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Pediatric Intensive Care

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THE MAJOR OBJECTIVE of intensive care is to provide maximum surveillance and support of vital systems in patients with acute, but reversible, life-threatening disease. In pediatric patients, the reversal of life-threatening conditions and the preservation of essential functions, especially those of the brain, may result in many years of useful life. For example, in the United States a 3-year-old white female child has a life expectancy of 78 years, and a 1-year-old black male child, although less favored, still has an expectancy of 61 years. However, if brain function becomes permanently impaired as a result of delayed or improper care, both the patient and society will suffer for many years. In this monograph we cite the elements and the special considerations involved in the intensive care of critically ill pediatric patients, and the common causes of acute major organ failure, and consider in detail the diagnosis and management of acute respiratory failure.

Elements of Pediatric Intensive Care

The elements[1] of intensive care have been defined as: 1) geographic full-time physician specialists in Anesthesiology, Medicine (Pediatrics), General Surgery and its subspecialties; 2) a full-time physician director of Intensive Care; 3) nursing and allied health personnel specially trained in care of the critically ill; 4) availability of resuscitation and respiratory therapy equipment and drugs; 5) monitoring and alarm systems for continuous assessment of vital functions; 6) a 24-hour laboratory service for the rapid determination of *p*H and blood-gas tensions, oxygen content, hemoglobin, blood sugar, and serum sodium, potassium, calcium, and osmolality; 7) a 24-hour radiology service responsive to the needs of the critically ill; 8) location of these facilities in one area of the hospital.

Pediatric intensive care involves these elements as they apply to critically ill patients ranging in age from 1 month to late adolescence. This requires that the personnel involved be thoroughly familiar with pediatric anatomy, physiology, pharmacology, pathology, psychology, and appropriate technical maneuvers. For example, a 7-month-old, 6-kg infant with bronchiolitis may develop life-threatening impairment of gas exchange (acute respiratory failure).[2] The infant's airways are of narrower caliber than those of the adult relative to body size and the requirements for gas exchange.[3] Thus, a minimal reduction in the bronchiolar lumen due to the edema and secretions associated with bronchiolitis results in a considerable decrease in compliance and increase in the work of breathing.[4,5] Decreased oral intake resulting from dyspnea and the infant's high basal metabolic rate culminate in dehydration and acute caloric deprivation. These, in turn, lead to fatigue of the respiratory muscles, a decrease in minute volume, and acute respiratory failure. Mechanical ventilation of the infant with bronchiolitis is facilitated by the use of nondepolarizing muscle relaxants, although the infant may have a prolonged response to these drugs.[6] Placement and care of the tracheal airway, insertion of intravascular cannulas in a 6-kg infant, etc., demand special technical skills, and specially designed equipment. Finally, the 7-month-old infant is cognizant of separation from his mother, requiring sedation during the most critical phases of the illness, and later, fondling, as well as other forms of maternal care.[7]

Causes of Acute Major Organ Failure

The common disorders resulting in acute major organ failure in infants and children

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are outlined in table 1 according to the primary systems involved.

Nervous System

Acute central nervous system failure poses an immediate threat to life due to: 1) coma, with upper airway obstruction or pulmonary aspiration of gastric contents; 2) disturbed respiratory control secondary to medullary depression; 3) increased intracranial pressure, causing brain-stem and cortical compression.

The clinical signs of increasing intracranial pressure include decreases in consciousness and response to pain, papilledema, increasing systemic arterial pressure, bradycardia, and irregular breathing. Unfortunately, the increase in intracranial pressure may considerably exceed the normal upper limit of 20 torr before these clinical signs are recognized. Therefore, in patients at risk from increased intracranial pressure, a subdural bolt[8] or intraventricular cannula[9] should be inserted. The latter, when attached to a transducer and slow-speed recorder with simultaneous recording of systemic arterial pressure and heart rate, monitors the pressure waves that precede the development of continuously increased intracranial pressure. Thus, an estimate of the cerebral perfusion pressure can be constantly assessed. Appropriate therapy with oxygen, hyperventilation, maintenance of normal body temperature, and intravenous corticosteroids, barbiturates, and mannitol can be instituted before irreversible brain damage occurs.[10] Reye's syndrome is a bizarre encephalopathy in children associated with cerebral edema and fat deposition in the liver and other viscera.[11] The monitoring and treatment of increased intracranial pressure due to cerebral edema appears to be a fruitful approach to therapy in this syndrome.‡

Disorders such as the Guillain-Barré syndrome and myasthenia gravis that produce peripheral neuromuscular failure may pose a threat to life because of respiratory depression and impairment of upper airway reflexes.[12]

‡Berman W: Personal communication.

TABLE 1. Common Pediatric Disorders Resulting in Acute Major System Failure

Nervous system
Central
Trauma to the head
Cerebral hypoxia
Encephalitis
Cerebral hemorrhage
Drug ingestion poisoning
Hydrocephalus
Tumor
Reye's syndrome
Status epilepticus
Peripheral
Polyneuritis (Guillain-Barré syndrome)
Myasthenia gravis
Tetanus
Poliomyelitis
Cardiovascular system
Congenital cardiac lesions
Myocarditis
Septic shock
Blood loss
Fluid overload
Renal system
Acute tubular necrosis
Renal-vein thrombosis
Hemolytic uremia syndrome
Goodpasture's syndrome
Hematopoietic system
Sickle-cell anemia
Leukemia

Cardiovascular System

Congenital cardiovascular lesions are among the leading causes of death in infants less than 1 year of age. Cyanotic lesions such as tetralogy of Fallot and transposition of the great vessels cause severe tissue hypoxemia and eventual myocardial and central nervous system failure. In addition, these lesions are associated with polycythemia and increased blood viscosity. Hematocrits in excess of 60 per cent may result in widespread vascular thromboses, especially in the brain. Many of these children require intensive care and surgical correction or palliation in the first year of life to prevent irreversible hypoxic damage.[13] Infants and young children with acyanotic lesions often have congestive cardiac failure, pulmonary edema,[14] or pulmonary-artery hypertension.[15] These infants are particularly susceptible to pulmo-

nary infections, especially pneumonitis caused by gram-negative organisms, which may be the precipitating factor in the development of congestive cardiac failure.

Myocarditis, most often viral in origin, can cause severe congestive cardiac failure with pulmonary edema, as well as potentially lethal arrhythmias. Sepsis caused by gram-negative organisms in infants less than one year of age commonly causes severe arterial hypotension, necessitating administration of large volumes of intravenous fluids and, occasionally, infusion of catecholamines to maintain adequate organ perfusion.

Renal System

Acute renal failure may be associated with bacterial or uremic pneumonitis, or with pulmonary edema, in infants and children.[16] The hyperkalemia that usually occurs may produce lethal arrhythmias and eventual myocardial depression with systemic arterial hypotension unless controlled by peritoneal dialysis. Goodpasture's syndrome is an uncommon disorder of unknown etiology affecting the basement membranes of the lungs and kidneys with consequent diffuse intra-aiveolar hemorrhage and glomerulonephritis; it occurs in older children.[17] Survival is possible if the pulmonary hemorrhages can be controlled.[18]

Hematologic System

The two most common pediatric hematologic disorders, sickle-cell anemia and leukemia, may produce acute life-threatening conditions that are reversible with appropriate care. Children with sickle-cell anemia can manifest severe high-output congestive cardiac failure, and respond to transfusions of erythrocytes, digitalis, diuretics, and control of the associated pulmonary edema. These patients may also develop cerebrovascular thromboses, from which they can recover with appropriate control of intracranial pressure, hydration, and erythrocyte transfusions. With modern chemotherapy, children seldom die of the direct effects of leukemia. Rather, sepsis, pneumonitis, or unrecognized expanding intracerebral metastatic lesions may be the cause of death. Most often, pneumonitis is the result of gram-negative organisms such as *Pseudomonas* or *Pneumocystis carinii* to which these children become susceptible because of the immunosuppressant effects of chemotherapy. Fortunately, many leukemic children have recovered from prolonged respiratory failure and returned to normal childhood activities for a number of years.

Acute Respiratory Failure

The majority of disorders requiring intensive care in infants and children are associated with impaired ventilation. When the impairment of ventilation is sufficient to pose an immediate threat to life, acute respiratory failure exists. The criteria for diagnosis of acute respiratory failure vary with the age of the patient and the primary disease. Although clinical observations are of considerable importance, the diagnosis should depend on repeated physiologic measurements, particularly arterial *p*H and blood-gas tensions.

Causes of Acute Respiratory Failure

The principal causes of acute respiratory failure have been outlined in table 2. Among the disorders causing life-threatening upper airway obstruction, infectious croup (viral laryngotracheobronchitis) is the most common. Infants less than 6 months of age who have clinical signs of severe croup often have associated congenital subglottic stenosis. Although less common than croup, epiglottitis results in much higher mortality and morbidity because of the rapid onset of intense supralaryngeal and laryngeal edema, in addition to the toxicity produced by *Haemophilus influenzae*, the usual infecting organism.

Life-threatening lower-airway obstruction in children is most commonly caused by status asthmaticus[19,20] or bronchopneumonia[2,21] due to bacterial or viral infection. Status asthmaticus is defined as intense bilateral wheezing, occurring in an atopic child, which fails to improve after three subcutaneous injections of epinephrine (0.01 mg/kg) at 20-minute intervals. The majority of children in status asthmaticus do not develop acute respiratory failure, but the

onset of severe hypercapnia and hypoxemia may be subtle and sudden.[19] Thus, these children require extraordinarily close surveillance. The signs of status asthmaticus in chilcren less than 1 year old may also be produced by a form of viral bronchopneumonia known as "bronchiolitis." Airway obstruction in these infants causes hyperinflation, tachypnea, and intense wheezing, persisting over a period of three to five days. Bronchodilators usually are not beneficial, and acute respiratory failure occasionally develops.[2] The pulmonary manifestations of cystic fibrosis may appear at any age, including the newborn.[22] The early onset of obstructive pulmonary disease in infants with cystic fibrosis carries an ominous prognosis for life beyond the age of 12 years.[23]

Bacterial and viral pneumonia are the most common alveolar disorders causing respiratory failure in the pediatric age group. Systemic infection with gram-negative organisms in infants and young children produces severe pneumonitis, leading to acute respiratory failure. Gram-negative sepsis in the advanced stages also causes alveolar hemorrhage. Ingestion of hydrocarbons by young children with resultant pulmonary aspiration produces an intense chemical pneumonitis, frequently associated with progressive hypoxemia, bleb formation, and pneumothorax.

Pulmonary edema most commonly is caused by congenital cardiac lesions with congestive cardiac failure. Long-term mechanical ventilation of infants and children with respiratory failure due to cardiopulmonary disease has been associated with interstitial pulmonary edema, evident radiographically and causing increases in the alveolar–arterial oxygen tension differences. Oxygen toxicity has been incriminated as a major factor in the development of bronchopulmonary dysplasia in the newborn.[24] However, there is no objective evidence to indicate this is associated with prolonged use of elevated inspired concentrations of oxygen in older infants and in children.

Space-occupying lesions such as pneumothorax, hemothorax, or diaphragmatic eventration must be excluded by chest x-ray in any infant or child manifesting acute respiratory failure. Pneumomediastinum can produce potentially fatal cardiac tamponade unless decompression occurs by air dissection into th facial planes of the neck, pleura, or peritoneum. Severe kyphoscoliosis in older children and adolescents has been associated with acute respiratory failure when these patients develop pneumonia, or following major orthopedic procedures for stabilization of the spine.[25]

TABLE 2. Common Pediatric Disorders Resulting in Acute Major System Failure of the Respiratory System

Upper airway obstruction
Croup
Infectious
Post-intubation
Epiglottitis
Congenital subglottic stenosis
Foreign body
Vocal-cord paralysis
Vascular ring
Granuloma
Burns
Lower airway obstruction
Status asthmaticus
Bronchopneumonia (bronchiolitis)
Smoke inhalation
Cystic fibrosis
Aspiration syndrome
Lobar emphysema
Alveolar disorders
Pneumonia
Infectious
Chemical
Pulmonary edema
Pulmonary hemorrhage
Trauma
Oxygen toxicity
Other
Pneumothorax
Hemothorax
Diaphragmatic eventration
Pneumomediastinum
Severe kyphoscoliosis

Diagnosis of Acute Respiratory Failure

The criteria for diagnosis of acute potentially fatal impairment of gas exchange in the lungs varies with the type of disease. Specific criteria have not been developed for all of the causes of acute respiratory failure in the

TABLE 3. Clinical Croup Score

	0	1	2
Inspiratory breath sounds	Normal	Harsh with rhonchi	Delayed
Stridor	None	Inspiratory	Inspiratory and expiratory
Cough	None	Hoarse cry	Bark
Retractions and flaring	None	Flaring and supersternal retractions	As under 1 plus subcostal, intercostal retractions
Cyanosis	None	In air	In 40 per cent O_2

TABLE 4. Clinical Asthma Score

	0	1	2
Cyanosis (Pa_{O_2})	None (70–100 torr)	In air (≤70 torr)	In 40 per cent O_2 (≤70 torr)
Inspiratory breath sounds	Normal	Unequal	Decreased to absent
Use of accessory muscles	None	Moderate	Maximal
Expiratory wheezing	None	Moderate	Marked
Cerebral function	Normal	Depressed or agitated	Coma

pediatric age group, but several scoring systems and lists of criteria can serve as guides in evaluating patients.

A clinical scoring system has been evolved to grade the severity of upper airway obstruction associated with croup due to viral infection (table 3). A score of 4 or more indicates moderately severe airway obstruction that may benefit from inhalation of racemic epinephrine. A score of 7 or more, particularly when associated with $Pa_{CO_2} \geq 45$ torr and $Pa_{O_2} \leq 70$ torr (breathing room air), persisting for more than 30 minutes despite therapy with intravenous fluids, mist, and epinephrine aerosol, indicates the need for an artificial tracheal airway.

A clinical asthma score, devised as a means of assessing the clinical severity of status asthmaticus in infants and children, is outlined in table 4. This score correlates significantly and directly with Pa_{CO_2} and inversely with Pa_{O_2} (in room air.)[26] In a child who has received oxygen, intravenous fluids, sodium bicarbonate, and aminophylline, a score of 5 or more with a Pa_{CO_2} of 55 torr or higher is an indication for continuous intravenous infusion of isoproterenol. A score of 7 or more with a Pa_{CO_2} of 75 torr or higher despite intravenous administration of isoproterenol is an indication for tracheal intubation, mechanical ventilation, and continuous neuromuscular blockade.

In cases of children who have other forms of acute pulmonary disease, the general criteria outlined in table 5 can be used to diagnose acute respiratory failure. In cases of infants and children with infectious polyneuritis (Guillain-Barrè syndrome) the

TABLE 5. Criteria for Diagnosis of Respiratory Failure in Infants and Children with Acute Pulmonary Disease

Clinical
Decreased or absent inspiratory breath sounds
Severe inspiratory retractions and use of accessory muscles
Cyanosis in 40 per cent ambient oxygen
Depressed level of consciousness and response to pain
Poor skeletal muscle tone
Physiologic
$Pa_{CO_2} \geq 75$ torr
$Pa_{CO_2} \leq 100$ torr in 100 per cent oxygen
Three clinical and one physiologic criteria = acute respiratory failure

criteria outlined in table 6 can prove effective in the diagnosis of impending respiratory failure and determination of the need for an artificial tracheal airway and mechanical ventilation.

Treatment of Acute Respiratory Failure

Initial Therapy.

Once the diagnosis of acute respiratory failure is made, steps should be taken immediately to improve pulmonary gas exchange. Evidence of severe hypoxemia, bradycardia, or systemic arterial hypotension calls for the use of 100 per cent oxygen by bag and mask with assisted ventilation, aspiration of upper-airway secretions, establishment of an adequate intravenous route with a plastic cannula, monitoring of precordial heart tones and electrocardiogram, and use of intravenous sodium bicarbonate to restore the *p*H of arterial blood to 7.20 or higher.

Consideration should be given to forms of therapy that may substantially improve gas exchange without an artificial tracheal airway or mechanical ventilation. In children who have severe upper airway obstruction from croup, inhalation of nebulized racemic epinephrine (0.25 per cent) by mask for 10 minutes every 2–4 hours has reduced the need for tracheal intubation and tracheostomy.[27] In 27 of 30 infants and children with status asthmaticus and Pa_{CO_2}'s between 55 and 100 torr after administration of epinephrine, aminophylline, and corticosteroids, intravenous infusion of isoproterenol in high doses (to as much as 3.6 μg/kg/min) decreased bronchospasm and resulted in significant reductions in Pa_{CO_2}.[28] This technique, however, has resulted in one instance of multifocal ventricular arrhythmia and four instances of severe hypoxemia, requiring more than 70 per cent inspired oxygen for correction. Thus, these children require care in an intensive care unit with ECG monitoring and an indwelling arterial catheter for repeated arterial blood sampling. However, the hazards of intravenous infusion of isoproterenol are less than those associated with mechanical ventilation in status asthmaticus.

Unless a dramatic improvement in ventilation and blood-gas tensions occurs as a result of the immediate resuscitative measures or other therapy, an orotracheal tube should be inserted and the lungs ventilated with 100 per cent oxygen. Orotracheal-tube dimensions appropriate for various ages are presented in table 7. Most of these patients have retained gastric secretions or food in their stomachs, and must be treated as such when inserting

TABLE 6. Criteria of Respiratory Failure in Infants and Children with Infectious Polyneuritis

Clinical
Weak to absent cough reflex
Incompetent swallowing mechanism
Weak to absent gag reflex
Physiologic
Vital capacity ≤ 12 ml/kg
$Pa_{O_2} \leq 70$ torr (in air)
Two clinical and one physiologic criteria = impending respiratory failure

TABLE 7. Pediatric Endotracheal-tube Dimensions*

Age	French Size	Internal Diameter (mm)	Length (cm)	15-mm Male Connector Size (mm I.D.)
Newborn (≤1.0 kg)	11–12	2.5	10	3
Newborn (≥1.0 kg)	13–14	3.0	11	3
1–4 months	15–16	3.5	11	4
4–8 months	17–18	4.0	12	4
8–12 months	19–20	4.5	13	5
12–36 months	21–22	5.0	14	5
3–4 years	23–24	5.5	16	6
5–6 years	25	6.0	18	6
6–7 years	26	6.5	18	7
8–9 years	27–28	7.0	20	7
10–11 years	29–30	7.5	22	8
12–14 years	32–34	8.0	24	8

* Clear polyvinylchloride endotracheal tubes that satisfy the U.S.P. standard implant test† and lightweight nylon connectors are recommended.

† Guess WL: Tissue testing of polymers. Int Anesthesiol Clin 8:787–804, 1970.

the tracheal tube. Following orotracheal intubation and ventilation with oxygen, secretions in the trachea and bronchi should be aspirated, utilizing sterile techniques. If the patient's general condition permits, chest physiotherapy and tracheobronchial toilet performed with the patient in both lateral positions will often reinflate atelectatic segments.

Tracheal Airway

Nasotracheal intubation and tracheostomy are elective procedures, to be accomplished after initial orotracheal intubation. If an artificial tracheal airway will be needed for longer than 12 hours, but less than 5 to 7 days, we prefer a nasotracheal rather than an orotracheal tube. A properly placed nasotracheal tube provides more stable fixation, less danger of accidental extubation, fewer oropharyngeal secretions, and permits care of the mouth and oropharynx. With rare exceptions, a tube of the same diameter used for the oral route can be inserted nasally. Cuffed tubes are not necessary in infants and small children, whose narrow subglottic tracheal diameters insure an adequate tracheal seal. Unless very high pressures are necessary for adequate mechanical ventilation, a tracheal tube permitting a slight leak at 40 cm H_2O airway pressure should be used to minimize trauma to the subglottic area. Children more than 9 years old may require tubes with low-pressure, large-volume cuffs when high airway pressures are necessary to provide adequate alveolar ventilation (*e.g.*, in status asthmaticus). Once the nasotracheal tube has been inserted, a roentgenogram of the chest should be obtained to exclude bronchial intubation and pneumothorax. The tube should be fixed by careful application of waterproof adhesive tape after drying and preparation of both tube and skin with tincture of benzoin. Meticulous attention to these details will protect the patient against the disaster of accidental extubation.

Tracheostomy is preferable to nasotracheal intubation when an artificial tracheal airway is needed for more than seven days, or in cases of intrinsic laryngeal disease, extremely thick tracheal secretions, or pulmonary hemorrhage with blood clots. Appropriate surgical techniques for tracheostomy in infants and children have been described.[29] Experience with polyvinyl pediatric tubes,§ proper humidification, and airway care have made metal tubes with inner cannulas unnecessary. A flexible attachment¶ from the tracheostomy tube to a ventilator or T piece is essential. Cuffed tracheostomy tubes are rarely necessary in the care of infants and children. If a cuff must be used, it should be a low-pressure, high-volume cuff inflated to the minimal pressure required to insure a satisfactory seal. Cuffed tracheostomy tubes can lead to severe tracheal stenosis, and their use in pediatric patients should be discouraged.

Airway Care

An artificial tracheal airway in a child requires expert care. Inspired gas should be humidified to provide 35 to 44 mg of water vapor per liter of gas flow at 37 C (80 to 100 per cent relative humidity) in the trachea. Most commercial humidifiers fail to accomplish this; therefore, instillation of sterile saline solution (1 to 3 ml) at hourly intervals is necessary to prevent occlusion of the tracheal airway and bronchi by dried sections. Secretions in the smaller bronchi and bronchioles can be mobilized and moved toward the trachea by changes in the patient's position, and chest vibration and percussion every one to two hours. These maneuvers are followed by sterile tracheal aspiration utilizing a molded-tip, end-hole catheter (Argyle Aero-Flo) inserted to the maximum depth. Artificial coughing, a technique involving manual inflation of the lungs followed by application of manual pressure to the thorax and sudden deflation of the lungs by release of pressure in the airway, can be very useful in raising thick secretions. Before and after tracheal aspiration, the patient's lungs should be intermittently inflated to a volume near vital capacity with oxygen for a period of 1 to 3 minutes to protect against hypoxemia and to re-expand atelectatic segments.

§Aberdeen pediatric tubes, Harlake Co., Cleveland, Ohio.

¶Pediatric Swivel, Clarence B. Smith Company, Arlington, Massachusetts.

Continuous Positive Airway Pressure (CPAP)

In the child who has a large venoarterial shunt through unventilated but perfused segments of lung resulting in $Pa_{O_2} \leq 100$ torr at 50 per cent inspired oxygen, tracheal intubation and continuous elevation of the end-expiratory pressure to 5–10 cm H_2O above atmospheric can substantially improve arterial oxygenation. This can be applied with the patient breathing spontaneously and unassisted, or as an adjunct to mechanical ventilation. In a wide variety of patients who have diffuse alveolar collapse, continuous positive airway pressure (CPAP) or positive end-expiratory pressure (PEEP) during mechanical ventilation has been shown to increase functional residual capacity and decrease venoarterial shunt, apparently resulting from the inflation of previously collapsed alveoli, which can then participate in gas exchange throughout the ventilatory cycle.[30] In normovolemic children, CPAP causes minimal or no depression of systemic arterial pressure. With CPAP many patients develop an increase in Pa_{O_2} that permits reduction of the inspired oxygen concentration to lower and potentially less toxic levels.

Mechanical Ventilation

Once the tracheal airway has been established and secretions removed, mechanical ventilation can be instituted. A mechanical ventilator simply replaces the bellows function of the diaphragm and thoracic wall muscles. Positive-pressure mechanical ventilation, when properly applied, also tends to improve the distribution of gas within the lung and expand atelectatic segments. The ultimate test of a mechanical ventilator is its ability to provide adequate ventilation under conditions of increased airway resistance and decreased lung compliance. In the presence of adverse changes in pulmonary mechanics, volume preset ventilators are far more effective than those that are pressure-preset.[31] Although pressure-preset ventilators can compensate to a limited extent for minor leaks in the tracheal airway, this advantage is of minimal importance when the proper-sized airway has been inserted. The assist mode is provided with most pressure-preset ventilators, but is of minimal practical value in infants and small children. The volumes that an infant must inspire in order to trigger the device vary widely from model to model.[32] In the management of acute respiratory failure in infants and children, including the period when the patient is being "weaned" from the ventilator, the control mode has proven effective and adequate.

A major problem associated with the use of most commercially available volume-preset ventilators in infants and children is their large internal compliance (compression volume).[33] Internal compliance can be defined as the volume of gas compressed in the ventilation system per unit mean airway pressure; this gas does not participate in the minute ventilation of the patient. For example, if the internal compliance is 4 ml per cm H_2O pressure, and an infant is ventilated with a tidal volume that results in a mean airway pressure of 15 cm H_2O, 60 ml of gas are compressed within the ventilator tubing, humidifier, water trap, and alarm system. Thus, a volume setting of 60 ml would result in virtually no inspired gas passing through the tracheal tube to the patient's lungs. The ventilator volume setting must be increased to compensate for this internal compliance. Apparatus deadspace has received considerable attention in the past, but is not of critical importance in clinical care. Apparatus deadspace may be 10 ml or more, yet is readily compensated for by a small increase in the tidal volume. The patient's minute volume requirement may be difficult to predict unless the amount of wasted ventilation is known. Frequently one must rely on visual and auscultatory evidence of ventilation during the initial adjustments, and readjust the tidal volume and respiratory frequency according to serial Pa_{CO_2} and Pa_{O_2} determinations. Initial tidal volumes of 10 to 15 ml/kg at a respiratory frequency approximately two thirds normal for the patient's age will serve to correct arterial blood-gas tensions toward normal.

Coordination of the patient with the mechanical ventilator can often be achieved

merely by providing adequate alveolar ventilation and oxygenation. Diazepam (0.1 mg/kg intravenously) or morphine (0.1 mg/kg intravenously), in repeated doses or continuous infusion, will produce adequate sedation and depression of the respiratory drive in the restless infant or child. In patients who have severe bronchospasm and air trapping (*e.g.*, status asthmaticus or bronchiolitis) or in whom severe pneumonia has resulted in a marked reflex tachypnea, neuromuscular blockade by continuous infusion of pancuronium provides minimal chest-wall resistance to ventilation, and appears to reduce the hazards of pneumothorax or pneumomediastinum. When muscle relaxants are used, the patient must be cared for by personnel who have considerable experience in handling apneic, paralyzed infants and children. We prefer the continuous infusion of sedatives and neuromuscular blockers for evenness of effect. However, infusions should be stopped daily and not restarted until signs of recovery from pharmocologic effects are evident.

Discontinuing Mechanical Ventilation

A program for discontinuing mechanical ventilation in stages can generally be initiated when the Pa_{CO_2} remains less than 50 torr and Pa_{O_2} more than 100 torr at 50 per cent inspired oxygen concentration with peak inflating pressures less than 25 cm H_2O. In children who have cyanotic congenital heart disease, a Pa_{O_2} of more than 35 torr appears adequate. Unfortunately, there are no reliable physiologic criteria by which one can predict whether a patient can definitely have ventilator support withdrawn. For that reason, and not because of psychic dependence on the ventilator, a program of intermittent mandatory ventilation (IMV), or progressively longer intervals without mechanical ventilation, usually must be carried out. IMV[34] is a technique in which the gas flow through the ventilator system is sufficient to prevent rebreathing during spontaneous ventilation. Mandatory lung inflations provided by the ventilator at preset tidal volumes are delivered 1–20 times per minute, depending on the patient's ability to maintain adequate minute ventilation. During this period frequent determinations of arterial blood-gas tensions and *p*H and daily roentgenograms of the chest are essential. A progressively rising Pa_{CO_2} or falling Pa_{O_2} even though not to levels considered diagnostic of respiratory failure, indicates the patient has not recovered sufficiently, and continued mechanical ventilation is necessary. Clinical evidence of excessive work of breathing, tachypnea, and tachycardia frequently are seen prior to changes in blood gas tensions, and in themselves may be an indication for further ventilatory support.

Once the patient can tolerate spontaneous ventilation for at least 12 to 24 hours, consideration should be given to removal of the tracheal airway. It is preferable to maintain 2–3 cm H_2O CPAP while the tracheal tube is in place to achieve optimal Pa_{O_2} levels. Extubation should not be done until secretions are minimal and of thin consistency, the chest roentgenogram shows continuing improvement, and the patient's general condition is stable. For at least 12 hours prior to and 24 hours after removal of nasotracheal tube, oral intake should be restricted and hydration maintained with intravenous fluids. Because of the temporary incompetence of the larynx and impaired subglottic ciliary function following removal of a nasotracheal tube, tracheobronchial suction with a sterile catheter under direct laryngoscopy may be needed once or even twice daily for one or two days.

Supportive Therapy

Normal body temperature should be maintained by utilizing automated heating and cooling blankets, as well as vasodilating drugs such as chlorpromazine. Appropriate intravenous fluids and electrolytes, calories to prevent further catabolism, and antibiotic therapy for infection are essential to the recovery of the child with respiratory failure. Caloric deprivation is a common consequence of critical illness. Since 5 per cent glucose, intravenously, can provide only a fraction of the basal caloric needs, supplementary gastrointestinal feedings or intravenous hyperalimentation must be

utilized. Careful attention to washing and general care of the skin, frequent changes of position, passive range of motion exercises, and judicious use of orthopedic splints will prevent skin breakdown and contractures.

COMPLICATIONS

Airway Complications

Accidental tracheal extubation or occlusion of the lumen of the tracheal airway by secretions usually can be prevented by attention to the details of airway care (as described above), and proper humidification of the inspired gas. Prolonged nasotracheal intubation has been associated with post extubation subglottic stenosis, granuloma formation, and fibrotic bands,[35] although the incidences vary considerably with different diseases and from one institution to another. Subglottic obstruction has been reported to occur in 3 to 5 per cent of children intubated for more than 24 hours. These subglottic complications cause partial upper-airway obstruction, and may appear as late as six weeks after extubation. However, with the use of polyvinylchloride tubes that are not sterilized with ethylene oxide, atraumatic insertion, and an appropriate tracheal fit, such complications have become relatively rare.[36]

Tracheostomy may lead to granuloma formation distal to the end of the tracheostomy tube and at the cephalad margin of the tracheostomy incision. Difficulties in extubation of small infants following tracheostomy can result from instability of the anterior tracheal wall. Tracheal stenosis secondary to tracheostomy is extremely rare in infants and children because cuffed tracheostomy tubes are not ordinarily necessary and the surgical technique does not involve excision of cartilage.

Pulmonary Complications

The major intrapulmonary complications are caused by retention of tracheobronchial secretions, contamination of the airways with pathogenic organisms,[37] overdistention of the lungs associated with maldistribution of gas at high airway pressures, excessive pulmonary extravascular water,[38] and oxygen toxicity.[39] Sudden deterioration in the circulatory status or blood-gas tensions in a patient receiving mechanical ventilation necessitates an immediate roentgenogram of the chest to exclude tension pneumothorax. The hazard of pneumothorax probably is increased when the peak airway pressure exceeds 40 cm H_2O or when CPAP exceeds 10 cm H_2O. In a number of children undergoing prolonged mechanical ventilation, we have seen decreases in urinary output and water retention, with or without roentgenologic evidence of interstitial pulmonary edema, and decreasing Pa_{O_2}'s similar to those observed in adults. Fluid restriction and diuretics such as furosemide will often result in a brisk diuresis, an increase in Pa_{O_2}, and radiographic evidence of improvement. Most of these patients also require large tidal volumes and CPAP to maintain an optimal Pa_{O_2}. The etiology of the fluid retention remains obscure. Ultrasonic nebulizers should not be used with mechanical ventilation because they can provide an excess volume of water,[40] resulting in uptake of an unpredictable volume of water from the lungs.

Oxygen has the ability to damage human alveolar tissue with prolonged exposure at high inspired concentrations.[41] Exposure to concentrations of less than 50 per cent for many days does not appear to harm the normal human lung,[41] but the effect on diseased lungs remains uncertain. The most sensitive indicator of impaired lung function due to oxygen toxicity is a decrease in Pa_{O_2} during breathing of 100 per cent oxygen.[42] Arterial hypoxemia does not appear to protect the lungs against injury from high alveolar oxygen tensions.[43] However, fear of pulmonary oxygen toxicity should not preclude the use of sufficiently high inspired oxygen concentrations to maintain the Pa_{O_2} at a level compatible with adequate oxygenation of the brain and other organs. The term "respirator lung" has been incorrectly applied to fatal pulmonary oxygen toxicity or to other pulmonary conditions.[44] Mechanical ventilation itself, when properly applied to normal mammalian lungs, does not result in serious physiologic dysfunction or histologic damage.[45]

Infection

Infection poses one of the major risks involved in the treatment of acute respiratory failure. Trauma to the tracheal mucosa associated with catheter aspiration[46] and inadvertent breaks in aseptic technique account for the high incidence of tracheitis, associated with pathogenic bacteria found on smear and culture.[47] However, because of the danger of emergence of virulent gram-negative pathogens or antibiotic-resistant strains, we do not advocate the use of antibiotics unless there is evidence of associated bronchopneumonia,[37] fever not readily explained by other causes, leukocytosis, or the development of copious, thick, purulent secretions. Gram-negative septicemia often occurs in critically ill patients in whom the airway and multiple intravascular catheters represent the probable routes of contamination, and the hands of professional personnel the major vector.[47] Careful hand washing with a bactericidal agent by physicians and nurses, limited examination of the patient by personnel not immediately involved with patient care, strict aseptic practice in the handling of the patient's airway and vascular catheters, frequent changing or chemical decontamination of respiratory therapy equipment, proper care of the patient's skin, and the meticulous handling of airway connections can substantially reduce the incidence of infection.

Mechanical Complications

Complications associated with mechanical ventilation can be virtually eliminated by detailed attention on the part of the nurses and physicians to the ventilator system itself, and by the use of reliable, tested equipment. Mechanical complications are quite common when a team of physicians and nurses first embarks on a program of intensive respiratory care; as they gain experience, these become much less frequent, and the complications mentioned previously emerge as the most difficult problems to solve.

Other Complications

The circulatory consequences of mechanical ventilation are seldom a serious problem in infants and children. Hypocapnia ($Pa_{CO_2} \leqslant 28$ torr) and alkalosis ($pH \geqslant 7.45$) may decrease cerebral blood flow[48] and cardiac output,[49] although quantitative data in infants and children are lacking. Our clinical experience indicates that induced respiratory alkalosis produces little harm, even when tetany has occurred, whereas inadequate alveolar ventilation has dire consequences. Overheating of inspiratory gas resulting in fever, and muscular work associated with discoordination of the patient and ventilator, will increase the patient's metabolic rate. Vigorous treatment of fever, regardless of the cause, and careful attention to coordination of the patient with the ventilator serve to minimize metabolic stress.

Results of Pediatric Intensive Care

A survey of eight major pediatric intensive care units admitting all types of critically ill infants and children indicates an average survival rate of 88 per cent.** The detailed statistics for survival rates associated with acute respiratory failure from various causes are not available. In the Pediatric Intensive Care Unit at the Children's Hospital of Philadelphia, acute respiratory failure from all causes treated with mechanical ventilation was associated with a 64 per cent survival rate. The major causes of death were irreversible central nervous system damage and irreparable congenital heart disease. In patients with respiratory failure (mean Pa_{CO_2} 103 torr) due to status asthmaticus requiring mechanical ventilation, there was one death in 37 episodes, a mortality rate of less than 3 per cent. Intensive care services are extremely expensive and should not be duplicated in every hospital. Therefore, a pressing need for regional distribution of Pediatric Intensive Care Centers exists. These centers should be serviced by an effective and safe transportation network to facilitate the transfer of patients from other hospitals. Only with this type of system can we provide every critically ill or injured child an optimal chance for full recovery at the least cost to his family and community.

**Downes JJ: Personal communication.

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