

## RESPIRATORY DISTRESS IN NEWBORN: TREATED WITH VENTILATION IN A LEVEL II NURSERY

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### ABSTRACT

*Fifty consecutive neonates with respiratory distress persisting beyond 6 h of age were studied during a 18 month period (total deliveries 2000/y). Twenty two neonates were managed with oxygen hood with increasing oxygen concentration, 28 with continuous positive airway pressure (CPAP) ventilation using a nasal cannula. Of these babies on CPAP, 10 were shifted to intermittent positive pressure ventilation (IPPV) on a pressure limited, time cycled ventilator (Neovent, Vickers). Babies were monitored with continuous hemoglobin oxygen saturation (SaO<sub>2</sub>), hourly blood pressure and vital charting. Radial arterial blood gas analysis (ABG) was done when feasible and especially on clinical deterioration. Oxygen (FiO<sub>2</sub> 0.95) from an oxygen concentrator was used as a source of continuous supply of oxygen. Commonest cause of respiratory distress was hyaline membrane disease (18%), followed by wet lung syndromes (14%), meconium aspiration (12%), asphyxia (12%) and septicemia (8%). In 8 babies, a lung biopsy (postmortem) was done to confirm the diagnosis. Nineteen of the 50 babies with respiratory distress died, there was a survival of 50% on CPAP and 30% on IPPV. No case of oxygen toxicity or other major complications was encountered. Even with moderate resources, neonatal ventilation in a Level II nursery is a*

Respiratory distress is the most common problem in neonatal nurseries. It results from a variety of causes and an urgent work up is essential. Yet for hypoxemia some form of assisted ventilation is immediately warranted. The outlook of babies with respiratory distress syndrome has changed after the first use of continuous positive airway pressure (CPAP) by Gregory *et al.*(1). It is now an established modality for neonates for many years.

In India, a survival of 100% in babies more than 1.5 Kg on CPAP mode is reported(2). It is also recommended that neonatal ventilation should be ventured in centres where basic facilities for Level II care already exist. Ours is a well equipped nursery recognized by the National Neonatology Forum (NNF). We treated 50 babies with respiratory distress, of which 31 survived.

### Material and Methods

In a prospective study, we analyzed the causes of respiratory distress and indications for ventilation in newborns during a 18 month period (Jan '92-June '93). There were a total of 2931 booked deliveries during this period in our hospital which caters to a mixed Indian

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*challenging task. Babies less than 1000g require aggressive measures which is not very economical in a special care baby unit (SCBU).*

**Key words:** Neonatal ventilation, Respiratory distress.

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population. For the diagnosis of respiratory distress, the important signs are respiratory rate more than 60 per minute, grunt and sub-costal recessions. These signs being non-specific, we consider two out of three of these as confirmative of respiratory distress(3). Rarely, apneic attacks may present as a sole manifestation of ventilatory failure.

As the spectrum of respiratory distress in newborns is large, we start a workup at four hours of age and give a provisional diagnosis at 6 hours of post natal life. The initial workup included a gastric aspirate shake test, chest X-ray and an arterial blood gas (ABG) analysis. Blood pressure and vital signs were recorded. Oxygen saturation (SaO<sub>2</sub>) and heart rate were monitored using a pulse oximeter (Ohmeda Biox 3760). Oxygen from a oxygen concentrator (Air Sep Forlife) with FiO<sub>2</sub> 0.95 was used to supply oxygen. Intravenous fluids were started at the rate of 60 ml per kg body weight of 10% dextrose solution. If clinical condition warranted antibiotics, blood cultures were taken prior in 10 ml culture bottles. Downes RDS Score(4) was recorded and babies with a score of six or more were put on CPAP with a nasal cannula (Argyle). Babies with a score of five or less were managed with oxygen hood with increasing FiO<sub>2</sub> concentrations.

The indication for giving CPAP were (i) Downes score of 6 or more; (ii) inability to maintain a SaO<sub>2</sub> of 87% with oxygen hood; (iii) PaO<sub>2</sub> of less than 50 mm Hg; and (iv) radiological evidence of severe hyaline membrane disease (HMD) with a negative shake test. CPAP is considered a failure if a baby has (i) inability to maintain a SaO<sub>2</sub> of

87% with CPAP of 12 cm H<sub>2</sub>O and FiO<sub>2</sub> of 0.9; (ii) PaO<sub>2</sub> <50 mm Hg with FiO<sub>2</sub> 0.9; (iii) pH <7.25, PaCO<sub>2</sub> >60 mm Hg; and (iv) recurrent (more than 3) apneic attacks as a manifestation of respiratory failure. CPAP failures were shifted to intermittent positive pressure ventilation (IPPV) mode on a time cycled, pressure limited continuous flow neonatal ventilator (Neovent, Vickers). We use minimal ventilatory settings depending on the lung pathology to achieve a SaO<sub>2</sub> of 90±3% or PaO<sub>2</sub> 60-80 mm Hg. Subsequent ABG was done through repeated radial arterial punctures at 6-12 hourly intervals. The definitions suggested by NNF Group on Neonatal Nomenclature(3) were accepted. Babies delivered with a thick meconium stained liquor were intubated prior to first breath. Sepsicemia was diagnosed if the blood culture grew pathogenic organisms. Immaturity was labelled when a baby less than 1000 g had no other primary cause of death.

## Results

Out of the 50 babies enrolled in the study, 26 were males and 24 females. There were 40 babies with low birth weight; of these 10 babies were less than 1000g. Mean birth weight was 1823 g (range 740-3900 g) and mean gestational age 33 wk (range 26-42 wk). The smallest baby a non survivor was 740 g with a gestational age of 26 weeks. *Table I* depicts the modes of oxygenation. Twenty two babies were managed with oxygen hood and 28 required CPAP ventilation. Of these 10 babies had a failure of CPAP ventilation and were shifted to IPPV mode. The mean duration of CPAP mode was 53 h (range 11-156 h) and IPPV mode 46 h (range 7-74 h).

Hyaline membrane disease (18%) was the commonest cause of respiratory distress, followed by wet lung syndromes (14%), meconium aspiration syndrome (12%), asphyxia (12%) and

septicemia (8%) (*Table II*). There was one baby 1000 g who developed chronic pulmonary insufficiency of prematurity (CPIP) and was discharged with a weight of 1750g. In 8 cases (birth weight

TABLE I—Birth Weight in Relation to Mode of Ventilation

| Weight (g) | CPAP only |           | IPPV |           | Oxygen hood |           |
|------------|-----------|-----------|------|-----------|-------------|-----------|
|            | n         | Survivors | n    | Survivors | n           | Survivors |
| <1000      | 6         | 1         | 2    | -         | 2           | 2         |
| 1001-1500  | 5         | 4         | 3    | 1         | 1           | 1         |
| 1501-2000  | 4         | 2         | 5    | 2         | 5           | 3         |
| 2001-2500  | -         | -         | -    | -         | 7           | 6         |
| >2500      | 3         | 2         | -    | -         | 7           | 7         |
| Total      | 18        | 9         | 10   | 3         | 22          | 19        |

TABLE II—Etiology and Incidence of Respiratory Distress

| Etiology                     | n  | Incidence (%) | Survivors |
|------------------------------|----|---------------|-----------|
| Hyaline membrane disease     | 9  | 18            | 1         |
| Wet lung syndromes           | 7  | 14            | 7         |
| Meconium aspiration syndrome | 6  | 12            | 6         |
| Asphyxia                     | 6  | 12            | 4         |
| Apnea                        | 5  | 10            | 5         |
| Septicemia                   | 4  | 8             | 2         |
| Immaturity                   | 3  | 6             | 0         |
| Congenital heart disease     | 2  | 4             | 0         |
| Congenital pneumonia         | 2  | 4             | 2         |
| Miscellaneous*               | 6  | 12            | 4         |
| Total                        | 50 | 100           | 31        |

\* 1 case each of aspiration pneumonia, chronic pulmonary insufficiency of prematurity, hypothermia, hypoglycemia, laryngotracheomalacia and laryngeal stridor.

900-2000g) with a clinical diagnosis of HMD, the cause of death was confirmed by a postmortem lung biopsy. In all these cases, the histopathological features were consistent with hyaline membrane disease. *Table III* depicts the survival in relation to the birth weight and mode of ventilation. There was a 50% survival on CPAP and 30% on IPPV mode. Survival of babies less than 1000g was 30%. Three babies had a symptomatic ductus arteriosus (PDA) and were managed on standard lines. No baby developed pneumothorax or other major complications of ventilation or oxygen therapy.

### Discussion

Majority of the medical colleges in our country lack the basic infrastructure to ventilate babies(2). Ventilation has only been reported from tertiary centres and experience from Level II nurseries is lacking. We have the expertise but the resources are limited. At the same time, IPPV and aggressive monitoring results in a significant iatrogenic morbidity. Within the frame work of the physics of ventilation every nursery evolves its protocols and settings for ventilation.

As oxygen is a drug, we choose a mid-line path whereby the complications are minimum. The CPAP mode with nasal cannula is an easy way to improve oxygenation. This makes the routine endotracheal intubation of infants requiring only continuous airway distending pressures no longer justifiable(5).

Once oxygenation is adequate and signs of respiratory distress decrease, an optimal CPAP is achieved. CPAP failures appear to have an ominous prognosis regardless of the birth weight(5). In our series, we had 10 babies less than 1000g and the mortality in them was 70%. CPAP is not an ideal treatment for them and they may require IPPV at the outset(2). Chronic pulmonary insufficiency of prematurity is a distinct entity as these babies are less than 1000g and have a normal chest X-ray at birth but are oxygen dependent at 3-4 weeks of age. We agree with Singh *et al.* in considering a cut off of immaturity at 750 g(2). In a review of literature(5) the incidence of pneumothoraces was 0 to 14%. We had no case of pneumothorax or other major complications of oxygen toxicity.

TABLE III—Survival in Relation to Birth Weight

| Weight (g) | n  | Survival | Survival (%) |
|------------|----|----------|--------------|
| <1000      | 10 | 3        | 30.0         |
| 1000-1500  | 9  | 6        | 66.6         |
| 1501-2000  | 14 | 7        | 50.0         |
| 2001-2500  | 7  | 6        | 85.7         |
| >2500      | 10 | 9        | 90.0         |
| Total      | 50 | 31       | 62.0         |

Pulse oximetry is a reliable technique for monitoring of oxygenation in newborns(6,7). It is a handy tool in a Level II nursery as apart from SaO<sub>2</sub> it displays the heart rate. It obviates the use of an electrocardiograph and apnea monitors. In managing cases of respiratory distress, it has few limitations and the artifacts are avoidable. Fanconi *et al.*(8) constructed oxygen dissociation curves with pulse oximeter SaO<sub>2</sub>, measured SaO<sub>2</sub> and calculated SaO<sub>2</sub>; they were all similar. A pulse oximeter reading of 85% to 90% is a clinically safe target. We maintain a SaO<sub>2</sub> level of 90±3% and titrate FiO<sub>2</sub> to that level. On clinical deterioration and failure to maintain saturation, an ABG helps in correcting the acid base defect. As oxygen is always at a premium in a Level II nursery, oxygen (FiO<sub>2</sub> 0.95) from an oxygen concentrator is a useful equipment to supply oxygen.

Getting a neonatal autopsy in India is a difficult task. We have found a lung biopsy done immediately after death an easy method to come to a diagnosis. In 8 of our babies, biopsy showed features suggesting HMD. Considering babies more than 1000g, we have a survival of 70% and only 30% in less than 1000g. These babies require aggressive measures which is not very economical in a Level II nursery.

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