**FIGURE 6-4**

Main components of the ventilatory system. The ventilatory system is responsible for maintaining the arterial carbon dioxide tension (PaCO_2) within normal limits by adjusting minute ventilation (\dot{V}) to match the rate of carbon dioxide production. The main elements of ventilation are the respiratory pump, which generates a pressure gradient responsible for air flow, and the loads that oppose such action. The machinery of the respiratory pump includes the cerebrum, brain stem, spinal cord, phrenic and intercostal nerves, and the muscles of respiration. Inspiratory muscle contraction lowers pleural pressure (Ppl) thereby inflating the lungs (ΔV). The diaphragm, the most important inspiratory muscle, moves downward as a piston at the floor of the thorax, raising abdominal pressure (Pabd). The inspiratory decrease in Ppl by the respiratory pump must be sufficient to counterbalance the opposing effect of the combined loads, including the airway flow resistance, and the elastic recoil of the lungs and chest wall. The ventilatory requirement influences the load by altering the frequency and depth of the ventilatory cycle. The strength of the respiratory pump is evaluated by the pressure generated ($\Delta P = \text{Ppl} - \text{Pabd}$).

DETERMINANTS AND CAUSES OF CARBON DIOXIDE RETENTION

Respiratory Pump		Load	
Depressed Central Drive	Abnormal Neuromuscular Transmission	Increased Ventilatory Demand	Lung Stiffness
Acute	Acute	High carbohydrate diet	Acute
General anesthesia	High spinal cord injury	Sorbent-regenerative hemodialysis	Severe bilateral pneumonia or bronchopneumonia
Sedative overdose	Guillain-Barré syndrome	Pulmonary thromboembolism	Acute respiratory distress syndrome
Head trauma	Status epilepticus	Fat, air pulmonary embolism	Severe pulmonary edema
Cerebrovascular accident	Botulism	Sepsis	Atelectasis
Central sleep apnea	Tetanus	Hypovolemia	Chronic
Cerebral edema	Crisis in myasthenia gravis	Augmented Airway Flow Resistance	Severe chronic pneumonitis
Brain tumor	Hypokalemic myopathy	Acute	Diffuse infiltrative disease eg alveolar proteinosis
Encephalitis	Familial periodic paralysis	Upper airway obstruction	Interstitial fibrosis
Brainstem lesion	Drugs or toxic agents eg curare, succinylcholine, aminoglycosides, organophosphorus	Coma-induced hypopharyngeal obstruction	Chest Wall Stiffness
Chronic	Chronic	Aspiration of foreign body or vomitus	Acute
Sedative overdose	Poliomyelitis	Laryngospasm	Rib fractures with flail chest
Methadone or heroin addiction	Multiple sclerosis	Angioedema	Pneumothorax
Sleep disordered breathing	Muscular dystrophy	Obstructive sleep apnea	Hemothorax
Brain tumor	Amyotrophic lateral sclerosis	Inadequate laryngeal intubation	Abdominal distention
Bulbar poliomyelitis	Diaphragmatic paralysis	Laryngeal obstruction after intubation	Ascites
Hypothyroidism	Myopathic disease eg polymyositis	Lower airway obstruction	Peritoneal dialysis
	Muscle Dysfunction	Generalized bronchospasm	Chronic
	Acute	Airway edema and secretions	Kyphoscoliosis, spinal arthritis
	Fatigue	Severe episode of spasmodic asthma	Obesity
	Hyperkalemia	Bronchiolitis of infants and adults	Fibrothorax
	Hypokalemia	Chronic	Hydrothorax
	Hypoperfusion state	Upper airway obstruction	Chest wall tumor
	Hypoxemia	Tonsillar and peritonsillar hypertrophy	
	Malnutrition	Paralysis of vocal cords	
	Chronic	Tumor of the cords or larynx	
	Myopathic disease eg polymyositis	Airway stenosis after prolonged intubation	
		Thymoma, aortic aneurysm	
		Lower airway obstruction	
		Airway scarring	
		Chronic obstructive lung disease eg bronchitis, bronchiolitis, bronchiectasis, emphysema	

FIGURE 6-5

Determinants and causes of carbon dioxide retention. When the respiratory pump is unable to balance the opposing load, respiratory acidosis develops. Decreases in respiratory pump strength, increases in load, or a combination of the two, can result in carbon dioxide retention. Respiratory pump failure can occur because of depressed central drive, abnormal neuromuscular transmission, or respiratory

muscle dysfunction. A higher load can be caused by increased ventilatory demand, augmented airway flow resistance, and stiffness of the lungs or chest wall. In most cases, causes of the various determinants of carbon dioxide retention, and thus respiratory acidosis, are categorized into acute and chronic subgroups, taking into consideration their usual mode of onset and duration [2].

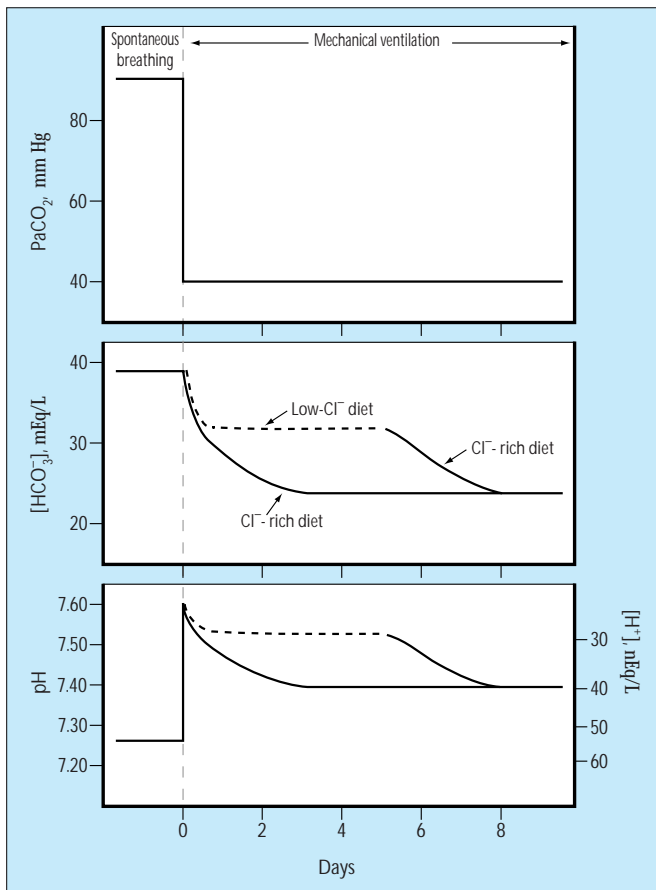


FIGURE 6-6

Posthypercapnic metabolic alkalosis. Development of posthypercapnic metabolic alkalosis is shown after abrupt normalization of the arterial carbon dioxide tension (PaCO_2) by way of mechanical ventilation in a 70-year-old man with respiratory decompensation who has chronic obstructive pulmonary disease and chronic hypercapnia. The acute decrease in plasma bicarbonate concentration ($[\text{HCO}_3^-]$) over the first few minutes after the decrease in PaCO_2 originates from alkaline titration of the nonbicarbonate buffers of the body. When a diet rich in chloride (Cl^-) is provided, the excess bicarbonate is excreted by the kidneys over the next 2 to 3 days, and acid-base equilibrium is normalized. In contrast, a low-chloride diet sustains the hyperbicarbonatemia and perpetuates the posthypercapnic metabolic alkalosis. Abrupt correction of severe hypercapnia by way of mechanical ventilation generally is not recommended. Rather, gradual return toward the patient's baseline PaCO_2 level should be pursued [1,2]. $[\text{H}^+]$ —hydrogen ion concentration.

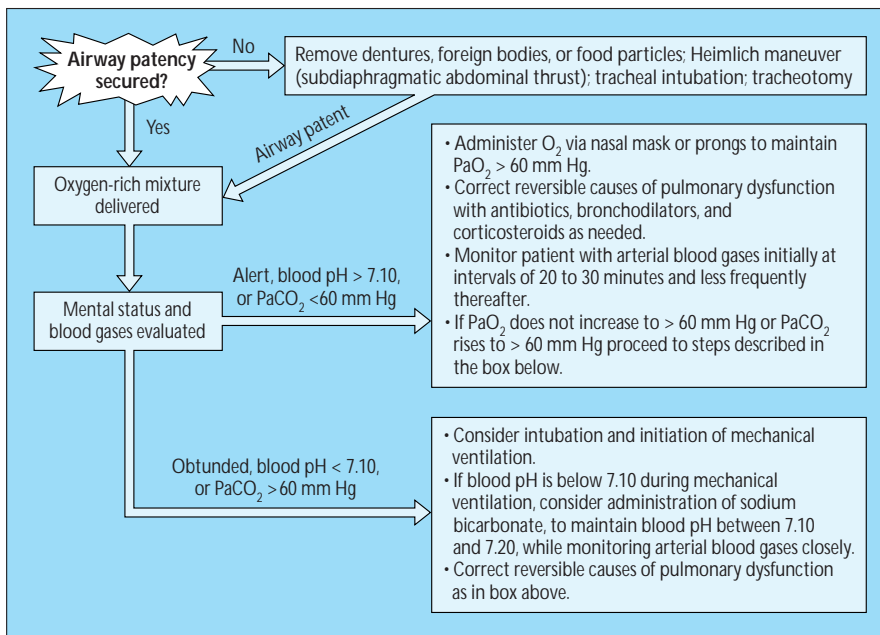


FIGURE 6-7

Acute respiratory acidosis management. Securing airway patency and delivering an oxygen-rich mixture are critical initial steps in management. Subsequent measures must be directed at identifying and correcting the underlying cause, whenever possible [1,9]. PaCO_2 —arterial carbon dioxide tension.