Proceeding S.Z.P.G.M.I vol: 9(1-2) 1995, pp. 1-9.

Editorial Review

Acute Respiratory Failure in Children

Fouzia Shaukat, Sajid Maqbool
Department of Paediatrics, Shaikh Zayed Hospital, Lahore.

Acute respiratory failure (ARF) continues to be a major problem in paediatric critical care[1]. It is defined as the inability of the respiratory system to deliver adequate oxygen or to remove CO_2 from the pulmonary circulation thereby leading to arterial hypoxia, hypercapnia or both[2]. In practical terms acute respiratory failure is present when the PaO_2 is < 8 kPa (60 mmHg) or the $PaCO_2$ is > 7 kPa (55 mmHg)[3]. It is a common paediatric problem and is responsible not only for morbidity in children but also a high death rate. The clinical findings in respiratory failure are determined by the adverse effects of low oxygen and high CO_2 on the function of susceptible organ systems, chiefly the lung, heart, kidneys and brain[2].

In a recent report from a typical, large city tertiary paediatric intensive care unit, patients with ARF comprise nearly 3% of all admissions and 8% of total patient days[4]. Mortality in this group was 62% and accounted for 33% of all unit deaths in the 24 month surveillance period. Others have reported a similar prevalence with comparable mortalities ranging from 40% to 75%[5-8], which appear not to have changed over the past 15 to 20 years[1].

INFANTS - AT HIGH RISK OF RESPIRATORY FAILURE

Infants are at a higher risk of respiratory failure because of physiologic, anatomic and mechanical differences between their respiratory systems and those of adults.

1. In infants the thoracic cage is soft and therefore, provides an unstable base for the ribs.
2. Intercostal muscles are poorly developed in children, therefore, cannot achieve the classic bucket handle motion that characterizes adult breathing.
3. The diaphragm is less effective in infants because it is relatively flat and short and has fewer type I muscle fibres.
4. During REM sleep, the ventilatory movements of the rib cage become uncoordinated and out of phase with those of the diaphragm.
5. The infant trachea is small, only one-third the diameter of the adult trachea. Therefore, according to Poiseuille's Law, a 1-mm thickening of the respiratory mucosa in an infant causes a 75% reduction in cross-sectional area in the infants airways compared to only a 20% reduction in the adult airway.
6. The children's alveoli are smaller and have less collateral ventilation with fewer pores of Kohn, resulting in a greater tendency for the alveolus to collapse and thus cause atelactasis[2].

RESPIRATORY FAILURE - CLASSIFICATION

Respiratory failure can be classified into two types though it should always be kept in mind that paediatric patients with respiratory failure usually display a variable combination of the two[2].

- **Type I Respiratory Failure**: Patients with type I respiratory failure generally have a low PaO_2 with a normal to low $PaCO_2$.
- **Type II Respiratory Failure**: In type II respiratory failure the patients have a high $PaCO_2$ with a low PaO_2.

Type I respiratory failure is the failure of the lung to oxygenate the blood and occurs in three situations;

1. The most frequent cause is a **ventilation/ perfusion defect** (V/Q mismatch) which occurs when blood flows to parts of the lungs that are poorly ventilated or underventilated i.e. when blood flow and alveolar ventilation are mismatched.
2. **Diffusion defects** result from disturbances such as a thickened alveolar membrane or a build up of interstitial fluid at the alveolar capillary junction.

3. **Intrapulmonary shunt** occurs when blood flows through areas of the lung that are never ventilated.

Type II respiratory failure, characterized by a high $PaCO_2$ and a low PaO_2 can be viewed as respiratory "pump" failure and is generally the result of alveolar hypoventilation and not of a primary disease of the lung. Numerous disease processes can contribute to this hypoventilation[2].

The types of respiratory failure and the various disease processes contributing either to hypoxemic or ventilatory failure are shown in Table 1.

Table 1: Types of respiratory failure

Findings	*Causes*	*Examples*
Type I		
Hypoxia	(V/Q defect	Positional (supine in bed)
Decreased PaO_2	ventilation	ARDS
Normal $PaCO_2$	perfusion	Atelactasis
	defects)	Pneumonia
		Pulmonary embolus
		Bronchopulmonary dysplasia
	Diffusion impairment	Pulmonary oedema
		ARDS
		Interstitial pneumonia
	Shunt	Pulmonary arterio-venous malformation
		Congenital adeno-matoid malformation
Type II		
Hypoxia	Hypoventilation	Neuro-muscular disease
Hypercapnia		(Polio, Guillain Barre syndrome)
Decreased PaO_2		
Increased $PaCO_2$		Head trauma, sedation
		Chest wall dysfunction (burns)
		Severe reactive airway disease

It is important to remember that hypoxemia is not always related to respiratory failure. Right to left cardiac shunts, high altitude with its low ambient oxygen concentration and the production of methemoglobin, all may produce severe hypoxemia with normal respiratory function[2].

RESPIRATORY FAILURE - CLINICAL ASSESSMENT

Clinical findings in respiratory failure are determined by the adverse effects of low oxygen and high CO_2 and pH on the function of susceptible organ systems chiefly the lung, heart, kidneys and brain[2]. The most sensitive clinical indicator of increasing respiratory difficulty is a rising respiratory rate[3]. As respiratory failure ensues, physical findings such as tachypnea, retraction, cyanosis restlessness, or even somnolence can be seen. The hypoxemia or hypercapnia that results from ventilation/perfusion mismatch, shunt, or hypoventilation may interfere with brain metabolism, depress the myocardium, or cause pulmonary hypertension. Hypercapnia depresses the central nervous system, and the resulting acidemia depresses myocardial function. Thus patients in respiratory failure can exhibit significant changes in central nervous system and cardiac function[2]. Table 2 depicts the various clinical criteria for respiratory failure.

Table 2: Clinical criteria for respiratory failure

Respiratory
- Wheezing
- Expiratory grunting
- Decreased or absent breath sounds
- Flaring of alae nasi
- Retractions of chest wall
- Tachypnea, bradycardia, or apnea
- Cyanosis.

Cerebral
- Restlessness
- Irritability
- Headache
- Mental confusion
- Convulsions
- Coma

Cardiac
- Bradycardia or excessive tachycardia
- Hypotension or hypertension

General
- Fatigue
- Sweating

ACUTE RESPIRATORY FAILURE - MONITORING

Monitoring is often helpful in guaging the severity and acuity of respiratory failure.

The *Pulse oxymeter* may be used to evaluate "arterial" blood oxygen saturation[2,9-18]. This instrument provides the clinician with a tool to rapidly and non-invasively assess and continuously monitor oxygen saturation in the patient with acute or impending respiratory failure. It should be routinely used in the assessment and management of all patients with suspected respiratory failure. However, pulseoxymeter provides no information on the CO_2 or acid base status of the patient[2,9,19-37].

Arterial blood gases (ABGs) measurement remains the best means for assessment of acute respiratory failure[38-51].

Arterial blood gas analysis gives information on the acid base status (with a measured pH and calculated bicarbonate level) as well as information on the status of oxygenation (PaO_2) and ventilation($PaCO_2$) in the patient[52-61]. Arterial blood gas determinations are the best indicator of how well the respiratory system is performing its gas exchanging function and how well acid-base homeostasis is being maintained.

Abnormalities in blood gas tension may occur with dysfunction of any part of the respiratory system including the respiratory controller, the conducting airway, the gas exchanging portions of the lung, the pulmonary circulation, the respiratory muscles, and the chest wall.

Blood gases may be sampled by two methods by intermittent arterial puncture or through indwelling arterial lines[38-51]

Table 3 gives normal values of arterial pH, PaO_2 and $PaCO_2$ at sea level and at an altitude of 5000 feet[2,62]

Table 3: Normal arterial blood gas values on room air

	Sea level	*5000 feet*
pH	7.38 - 7.42	7.36 - 7.40
PaO_2 (mmHg)	85 - 95	65 - 75
$PaCO_2$ (mmHg)	36 - 42	35 - 40

$PaCO_2$ is a sensitive measure of ventilation and is inversely related to the minute ventilation[2].

When interpreting the PaO_2 it is important to remember that it is the oxygen content of the arterial blood that matters and that this is determined by the percentage saturation of haemoglobin with oxygen. The relationship between the latter and the PaO_2 is determined by the oxyhaemoglobin dissociation curve. In general, if the saturation is greater than 90%, oxygenation can be considered to be adequate. It must be remembered, however, that on the steep portion of the oxygen dissociation curve small falls in PaO_2 will cause significant reductions in oxygen content. PaO_2 is also influenced by factors other than pulmonary function including alterations caused by changes in the metabolic rate or/and cardiac output[3].

Knowing the ABG values and the inspired oxygen concentration enables one to calculate several parameters that may be helpful in determining the efficiency of gas exchange.

a. The difference between alveolar oxygen concentration and the arterial oxygen value is the *alveolar arterial oxygen difference ($AaDO_2$)*. The $AaDO_2$ is less than 15 mmHG under normal conditions and it increases with increasing inspired oxygen concentrations to about 100 mmHg in normal patients breathing 100% oxygen.
Diffusion impairment, shunts and ventilation/perfusion mismatches all cause increased $AaDO_2$.
The alveolar arterial oxygen gradient ($AaDO_2$) is used as a predictor of outcome associated with acute respiratory failure among neonates and older children[65].

$AaDO_2$ = $PiO_2 - (PaCO_2 / R) - PaO_2$ (normal = 5-15 mmHg)
Aa DO_2 = Alveolar arterial oxygen difference (mmHg)
PiO_2 = Partial pressure of oxygen in inspired air mmHg.
PiO_2 = (Barometric pressure - 47) x % inspired oxygen concentration.
$PaCO_2$ = Partial pressure of carbon dioxide in arterial blood (mmHg).
R = Respiratory quotient (usually 0.8)
PaO_2 = Partial pressure of oxygen in arterial blood (mmHg).
47 = Vapor pressure of water.

b. In addition to the calculation of the $AaDO_2$, assessment of the *intrapulmonary shunting of blood* may be helpful.
The intrapulmonary shunt is the percentage of

pulmonary blood flow which passes through non-ventilated areas of the lung. It has the same effect as a right-to-left cardiac shunt in that oxygen saturation are lowered as shunting increase[2].

Normal individuals have less than a 5% physiologic shunt from bronchial, thebesian, and coronary circulation.

Shunt fractions greater than 15% usually indicates the need for aggressive respiratory support.

When intrapulmonary shunt reaches 50% of pulmonary blood flow PaO_2 does not increase regardless of the amount of supplemental oxygen used.

$$\frac{Q_s}{Q_t} = \frac{Cco_2 - CaO_2}{Cco_2 - Cvo_2} \quad \text{(Normal < 5\%)}$$

$$\frac{Q_s}{Q_t} = \frac{\text{Intrapulmonary shunt (\%)}}{\text{(in patients without cardiac shunt)}} \quad \text{(Normal < 5\%)}$$

Cco_2 = Oxygen content of pulmonary capillary blood (ml/dl)
CaO_2 = Oxygen content of arterial blood (ml/dL)
Cvo_2 = Oxygen content of mixed venous blood (ml/dl)

c. Dead space ventilation (DSV) is that part of the breath in the conducting air passages plus the alveolar volume which is ventilated but not perfused by the pulmonary circulation.

DSV is increased in conditions like, bronchopulmonary dysplasia, V/Q mismatches, pulmonary interstitial emphysema, pulmonary embolism and many other entities

Decreasing DSV is dependent on its cause but such methods as tracheostomy in patients with chronic respiratory failure or streptokinase therapy in persons with pulmonary emboli are examples[2].

$$V_d = \frac{(PaCO_2 - PeCO_2)}{PcCO_2} \text{ (Normal approximately 2 ml/kg)}$$

V_d = Physiologic dead space (anatomic dead space + alveolar dead space) (ml)
$PaCO_2$ = Partial pressure of carbondioxide in arterial blood (mmHg).
$PeCO_2$ = Partial pressure of carbondioxide is expired air (mmHg).
$PcCO_2$ = Partial pressure of carbondioxide in capillary blood (mmHg).

MANAGEMENT OF RESPIRATORY FAILURE

A. Conventional Management

Conventional management of patients with respiratory failure includes the following:

a) Administration of supplemental oxygen.
b) The control of secretions
c) The treatment of pulmonary infection,
d) The control of bronchospasm
e) Measures to limit pulmonary oedema[3].

B. Mechanical Ventilation

1. O_2 supplementation: Patients with hypoxemia induced by respiratory failure may respond to supplemental oxygen administration alone (Table 4).

- Those patients with hypoventilation and diffusion defects respond better than patients with shunts or V/Q mismatches.
- Severe V/Q mismatches often do not respond to anything but aggressive airway management and mechanical ventilation.
- Patients with a decreased functional residual capacity (FRC - the amount of air left in the lungs at the end of passive expiration) often respond to the delivery of continuous positive airway pressure (CPAP) 5-10 cm H_2O by either mask or endotracheal tube.
- Patients with severe hypoxemia, hypoventilation, or apnea require assistance with bag and mask ventilation until the airway is intubated.
- Ventilation may be maintained for sometimes with a mask of the proper size but gastric distention, emesis, and inadequate tidal volumes are possible complications.
- In patients who fail to respond to simple oxygen supplementation establishment of an artificial airway is often life saving[2]. Therefore, if, despite the conventional management the patient with respiratory failure (impending or evident) continues to deteriorate or fails to improve, endotracheal intubation should be performed and the institution of some form of respiratory support should be considered.

Table 4: Supplemental oxygen therapy

Source	*Max. % O_2*	*Range of flow rates*	*Advantages*	*Disadvantages*
Nasal cannula	35-40%	0.125-4 L/min	Easily applied, relatively comfortable	Uncomfortable at higher flow rates, requires open nasal airways, easily dislodged, lower % O_2, nosebleeds
Simple mask	50-60%	5-10 L/min	Higher % O_2, good for mouth breathers	Uncomfortable, dangerous for patients with poor airway control and at risk for emesis, hard to give airway care, unsure of % O_2.
Face tent	40-60%	8-10 L/min	Higher % O_2, good for mouth breathers, less restrictive	Uncomfortable, dangerous for patients with poor airway control and at risk for emesis, hard to give a airway care, unsure of % O_2.
Rebreathing mask	80-90%	5-10 L/min	Higher % O_2, good for mouth breathers, higher O_2 concentration	Uncomfortable, dangerous for patients with poor airway control and at risk for emesis, hard to give airway care, unsure of % O_2.
Oxyhood	90-100%	5-10 L/min (mixed at wall)	Stable and accurate O_2 concentration	Temperature regulation, hard to give airway care.

(Reproduced from Stenmark KR, et al. Acute respiratory failure: Current Paediatric Diagnosis and Treatment 1994: pp. 346).

2. *Endotracheal Intubation*

- Intubation of the trachea in infants and children requires experienced personnel and the right equipment.
- A patient in respiratory failure whose airway must be stabilized requires many interventions before the actual intubation.
- The patient must be properly positioned to facilitate air exchange while supplemental oxygen is given. The sniffing position is used in infants while head extension with jaw thrust is used in older children without neck injuries.
- If obstructed by secretions or vomitus the airway must be cleared by suction.
- When not obstructed by a foreign body or epiglottitis, airway should open with proper positioning and placement of an oral or nasopharyngeal airway of the correct size. Nasal airways are better tolerated than oral airways by conscious patients.
- As each step is taken, it is imperative to monitor changes in chest movements airway and breath sounds, skin color, and mental status.
- In patients with normal airway, an intravenous anaesthesia induction for intubation may be performed by those experienced with the drugs and the intubation procedure.
- Drugs commonly used for controlled intubation include:

  - Atropine 0.02 mg/kg/dose
  - Thiopental 3-5 mg/kg/dose
  - Ketamine 1-2 mg/kg/dose (IV)
    4-8 mg/kg/dose (IM)
  - Succinylcholine 1-2 mg/kg/dose
  - Pancuromium 0.1 mg/kg/dose

- Patients with obstructed upper airways (e.g. patients with croup, epiglottilis, obstruction by

foreign bodies or subglotic stenosis) should be awake when intubated unless trained airway specialists decide otherwise.

- Insertion of an endotracheal tube of the correct size is of critical importance in pediatrics.
- A tube that is too large for a pediatric patient may cause pressure necrosis of the tissues in the subglottic region.
  (The subglottic region is the narrowest portion of the upper airway in children, in contrast to the glottis in adults)
  Insertion of inappropriately large endotracheal tube (ETT) has been associated with scarring and in some cases permanent stenosis of the subglottic region, requiring tracheostomy or cricoid split for repair[2].
- Too small an endotracheal tube can result in inadequate pulmonary toilet and excessive air leak around the endotracheal tube, making optimal ventilation and oxygenation difficult.
- There are many ways to calculate the size of endotracheal tube that is appropriate for a child[77]. A useful method is the following:
  Tube size = (16 + age in years) / 4
  Patients under 8 years of age should have uncuffed endotracheal tubes[78-80] (Table 5).

Table 5: Tracheal tube size and estimated weight by age.

Age (Yrs)	*Weight (kg)*	*Endotracheal tube size (mm internal diameter*
Premature	1-2.5	2.5 uncuffed
Term newborn	3	3.0 uncuffed
1	10	3.5-4.0 uncuffed
2	12	4.5 uncuffed
3	14	4.5 uncuffed
4	16	5.0 uncuffed
5	18	5.0-5.5 uncuffed
6	20	5.5 uncuffed
7	22	5.5 - 6.0 uncuffed
8	24	6.0 cuffed
10	32	6.0-6.5 cuffed
Adolescent	50	7.0 cuffed

(Reproduced from Batten FK. Emergencies and Accidents. Current Paediatrics Diagnosis and Treatment 1994; pp. 331.)

However, in many paediatric patients decreased pulmonary compliance develops as a result of the disease process and high positive end-expiratory pressure is required to maintain open alveoli and relatively high pressures so as to provide adequate tidal volume. In these patients the use of cuffed ETT is helpful, if not essential to provide optimal and consistent levels of ventilation and positive end-expiratory pressure. A leak around the ETT often hampers or defeats these two goals of assisted ventilation. Cuffed ETTs are also useful in severe bronchiolitis with high airway resistance and in decreasing the incidence of aspiration[81].

Use of cuffed endotracheal tubes has been associated with several problems and complications[83-88], but it has recently been studied that cuffed endotracheal intubation in children younger than 8 years (age range, 1 year - 8 years) is not associated with increased risk of post extubation stridor or significant long term sequelae[77,89,90].

- After placement of the endotracheal tube breath sounds should be evaluated for bilateral equality.
- One should then check for a leak between the endotracheal tube and the larynx. To do this, connect a pressure - monitoring anaesthesia bag to the circuit and allow it to inflate creating positive pressure. Check for the leak by auscultating over the throat, noting the pressure at which air escapes around the endotracheal tube.
- Leaks of 15-20 cm H_2O are acceptable.
- Larger leaks (>20 cm H_2O) are acceptable only in patients having severe lung disease and poor compliance and requiring high pressures to achieve ventilation. In this situation, one must be aware of the possible post extubation complications of subglottic stenosis in the patient.
- A chest x-ray is necessary for final assessment of endotracheal tube placement[2].

3. *Mechanical ventilation* can be life saving in patients with acute severe hypoxemia or worsening respiratory acidosis (or both) that is refractory to more conservative measures. In patients with severe cardiopulmonary distress for whom the effort of breathing is intolerable, mechanical ventilation substitutes for the action of respiratory muscles. In some patients the respiratory muscles account for as much as 50

percent of total oxygen consumption[66,67]. In such circumstances, mechanical ventilation allows precious stores of oxygen to be rerouted to other tissue beds that may be vulnerable. In addition, the reversal of respiratory muscle fatigue, which may have a role in the development of acute ventilatory failure depends on adequate rest of the respiratory muscles. Positive pressure ventilation can reverse and prevent atelectasis, and by allowing inspiration at a more compliant region of the pulmonary pressure volume curve, it can decrease the work of breathing[68]. Improvements in pulmonary gas exchange and pressure volume relations and relief from excessive respiratory work provide an opportunity for the lungs and airways to heal[66].

That positive pressure mechanical ventilation can save lives was proved during the poliomyelitis epidemics in the 1950s[69]. Since that time there has been a growing increase in the use of ventilatory support, and it has been closely associated with the development of critical care medicine. Early ventilators were used in conjunction with neuromuscular blocking agents to provide "controlled" ventilation. Today, most machines are triggered by the patient, and there is growing awareness of the complexity of patient ventilator interaction[70-72]. There is also increasing recognition that ventilators can induce subtle forms of lung injury[73], which has led to a reappraisal of the goals of ventilatory support[74]. With advances in computer and electronic technology, ventilators have changed markedly in appearance and there is an array of options that is increasingly intimidating[75,76]. However, the fundamental principles of ventilatory treatment of the critically ill patient remain unchanged, although there are several new naunces in their application[66].

REFERENCES

1. **Ring JC, Stidham GL.** Novel therapies for acute respiratory failure. *Pead Clin N Am 1994;* **41**: 1325-63.
2. **Stenmark KR, Dobyns EM, et al.** Acute respiratory failure: *Current Paediatric Diagnosis and Treatment 1994;* pp. 343-347.
3. **Kumar PJ, Clark ML.** Respiratory failure. *Clinical Medicine 1994;* pp. 726-732.
4. **Davis SL, Furman DP, Costarino AT Jr.** Adult respiratory distress syndrome in children: Associated disease, clinical course, and predictors of death. *J Pediatr 1993;* 123: 35.
5. **Lyrene RK, Truog WE.** Adult respiratory distress syndrome in a pediatric intensive care unit: Predisposing conditions, clinical course; and outcome. *Pediatrics 1981;* **67:** 790.
6. **Rivera RA, Butt W, Shann F.** Predictors of mortality in children with respiratory failure. Possible indications for ECMO. *Anaesth Intensive Care 1990;* **18:** 385.
7. **Tamburro RF, Bugnitz MC, Stidham GL.** Alveolar-arterial oxygen gradient as predictor of outcome in patients with non-neonatal pediatric respiratory failure. *J Pediatr 1992;* **121:** 330.
8. **Timmons OD, Dean JM, Vernon DD.** Mortality rates and prognostic variables in children with adult respiratory distress syndrome. *J Pediatr 1991;* **119**: 896.
9. **Tremper KK, Barker SJ.** Pulse oximetry. *Anesthesiology 1989;* **70:** 98-108.
10. **Pologe JA.** Pulse oximetry: Technical aspects of machine design. *Int Anesthesiol Clin 1987;* **25:** 137-53.
11. **Nickerson BG, Sakrison C, Tremper KK.** Bias and precision of pulse oximeters and arterial oximeters. *Chest 1988;* **93:** 515-17.
12. **Mihm FG, Halperin DH.** Non-invasive detection of profound arterial desaturations using pulse oximetry device. *Anesthesiology 1985;* **62:** 85-7.
13. **Cecil WT, Thorpe KJ, Fibuch EF, Tuohy GF.** A clinical evaluation of the accuracy of the nellcor N 100 and the Ohmeda 3700 pulse oximeters. *J Clin Monit 1988;* **4:** 31-36.
14. **Fait CD, Wetzel RC, Dean JM, Schleien CL, Gioia FR.** Pulse oximetry in critically ill children. *J Clin Monit 1985;* **1:** 232-35.
15. **Boxer RA, Gottesfeld I, Singh S, Lacrote MA, Parnell VA, Walker P.** Non-invasive pulse oximetry in children with cyanotic congenital heart disease. *Crit Care Med 1987;* **15:** 1062-64.
16. **Mok J, Pintar M, Benson L, McLaughlin FJ, Levinson H.** Evaluation of non-invasive measurements of oxygenation in stable infants. *Crit Care Med 1986;* **14:** 960-63.
17. **Duran M, Ramanathan R.** Pulse oximetry for continuous oxygen monitoring in six newborn infants. *J Pediatr 1986;* **109:** 1052-55.
18. **Cote CJ, Goldstein EA, Cote MA, Hoaglin DC, Ryan JF.** A single blind study of pulse oximetry in children. *Anesthesiology 1988;* **68:** 184-88.
19. **The technology assessment task force of the society of critical care medicine.** A model for technology assessment applied to pulse oximetry. *Crit Car Med 1993;* **21:** 615-24.
20. **Emergency Care Research Institute:** Pulse oximeters evaluation. *Health Devices 1989;* **18:** 185-230.
21. **New W.** Pulse oxymetry. *J Clin Monit 1985;* **1:** 126-29.
22. **Dautzenberg B, Gallinari C, Moreau A, et al.** The advantages of real-time oximetry over intermittent arterial blood gas analyses in a chest department. In: *Pulse Oximetry*, **Payne JP, Severinghaus JW** (eds.). Berlin, Springer-Verlag, 1986; pp. 63-5.
23. **Fanconi S.** Pulse oximetry for hypoxemia: A warning to users and manufacturers. *Intensive Care Med 1989;* **15:** 540-42.
24. **Kelleher JF.** Pulse oximetry. *J Clin Monit 1989;* **5:** 37-62.
25. **Mariani JJ.** Monitoring during mechanical ventilation. *Clin Chest Med 1988;* **9:** 73-100.
26. **King T, Simon RH.** Pulse oximetry for tapering supplemental oxygen in hospitalized patients: Evaluation

of a protocol. *Chest 1987;* **92:** 713-16.

27. **Severinghaus JW, Naifeh KH, Khoh SO.** Errors in 14 pulse oximeters during profound hypoxia. *J Clin Monit 1989;* **5:** 72-81.
28. **Severinghaus JW, Naifeh KH.** Accuracy of six pulse oximeters to profound hypoxia. *Anesthesiology 1987;* **67:** 551-58.
29. **Chapman KR, Liu FLW, Watson RM, et al.** Range of accuracy of two wavelength oximetry. *Chest 1989;* **4:** 540-42.
30. **Nickerson BG, Sarkisian C, Tremper K:** Bias and precision of pulse oximeters and arterial oximeters. *Chest 1988;* **93:** 515-17.
31. **Bowton DL, Scueri PE, Harris L, et al.** Pulse oximetry monitoring outside the intensive care unit: progress or problem? *Ann Intern Med 1991;* **115:** 450-54.
32. **Rotello LC, Jastremski MS.** Continous oximetry: Is it beneficial? *Perspective in Critical Care 1990;* **3:** 1-29.
33. **Russell RIR, Helms PJ.** Comparative accuracy of pulse oximetry and transcutaneous oxygen in assessing arterial saturation in pediatric intensive care. *Crit Care Med 1990;* **18:** 725-27.
34. **New W.** Pulse oximetry. *J Clin Monit 1985;* **1:** 126.
35. **Fanconi S, Doherty P, Edwards JF, et al.** Pulse oximetry in pediatric intensive care: comparison with measured saturations and transcutaneous oxygen tension. *J Pediatr 1985;* **107:** 362.
36. **Southall DP, Bignall S, Stebbens VA, et al.** Pulse oximeter and transcutaneous arterial oxygen measurements in neonatal and pediatric intensive care. *Arch Dis Child 1987;* **62:** 882.
37. **Mok J, Pintar M, Benson L, et al.** Evaluation of non-invasive measurements of oxygenation in stable infants. *Crit Care Med 1986;* **14:** 960.
38. **Zimmerman JL, Dellinger RP.** Initial evaluation of a new intra-arterial blood gas system in humans. *Crit Care Med 1993;* **21:** 495-99.
39. **Misiano DR, Meyerhoff ME, Collison ME.** Current and future directions in the technology relating to bedside testing of critically ill patients. *Chest 1990;* **97:** 204S-14S.
40. **Shapiro BA, Cane RD.** Blood gas monitoring: Yesterday, today and tomorrow. *Crit Care Med 1989;* **17:** 573-81.
41. **Gehrich JL, Lubbers DW, Opitz N.** Optical fluorescence and its application to an intravascular blood gas monitoring system. IEEE *Trans Biomed Eng 1986;* **33:** 117-32.
42. **MacGregor DA, Scuderi PE, Bowton DL, et al.** A side-by-side comparison of four blood gas analyzers using tonometered human blood. Abstr. *Chest 1990;* **98:** 33S.
43. **Metzger LF, Stauffer WB, Krupinski AV, et al.** Detecting errors in blood gas measurement by analysis with two instruments. *Clin Chem 1987;* **33:** 512-17.
44. **Shapiro BA, Cane RD, Chomka CM.** Preliminary evaluation of an intra-arterial blood gas system in dogs and humans. *Crit Care Med 1989;* **17:** 455-60.
45. **Larson CP, Daviers GA, Ricatelli SD.** Continuous monitoring of PaO_2, $PaCO_2$ in surgical patients. Abstr. *Crit Care Med 1991;* **19:** S25.
46. **Mahutte K, Sassoon SH, Muro JR, et al.** Progress in the development of a fluorescent intravascular blood gas system in man. *J Clin Monit 1990;* **6:** 147-57.
47. **Barker SJ, Hyatt J.** Continuous measurement of intra-arterial pH, $PaCO_2$ and PaO_2 in the operating room. *Anesth Analg 1991;* 43-48.
48. **Biswas CK, Ramos JM, Agroyannis B, et al.** Blood gas analysis: Effects of air bubbles in syringe and delay in estimation. *Br Med J 1982;* **284:** 923-27.
49. **Zaloga GP.** Evaluation of bedside testing options for the critical care unit. *Chest 1990;* **97:** 185S-190S.
50. **Thorson SH, Marini JJ, Pierson DJ, et al.** Variability of arterial blood gas values in stable patients in the ICU. *Chest 1983;* **84:** 44-8.
51. **Salem M, Chernow B, Burke R, et al.** Bedside diagnostic blood testing-Its accuracy, rapidity and utility in blood conservation. *JAMA 1991;* **266:** 382-89.
52. **Shapiro BA, Mahutte CK, Cane RD, Gilmour IJ.** Clinical peformance of a blood gas monitor: A prospective, multicenter trial. *Crit Care Med 1993;* **21:** 487-94.
53. **Shapiro BA.** Quality improvement standards for intensive care unit monitors: We must be informed and involved. *Crit Care Med 1992;* **20:** 1629-30.
54. **Shapiro BA.** pH and blood gas measurements: Discerning innovation from sophistication. *Crit Care Med 1989;* **17:** 966.
55. **Eichorn J, Moran R, Cormier A.** *Blood gas pre-analytical considerations: Specimen collection, calibration and control.* NCCLS Publication C-27. Villanova, PA, 1985.
56. **Shapiro BA.** Monitoring gas exchange in acute respiratory failure. *Respir Care 1983;* **28:** 605-7.
57. **Maukkassa FF, Rutledge R, Fakhry SM, et al.** ABGs and arterial lines: The relationship to unnecessarily drawn arterial blood gas samples. *J Trauma 1991;* **30:** 1087-95.
58. **Shapiro BA.** In vivo monitoring of arterial blood gases and pH. *Respir Care 1992;* **37:** 165-69.
59. **Mahutte CK.** On line blood gas monitoring. In: *Contemporary management in critical care: respiratory monitoring.* Chapter 2, vol. 4. **Tobin MJ (ed).** New York Churchill Livingstone, 1991.
60. **Greene GE, Hassell RT, Mahutte CK.** Comparison of arterial blood gas with continuous intra-arterial and transcutaneous Po_2 sensors in adult critically ill patients. *Crit Care Med 1985;* **13:** 859-63.
61. **Wickers EK, Teunissen AJ, Van den Camp RA, et al.** A comparative study of the electrode systems of three pH and blood gas apparatus. *J Clin Chem Biochem 1978;* **16:** 175-185.
62. **Miller WW, Yafuso M, Yan CF, et al.** Performance of an in vivo, continuous blood gas monitor with disposable probe. *Clin Chem 1987;* **33:** 1538-42.
63. **Thorson SH, Marini JJ, Pierson DJ, et al.** Variability of arterial blood gas values in stable patients in the ICU. *Chest 1983;* **4:** 14-18.
64. **Clausen JL, Murray KM.** Clinical application of arterial blood gases: How much accuracy do we need? *J Med Tech 1985;* **2:** 19-32.
65. **Tamburro RF, Bugnitz MC, Stidham GL.** Aveolar-arterial oxygen gradient as a predictor of outcome in patients with non-neonatal pediatric respiratory failure. *J Peadiatr 1991;* **119:** 935-38.
66. **Tobin MJ.** Mechanical ventilation. *New Engl J Med 1994;* **330:** 1056-61.
67. **Field S, Kelly SM, Macklem PT.** The oxygen cost of breathing in patients with cardiorespiratory disease. *Am Rev Respir Dis 1982;* **126:** 9-13.
68. **Kntz JA, Marks JD.** Inspiratory work with and without continuous positive airway pressure in patients with acute respiratory failure. *Anesthesiology 1985;* **63:** 598-607.
69. **Ibsen B.** The anaesthetist's viewpoint on the treatment of respiratory complications in poliomyelitis during the epidemic in Copenhagen, 1952. *Proc R Soc Med 195;* **47:** 72-4.
70. **Gibney RTN, Wilson RS, Pontoppidan H.** Comparison of work of breathing on high gas flow and demand valve continuous positive airway pressure systems. *Chest 1982;* **82:** 692-5.
71. **Marini JJ, Capps JS, Culver BH.** The inspiratory work

of breathing during assisted mechanical ventilation. *Chest 1985;* **87:** 612-8.

72. **Hubmayr RD, Abel MD, Rehder K.** Physiologic approach to mechanical ventilation. *Crit Care Med 1990;* **18:** 103-13.
73. **Dreyfuss D, Soler P, Basset G, Saumon G.** High inflation pulmonary edema: respective effects of high airway pressure, high tidal volume, and positive end-expiratory pressure. *Am Rev Respir Dis 1988;* **137:** 1159-64.
74. **Slutsky AS.** Mechanical ventilation. *Chest 1993;* **104:** 1833-59.
75. **Kacmarek RM, Meklaus GJ.** The new generation of mechanical ventilators. *Crit Care Clin 1990;* **6:** 551-78.
76. **American Association for Respiratory Care.** Consensus statment on the essentials of mechanical ventilation 1992. *Respir Care 1992;* **37:** 1000-8.
77. **Deakers TW, Reynolds G, Stretton M, et al.** Cuffed endotracheal tubes in pediatric intensive care. *J Pediatr 1994;* **125:** 57-62.
78. **Schleien CL, Rogers MC.** *Cardiopulmonary resuscitation in infants and children.* In: **Rogers MC,** ed. Textbook of pediatric intensive care. Baltimore: Williams and Wilkins, 1987: 7-51.
79. **Chameides L, ed.** *Pediatric airway management.* In: Textbook of pediatric life support. Dallas: American Heart Association, 1990.
80. **Fisher DM.** *Anesthesia equipment in pediatrics.* In: **Gregory GA,** ed. pediatric anesthesia. 2nd ed. New York: Churchill Livingstone, 1987.
81. **Mehta S.** The risk of aspiration in presence of cuffed endotracheal tubes. *Br J Anaesth 1972;* **44:** 601-5.
82. **Gregory GA.** Respiratory care of the child. *Crit Care Med 1980;* 8: 582.
83. **Copper JD, Grillo HC.** Analysis of problems related to cuffs on endotracheal tubes. *Chest 1972;* **62:** 21S-27S.
84. **Stauffer JL, Olson DE, Petty TL.** Complication and consequences of endotracheal intubation and tracheostomy. *Am J Med 1981;* **170:** 65-76.
85. **Pollack MM, Ruttimann U, Getson PR.** Pediatric risk of mortality (PRISM) score. *Crit Care Med 1988;* **16:** 1110-6.
86. **Tellez D, Galvis A, Storgion, et al.** Dexamethasone in the prevention of postextubation stridor in children. *J Pediatr 1991;* **118:** 289-94.
87. **super DMM, Catelli NA, Brooks LJ, Lembo RM, Jumar ML.** A prospective randomized double-blind study to evaluate the effect of dexamethasone in aute laryngotracheitis. *J Pediatr 1989;* **115:** 323-9.
88. **Bishop MJ.** Mechanisms of laryngotracheal injury following prolonged tracheal intubation. *Chest 1989;* **96:** 185-6.
89. **Smith PG, Blumer JL.** Absence of adverse outcomes from the use of cuffed endotracheal tubes in pediatric patients. *Crit Care Med 1989;* **17:** S108.
90. **Kemper K, Benson M, Bishop M.** Predictors of postextubation stridor in pediatric trauma patients. *Crit Care Med 1991;* **19:** 352-5.

The Authors:

Fouzia Shaukat,
Medical Officer,
Department of Paediatrics,
Shaikh Zayed Hospital,
Lahore.

Sajid Maqbool
Professor & Head of
Department of Paediatrics,
Shaikh Zayed Hospital,
Lahore.

Address for Correspondence:

Fouzia Shaukat,
Medical Officer,
Department of Paediatrics,
Shaikh Zayed Hospital,
Lahore.