: pneumonia, near drowning, adult respiratory distress syndrome (ARDS), pneumonitis, vasculitis, pulmonary edema, cystic fibrosis, and tuberculosis. Respiratory failure also may be caused by control abnormalities or by abnormalities in the mechanical function of the lungs. Control abnormalities are the result of decreased respiratory drive and, thus, there are few or no signs of respiratory distress even when there is significant hypercapnia and/or hypoxemia.

Respiratory failure caused by abnormalities in the mechanical function of the lungs and/or chest wall generally raise the ventilatory requirements and increase the work of breathing so the patient has to expend more physical effort to breathe. The patient will have air hunger; complain of dyspnea (secondary to chemoreceptor stimulation); and have an increased respiratory drive with physical signs and symptoms of respiratory distress such as tachypnea, retractions, etc. In children, respiratory failure more commonly is caused by mechanical abnormalities than by control abnormalities.[11]

Case No. 2

An 8-year-old male with muscular dystrophy is seen in his pediatrician's office because of increasing weakness. He is referred by his primary care physician to the ED for evaluation. The parents tell you that everyone in the family, including his sisters, had colds recently.

You observe a boy, who is thin and small for his age, sitting in a wheelchair. The parents tell you he usually walks with assistance but has been too weak to do so for the last day. The child nods appropriately but can speak only one or two syllables at a time, although he usually can talk in full sentences. His speech is slurred.

His vital signs are:

- T = 100.2°F;
- P = 120 beats/min;
- R = 12 breaths/min; and
- BP = 100/70 mmHg; and
- Weight = 20 kg.

A head, ears, eyes, nose, and throat (HEENT) examination reveals rhinorrhea and excessive secretions in the oropharynx. There are scattered rhonchi in the lungs bilaterally. The abdomen is benign. There is no peripheral edema or cyanosis. The neurologic exam is consistent with his diagnosis of muscular dystrophy with muscle weakness.

The respiratory therapist tells you that his maximum inspiratory pressure is 10 cm H_2O, and his vital capacity is 160 cc. Based on these pulmonary function parameters (see Table 4), you diagnose respiratory insufficiency with acute exacerbation of muscle weakness from an upper respiratory infection (URI) in a patient with chronically impaired muscle function secondary to his muscular dystrophy. Other tests confirm your diagnosis. A chest roentgenogram shows areas of plate-like atelectasis.

The ABG is: pH = 7.17; pO_2 = 46; pCO_2 = 78; HCO_3 = 32; and O_2 saturation = 71% on room air. You place him on BiPAP and admit him to the pediatric intensive care unit (PICU) for observation.

This patient has Type II hypercapnic respiratory failure secondary to failure of the respiratory muscles from a primary muscle disorder. He may be able to maintain himself in mild respiratory acidosis with a partially compensated metabolic alkalosis (as noted by the mildly increased bicarbonate) until an acute exacerbation (caused by the URI) weakens his already limited muscle strength and sends him into ARF with a rising pCO_2 and respiratory acidosis.

BiPAP is a reasonable therapy for the patient since he is awake and cooperative. BiPAP has been used in children as young as 4 years of age, although most pediatric patients on BiPAP are older than 5 years of age.

Signs and Symptoms of Respiratory Distress

History. The parents or caregivers of an infant or child who is having respiratory difficulty may report a decreased level of activity (i.e., not playing, not feeding, "not acting right") and a change in mental status (i.e., lethargy, listless, somnolent, restless, agitated, sleepy). Excessive sleepiness or somnolescence may occur with a pCO_2 of more than 45 mmHg and restlessness from hypoxia may occur at a PO_2 of less than 75 mmHg. Older children or adolescents also may report shortness of breath or dyspnea. The older child or adolescent may complain of a headache. Dilatation of the cerebral blood vessels from acute hypoxemia and hypercapnia is responsible for the headache symptoms.

Depending on the etiology of the respiratory failure, there may be a history of a foreign body ingestion, a previous URI, or another infection. The parents may mention noisy breathing or wheezing. They may relate the presence of a fever, cough, sputum, or hemoptysis. The patient may be tired or fatigued due to the increased work of breathing.

Table 2. Causes of Hypoxemia (Type I) Normocapnic Respiratory Failure*

VENTILATION/PERFUSION (V/Q) ABNORMALITIES
Pulmonary edema, meconium aspiration, pneumonia
SHUNTING
Cyanotic congenital heart disease: right to left shunts (ventricular septal defect, endocardial cushion defect)
Intrapulmonary shunts: alveoli are ventilated but not perfused (pneumonia, ARDS)
DIFFUSION ABNORMALITIES
Interstitial fibrosis
INADEQUATE SYSTEMIC BLOOD FLOW
Inadequate cardiac output: shock, cardiomyopathy
INADEQUATE OXYGEN CARRYING CAPACITY
Abnormal hemoglobin (methemoglobinemia, cyanide poisoning), severe anemia
INADEQUATE CELLULAR UPTAKE/UTILIZATION OF OXYGEN
Cyanide poisoning

** This is not an inclusive list. Some disorders are included in each group.*

Physical Examination

The patient may be confused, combative, restless, irritable, somnolent, lethargic, unresponsive, or comatose. The patient may have assumed a certain posture in an attempt to maintain an open airway or to help decrease the work of breathing. A child who is sitting up and leaning forward in the tripod position is attempting to keep his/her airway open. Drooling, which indicates an inability to swallow one's saliva, can occur with upper airway obstruction or can be caused by a foreign body, or an infection such as epiglottitis or a retropharyngeal abscess.

The vital signs will be abnormal if significant respiratory distress is present. Tachycardia and tachypnea are usual. Bradycardia and bradypnea are ominous signs of impending respiratory failure and/or cardiopulmonary arrest. Hypertension and diaphoresis reflect the body's attempt to compensate by the adrenergic "flight or fight" response. Hypotension suggests significant dehydration or shock. If an infection is the underlying cause of the respiratory failure, then fever may be present, although hypothermia also can occur.

Table 3. Causes of Hypercapnic (Type II) Respiratory Failure: Hypoxemia (Low PO_2) with Hypercapnia (High PO_2)

ALVEOLAR HYPOVENTILATION

NEUROLOGIC DISORDERS

- Central nervous system inhibition (control abnormalities)
  - Coma
  - Overdose
  - CNS disease
- Spinal/anterior horn cell disorders
  - Cervical spinal cord trauma
  - Myelitis
- Peripheral nerve disease
  - Guillian-Barré
- Neuromuscular junction disorders
  - Myasthenia gravis
  - Paralytic drugs

RESPIRATORY MUSCLE DISORDERS

- Respiratory muscle failure
  - Primary muscle diseases (i.e., muscular dystrophy, poliomyelitis)
- Respiratory muscle fatigue (i.e., excessive work of breathing)

CHEST WALL/PLEURA DISORDERS

- Decreased chest wall compliance
  - Flail chest
  - Diaphragmatic hernia
  - Severe kyphoscoliosis
- Disrupted pleural space
  - Limited movement of lungs: pneumothorax, hemothorax, pleural effusion

AIRWAY DISORDERS

- Upper airway obstruction:
  - Foreign body
  - Edema
  - Infection (croup, laryngotracheobronchitis, epiglottitis, retropharyngeal abscess)
  - Congenital web
  - Subglottic stenosis
  - Tracheomalacia
- Lower airway obstruction (edema, foreign body, tracheobronchomalacia, tumor)

PULMONARY DISEASES

- Increased airway resistance
  - Bronchiolitis, asthma
- Decreased lung compliance
  - Fibrosis, pulmonary edema, vascular diseases (polyarteritis), interstitial disease (sarcoidosis)
- Ventilation/perfusion (V/Q) abnormalities:
  - Pneumonia
- Increased dead space ventilation
  - Alveoli overdistention
  - Asthma, COPD
- Decreased pulmonary blood flow
  - Severe pulmonic stenosis, pulmonary hypertension

INCREASED CARBON DIOXIDE PRODUCTION

- Increased metabolic rate
  - Infection
  - Sepsis
  - Fever
  - Burns
- Abnormal respiratory exchange quotient

Increased work of breathing is reflected by: intercostal retractions, use of accessory muscles in the neck, flaring of the alae nasi, "see-saw" respirations, paradoxical breathing, abdominal breathing, head bobbing (especially in infants), and pursed lips.[12,13]

Noisy, abnormal respiratory sounds, from audible wheezing to stridor, or crowing or gurgling sounds may be present. Inspiratory stridor indicates obstruction in the upper airway, while prolonged expiration with wheezing is due to lower airway obstruction at the bronchial or bronchiolar level. Stridor may be due to infection (epiglottitis, retropharyngeal abscess or laryngotracheobronchitis), foreign body aspiration, congenital anomalies (i.e, laryngeal web and vocal cord cyst), or laryngomalacia. Wheezing usually occurs with asthma and bronchiolitis. Expiratory grunting is an attempt to increase airway pressure, thereby maintaining or increasing the functional residual capacity. In early inspiration, premature closure of the glottis, along with active contraction of the chest wall, results in grunting.

The rate (tachypnea, normal, or bradypnea), depth (shallow vs deep), and pattern (irregular, apnea, etc.) of respiration should be noted. Respiratory alternans may be present. Cyanosis is a late sign of respiratory failure. The percentage of desaturated hemoglobin in the blood determines whether cyanosis is present. Weak, thready pulses and delayed capillary fill of the skin suggest decreased peripheral perfusion and shock.

Observation of the chest may reveal an abnormal shape, appearance, or asymmetrical movement. Lung auscultation may detect rales, rhonchi, wheezing, and/or prolonged expiration. Fremitus and dullness to percussion are suggestive of consolidation as with pneumonia. Hyperresonance to percussion with decreased breath sounds is consistent with a pneumothorax.

Physical examination may reveal disorders that increase the risk for respiratory failure (see Table 5) or an underlying etiology for ARF.

Case No. 3

A 4-month-old female who is having trouble breathing is brought into the ED by her mother. She has had a cold for several days. Today she developed a fever, cough, and breathing difficulties.

Neonatal history is significant for prematurity (30 weeks), which was complicated by respiratory distress syndrome requiring a ventilator for two weeks and supplemental oxygen for an additional five weeks. She also had a congenital gastrointestinal problem requiring surgery at 6 weeks of age and has continued to have gastrointestinal problems. She has bronchopulmonary dysplasia and has not received the immunization for respiratory syncytial virus.

Her vital signs are:

- T = 103.5° F;
- P = 190 beats/min;
- R = 64 breaths/min;
- BP = 80/50 mmHg; and
- Pulse oxygen saturation = 82% in room air.

She is small for her age. The infant is in marked respiratory distress with retractions, grunting, flaring, head bobbing, abdominal respirations, and the use of accessory muscles for respiration. Her skin is pale, sweaty, and cyanotic with delayed capillary fill. There are rales in both lung fields. The chest roentgenogram shows diffuse bilateral infiltrates.

The ABG on room air is: pH = 7.61; pO_2 = 56; pCO_2 = 24;

Table 4. Criteria for Acute Respiratory Failure

CLINICAL FINDINGS OF ACUTE RESPIRATORY DISTRESS (IMPENDING RESPIRATORY FAILURE)

- **Laboratory parameters**
  - Hypoxemia
  - PaO_2 < 50-60 mmHg
  - SaO_2 < 90%
  - PaO_2/FiO_2 ratio < 300
  - PaO_2 < 60 mmHg on FiO_2 > 40
- **Hypercapnia**
  - pCO_2 > 55
  - pCO_2 > 50 with acidosis (pH < 7.25)
  - pCO_2 > 40 with severe distress
- **Pulmonary function parameters**
  - Vital capacity < 15 mL/kg
  - Maximum inspiratory force (pressure) < 20-25 cmH_20
  - VD/VT = Dead space/tidal volume > 0.60

HCO_3 = 27; and oxygen saturation is 78%.

This infant has multiple risk factors for ARF: prematurity with bronchopulmonary dysplasia, congenital anomalies, gastrointestinal disorder (with the potential for aspiration), malnutrition with failure to thrive, and her young age (infants have less pulmonary reserve and impaired immunologic capability compared to older children and adults).

She also has all the classic signs of air hunger and is struggling to breathe with increased work of breathing. She has respiratory failure due to pulmonary disease from an alveolar parenchymal disorder. Due to her pneumonia, she has V/Q abnormalities from collapsed alveoli that are perfused but not ventilated (and fail to pick up oxygen), creating an intrapulmonary right to left shunt. She has Type I respiratory failure characterized by hypoxia with low pCO_2. In this case, her CO_2 is low because she is compensating with increased work of breathing and tachypnea.

The cause of her hypoxia by mechanism includes: V/Q abnormalities, and intrapulmonary shunting. She has numerous criteria for ARF, including acute respiratory distress and laboratory parameters of hypoxemia. She has multiple indications for intubation (see Table 6), including: hypoxia, respiratory distress and need for special therapy; prolonged ventilation (until her pneumonia improves) and need for pulmonary care (suctioning, etc.); and delivery of positive end expiratory pressure (PEEP) to open her collapsed alveoli.

Diagnosis of Respiratory Failure

Respiratory failure can be determined by clinical, laboratory (e.g., ABG), and pulmonary function parameters.

Classically, the hallmark of respiratory failure is hypoxemia and/or hypercapnia.[20] Hypoxia is a decreased amount of oxygen supplied to or utilized by the body's tissues and cells, while hypoxemia refers to a decreased amount of oxygen in the blood or less than the physiologically normal amount of oxygen in the blood.

Normal ABG values are: pO_2 of 80-100 mmHg; pCO_2 of 35-45 mmHg; pH of 7.35-7.45; and SaO_2 of 95-100%. Hypoxemia is any value less than normal for the PO_2 and/or oxygen saturation. The arterial pO_2 in healthy young adults is usually 95-100 mmHg, although some healthy young adults have an arterial pO_2 of 80-90 mmHg. Therefore, the accepted range of normal for arterial pO_2 varies, and some allow a value as low as 80 for a normal pO_2.

Another commonly used method for evaluating oxygenation is the alveolar-arterial oxygen gradient:

$PAO_2 - PaO_2 = P(A\text{-}a)O_2$.

The alveolar-arterial difference ($PAO_2 - PaO_2$) is normally less than 15 mmHg. In a person breathing room air at sea level, the alveolar PO_2 or PAO_2 can be calculated by subtracting the arterial pCO_2 measured in the blood from 150 ($PAO_2 = 150 - pCO_2$) where there is a normal pCO_2 of 40 (the PAO_2 = 150- 40 = 110 mmHg). In patients given supplemental oxygen, a rough approximation of the expected or normal alveolar pO_2 is calculated by multiplying the actual delivered percentage of oxygen by 6. In a patient receiving 100% oxygen, the PAO_2 would equal 600 (100×6), while the PAO_2 would equal 360 (60×6) in a patient receiving 60% oxygen.

The PaO_2/FiO_2 ratio is yet another method for estimating the oxygenation. The normal PaO_2/FiO_2 ratio is 500-600, which is the arterial pO_2 measured in the blood divided by the fraction of inspired oxygen. This ratio is then used to determine the amount of shunting in the lungs.

The shunt (Qs/QT) represents the fraction of blood that passes through the lung without being oxygenated. The normal amount of physiologic shunting in the lung (also known as venous admixture) is 2-3% and represents the small amount of blood in the bronchial veins that drains into the pulmonary veins and the thebesian veins, which drain into the left atrium.

There are several variables that affect the arterial PO_2 or PaO_2. These are: the concentration or fraction of oxygen in the inspired gases (FiO_2), the amount of alveolar ventilation, the oxyhemoglobin dissociation curve, and the functional capability of the lungs.

The FiO_2 when breathing room air is 21% (or .21), and the addition of supplemental oxygen can increase the FiO_2 up to 100% (or 1.0). At a given FiO_2, the PaO_2 changes with the altitude or height above sea level. The higher the altitude, the lower the PO_2 in the air. For each 10,000-foot increase above sea level, the PaO_2 decreases approximately 3-4 mmHg.

The patient's age also affects pulmonary function. The normal PaO_2 is lower at the extremes of age. A normal, healthy older infant, child, or young adult has an arterial PO_2 of 80-100 mmHg, while a newborn has an arterial PO_2 of 60-90 mmHg. An 80-year-old has an arterial PO_2 of 75-80 mmHg. After age 30, the PaO_2 decreases by approximately 3-4 mmHg in each decade.

The generally accepted values of hypoxemia that define ARF are: PaO_2 less than 50-60 mmHg; SaO_2 less than 90%; PaO_2 less than 60 mmHg on FiO_2 = 40% or PaO_2/FiO_2 less than 300.

Hypercapnia or hypercarbia is an elevated level of carbon dioxide in the blood. ARF is indicated by the following pCO_2 values: pCO_2 higher than 50 with acidosis (pH < 7.25) or pCO_2 higher than 40 with severe distress, or pCO_2 higher than 55. This assumes that there are no pre-existing pulmonary disorders, such as cystic fibrosis, which cause chronic retention of carbon dioxide. Pulmonary function abnormalities consistent with ARF are: vital capacity less than 15 mL/kg or inspiratory

Table 5. Patients at High Risk for Acute Respiratory Failure

PRE-EXISTING DISORDERS

- Pre-existing pulmonary disease (Cystic fibrosis, bronchopulmonary dysplasia, etc.)
- Congenital cardiovascular disease
- Immunocompromised state (Malignancy, immunosuppression, HIV, etc.)
- Genetic and/or congenital defects

CO-EXISTENT DISEASE STATES

- Coma
- Shock
- Multiple organ system dysfunction (i.e., hypoxic-ischemic encephalopathy)
- Hypermetabolic states (i.e., thyroid storm)

CO-EXISTENT STRESSFUL CONDITIONS

- Multiple trauma
- Central nervous system trauma
- Postoperative (following major surgery)
- Post anesthesia (following general anesthesia)
- Post sedation (following general sedation)

HOST FACTORS

- Malnutrition
- Extremes of age (infants, geriatric patients)

pressure less than 25-30 cmH_20.

ABGs should be interpreted in a clinical setting.[11,20] For example, in a child with an intracardiac right to left shunt, as with a ventricular septal defect and pulmonic stenosis, the arterial PO_2 may be low, yet the patient is not in ARF. In a patient receiving supplemental oxygen, the arterial PO_2 may be in the normal range (pO_2 = 90-100), yet the patient is in respiratory failure. In patients with chronic metabolic alkalosis, the arterial pCO_2 may be elevated although the patient is not in respiratory failure. Patients with a respiratory disease such as asthma may be able to maintain normal blood gas values temporarily by increasing their work of breathing yet be in impending respiratory failure. Furthermore, it takes time to obtain and analyze an ABG or any other test. Life-saving therapy should not be delayed in order to obtain a blood gas or any other test. Clinical assessment may be the most important parameter in determining respiratory failure, and treatment often should be initiated before the specific laboratory criteria for ARF can be obtained or are fulfilled.

Case No. 4

A 2-month-old is brought to the ED by her mother with a chief complaint of not eating for several days. Vital signs are:

- T = 36.8°C (R);
- P = 180 beats/min;
- R = 58 breaths/min
- BP = 55/30 mmHg; and
- Pulse oxygen saturation is 78% on room air.

Past medical history was unremarkable. She was the full-term product of an uncomplicated pregnancy, labor, and delivery. Her birth weight was 7 lbs, 10 oz, and she had no neonatal problems.

Physical examination reveals a 2-month-old female in respiratory distress with tachypnea, retractions, and cyanosis. The HEENT examination is within normal limits. The neck is supple. The lungs are clear. The heart is tachycardic with no murmurs. The liver edge is down 2 cm. The abdomen is non-tender. There is no edema and no rash.

The chest roentgenogram has no infiltrates and the heart size is enlarged. The WBC is 12,000, with 60% polymorphonuclear leukocytes, 30% lymphocytes, 8% monocytes, 2% eosinophils, and no bands. The urinanalysis is labstick negative, with no WBCs, no RBCs, and no casts.

An ABG reveals: pH = 7.48; pO_2 = 62; pCO_2 = 34; and HCO_3 = 23.

The electrocardiogram shows sinus tachycardia with right ventricular hypertrophy. Because she is afebrile without a marked leukocytosis or a leftward shift, the chest roentgenogram is without infiltrates, and she has a negative urinalysis; your suspicion for sepsis is low. Since there are no pediatric cardiologists or pediatric intensivists at your community hospital, an echocardiogram is not available.

The next test you order will confirm your diagnosis of congenital heart disease. An ABG drawn on 100% FiO_2 shows essentially no change from the room air blood gas: pH = 7.48; pO_2 = 64; pCO_2 = 35; HCO_3 = 23; and O_2 saturation is 79%.

The only disease mechanism that shows essentially no response to supplemental oxygen is a right to left shunt. You already have placed a foley catheter to measure urine output and given furosemide with a good response. On repeat examination: P = 150 beats/min; R = 40 breaths/min; BP = 60/40 mmHg; O_2 saturation is 82%; the liver edge is down 1 cm; and a murmur is heard. After appropriate stabilization, you transfer the patient to a tertiary care hospital. At the tertiary care hospital, after an evaluation by the pediatric cardiologist, the infant is diagnosed with cyanotic congenital heart disease (i.e, pulmonic stenosis and ventricular septal defect with a right to left shunt) and congestive heart failure.

Infants may not have all the classic signs of heart failure, such as peripheral edema or jugular venous distention. Since the cardiac output was poor, no murmur was heard initially. Later, after treatment, she improved clinically and a murmur was heard.

Discussion

Failure to respond to supplemental oxygen is pathognomonic for a right to left shunt of more than 30%. If other tests are not immediately available, ABGs done on room air and 100% oxygen will allow the diagnosis to be made. A shunt can be non-pulmonary, as in this case, with cyanotic congenital heart disease, from a pulmonary arteriovenous fistula, or a shunt can be from a severe pulmonary shunt such as extensive lung disease (although this is uncommon). Whatever the etiology, with a shunt, there is bypass of the pulmonary capillaries bed because of shunting of systemic venous blood to the systemic arterial system. The amount of the shunt can be calculated by the formula for QS/QT.

Respiratory Physiology: Hypoxia

Respiration involves several key physiologic events. Pulmonary ventilation is the movement of air (inflow and outflow) between the environment or atmosphere and the alveoli. Gaseous exchange in the alveoli involves diffusion of oxygen

Table 6. Causes of Hypoxia by Mechanism

LOW INSPIRED FIO_2

- Deficiency of oxygen in atmosphere (i.e., high altitude)
- Low oxygen content of gas mixture: Suffocation, smoke inhalation, iatrogenic (problem with oxygen inflow during anesthesia, etc.)

HYPOVENTILATION

- Control disorders/central nervous system diseases (i.e., infection, trauma, infarct, encephalopathy)

NEUROMUSCULAR DISEASES

- Spinal disorders/diseases (i.e., spinal cord injury)
- Peripheral nerve disorders (i.e., Guillain-Barré disease)
- Neuromuscular junction (i.e., botulism, tetanus)

STRUCTURAL DISEASES: CHEST WALL OR PLEURAL DISORDERS

- Flail chest
- Hemothorax
- Pneumothorax

PULMONARY DISORDERS

- Increased airway resistance
  - Airway obstruction (i.e., bronchospasm, mucous plugging foreign body, etc.)
  - Pulmonary edema
- Decreased compliance
  - Decreased chest wall compliance (spinal/chest wall deformities)
  - Decreased lung compliance (i.e., pulmonary edema)
- Impaired alveolar diffusion of oxygen
  - Pulmonary fibrosis
- Ventilation/perfusion abnormalities (V/Q mismatch)
  - Asthma, pneumonia
- Intrapulmonary shunts
  - Blood goes through pulmonary circulation without being oxygenated (i.e., severe pneumonia, pulmonary arteriovenous fistula)

INADEQUATE TRANSPORT/DELIVERY OF OXYGEN

- Hematologic
  - Anemia, abnormal hemoglobins
- Cardiac/circulation
  - Shock, low output states, local circulation abnormality

SHUNTS: CARDIAC

- Ventricular septal defect with pulmonic stenosis

TISSUE/CELL INCAPABLE OF UTILIZING OXYGEN

- Cyanide poisoning, beriberi

and carbon dioxide across the pulmonary capillary membrane. Oxygen and carbon dioxide are then transported in the blood to and from the cells. The tissues and cells must be able to utilize the oxygen. Finally there is a control process or regulation of the physiologic events involved in respiration.[21,22]

There are numerous causes of hypoxia.[2,23] *(See Table 6.)* The more common causes are: hypoventilation (whether secondary to control abnormalities or pulmonary disorders involving increased airway resistance or decreased compliance), ventilation/perfusion abnormalities, and shunts.[5] Less common etiologies involve: inadequate delivery of oxygen (hematologic or circulation abnormalities), tissue/cellular inability to use oxygen, and oxygen deficiency in the inhaled gases.[5] V/Q abnormalities may be the most common cause of hypoxemia. In the normal lung, ventilation nearly matches perfusion (V/Q = .8), or alveoli that are ventilated also have adequate blood flow. Where there is no alveolar blood flow to a ventilated alveoli, ventilation is wasted and there is increased dead space with increased work of breathing. When there is an alveoli that has normal blood flow but is not ventilated, there is an intrapulmonary shunt whereby some pulmonary blood flow fails to pick up oxygen, which can cause hypoxemia if severe.

Thus, lung units that are perfused but poorly ventilated cause desaturation, while lung units that are well ventilated but poorly perfused (e.g., high V/Q) cause physiologic dead-space, but not hypoxemia. When alveoli that have no blood flow are ventilated, the ventilation of these alveoli is wasted, which adds to the dead space. Dead space refers to areas of the lung that are ventilated but not perfused. Ventilation of the respiratory passageways (conducting airways such as the larynx, trachea, and bronchi) where gas exchange does not occur is anatomic dead space. When there is a large increase in dead space, much of the work of breathing becomes a wasted effort, since most of the ventilated air never reaches the bloodstream. When an area of the lung is perfused but is not ventilated, blood is not oxygenated and an intrapulmonary shunt is present. The shunt fraction (or venous admixture) estimates the amount of pulmonary blood flow that perfuses non-ventilated or underventilated lung.

Alveolar dead space is that part of the inspired air that passes through the anatomic dead space to the alveoli but does not take part in the gas exchange in the pulmonary capillary bed. In the normal lung, alveolar dead space is negligible; however, it is markedly increased in diseases such as ARDS and with pulmonary emboli.

Under normal conditions, there is a small intrapulmonary shunt (2-3%) due to the drainage of bronchial veins into the pulmonary vein and the thebesian veins into the left ventricle. Venous admixture (or pulmonary shunt) of more than 5% is abnormal. Pathologic admixture can be caused by: intracardiac right to left shunts (cyanotic congenital heart disease, such as ventricular septal defect or endocardial cushion defect), or by intrapulmonary shunting from pulmonary diseases such as pneumonia, pulmonary edema, and atelectasis.

Impaired diffusion across the alveolar-capillary membrane can lead to hypoxia. Diseases that impose a barrier to diffusion across the alveolar-capillary membrane include pulmonary fibrosis and pulmonary edema.

Oxygen transport failure results from the inability to deliver adequate amounts of oxygen to the tissues/cells. Oxygen transport failure can occur with hematologic disorders. These include: severe anemia, the presence of abnormal hemoglobins (e.g., methemoglobinemia), and lack of hemoglobin binding to oxygen (e.g., carbon monoxide poisoning). Cardiac diseases with low output (including cardiomyopathies and severe heart

Table 7. Indications for Intubation

CATEGORY	CLINICAL EXAMPLE OF DISEASES
Failure of respiratory drive (Control mechanism abnormalities)	CNS disorders (infection, trauma, malformations, infarcts), unresponsiveness, altered mental status, coma
Airway protection	Anesthesia Sedation, alcohol or drug intoxication Loss of gag reflex Severe emesis during procedures (such as gastric lavage)
Cardiac	Cardiac arrest Shock Peripheral vascular collapse
Pulmonary (Clinical signs: tachypnea, retractions, etc.)	Respiratory distress Ineffective ventilation, 2° respiratory muscle injury/disorders (trauma, spinal/peripheral nerve disease, neuromuscular junction disease), etc. Chest wall/pleural disease (flail chest, pneumothorax, etc.) Pulmonary disorders causing hypoxia and/or hypercapnia, ARF
Need for special therapy	Hyperventilation Delivery of PEEP, special need pulmonary care (suctioning, etc.), need for prolonged ventilation

failure) and circulatory disorders (e.g., shock) can lead to inadequate oxygen transport to the tissues/cells. Hypoxia secondary to low inspired FiO_2 most often is encountered in cases involving high altitudes, suffocation, or smoke inhalation.

Disorders in which the cells and tissues are presented with oxygen but are unable to use or metabolize oxygen are rare. This does occur with cyanide poisoning and in deficiencies of the oxidative enzymes or tissue oxidation system (e.g., berberi).

Hypoventilation is a common cause of hypoxia and usually also causes hypercapnia (Type II respiratory failure). Hypoventilation can be due to ventilatory pump failure, caused by either control abnormalities (nervous system) or chest wall/pleural mechanical pulmonary abnormalities leading to increased airway resistance or decreased compliance. Airway obstruction and asthma are causes of increased airway resistance.

Case No. 5

A 5-year-old male is seen for a cough of several days duration that is not improving. He developed a cough three days ago after playing outside on a hot, humid day while his father mowed the lawn. Past medical history is unremarkable except for a history of bronchiolitis as an infant, for which he was hospitalized for two days. Family history is positive for asthma in an older sister, two paternal cousins, and the mother. His mother noted that he is usually quite active but hasn't been playing much, and hasn't been eating for the past three days. Tonight he seems worse.

The nursing triage note reads "chief complaint: cough; no fever, emesis, or diarrhea."

Vital signs are:

- T = 96.8°F (O);
- P = 170 beats/min;
- R = 44 breaths/min; and
- Pulse oximetry is 94% on room air.

On physical examination, there is an alert 5-year-old male sitting up and leaning forward. A lung examination reveals wheezing bilaterally. He is tachypnic with intercostal retractions. Three continuous albuterol aerosols were given by respiratory therapy. An ABG was drawn because of his poor response to aerosol.

An hour after he was triaged, you notice a lethargic 5-year-old male. His lungs are clear, no wheeze or rales, and no retractions. He has dry mucous membranes and pale skin with tenting. You order repeat vital signs and put him on a monitor.

Vital signs are now:

- T = 96.8°F (O);
- P = 102 beats/min;
- R = 16 breaths/min;
- BP = 65/40 mmHg; and
- Pulse oxygen saturation = 86% on room air.

The monitor shows sinus tachycardia with occasional premature ventricular contractions (PVCs). You order an IV, electrolytes, blood urea nitrogen, creatinine, complete blood count, chest x-ray, ABG, repeat aerosol, 100% FiO_2, a bolus of normal saline at 20 cc/kg, and IV steroids. A terbutaline drip also is started.

After the second aerosol, there is bilateral wheezing but poor air entry/exchange and no retractions:

- P = 94 beats/min;
- R = 14 breaths/min;
- BP = 75/45 mmHg; and
- Pulse oximetry is 91% on 100% FiO_2.

He is lethargic, but when aroused by a needle-stick, he is confused and combative. You give a second bolus of normal saline to treat his hypotension.

You decide you need to intubate the patient and perform a rapid sequence induction. You premedicate with atropine and lidocaine, followed by ketamine as the sedating agent and vecuronium as the paralytic agent. He is given an aerosol and 100% FiO_2 via the endotracheal tube. Post intubation, he is difficult to ventilate by bagging. However, the endotracheal tube is checked for tube placement and is documented to be in good position (a chest roentgenogram shows no pneumothorax or infiltrate), and there is some improvement after the aerosol. He is admitted to the PICU.

The laboratory data arrives after the patient is in the PICU. Laboratory results include:

- WBC = 14,600;

- Hematocrit = 52;
- BUN = 60;
- Creatinine = 1.5;
- Sodium = 150; and
- Potassium = 4.0.

The first ABG on room air shows: pH = 7.52; pO_2 = 58; pCO_2 = 24; HCO_3 = 14; and oxygen saturation = 88% on room air.

The second ABG shows: pH = 7.12; pO_2 = 68; pCO_2 = 70; HCO_3 = 14; and oxygen saturation is 90% on 100% FiO_2.

Five days later, he was doing well and was discharged home from the pediatric floor.

Discussion

The patient had hypotension/shock caused by several days of poor oral intake and increased fluid losses from his tachypnea (secondary to the asthmatic attack) and the hot weather. The laboratory data indicate hypernatremic dehydration with an elevated BUN, sodium, and hematocrit (hemoconcentration). The WBC was elevated from the stress of an acute asthmatic attack.

Even without an ABG, the patient had multiple indications for emergent intubation. On initial presentation, he had numerous indications that he might require intubation: signs of severe respiratory distress; wheezing with tachypnea; intercostal retractions; and use of accessory muscles. He was struggling to breathe and had air hunger. Therapy did not improve his air exchange.

A short time later (~ 1 hour), his respiratory rate dropped to the normal range (from 44 to 16). He was so exhausted from the three days of doing excessive work to breathe that he no longer had the energy to struggle to breathe and no longer had retractions or the use of accessory muscles. He no longer had wheezing since he was moving so little air, also indicating a worsening clinical condition. He was no longer alert but now was lethargic, suggesting somnolence due to a rising pCO_2. The confusion and combativeness that occurred when an IV was started was probably due to cerebral hypoxia. He had multiple clinical indicators of respiratory distress. He also had shock secondary to severe dehydration.

In addition to these clinical parameters, he also had laboratory criteria for ARF. The ABG obtained when he was struggling to breathe showed respiratory alkalosis (low pCO_2), and low bicarbonate from his dehydration, shock, and hypoxia (paO_2 < 60 mmHg; SaO_2 < 90%; PaO_2/FiO_2 = 58/0.21 = 276 < 300). When he was too tired to continue the extra effort of breathing, the second ABG demonstrated hypercapnia with respiratory acidosis and hypoxia.

His underlying pulmonary disorder (asthma) caused increased airway resistance, which resulted in increased work of breathing, which led to impaired movement of air from the atmosphere to the alveoli (ventilation). His respiratory failure was due to respiratory muscle fatigue from increased work of breathing and a cardiovascular disorder (e.g., shock). He had many indications for intubation. *(See Table 7.)* Ketamine is considered the sedative of choice with an acute asthmatic attack because of its bronchodilating effects.

Table 8. Methods for Administration of Supplemental Oxygen*

METHOD	MAXIMUM POSSIBLE FIO_2
• Isolette (with supplemental oxygen through port)	~ 100%[†]
• Oxygen hood (head box)[α]	~ 95%
• Face shield (face tent)	40%
• Oxygen tent	40-50%[β]
• Nasal cannula (various sizes)	Flow rate (liters/min)
Infant (< 1 year)	
Toddler (1-5 years)	
Pediatric (5-13 years)	
Adolescent (> 13 years)	
Adult	24-25% - 44-45%
• Mouth to mask (with supplemental oxygen)	50-80%
• Face mask	
Venturi face	24, 28, 31, 35, 40, 50%
Simple face mask	40-50%
Reservoir (partial re-breather, no one-way valves)	60-80%
Reservoir bag (non-breather, has two one-way valves)	90-100%
• Bag valve mask ventilation (without reservoir)	40%
• Bag valve mask ventilation (with reservoir)	90-100%
• Anesthesia bag (depending on source gas)	21-100%

FiO_2 depends on the rate, depth and respiration. A rough guideline is to add ~ 4% to FiO_2 of 21% for room air for each liter per minute.

Legend

*Methods preferred depend on ease of administration, consistency of FiO_2, tolerance by patient, and ability to achieve a specific FiO_2.

[†] Use with oxygen analyzer

[α] Difficult to maintain a consistent or stable FiO_2

[β] Size allows for use in patients up to about 1 year of age

Treatment

The first step is to ensure patency of the airway.[24] If the child has adequate air exchange, then he or she should be allowed to maintain a position of comfort with the head in a neutral sniffing position. Hyperextension of the neck may worsen upper airway obstruction and should be avoided. Similarly, a child may have better ventilation if left in an upright position sitting in his or her parent's lap than if forced to lie down. An upright position on the examining table is usually preferred since the supine position increases the work of breathing by placing the weight of the abdominal organs on the diaphragm.

Suctioning of the airway, clearing the airway of an obstruction, and appropriately positioning the head and neck of the patient may help maintain airway patency.

After a patent airway is obtained, oxygenation and/or ventilation must be assessed and appropriate intervention completed. Respiratory failure may be categorized as a failure of oxygenation, a failure of ventilation, or both. Hypoxia is treated by

administering supplemental oxygen by various devices ranging from facemasks or nasal cannulas to hoods. *(See Table 8.)* Administration of supplemental oxygen is indicated in all pediatric patients at risk for respiratory failure, even if there is no obvious hypoxemia or impending ARF. Such aggressive therapy may prevent the at risk patient from proceeding to ARF. Patients with ARF may need assisted ventilation in addition to supplemental oxygen.

Additional therapies also are beneficial in the ARF patient. These include careful attention to fluids and electrolytes; treatment of dehydration; appropriate antibiotics for infection; correcting electrolyte abnormalities; and transfusion if the patient is severely anemic.

Treatment of the underlying cause of the respiratory distress may prevent respiratory failure or reverse/correct the respiratory failure if it is already present. Treatment of an infection such as pneumonia with appropriate antibiotics, relieving an airway obstruction, or initiating bronchodilator therapy in asthma is essential in averting or reversing ARF in specific cases.

In those patients who do develop ARF, various newer therapies, such as high frequency jet ventilation, BiPAP, surfactant, liquid ventilation, nitrous oxide, or extra-corporeal membrane oxygenation, may be beneficial in selected cases.[25-28]

Summary

Pulmonary disease remains a leading cause of mortality in pediatric patients in spite of recent advances in treatment. ARF is a significant cause of morbidity and mortality in infants and children. Careful assessment of pediatric patients can lead to early recognition of respiratory distress, and institution of appropriate therapy may prevent ARF in some patients. The advent of newer therapies also may lead to a decrease in the high morbidity and mortality associated with ARF.

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Physician CME Questions

19. A 3-month-old has grunting, flaring, retracting, and tachypnea with a pulse oxygen saturation of 88% on 50-1 oxygen. The infant most likely has:
    A. Type I respiratory failure.

B. Type II hypercapnic respiratory failure.
C. Both of the above
D. None of the above

20. The 3-month-old infant from Question 19:
A. does not have any indication for intubation.
B. does meet some criteria for intubation.
C. requires an ABG before a decision to intubate is made.
D. None of the above

21. Which of the following is an acceptable indication for intubation?
A. Coma
B. Respiratory distress
C. Shock
D. All of the above

22. Of the following, the most common mechanism for hypoxemia in patients with pneumonia is:
A. decreased pulmonary blood flow.
B. increased upper airway resistance.
C. diffusion abnormalities.
D. ventilation/perfusion (V/Q) abnormalities.

23. Which of the following processes involved in respiration usually is impaired in patients in hemorrhagic shock?
A. Ventilation
B. Intrapulmonary gas exchange
C. Gas transport to the tissues or cells
D. Tissue or cellular gas exchange

24. A patient with cyanide poisoning has difficulty with which of the following process involved in respiration?
A. Ventilation
B. Intrapulmonary gas exchange
C. Diffusion abnormalities
D. Tissue or cellular exchange

25. A 14-year-old male is unresponsive to verbal or painful stimuli. His vital signs are:
- T = 98.6° F;
- P = 84 beats/min;
- R = 6 breaths/min; and
- BP = 110/70 mmHg.

The ABG is: ph = 7.15; pCO_2 = 80; pO_2 = 70; HCO_3 = 26; and O_2 saturation is 90% on room air. This patient has:
A. Type I respiratory failure.
B. normocapnic respiratory failure.
C. hypoxemia non-ventilatory respiratory failure.
D. Type II respiratory failure.

26. The respiratory failure in the patient in Question 25 is due to:
A. increased CO_2 production.
B. increased dead space ventilation.
C. alveolar hypoventilation from impaired central ventilatory drive.
D. intrapulmonary right to left shunt.

27. A patient has a 50% intracardiac right to left shunt. When given 100% supplemental oxygen (100 FiO_2) there would be:
A. an increase in pO_2 and oxygen saturation.
B. a decrease in pO_2 and oxygen saturation.
C. no change in pO_2 and oxygen saturation.
D. All of the above would occur.

28. Which of the following factors increases the body's production of carbon dioxide by increasing the body's metabolic rate?
A. Fever
B. Infection
C. Burns
D. All of the above

In Future Issues:

Antibiotic Resistance