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Respiratory failure in newborns, infants and children

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An atmosphere containing sufficient 0_2 and relatively free of CO_2 is essential for human survival. Following birth, the adaptation of the human organism from intra-uterine to this environment must occur quickly and normal function requires an intact ventilatory pathway[1] and profound physiological changes.[2] Abnormal intra-uterine development or lack of appropriate physiologic responses can result in ventilatory inadequacy. A deleterious influence (e.g. absence of surfactant, sepsis, maternal smoking, meconium aspiration) before, during or after birth can also interfere with normal respiratory function. Similarly genetic predisposition (as in cystic fibrosis, immotile cilia syndrome) can lead to respiratory failure early in childhood.[3]

This article will briefly discuss the ventilation modalities available in the management of respiratory failure in newborns, infants and children; its recognition and management in this age group along with a concise review of the vicious cycle of circulatory and metabolic changes accompanying neonatal res-

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Abbreviations used

O_2 : oxygen
CO_2 : carbon dioxide
$AaDo_2$: alveolar arterial difference for oxygen
$PaCo_2$: arterial Co_2 tension
PaO_2 : arterial oxygen tension
V/Q : ventilation perfusion ratio
V_D : dead space (anatomic)
V_T : tidal volume
Qs/Qt : pulmonary R-L shunt
RDS : respiratory distress syndrome
CMV : continuons mechanical ventilation
pH : negative logarithm of the hydrogen ion concentration
CDP : continuous distending pressure
CPAP : continuous positive airway pressure
NCDP : nasal CDP
F_IO_2 : fraction of inspired oxygen
CF : cystic fibrosis
PEEP : positive end expiratory pressure
IMV : intermittent mandatory ventilation
I/E : inspiratory expiratory time ratio
IC : inspiratory capacity
MIP : maximum inspiratory pressure
HFPPV : high frequency positive pressure ventilation
HFO : high frequency oscillation
HZ : hertz
SIMV : synchronized intermittent mandaoryt ventilation
IDV : intermittent demand ventilatian

piratory failure; a brief discussion on the 'pros' and 'cons' of intubation vs tracheostomy; and an update of the recent advances in the management of respiratory failure.

Ventilation modalities

The mechanical treatment of respiratory failure in children has advanced rapidly during the past 10 years. The early limited and questionably efficacious use of continued mechanical ventilation utilizing adult ventilators has given way to judicious employment of several different mechanical devices to assist ventilation under selected circumstances. Partially because of these newer techniques and partially because of increased understanding of infant cardiopulmonary physiology, the mortality and morbidity of respiratory failure in children and especially in newborns have improved markedly. In this section we will discuss in a necessarily cursory fashion the newer concepts of ventilation in children.

One must recognize at the outset that up to age eight years, children are not just small adults. This is true particularly for pulmonary anatomy and physiology. The difference between infants and adults is perhaps best demonstrated by the increased A-aDO_2 and lower $PaCO_2$ in the infant reflecting less effiicient matching of ventilation and perfusion. Accordingly, management of respiratory failure in infants and children requires two things: specialized knowledge and specialized equipment. Need for the former is self-evident. For the latter equipment with a minimum dead space, minimum resistance, maximum accuracy and sensitivity is needed.

In children as in adults, most abnormalities of gas exchange relating to pulmonary dysfunction (as distinguished from cardiac shunting) result from mismatching of ventilation and perfusion. Abnormal diffusion across the alveolar capillary membrance appears to be much less common.

Ventilation/perfusion abnormalities in infants are a result of pathopysiologic changes in perfusion as well as in ventilation in contrast to the typical picture in adults where perfusion abnormalities play a lesser role. The increased contribution of perfusion problems in the neonate is the result of the exquisite sensitivity of pulmonary vasculature to changes in hydrogen ion and oxygen tension. This section, however, will focus only on manipulations of ventilation.

The objective of all the various manipulations we well discuss is to minimize wasted ventilation (high V/Q, high V_D) and wasted perfusion (low V/Q, increased Q_s/Q_t). Wasted perfusion is usually associated with regional volume loss (atelectasis) which results from a variety of causes including extremely low compliance as seen in RDS, airway obstruction noted with meconium aspiration, and suboptimal V_T. Many of the commonly used therapeutic modalities are aimed directly at treating and preventing this volume loss. When being mechanically ventilated, infants as well as adults should be ventilated at a V_T of approximately 10-15 ml/kg. This 'continuous sighing' technique offsets the less optimal distribution of ventilation associated with positive pressure ventilation and helps to treat and prevent atelectasis. Ventilatory rate is manipulated to maintain normal

pH; the rate tends to decrease with age as matching of ventilation to perfusion becomes more effective.

Continuous mechanical ventilation: Although mentioned first here, CMV is parhaps the least desirable of the various modalities used in children; it is mentioned first primarily because it was the first therapy attempted. CMV has the disadvantage of requiring intubation of the trachea, increased risk of barotrauma and circulatory side effects. Currently CMV is utilized in children requiring muscle relaxants during surgery, for ventilatory failure ($pH < 7.25$, $PCO_2 > 60$ torr), for elevated intracranial pressure, following cardiac arrest, or where the work of breathing is excessive (very high resistance, very low compliance).

Continuous distending pressure (CDP). It rapidly became obvious that many infants, especially those with RDS, had much less problem of air into and out of the lung than they did with matching ventilation and perfusion. Recognizing this, Gregory, et al.,[4] introduced the concept of CDP (CPAP, PEEP) to increase FRC. With CDP, the airway is subjected to a supra-atmospheric pressure usually expressed as cm H_2O above atmospheric pressure. In this way, an attempt is made to recruit atelectatic alveoli and then to hold them open, thus allowing improved matching of ventilation and perfusion. Because neonates are obligate nose breathers until about six weeks of age, CDP can be applied without endotracheal intubation by means of nasal prongs or a nasopharyngeal tube, thus allowing for non-invasive therapy of hypoxemia in the absence of ventilatory failure and respiratory acidosis; it may also be utilized with endotracheal tubes. CDP is not without its hazards, the most prominent of these being barotrauma and cardiovascular side effects. One must keep in mind that the areas of lung most affected by CDP will be the relatively normal areas. Accordingly, these portions of the lung may become over-distended and rupture creating a pneumothorax and other forms of barotrauma. The use of CDP may also cause an elevation of intrathoracic (pleural) pressure which in turn will decrease venous return and eventually cardiac output. Fortunately, the cardiovascular effects of CDP are directly related to pulmonary compliance: the sicker the lung, the less the effect on intrathoracic pressure.[5]

A CDP system should be set up in such a way that ventilatory work is minimized and yet flows and volumes are optimal and $F_I O_2$ constant. Two types of systems are available—those which utilize demand valves and those which utilize one-way valves and reservoirs. Although demand valves have significant advantages, they are more expensive. With either system, an oxygen mixing device is necessary because the back pressure will cause significant fluctuations in the oxygen concentration delivered by a Venturi device. The supra-atmospheric pressure may be exerted by a threshold resistor or by a flow resistor. The threshold resistor (Fig 1), the most common example of which is a wide-bore exhalation tube placed under a pre-determined depth of water, allows normal expiratory flow rates and simply elevates the pressure at which the respiratory system operates. A flow resistor (Fig 1) decreases the expiratory flow rate (expiratory retard); this may have some advantages in RDS where the absence of surfactant results in very high elastic recoil of the lung and a tendency to airway collapse; it has no part, however, in the treatment of patients with intrinsic increases in airway resistance (asthma, CF) because of the tendency towards gas trapping. For mechanical details of these systems, the reader may refer to

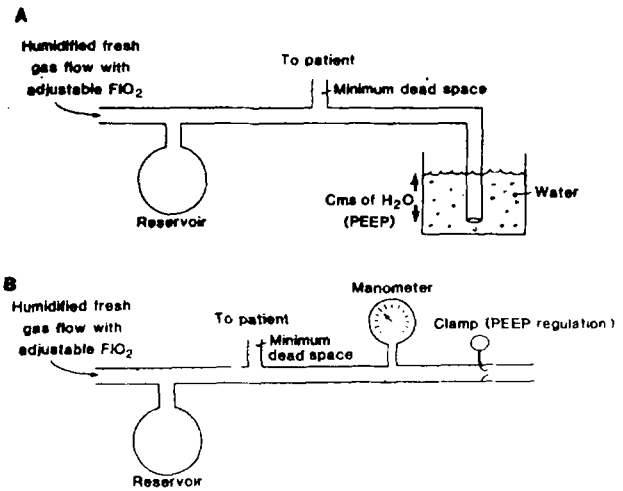

Fig. 1. Diagram showing apparatus used to deliver continuous positive airway pressure by means of either a threshold resistor (as shown in A) or a flow resistor (as shown in B).

Graybar and Smith.[6] Less commonly, the thorax is closed by subatmospheric pressure[7] (a Cuirass or an Ohio incubator ventilator); pleural pressure is lowered by decreasing the recoil of the chest wall thereby increasing transpulmonary pressure.

Positive end expiratory pressure (PEEP). It is important to recognize that PEEP and CDP are not equivalent in the spontaneously breathing patient. PEEP is applied only during expiration whereas CDP is applied throughout the ventilatory cycle, usually by supplying gas at flow rates greatly in excess of the patient's inspiratory flow (200-300 liters/minute). Because PEEP appears to increase the work of breathing in spontaneously breathing patients, it is not commonly used for CDP in infants and children. This is not true, when PEEP is applied during positive pressure ventilation.

Not only may excessive CDP have significant cardiovascular side-effects and barotrauma but it may paradoxically increase intrapulmonary shunt. This occurs because over-distension of the more normal portions of the lung may by a Starling resistor effect (Figure 2) decrease blood flow around these alveoli and redistribute the flow to atelectatic lung, another example of "too much of a good thing."

Intermittent mandatory ventilation (IMV). Not all infants with abnormalities of gas exchange are capable of adequate ventilation or $C0_2$ elimination. As noted above, initially the only option for treatment of this problem was with CMV. However, more recently, the focus has been made on supplementing spontaneous ventilation with such additional mechanical ventilation as necessary to maintain a normal pH. This is called IMV. Most modern ventilators have built-in-circuitry which allows IMV; however, when these are not available an IMV circuit is relatively simple to construct (Fig. 3).

Although several commercial IMV systems are set up such that the mechanical breath is synchronized with the patient's inspiration (IDV, SIMV) there is no evidence that this synchronization is beneficial.[6] Superficially, IMV may appear to offer several advantages over CMV. Even though it shares with CMV the necessity for endotracheal intubation, IMV is thought to have less cardiovascular side-effects, to have a lower incidence of barotrauma, to make weaning easier by allowing a gradual withdrawal of mechanical support, and it may allow for better

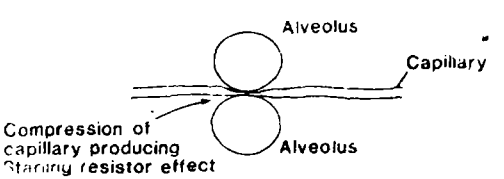

Fig. 2. Diagrammatic demonstration of the Starling resistor effect.

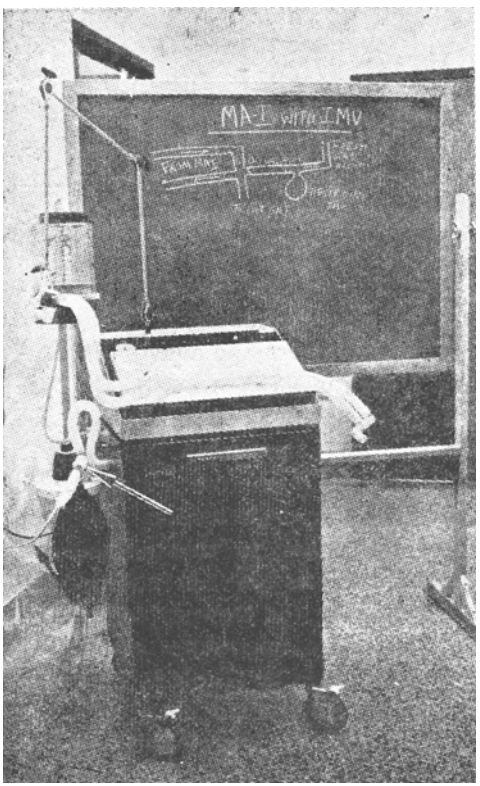

Fig. 3. Photograph of the MA-1 adult respirator demonstrating the incorporation of the IMV circuit. A new source of fresh gas flow (as indicated by the small arrow) is used which leads to a reservoir bag. This is connected to the inspiratory limb of the tubing coming from the machine by a 'T' piece (large arrow). A low resistance one-way valve prevents back flow into the reservoir bag. This arrangement has been used with some success in adult patients and a similar principle may be utilized in pediatric ventilators with minimum compliance tubing providing a higher accuracy of volumes and pressures.

control of acid-base parameters. It is by on means certain, however, that all of these benefits accrue with IMV, especially the latter. Nevertheless, IMV is being utilized increasingly. It would appear that as with CMV, the design and mechanics of the system are of paramount importance. Special attention is required to ensure minimal resistance, adequate flows, accurate inspirated oxygen concentration and precise control of mechanical volumes, As with CMV, IMV is usually utilized in infants and childern with CDP to maintain FRC during spontaneous breaths and between breaths. The amount of positive pressure required is determined by the PaO_2, F_IO_2 and the cardiovascular side-effects. In the event that cardiovascular side-effects become a limiting factor, the tendency now is to support cardiac output with volume or inotropic agents, and to use sufficient CDP to achieve a satisfactory PaO_2.

One effect of lung disease commonly found in neonates, such as in meconium aspiration, is mal-distribution of ventilation. Because the distribution of inspired gas during CMV is determined by regional resistance and compliance, ventilation will be least in those areas of the lung where resistance is highest and compliance lowest. Because resistance is increased by turbulent flow, *decreasing flow rates* might have a beneficial effect on ventilation distribution and this has been found to be true. Although Cournand[8] in early work on positive ventilation found that cardiovascular side-effects increased proportionately with inspiratory time, subsequent work has shown that side effects are lower in the presence of lung disease and the cause of these side-effects can be overcome, as noted previously, when potential benefits outweigh the isks. An extension of the slowinspiratory rflow rate is *the inspiratory pause or inspiratory hold*, a maneuver by which the lung is held at end-inspiration for a pre-

determined period of time with each breath. During this pause, redistribution of volume will occur between over-distended regions of the lung with low time constants (low resistance, low to normal compliance) to poorly ventilated regions with high time constants (high resistance, normal to high compliance). In pediatric patients especially in newborns, the results of this "increased I/E" can be very dramatic with improvement in both shunt and dead space; similar results have not been seen in adults. Although almost all ventilators allow for variability of inspiratory flow rates, only the more advanced have a mechanism for use of inspiratory hold. Because of the high possibility of serious side-effects, this therapeutic modality should be used only under careful observation.

Management of respiratory failure in newborns and infants

Respiratory failure may be *defined* as the inability of the pulmonary system to meet the metabolic demands of the body. With some variations for barometric pressure, a PaO_2 <50 torr (F_1O_2=0.21) and/or a $PaCO_2$ >50 torr with pH below 7.25 without an intracardiac shunt are indicative of respiratory failure. In newborns and infants, the causes for this inadequacy of gas exchange are numerous and well described.[1-3,9]

As with most diseases, *early recognition* of respiratory failure is mandatory for proper treatment and a favorable outcome. A high index of suspicion is necessary for diagnosis. All newborns of mothers with complicated pregnancies and/or labor, premature gestation, or delivered by Caesarian section should be carefully examined to rule out neonatal ventilatory depression. In addition to the Apgar score, the Downes' score[10] is a useful index to quantitate respiratory failure at the bedside (Table I). However, it should be emphasized that signs and symptoms alone are inadequate for accurate assessment and arterial or arterialized capillary blood gases must be drawn in these patients.[1,2,9,11,12] A chest

Table 1. Factors Used in the Bedside Evaluation of Respiratory Status. (Downes' Scoring System)*

Score	Respiratory rate (per minute)	Cyanosis	Retraction	Grunting	Air entry** (crying)
0	60	None	None	None	Clear
1	60-80	Present during air breathing	Mild	Audible with stethoscope	Decreased or delayed
2	>80 or apneic episode	Present when $F_1O_2 \geq 0.4$	Moderate to severe	Audible without stethoscope	Barely audible

* See reference 10 for details.

** Quality of inspiratory breath sounds in the mid-axillary line.

x-ray is also essential to aid in the diagnosis and follow-up.

Once the diagnosis is established, *therapy* is aimed at promoting optimal gas exchange, preventing hypoxemia and hypercapnia, and allowing recovery of the primary pathologic process responsible for the ventilatory failure.

For purposes of simplicity, respiratory failure in this age group may be divided into mild, moderate and severe (Table II). *In mild disease*, the baby is able to breathe spontaneously with minimal respiratory distress and needs only oxygen therapy with an $F_IO_2 < 0.5$ to maintain an abdominal aortic PaO_2 of 60-80 torr. At this aortic PaO_2, the PaO_2 in the retinal artery is usually $\geqslant$ 100 torr even if there is a patent ductus arteriosus with some right to left shunting.[11]

Moderate respiratory distress is characterized by a more severe hypoxemia with $PaO_2 \leqslant$ 50-60 torr with $F_IO_2 \geqslant$ 0.5-0.6. There is usually no CO_2 retention. This condition often can be managed by the administration of CDP via nasal prongs or nasopharyngeal tube.[4,9] CDP can also be applied effectively with a tight fitting face mask although nasal prongs are preferable. Although pressures up to 12 cm H_2O have been used with this system[9], it is preferable not to exceed 6 cm H_2O because of the risk of barotrauma and cardiovascular compromise. If the $PaO_2 \leqslant$ 50-60 torr with $F_IO_2 \geqslant 0.7$, then NCDP can be applied in increments of 2 cm H_2O and the infant's PaO_2 response observed. If PaO_2 improves dramatically, then F_IO_2 can be reduced gradually. A gradual reduction is necessary because of the increased sensitivity of the pulmonary vasculature to rapid decreases in F_IO_2. NCDP is also useful in the management of recurrent apneic spells.[2] By reducing physiologic V_D, CDP may also help to reduce $PaCO_2$ to normal levels in some babies with mild CO_2 retention.

Severe respiratory failure requires transtracheal intubation and assisted ventilation. Such patients usually have a $PaO_2 \leqslant$ 40-50 torr with $F_IO_2 \geqslant 0.7$ on CDP $\leqslant$ 6-7 cm H_2O or an $F_IO_2 = 1.0$ without CDP. These patients invariably have some CO_2 retention. The form of assisted ventilation will depend upon the severity of the disease and the period during which the severe failure is manifested. If it occurs soon after birth, as in RDS, then controlled ventilation with muscle relaxation is indicated. Pancuronium, a long-acting muscle relaxant, by

Table II. Classification of Respiratory Failure in Newborns and Infants

Type	Downes' score	$Pa0_2$	$PaCo_2$	Treatment
Mild	3-4	Decreased	Normal	0_2 therapy
Moderate	5-6	Moderately decreased	Normal to mild elevation	CPAP (CDP)
Severe	>6	Severely decreased	Usually elevated	Intubation and assisted ventilation

speculative improvement in the distribution of ventilation has been shown to improve PaO_2 and allow a reduction in F_IO_2.[13]

For purposes of *ventilation*, oral intubation is satisfactory in this age group. It is preferable to use anticholinergics (atropine or glycopyrrolate) prior to intubution to prevent bradycardia associated with airway manipulation and/or succinylcholine use. An effective V_T of 10-15 ml/kg with a rate of 25 $\pm$ 5 per minute is usuaily adequate. Both PaO_2 and $PaCO_2$ will have to be closely monitored to allow necessary adjustments in rate, V_T and F_IO_2. In the severest forms of respiratory failure in neonates, rates of upto 60-80 per minute are sometimes necessary (e.g. severe pulmonary vasoconstriction with or without HMD as dicussed in the next section). A high F_IO_2 may be necessary initially but PEEP, prolonged I/E ratio and inspiratory hold manipulations often allow this to be rapidly lowered. This again will require a close monitoring of physical status, blood gases and chest x-ray.

Weaning from respiratory assisting modalities provides a challenging opportunity in these patients. Until recently,[14-16] physical status including Downes' score,[10] blood gases and chest x-ray findings were used to reduce ventilation assistance and favor extubation. Both IC and MIP measurement have now been found to be useful to assess quantitatively spontaneous ventilatory function.[14-16] As a general rule of thumb, the first step should be to reduce F_IO_2 very gradually with ABG (or transcutaneous O_2) monitoring on an hourly basis or longer to prevent 'flip-flop'.[2] This is a serious drop in PaO_2 produced by a reduction in F_IO_2 due to an extremely sensitive pulmonary vasculature. Pulmonary vasoconstriction results and magnifies V/Q abnormalities. The early, slow reduction of F_IO_2 to minimum necessary levels will reduce both pulmonary oxygen toxicity[17,18] and retrolental fibroplasia[18]. The recent development of methods to measure continuously the PO_2 across the skin (trauscutaneous monitoring) should permit the more rational regulation of O_2 administration to small infants[18]. The next step should be to reduce levels of CDP to 2 or 4 cm of H_2O and if the patient is on controlled ventilation to gradually initiate spontaneous ventilation by changing to the IMV mode with initial respiratory rates of 18-20/minute. If the newborn or infant demonstrates an IC$\geqslant$150 ml/m^2 and MIP$\geqslant$33 torr, then the IMV rate is decreased to zero and the infant placed on endotracheal CDP of 2 cm H_2O. If IC and MIP do not meet these minimum requirements, then, along with blood gas measurement and clinical signs, IC and MIP measurements are now repeated every 2 to 4 hours. If there is progressive improvement in these parameters, then IMV rate is decreased by 2/minute sequentially. When the minimum required levels of IC and MIP are achieved, dexamethasone is administered to diminish edema associated with endotracheal intubation and the patient extubated. After extubation, recemic epinephrine may be nebulized, again to minimize subglottic edema. A chest x-ray should be taken after extubation to rule out right upper lobe collapse.[11] The F_IO_2 is increased by 10% after extubation and gradually decreased to atmospheric concentrations.

Additionally, on must *treat any extra pulmonary cause of respiratory insufficiency*, such as congenital heart disease, sepsis, or congestive heart failure. This will be discussed in the next section. Also adequate attention must be paid to caloric expenditure and replacement, tissue trauma associated with intensive care, psychologic needs, and fluid and electrolyte management. After recovery, a pulmonary rehabilitation program, similar to programs outlined for adults,[19] should be instituted if necessary.

The vicious cycle of circulatory and metabolic changes in neonatal respiratory failure[2,20,21]

During intra-uterine life, the fetal

lungs are filled with fluid and receive only 10-15% of the cardiac output. The low blood flow is a result not only of the fetal circulation, but of the high pulmonary vascular resistance resulting from vasoconstriction by muscular pulmonary arterioles. After a normal gestation and delivery, pulmonary vascular resistance decreases and by approximately seven days of age, the pulmonary arterial pressures are similar to those in the adult. Studies in fetal lambs by Cook, et al.,[20] suggest that after the first breath of the newborn, ventilation, increased PaO_2 and reduced $PaCO_2$, possibly associated with chemical mediators, were responsible for this decrease in pulmonary vascular resistance and increase in pulmonay blood flow. Both these changes in the lung and systemic circulation cause the closure of the patent ductus arteriosus and foramen ovale after birth. Gradual transition from fetal muscular pulmonary arterioles to the then adult structure follows more slowly.

However, *numerous possible factors can prevent this transition.* Consistent elevation of pulmonary venous pressure, transmission of systemic pressure to the pulmonary circuit, and condition associated with increased pulmonary blood flow from birth and low arterial oxygen tensions may dalay the maturation of the thick-walled fetal pulmonary vasculature.[21] In some infants, such as those with persistent fetal circulation, persistent pulmonary hypertension may result from pulmonary vasospasm or possibly from increased muscle mass in this vascular bed, due to increased intra-uterine asphyxial stresses. Increased pulmonary vasoconstrictor substance or decreased vasodilators such as bradykinin at the the time of birth may be contributing factors towards pulmonary vasoconstriction.[2] Pulmonary hypertension may result in persistent fetal circulation with severe hypoxemia secondary to right to left shunting at the foramen ovale and/or ductus arteriosus.[2] A vicious cycle results (Fig 4) as hypoxemia and acidosis perpetuate pulmonary vasoconstriction and ventilatory failure.

A common cause of the vicious cycle seen in the figure is *RDS.* In this disease, surfactant, a substance which decreases surface tension, functions inadequately.[2] The increased surface tension causes a decrease in lung compliance and consequently the work of breathing increases. Progressive atelectasis leads to hypoventilation and shunting, which in turn cause hypoxemia and hypercarbic acidosis which potentiate pulmonary hypertension and again result in a self-perpetuating vicious cycle (dark solid lines in Fig 4). This situation is further complicated by the fact that hypoxia and acidosis further compromises surfactant production by the lung.[2]

Blocking the vicious cycle of hypoxemia, acidosis and pulmonary hypertension is paramount in the management of respiratory distress in this age group. Other contributing factors (broken lines in Fig 4) to the vicious cycle include deviations from normal body temperature, hypoglycemia, septicemia and/or circulatory disturbances with or without shock.

High body temperature can raise O_2 consumption and CO_2 production in an infant and result in hypoxia and hypercarbia. This is not as commonly seen as is hypothermia which can cause peripheral systemic vasoconstriction and lead to tissue hypoxia with associated anaerobic metabolism and progressive acidosis. Severe hypothermia can also result in hypoglycemia from increased glucose uti-

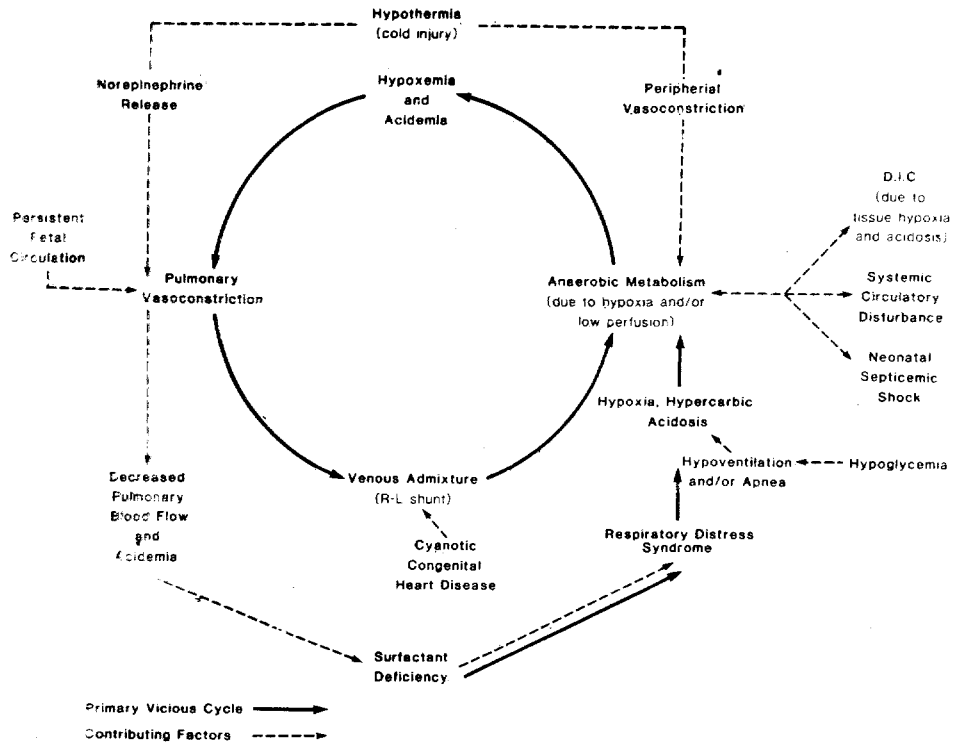

Fig. 4. Summary of the metabolic and circulatory vicious cycle associated with the newborn respiratory distress syndrome and respiratory failure.

lization, and reflex increases in norepinephrine which further complicates the vicious cycle. These problems make it essential to treat critically ill infants in a neutral thermal environment (30-33° C.) and this effort alone may be life-saving.

In addition to hypothermia, respiratory distress, perinatal asphyxia and maternal toxemia may cause transient hypoglycemia so commonly seen in the NICU which, as noted above, can accentuate the vicious cycle of respiratory distress. Hypoglycemia must, therefore, be prevented and/or treated. Dilute glucose solutions should be used to prevent a hyperosmolar load.

Hypovolemia, reduced cardiac output, CHF and pulmonary edema are commonly seen in critically ill babies. Hypovolemia, with or without shock, is often associated with the sepsis now thought to be a frequent occurrence in this age group.[22] All these obviously have a deleterious effect in the management of respiratory failure in this age group because hypoxia and anaerobic glycolysis will result in severe lactic acidosis, and DIC with a detrimental outcome.

Finally, if persistent pulmonary hypertension complicates respiratory failure, then in addition to ventilation

and oxygen therapy, pulmonary vasodilation (tolazoline) my be infused, preferably through a central vein. All drugs currently available for this purpose also cause systemic vasodilation and the baby must be observed for side-effects such as hypotension and hypertension, abdominal distention, GI hemorrhage and renal insufficiency.

Respiratory distress in children

Respiratory distress in children is usually secondary to some form of respiratory illness and may occur abruptly or insidiously depending upon the extent and rate at which the illness affects respiratory function. Respiratory distress is usually obvious clinically; the diagnosis of respiratory failure requires an arterial blood gas. By definition, a patient in ventilatory failure will have CO_2 retenwith acidosis. Although, all patients in respiratory failure breathing room air will be hypoxic,[23] there are many other causes of hypoxia. In children with normal lungs, the hypoxemia is secondary to hypoventilation (increased $PaCO_2$); in those with lung involvement, ventilation perfusion abnormalities contribute to the hypoxemia, and concomitant hypoventilation will accentuate the decrease in oxygen tension.

Anatomically, respiratory distress may be due to dysfunction of the respiratory center (bulbar tuberculosis, viral encephalitis), disease of the anterior horn cells innervating the respiratory musculature (e,g. poliomyelitis), weakness of the respiratory muscles (myasthenia gravis), or disease of the airways (e.g. bronchial asthma) or lung parenchyma (e.g. pneumonia, congenital emphysema), the latter two being most common. Parenchymal and airway disease can result in varying degrees of ventilation perfusion mismatch and hypoventilation which in turn cause hypercarbia. Uncommon causes of hypoxia in this age group are defects in diffusion across the alveolar capillary membrane as may be seen in children with bronchopulmonary dysplasias and interstitial pneumonitis.

Anatomic peculiarities of the pediatric respiratory tract predispose children to develop respiratory distress more easily than adults.[24] The small diameter of nasal cavity and airways, a relatively large tongue and a more cephalad larynx can result in airflow turbulence and increased work of breathing with minimal impediment (edema, mucous plugs, decreased ciliary function). A highly compliant chest wall and horizontally situated ribs make breathing less efficient. Although diaphragmatic fatigue is more commonly documented in neonates and infants, this entity can also exist in children. The best example of this is the hyperventilating asthmatic child with hyper-inflated lungs which put the flattened diaphragm at a mechanical disadvantage. Increased work of breathing may result in ventilatory muscle fatigue, hypoventilation and CO_2 retention. Increased oxygen consumption associated with excessive work of breathing is an additional detrimental factor in the already compromised patient.

The *clinical conditions resulting in respiratory distress* are numerous and have been differently classified by various authors.[23,25,26] In children, although respiratory failure may not be present at the onset of the illness, it is possible to

predict impending respiratory failure and treat this expectantly. Most commonly, these children suffer from bronchial, respiratory tract infections, congenital heart disease, CNS lesions, peripheral polyneuritis, foreign body aspiration, poisoning, trauma and in the western hemisphere, cystic fibrosis. Most of the conditions listed above can be treated conservatively i.e. without mechanical ventilation provided the children are closely monitored.

The main causes of *upper airway problems* in children are acute epiglottitis (supraglottic obstruction), and laryngotracheobronchitis (subglottic obstruction).[27] In supraglottic infections, an artifical airway (as discussed elsewhere in this article) to overcome the obstruction is the safest procedure.[28,29] In subglottic obstruction, unrecognized hypoxemia which can result in sudden death should be prevented. If conservative management with humidified oxygen, corticosteroids and inhaled racemic epinephrine does not improve the conditions, then a artifical airway is mandatory.[29–31]

The patient with *bronchial asthma* and impending respiratory failure needs aggressive management with hydration, systemic steroids and theophylline along with inhaled and parenteral β-adrenergic drugs.[32] In selected cases, IV isoproterenol may arrest the need for mechanical ventilation.[33,34] However, if the child shows signs of fatigue (suggested by $PCO_2 \geqslant 40$ torr, or a rise in $PaCO_2 > 5$ torr/hr) and there is no progressive improvement with this aggressive management, intubation and mechanical ventilation is indicated.

Management of the pediatric airway[35–38]

The two *indications for an artifical tracheal airway* in pediatric patients are as discussed in preceding sections, *acute respiratory failure* with the need for mechanical ventilation and *respiratory obstruction* for control and access to the airway. The most common causes of obstruction in the United States are epiglottitis, laryngotracheobronchitis and foreign bodies. Infectious disease may be more important in other populations.

Emergency care of an obstructed airway begins with the administration of oxygen. A concentration of 40-60% should be given to relieve the accompanying hypoxemia. It is the progressive hypoxemia that causes cardiac arrest, brain damage and death in these patients. Oxygen administration should be continued throughout the evaluation, transport and initial therapy of patients with airway obstruction. Oral intake should be prohibited and an intravenous infusion started to assure hydration. Secretions must be suctioned cautiously and and physical examination completed for as accurate a diagnosis as possible.

The next step is the *insertion of an artificial airway.* Emergency tracheostomy without prior endotracheal intubation and accurate assessment of ventilation is dangerous and associated with excessive mortality and morbidity. Intubation should be performed by experienced personnel in an orderly manner with oxygen therapy, adequate suction, atropine, functioning laryngoscopes, a variety of endotracheal tubes, anesthesia if necessary and facilities for emergency tracheostomy, all immediately available.

Debate continues over the relative merits of *endotracheal intubation vs tracheostomy* as the best method of airway management. For the emergency, an orotracheal tube is inserted. This can be replaced by a nasotracheal tube later. Downes[35] has stated that both nasotracheal intubation and tracheostomy have roles in airway management of infants and children. He summarized the arguments and indications as shown in Table III.

Table III. Relative Indications for Nasotracheal Tubes and Tracheostomy

Nasotracheal tube	Tracheostomy
Infants	Older children
Duration < 10-14 days	Duration > 14 days
Epiglottitis	Croup
Croup?	Congenital airway anomalies
Acute cardiopulmonary failure	Chronic cardiopulmonary failure
Reported need for an airway	
	One time need for airway (chronic)

Battersy, et al.,[36] reported on the results of *nasotracheal intubation* in 435 patients following cardiac surgery. They encountered only 24 complications; 21 were post-extubation stridor and three cases of subglottic stenosis. They recommended tracheostomy only in unusual circumstances such as congenital or acquired abnormalities of the upper airway or where there was little prospect of extubation in the foreseeable future.

To achieve good results, *proper care of the artifical airway* is essential. Humidity, so necessary to prevent drying of the mucosa, is provided by aerosols and isotonic saline instillations (0.5-3 cc). Chest physiotherapy is useful for moving secretions to a level where they can be removed by a sterile suction technique. The airway must be kept clear. Functional residual capacity can be maintained with positive airway pressure with 2 or more cm H_2O.

The size of the airway tube is crucial. A small leak should be audible when a positive pressure of 20-30 cm H_2O is applied to the endotracheal tube. If such a leak is not heard or disappears with time, a smaller tube should be considered. Routine changing of the tube is not needed if the tube is constructed of implant-tested (safe) materials. The above technic will nearly eliminate subglottic stenosis and most laryngeal problems.

When the patient has recovered, the airway may be removed. Direct examination under anesthesia may help decide when extubation should be done. Respiratory therapy will be required following extubation to assure secretion removal and to prevent atelectasis.

Conclusions

The technology of mechanical ventilation has, with the rest of medicine, continued to advance. Among the more interesting *recent innovations* has been HFPPV[39] and HFO.[40] HFPPV utilizes small tidal volumes (less than dead space) at respiratory rates of 60-300 breaths/minute. HFO uses a much smaller V_T (less than 20 ml) at extremely high rates (up to 50 Hz). With tidal volume smaller than the dead space, it is obvious that convective flow is not responsible for the gas exchange—and gas exchange does

occur; both animals and human subjects not only tolerate this treatmeat, but cease spontaneous ventilatory efforts. It has been postulated that these machines enhance the diffusion responsible for movement of O_2 into and CO_2 out of the alveoli. Both HFPPV and to a lesser extent HFO are being utilized clinically in Europe, with current emphasis being placed on utilization in the operating room during such procedures as bronchoscopy, micro-surgery of the larynx and thoracic surgery. Modified forms of high frequency ventilation are also being used to a certain extent in neonatal intensive care.

Another innovation in acute ventilatory care has been *ventilation of each lung separately* by a double lumen tube and endobronchial intubation. It was noted previously that most of our therapeutic efforts are relatively non-specific and they affect relatively normal lung more than diseased lung. If the pulmonary disease happens to be unilateral, by establishing a separate airway to each lung with an endobronchial tube, we can to some extent, tailor our therapy to each lung by adding more or less PEEP (CPAP) and using different tidal volumes. Unfortunately, the minimum size of such endotracheal tubes currently available for therapy is so large (7 mm, internal diameter) that it eliminates the use of these tubes in all but the largest children. Obviously, a great deal may be expected in the future from these new modes of therapy.

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Chemotherapeutic treatment of dental caries

The basic mechanism of dental caries is that dietary carbohydrate is taken up by the bacterial layer on the tooth surface (dental plaque) and metabolised, producing organic acid, which attacks the enamel surface, and removes the calcium phosphate mineral. Frequent attacks produce the first stage of dental caries-the white spot lesion which appears chnically as a chalky white patch on the enamel and microscopically a shallow of partial demineralisation. Later the surface cavitates, and the tooth is progressively destroyed.

In recent years factors which will favourably influence natural remineralisation of lesions have been investigated. These factor fall into two groups : firstly, factors which reduce the amount of plaque, or the proportion of sugar forming organisms in the plaque, or the ability of organisms to complete the glycolysis cycle, or the amount and frequency of fermentable substrate in the diet. The second group of factors include those which make more calcium and phosphate ions available at the plaque enamel surface. Dietary supplements of milk or organic phosphates are important. Ability of fluoride ions contained in drinking water, or in a mouth-rinse (250 parts/ 10^6 F), to promote remineralisation is undoubted. Even better is the Koulourides solution containing dicalcium phosphate dihydrate and fluoride. The use of these agents may make chemotherapeutic treatment of dental caries a reality.

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