THE MANAGEMENT OF RESPIRATORY ACIDOSIS IN THE PAEDIATRIC AGE-GROUP

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The primary function of the lung is related to adequate uptake of oxygen and elimination of carbon dioxide in sufficient amounts to maintain pressures of oxygen (PO_2) and carbon dioxide (PCO_2) in arterial blood within certain limits. Impairment of respiratory function to such a degree that the PCO_2 rises above, or the PO_2 falls below, normal limits, would lead to respiratory failure. The respiratory system also plays a vital role in acid-base homeostasis as rapid changes in pH are possible by changes in the degree of ventilation. It is useful to consider bicarbonate as being under renal regulation and carbon dioxide as being controlled by the lung.

The purpose of this article is to discuss respiratory acidosis and its management in general terms and to illustrate some features with appropriate case histories.

Classification and Causes of Respiratory Acidosis

Respiratory acidosis (CO_2 retention) is commonly the result of ventilatory failure, which causes not only the alveolar PCO_2 to be high but invariably lowers the alveolar PO_2. The low PO_2 is probably the more serious defect in respiratory acidosis.

In a small number of patients there may be hypoxaemia *without* CO_2 retention. This occurs when there is a so-called alveolar-capillary block or, more frequently, a ventilation-perfusion imbalance. The discrepancy is due to the much greater diffusion capacity of carbon dioxide which is 20 times that of oxygen.

In childhood, respiratory acidosis occurs at all ages and is related to inadequate ventilation in the majority of cases. Since respiration depends on the harmonious balance between several systems and structures, failure may occur at any of the following levels: central nervous system, peripheral nerves, respiratory muscles, thoracic cage, upper respiratory tract, conducting airways, lung parenchyma, pleural cavities and pulmonary blood supply. The more common conditions encountered at various age periods include:

Neonatal period—asphyxia neonatorum; hyaline membrane disease; aspiration pneumonia; cerebral birth trauma; tetanus; and abdominal surgical conditions.

Infancy—stridor, occurring from infections or foreign bodies; bronchiolitis; pneumonia; and CNS depression from poisons.

Childhood—encephalitis; poliomyelitis; polyneuritis; asthma; and pulmonary oedema.

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Effects of Respiratory Acidosis

The hazards arising from the above conditions depend on certain factors and the significance of each may be considered in turn.

Hypoxaemia. The most disastrous effect of a low PO_2 arises from the damage to tissues, particularly of the heart and brain. Anaerobic metabolism produces a metabolic acidosis due to accumulation of organic acids, especially lactic acid, in the blood.[1] A low PO_2 also causes vascular constriction of the pulmonary bed with rise in the pulmonary artery pressure,[2] which places an added load on the right ventricle with eventual right heart failure.

Hypercapnia. Toxic effects (such as confusion and coma) are first noticed on the nervous system and probably follow cerebral oedema which occurs as a result of vasodilatation and increased cerebral blood flow.

The cardiovascular system is also adversely affected, both indirectly by the liberation of nor-adrenaline and adrenaline, and directly by a depressant action which may result in shock.[3]

Acidosis. Initially the retention of CO_2 gives rise to a respiratory acidosis with lowering of the blood pH, but later a superimposed metabolic acidosis appears for the reasons described above.

The depletion of glycogen stores and catabolism of tissue occurring in conditions in which the work of breathing is greatly increased, e.g. hyaline membrane disease,[4] may indirectly result in further accumulation of acids.

DIAGNOSIS

The diagnosis of respiratory failure depends both on clinical and laboratory findings.

Hypoxaemia

Signs of hypoxaemia may be present before cyanosis is obvious and vary from aggressive behaviour to later coma and death.[5] In the newborn infant, however, the early restlessness is often absent, even in cases of severe hypoxia, and probably reflects the limited role of the baby's cerebral hemispheres.[6]

In most cases cyanosis may be detected clinically but it can only be recognized confidently when the oxygen saturation is below 75%.[7]

The laboratory determination of oxygen saturation may be made by means of the Van Slyke manometric apparatus, or calculated from the PO_2 determined by means of an oxygen electrode. Both investigations require an arterial sample of blood which is collected in a heparinized syringe, immediately sealed off and placed on ice.

The collection of such a sample poses no problem in the newborn infant, since a polythene feeding catheter (size 32, French) can be passed via the umbilical artery and left in place for several days. In the older child, however, collection becomes more difficult and a temporal or femoral artery may be punctured to obtain the blood specimen.

Hypercapnia

The patient with CO_2 retention often appears drowsy or may develop coma with associated papilloedema and a raised cerebrospinal fluid pressure. A rise in blood pressure, tachycardia and strengthening of the heart beat is indicative of cardiovascular involvement and these signs may be followed by a fall in the blood pressure and shock. Often the signs of CO_2 retention are masked by the underlying disease and clinical diagnosis can be extremely difficult. The features only appear when the PCO_2 is above 100 mm.Hg[8] and can only occur when the inspired air is enriched with oxygen, as the highest PCO_2 possible while breathing air is about 90 mm.Hg. This degree of hypercapnia by itself does not appear to be dangerous and death usually results from the associated anoxia.

The calculation of PCO_2 is made on arterialized capillary blood using a suitable microtechnique[9] but should the necessary apparatus not be available, the rebreathing method for the measurement of mixed venous PCO_2 described by Campbell and Howell[10] may be employed.

The aim of this method is to bring the CO_2 in a small bag into pressure equilibrium with the blood so that the CO_2 tension can be measured in a gas rather than in a liquid. The simplicity and reliability of this technique for the estimation of mixed venous PCO_2 in apnoeic children of all ages has been confirmed.[11]

Acidosis

The signs of a superimposed metabolic acidosis are variable and depend both on the age of the child and the severity of the condition.

Deep respirations may be marked by underlying lung pathology and newborn infants often have a pale grey colour without other specific features.

As with the estimation of hypercapnia, laboratory confirmation is essential to establish the diagnosis and this too is determined on an arterialized sample of blood using a microtechnique.[9]

TREATMENT OF RESPIRATORY ACIDOSIS

The primary aim of therapy in most cases is to restore adequate alveolar ventilation. This includes the following:

1. *Removal of the Underlying Cause (where possible)*

Removal of an underlying cause may be sufficient to reverse the signs of insufficiency and this is particularly seen in cases of acute respiratory embarrassment, e.g. tension pneumothorax or a foreign body in the trachea.

Case 1—Spontaneous tension pneumothorax. Baby C was delivered by forceps because of the slowing of the foetal heart and the presence of meconium-stained liquor. The child weighed 7 lb. 12 oz. and had the appearance of postmaturity.

Shortly after birth the baby developed a rapid respiratory rate and became extremely irritable and cyanosed. A bulge of the right chest was observed and air entry into the right side was diminished, the diagnosis of a right tension pneumothorax being made and confirmed by X-ray.

Acid-base findings at 2 hours of age showed a pH of 7·18 and PCO_2 of 53 mm.Hg. Further deterioration necessitated tube drainage of the pneumothorax at 3 hours of age with relief of all abnormal clinical signs. The cyanosis disappeared, the respiratory rate dropped from 80/min. to 60/min. and air entry into the right chest improved. A repeat of the acid-base figures showed pH 7·40 and PCO_2 38 mm.Hg.

2. *Correction of Hypoxaemia*

Although probably the most dangerous result of respiratory failure, hypoxaemia is usually easily corrected by the administration of oxygen in either a tent or incubator. Since oxygen is extremely irritating and has a drying effect on mucous membranes, it is essential to humidify the gas either by passing it through a water bottle or a nebulizer situated as close as possible to the patient.

The amount of oxygen given should be sufficient to maintain the PO_2 within normal limits. From the clinical application, Warley and Gairdner[12] have indicated that oxygen given at concentrations sufficient only to abolish cyanosis, produces PO_2 levels far below normal. As a working rule, they state that oxygen should be administered at 25% higher concentrations than the minimum needed to abolish cyanosis. This level appears to be safe at all ages. Should cyanosis persist despite clear airways and a high environmental oxygen concentration, the administration of 100% oxygen by face mask or assisted mechanical ventilation is indicated.

3. *Correction of Hypercapnia*

Hypercapnia may prove difficult to control, particularly if oxygen is being administered to counteract cyanosis. Should PCO_2 levels reach dangerous heights, viz. 90 - 100 mm.Hg, tracheostomy may prove beneficial, not only by substantially reducing the deadspace but by facilitating aspiration of the airways. At this stage, failure to improve ventilation and reduce PCO_2 is a further indication for mechanically assisted respiration.

4. *Keeping Airways Clear*

Clear airways are essential for adequate ventilation, as no amount of oxygen or artificial respiration can remove a large plug of mucus. It is therefore necessary to keep the air passages free from obstruction by frequent coughing and suction, routine posturing of the patient and physiotherapy to the chest. These principles apply to all age-groups.

Adequate oral fluids must be administered in all cases of chest pathology to maintain hydration, as water is still the best expectorant. The dangers of underhydration are well illustrated in cases of status asthmaticus and reverse pneumonia where children may refuse fluid and within a few days thick plugs of dry sputum clog the airways with resultant collapse of lobes.

Conversely, overhydration from the administration of excessive amounts of intravenous fluid must be avoided, particularly in cases of abnormal, already congested lungs. A tracheostomy may be required in cases with severe obstruction of the upper respiratory tracts as seen in congenital or acquired laryngeal stridor. A humidified atmosphere is essential when air bypasses the upper respiratory tract in tracheostomy cases and may be achieved either by the use of steam from a kettle or the passage of compressed air through a nebulizer.

Should thick secretions form, despite these precautions, they may be loosened by the instillation of a few drops of 4% sodium bicarbonate or normal saline, down the tracheostomy tube.

5. *Assisting Ventilation*

Should the above measures fail to correct the effects of inadequate ventilation or should respirations cease for any reason, assisted mechanical ventilation may be embarked upon. Although fraught with difficulties, the results are often most rewarding as illustrated below.

Case 2—Brain stem encephalitis and polyneuritis. J.T., aged 5 years, was admitted with a 2-day history of unsteady gait and gross ataxia. No poisons had been ingested and no disease contacts were reported.

Examination revealed a fully conscious apyrexial Coloured girl; weakness of all limbs was present, palatal movements were poor and the cervical muscles appeared weak. The condition progressed over the next few days and she developed complete lower motor neurone paralysis of the limbs and respiratory muscles. Diaphragmatic movements remained intact and cerebrospinal fluid was normal.

The bulbar signs now became more gross with pooling of saliva in the pharynx and difficulty in swallowing. Despite postural drainage for removal of secretions and later a tracheostomy, she developed evidence of gross underventilation and became extremely confused and then semiconscious.

Artificial ventilation was then applied by means of an Engström respirator. In spite of complete paralysis she became coherent, well orientated and cyanosis disappeared.

She was maintained on intermittent positive-pressure respiration for 3 weeks and over this period of time complete recovery of power in all limbs and chest muscles returned.

This case illustrates several points. In particular it may be noted that the mental changes were solely due to respiratory failure and not to the underlying encephalitis and, secondly, that the cyanosis was corrected by ventilating her normal lungs with room air. She did not require an oxygen-enriched atmosphere and had this been administered initially without correcting the inadequate ventilation, CO_2 retention would have occurred with probable further aggravation of her mental state.

Although this case demonstrates the efficacy of assisted ventilation in selected patients, this form of therapy is not synonymous with adequate alveolar ventilation and must not be embarked upon lightly. Adequate laboratory and specialized nursing facilities are necessary to prevent both underventilation and overventilation.

6. *Correcting Acidosis*

A respiratory acidosis *per se* requires no specific therapy, but in advanced cases of pulmonary insufficiency the associated metabolic acidosis can be corrected by the intravenous administration of sodium bicarbonate.

The following case illustrates several parameters of respiratory failure and successful correction of each aspect:

Case 3—Hyaline membrane disease. A premature baby, B.K., weighing 5 lb. 3 oz., was delivered at 36 weeks gestation by caesarean section. The mother had a type 4 placenta praevia and had experienced severe antepartum haemorrhages.

Ten minutes after birth the baby was noted to have rapid respirations, associated rib recession and generalized oedema. Expiratory grunting became audible and an X-ray of the chest at this stage showed a pattern in keeping with hyaline membrane disease.

At the age of 1 hour clinical cyanosis was obvious, and an incubator flow of 4 l. O_2/min. was necessary to maintain a good colour. At the age of 2 hours the baby appeared more distressed, mucous membranes were a pale grey colour and acid-base studies revealed pH 7·14, PCO_2 of 61 mm.Hg and BE −9·7. This was consistent with a combined respiratory and metabolic acidosis and over the next 6 hours the infant received 15 mEq. of sodium bicarbonate in 20% dextrose water intravenously. Acid-base figures now showed pH 7·34, PCO_2 50 mm.Hg, BE −1·5 and PO_2 140 mm.Hg.

This metabolic treatment of respiratory distress is based on the assumption that the lethal effects of the disease are due largely to disturbances secondary to pulmonary failure. The correction of pH and the provision of carbohydrate enables the infant to survive[13] while the pulmonary disorder improves either spontaneously or with further therapy.

At 30 hours of age the baby suddenly collapsed, with a period of apnoea and marked cyanosis. Peripheral pulses were poorly palpable and the right chest was noted to be hyperresonant, supporting the diagnosis of a superimposed tension pneumothorax. Acid-base studies revealed pH 7·2, PCO_2 58 mm.Hg and PO_2 45 mm.Hg.

Despite drainage of the pneumothorax, respirations remained inadequate to maintain ventilation and cyanosis persisted although 100% oxygen had been administered. An endotracheal tube was passed and ventilation was assisted by means of a Bird respirator. This led to an immediate improvement in the clinical state: pH now 7·42, PCO_2 42 mm.Hg and PO_2 178 mm.Hg. Respiratory therapy was continued for a further 5 days and the course thereafter was uneventful.

SUMMARY

The concept of respiratory acidosis is a functional one resulting in a lowered PO_2, a raised PCO_2 and an associated metabolic acidosis. The condition occurs at all ages of childhood and is related to inadequate ventilation. The diagnosis is based on both clinical and laboratory findings.

The aim of therapy is to maintain normal ventilation by relief of hypoxaemia, prevention of hypercapnia and correction of acidosis. The clinical application of this is related to removing the underlying cause where possible, administering oxygen, keeping the airways clear and in the more severe cases, reducing the airway deadspace by tracheostomy. Should these measures prove to be of no avail, assisted mechanical ventilation may be applied. This form of therapy is hazardous in unskilled hands and requires specialized nursing and laboratory facilities.

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