Bronchopulmonary dysplasia: a new look at management

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Bronchopulmonary dysplasia is an unacceptable but, at present, seemingly unavoidable complication of neonatal intensive care. There are many theories as to why this disorder arises, including lung immaturity, barotrauma from positive airway pressure ventilation, oxygen toxicity, respiratory infections after intubation, and increased lung fluid. Definitions vary, but based on the physiological definition of oxygen dependency at 28 days of age, Boynton has estimated that in the USA 3100 infants are suffering from chronic lung disease at any given time.[1] Based on similar calculations, there may be up to 730 in the UK. After initial hospital discharge, there is a high rate of readmission (up to 60%) and subsequent death (up to 20%).[2]

Having been referred over 90 infants with severe bronchopulmonary dysplasia over the last three years, we have updated our management of infants both in hospital and at home. This paper aims to survey conventional treatment in combination with the presentation of new ideas, including the maintenance of 'normal' oxygenation and the use of negative pressure ventilation as a non-invasive respiratory support.

Clinical management in hospital

(1) DIFFERENTIAL DIAGNOSIS

Infants undergo the following investigations to exclude additional problems where management may be affected: an echocardiogram (for example, to exclude patent ductus arteriosus or anomalous pulmonary venous drainage), sweat test, nasociliary studies, immunoglobulins (including IgE). In selected cases ventilation-perfusion scans may rarely confirm a localised lung defect.

(2) PREVENTION OF HYPOXAEMIA

Inadequate airway oxygenation, as reflected by arterial or transcutaneous measurements, will result in a greater tendency to develop pulmonary vascular hypertension and cyanotic episodes associated with a right to left arteriovenous intrapulmonary shunt.[3] Such episodes may be induced by crying or handling,[3][4] feeding,[5] and infection.[6] They are potentially dangerous and may result in a pulmonary vascular crisis (a major hypoxaemic episode) or sudden death.[7]

In addition to the acute and episodic development of a right to left intrapulmonary shunt, chronic intrapulmonary shunting from inadequate airway oxygenation may produce hypoxaemia over and above that directly due to alveolar hypoventilation. Such shunting may also lead to interstitial pulmonary oedema, simi-

lar to that arising from high altitude exposure[8] and may explain why diuretics are of value in some patients with bronchopulmonary dysplasia (see later). While we may use diuretics for established pulmonary hypertension and right heart failure, we would advocate the prevention of this complication. The presence of an accentuated pulmonary component of the second heart sound, right ventricular hypertrophy on electrocardiography or echocardiography, and cyanosis on crying or feeding indicate that airway oxygenation has been inadequate.

The persistent abnormalities in lung surfactant, which have been demonstrated in infants with bronchopulmonary dysplasia,[9] may also result from underperfusion or hypoxia of the alveoli. Thus an adequate airway oxygen tension and adequate effective pulmonary blood flow (that is, without a shunt) may also be important in maintaining function of the alveolar type I and type II cells.

Inadequate oxygenation may also be one cause of the failure to thrive seen in patients with bronchopulmonary dysplasia. There is evidence that additional inspired oxygen may reverse poor weight gain[10] and that weaning infants from additional inspired oxygen too early such that they sustain prolonged periods with arterial hypoxaemia (arterial oxygen saturation (SaO_2) <95%) may divert energy from growth (J R Groothius; Ross Laboratories Special Conference on Bronchopulmonary Dysplasia, Washington DC, December 1989).

Non-invasive and continuous monitoring of oxygenation would ideally involve both accurate transcutaneous oxygen tension $(TcPO_2)$ and SaO_2 measurements. Fanconi has shown that pulse oximeters from different manufacturers have different responses, and that before clinical use in neonates they should be validated against arterial line samples measured using a co-oximeter which compensates for fetal haemoglobin.[11] The Nellcor and Ohmeda pulse oximeters are the models most adequately validated in infants,[11–13] although the Ohmeda is less adequate at detecting hyperoxaemia[14] and may under-read the true SaO_2.[15]

In this age group $TcPO_2$ is far less accurate at detecting airway hypoxia than pulse oximetry, in part because of changes in skin blood flow with increasing age.[16] Moreover, the oxygen dissociation curve can be shifted to the right or left depending on 2,3-diphosphoglycerate and fetal haemoglobin concentrations,[17] thus altering the relationship between arterial oxygen pressure (PaO_2) and SaO_2. Although $TcPO_2$ is useful as a trend measurement (see later), we find pulse oximetry a more accurate measure of

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arterial oxygenation and therefore more useful in the definitive evaluation of airway hypoxia.

Before ensuring adequate oxygenation for any infant it is essential to know what is normal at that age and gestation.[18] Two recent investigations performed in our department are relevant. Overnight (12 hour) tape recordings of beat to beat SaO_2 (using a Nellcor N100 pulse oximeter), breathing movements and electrocardiogram have been performed on healthy full term infants at the following ages: in the first month of life (n=43), at around 6 weeks of age (n=67), at around 3 months of age (n=19), and around 6 months of age (n=18). At 6 weeks of age, the median value for the baseline SaO_2 during regular breathing, a state closely correlated with quiet sleep, was 99·8% (range 97·0 to 100%).

In a similar study on an unselected sample of 66 preterm infants at the time of discharge from special care, the median baseline SaO_2 during regular pattern breathing was 99·4%. Moreover, 63 of the 66 subjects had baseline values above 95%.

Thus most preterm and full term infants manifest baseline SaO_2 values between 97 and 100%. These values should therefore be considered as normal when providing additional inspired oxygen to compensate for airway hypoxia resulting from bronchopulmonary dysplasia. Because of the potential dangers of hyperoxaemia in the infant *before* term, the SaO_2 while on additional inspired oxygen must not be permitted to remain at 100% for any length of time. Allowing for an error of ± 2% on the Nellcor pulse oximeter,[12] we maintain SaO_2 between 95 and 98% in patients with bronchopulmonary dysplasia.

When the inspired oxygen requirement is 40% or less maintenance of an adequate inspired oxygen pressure (PO_2) can be achieved by neonatal nasal cannulae (for example, De Vilbiss, Feltham, Middlesex). More than 40% inspired oxygen is provided using a headbox. However, retention of the nasal cannulae may minimise swings in oxygenation that result from disturbances to the headbox oxygen content. It is also important to be aware that handling and medical or nursing procedures, which result in hypoxaemia,[4] may also interrupt the amount of oxygen supplied to the infant at a time when particularly needed. Constant non-invasive monitoring of oxygen concentrations may help prevent iatrogenic hypoxaemia:[19] [20] clinical detection based on cyanosis alone will detect only more severe disturbances. When a patient is demonstrating falls in SaO_2/PO_2 on feeding, we provide extra additional inspired oxygen before and during such events. This may mean temporarily increasing the inspired oxygen level from 40 to perhaps 70 or 80%. Noxious procedures, such as the taking of blood or passage of nasogastric tubes, produce crying and may result in severe hypoxaemia. We always provide additional oxygen before such procedures and, as clinically appropriate, adequate local or systemic analgesia or sedation. If an infant is severely upset by a procedure, return to the previous requirements of additional inspired oxygen may take up to several hours.

An additional way of improving oxygenation involves the use of continuous or intermittent negative pressure ventilation.[21] This is described below.

(3) CARBON DIOXIDE RETENTION

Some patients with bronchopulmonary dysplasia have few problems with carbon dioxide retention, suggesting that most of their disease process has selectively affected alveolar gas exchange. Some patients, however, have predominantly small airway disease, recognised by wheeze or evidence of air trapping. In any patient with bronchopulmonary dysplasia and a high arterial carbon dioxide pressure ($PaCO_2$) in the absence of severe oxygenation problems, one must ensure that upper airway obstruction, most commonly resulting from subglottic stenosis, is not present. Laryngeal injury, resulting from endotracheal intubation, has been reported to occur in up to 55% of intubated infants[22] and may be clinically silent. Diagnosis may be achieved by carefully watching the infant's pattern of breathing when asleep, by listening over the larynx with a stethoscope and by tape recordings of inspiratory waveform pattern (inductance plethysmography), SaO_2 and carbon dioxide pressure (PCO_2) concentrations during sleep.[23] Ultimately, endoscopy may be indicated.

Lower airway obstruction may be partly relieved by corticosteroids, methylxanthines, nebulised β agonists or anticholinergics, and occasionally by diuretics. We have also found continuous negative extrathoracic pressure (CNEP) to be of some value; perhaps by increasing the transthoracic pressure gradient, thereby improving airway patency during expiration and effectively replacing the auto positive end expiratory pressure (PEEP) or intrinsic PEEP caused by small airway closure at end expiration.[24] One promising new treatment is the use of nebulised budesonide, a corticosteroid (Astra Pharmaceuticals). Unlike systemic corticosteroids[25] (A Grant; Ross Laboratories Special Conference on Bronchopulmonary Dysplasia, Washington DC, December 1989) neither CNEP or nebulised budesonide have yet been subjected to randomised controlled trial.

(4) NEGATIVE PRESSURE VENTILATION

We use this treatment if more than 40% inspired oxygen is required to maintain SaO_2 at 95 to 98% when quiet or asleep, if there is evidence of noticeable respiratory distress with failure to thrive, and to help in reducing the deterioration that occurs when some infants are weaned from systemic corticosteroids. It is also valuable during intercurrent respiratory infections, particularly in situations where respiratory failure is worsening and further respiratory support is indicated. Being non-invasive it may be used earlier than one would subject an infant to intubation[21] and without the adverse haemodynamic effects produced by intermittent positive pressure ventilation (IPPV). Although positive airway pressure techniques increase

ventilation, they increase pulmonary vascular resistances and reduce cardiac output and effective pulmonary blood flow. Thus the gains from improved ventilation may not be fully reflected by improvements in tissue oxygenation. Negative extrathoracic pressure improves ventilation and pulmonary perfusion. Negative pressure may be continuous (CNEP -6 to -8 cm H_2O) or intermittent (intermittent negative extrathoracic pressure of $-35/-6$, rate 10-60/minute). The latter is initiated when there is increasing carbon dioxide retention or the infant has become tired. CNEP may be given overnight only; the patient breathing unaided during the daytime.

(5) AVOIDANCE OF ANAEMIA

There are two reasons why anaemia should be avoided. Firstly the adverse effects of anaemia on the delivery of oxygen to tissues may impair optimal tissue growth,[17] particularly in the lung. Secondly anaemia may increase the incidence of cyanotic episodes.[26] Correction of anaemia has been shown to reduce the incidence of hypoxaemic episodes,[26] reduce oxygen consumption,[27] and decrease the incidence of apnoeic pauses and periodic breathing.[28][29] We therefore aim to keep the haemoglobin concentration between 110 and 140 g/l. Regular iron and folic acid supplements are provided until the infant is on an adequate mixed diet. If the haemoglobin falls below 110 g/l a blood transfusion is given.

(6) DIURETICS

Controlled trials have shown that diuretics may improve respiratory function in patients with bronchopulmonary dysplasia.[30-32] We use a combination of frusemide (1 mg/kg/dose two to three times per day) and spironolactone (1-1·5 mg/kg twice daily) if right heart failure is present or if there is radiological evidence of interstitial fluid with a persistent oxygenation defect. As described above, however, it should be possible to prevent pulmonary oedema and right heart failure by maintaining adequate airway oxygenation thus avoiding the need for diuretics. Frusemide may cause hypercalcuria and thus in infants with a high alkaline phosphatase activity or radiological evidence of rickets or nephrocalcinosis, chlorthiazide (10–20 mg/kg twice a day) is preferable.

(7) SYSTEMIC CORTICOSTEROIDS

Controlled trials have indicated earlier extubation with corticosteroids, although the durations of oxygen dependency and hospital stay may not be helped.[33][34] Where there is increasing carbon dioxide retention or inability to wean from IPPV we use a three to five day course of dexamethasone at full dosage (0·25 mg/kg every 12 hours). If there is no initial improvement it is discontinued immediately. When an improvement has occurred it is tailed off over seven to 21 days. In those infants in whom deterioration occurs on stopping corticosteroids, we may use nebulised budesonide (0·5–1·0 mg 12 hourly) or alternate day steroids.

We are cautious in commencing corticosteroids because of their predisposition to and suppression of signs from infection. In addition long term effects of these drugs on the development of the fibrous tissue matrix within the lung warrant further study.[35][36]

(8) NUTRITION

As overall body growth is likely to be accompanied by increased lung development, we aim to optimise energy input and utilisation providing 0·63 MJ/kg/day (150 kcal) by supplementing feeds with additional carbohydrate (for example, Polycose (Abbott), Polycal (Cow and Gate), and Maxijul (Scientific Hospital Supplies)) and fat (for example, Liquigen, Calogen, and Duocal (all Scientific Hospital Supplies)). In the infant with persistent respiratory distress, energy consumption may be reduced by minimising the work of breathing, as for example using negative pressure ventilation.[21]

As a compliant ribcage will impair gas exchange in the lung and result in persistent respiratory distress, we monitor for the development of osteopenia of prematurity.[37] In addition to vitamin D supplements, we may also provide calcium and phosphate. Deficiency of the latter may also contribute to persistent respiratory distress from muscle weakness. The potential effects of diuretics and steroids on calcium metabolism also have to be considered (see above).

(9) INFECTION AND ITS TREATMENT AND PREVENTION

Intercurrent respiratory infection frequently begins as a viral infection. Respiratory syncytial virus may be a particular problem for preterm infants in neonatal units as well as the community,[38] with staff sometimes acting as the transmitter of infections.[39] What may simply be a cold or upper respiratory infection for the average baby can be devastating for a patient with bronchopulmonary dysplasia. It is therefore our policy to provide increased protection for all such patients. Parents and nursing or medical staff with an upper respiratory tract infection should rigidly avoid contact with such patients. In the most susceptible infants reverse barrier nursing may be provided. As soon as an upper respiratory tract infection is suspected, a nasopharyngeal aspirate should be taken for immunofluorescence in order to identify respiratory syncytial virus, parainfluenza, or adenovirus, for which it may be helpful to administer nebulised ribavirin. Adenovirus may be particularly problematic as bronchiolitis obliterans may make chronic lung changes irrepairable.

In the systemically ill infant (that is, with a fever, a leucocytosis, increased C reactive protein, and additional changes on chest radiography) we will administer a minimum five day course of broad spectrum intravenous antibiotics.

There is some evidence that *Ureaplasma urealyticum* infection may increase the likelihood of chronic lung disease in preterm infants.[40] We consider administering a seven day course of

erythromycin on an empirical basis in the presence of a fever, with or without respiratory symptoms after blood, endotracheal tube, throat, and urine cultures. In most instances, however, the cause of the fever is likely to be viral in origin and the results from serology may help identify the pathogen.

It is important to be aware of the potential danger of the less common pathogens such as monilia, particularly in the patient on corticosteroids. If there is a conjuctivitis, special swabs for chlamydia are taken and the infection treated with a minimum 10 day course of systemic erythromycin.

Early immunisation against pertussis is extremely important. This is begun at three months after delivery, whether or not the patient is in hospital.

(10) MONITORING

All of our patients with bronchopulmonary dysplasia are continuously monitored using a Nellcor pulse oximeter for assessment of baseline hypoxaemia and daily measurement of additional inspired oxygen requirement (see below).[41] During intercurrent respiratory infections, transcutaneous carbon dioxide pressure $TcPCO_2$) is monitored using a sensor heated to 42°C (Hewlett Packard/Draeger electrode) and changed every 12 hours. If there is any deterioration in $TcPCO_2$ or in oxygenation, capillary gas samples can be taken for an analysis of pH, PCO_2, standard bicarbonate, and base excess.

The measurement of additional oxygen requirement is used to assess respiratory function on a daily basis. In non-intubated patients this procedure involves placing the patient's head in a headbox, switching off the nasal cannula oxygen supply, providing oxygen in the headbox, and placing an oxygen analyser (calibrated in air and 100% oxygen) next to the infant's nose. The level of inspired oxygen required to keep the SaO_2 between 95 and 98% is assessed when the patient is quietly asleep. We expect to see oxygen requirements stable—that is, not changing by more than 0·25 l/min through the nasal cannulae, before discharge home is considered.

Home management

PREPARATION FOR HOME

Before discharge a planning meeting is held to which are invited key workers who have been, or will be, responsible for the care of the baby (see table 1). At this meeting the items listed in table 2 are discussed and channels of communication provided.

HOME MONITORING

Preterm infants are at increased risk of sudden, unexpected death (between 1/42 and 1/200).[42-46] Those with bronchopulmonary dysplasia are probably at greater risk, whether in hospital[7] or at home.[47] Although these deaths may not be classified as sudden infant death syndrome, the mechanism being thought by some

Table 1 Key workers invited to discharge planning meeting

Unit/ward nurses	Hospital paediatric staff
Family practitioner	Health visitor
Clinical nurse specialist	Liaison health visitor
Social worker	Parents

Table 2 Items for discussion at discharge planning meeting

(1)	Home oxygen supply: concentrators, cylinders
(2)	Home PO_2 monitor: instruction
(3)	Training in resuscitation
(4)	Training in respiratory support—for example, negative extrathoracic pressure
(5)	Training in nasogastric tube feeding and changing
(6)	Discussion of medications, immunisations
(7)	Notification of: Fire department Electricity department British Telecom (priority fault repair service) Insurance company (for building/equipment)
(8)	Identification of lines of communication for medical/equipment problems
(9)	Discharge transport
(10)	Respite care
(11)	Financial help
(12)	Follow up: Home visits (health visitor, clinical nurse specialist) Outpatient

to be different in infants with chronic lung disease, home monitoring is appropriate in this group of infants.[48] Clinical recognition of hypoxaemia is poor; it may easily be undetected[5] (JR Groothius; Ross Laboratories Special Conference on Bronchopulmonary Dysplasia, Washington DC, 1989), and this may be the precursor for more major life threatening events.[3] For the early detection of major cyanotic/apnoeic events, we use a $TcPO_2$ monitor (Kontron 821S). The sensor for this instrument is heated to 43°C and its site changed every eight hours.[49] Although skin PO_2 measurements do not accurately measure PaO_2, compared with SaO_2 measurements from validated pulse oximetry, they are less prone to movement artefact, produce fewer false alarms and may be used continuously when the infant is discharged home. They are also able to identify falls in baseline skin PO_2 indicative of arterial hypoxaemia, for example in the setting of an intercurrent respiratory infection. In 28 infants with bronchopulmonary dysplasia, seven had falls in baseline PO_2, identified by the monitor, and later proved to be hypoxaemia by a validated pulse oximeter in hospital.[49] Three subsequently needed respiratory support because of progressive respiratory failure. Parents rapidly become aware of changes in baseline PO_2 and respond by arranging medical assessment of the infant's clinical state and confirmation or otherwise of arterial hypoxaemia by pulse oximetry. The monitor is also an important tool for the surveillance of additional inspired oxygen supply at home (see below).

DOMICILIARY OXYGEN TREATMENT

To administer oxygen, a home based and a mobile supply both need to be available. The first is provided by an oxygen concentrator which is prescribable by the family practitioner.[50] These are provided by either De Vilbiss, Omnicare, or Rimer-Alco (depending

on area), who will visit the home and install the most appropriate system to suit the parents' living conditions. The cost and convenience of a concentrator makes this much more suitable than large cylinders for long term oxygen supply, even taking electricity costs into account. Cylinders are heavy, run out, have a delivery charge of £23, and ultimately are more expensive. When an infant requires 0·5 l/min, the cost per annum of a concentrator is £1030, compared with £3705–4901 for cylinders, depending on whether one or two deliveries per week are needed. Thus we invariably recommend a concentrator to be installed in the home. Parents may be reimbursed from the Family Practitioner Committee the cost of electricity used as this is metered and read by the concentrator supplier.

Secondly, small portable oxygen cylinders (usually size C or D, BOC), are required for when the baby is away from home, either in the car, out in the pram or at relatives' or friends' houses. They may allow mobility for up to 28 hours (see table 3). After negotiation BOC cylinders can usually be replaced when empty by the hospital pharmacy, but if used frequently this may be impractical. Small pin indexed cylinders are available (£494, SOS, Richmond, Surrey) which allow refilling at home from a single large cylinder. This is ultimately more convenient for parents, but involves a more expensive *initial* outlay for the hospital. Oxygen concentrators and small cylinders in most instances will need to be equipped with low flow meters. These are not prescribable by the family practitioner, so must be provided by the hospital and fitted by a qualified technician. De Vilbiss, however, supply them with their oxygen concentrator (for £150, payable by the hospital) and Omnicare may soon follow. Stocks of low flow meters are not large so one should allow ample time for ordering before discharge home.

We have found it preferable to supply oxygen through nasal cannulae as described earlier. Although lower flows can be used with a pernasal pharyngeal catheter, partial occlusion of a nostril and an increase in nasopharyngeal secretions may be a disadvantage of this technique. For emergency use the parents may also be provided with an Ambu bag and face mask of a size suitable for the baby.

Supervision of home oxygen treatment requires regular visits by a specialist nurse who can monitor the infant's oxygen requirement with a headbox and oxygen analyser (as described above), as well as give advice about the clinical aspects of adequate oxygenation. At the time of the visit the nurse will also measure, using a pulse oximeter, the SaO_2 of the baby while quiet and asleep and compare this with the $TcPO_2$ monitor values. As mentioned above, the SaO_2 of normal babies at home is between 97 and 100% when asleep. The aim of additional inspired oxygen should therefore be to keep values in this range. After allowing for a ±2% error on the pulse oximeter (Nellcor), SaO_2 should thus be kept 97–98% when asleep. As lung disease resolves, an overnight tape recording of SaO_2, breathing movements, and electrocardiography may guide the reduction in inspired oxygen concentrations.[23] Short term measurements of oxygen saturation (for example, for 20 minutes) may inadequately reflect the variations that may occur in such infants (JR Groothius; Ross Laboratories Special Conference on Bronchopulmonary Dysplasia, Washington DC, December 1989).

Parents are often worried that nasal cannulae may become displaced during their sleep. Use of a $TcPO_2$ monitor provides early warning that this has happened. Parents are also worried about electricity cuts. A new three pin plug (B and R Electrical Products, Harlow) that fits any household socket will alarm if this happens thus allowing parents temporarily to supply oxygen from a cylinder. Because of the high dependency of these infants on electricity we notify the local electricity board before their discharge from hospital.

AVOIDANCE OF INFECTION

All respiratory tract infections are potentially dangerous or lethal to the patient with bronchopulmonary dysplasia. It is therefore essential that, as far as possible, such infections are avoided.[51] We therefore do not advise parents to take their infants to the health visitor clinics, preferring the health visitor to visit the patient. Most 'well-baby' clinics, also accommodate preschool children, many of whom carry respiratory infections, especially in the winter months. It is also important that the health visitor herself does not visit a baby with bronchopulmonary dysplasia if she has a respiratory infection. It is impossible to separate older siblings from infants with bronchopulmonary dysplasia, but if they do contract respiratory infections, it is probably best for them temporarily to minimise their contact and handling of the baby. Parental and sibling contact cannot be prevented, but any visitors to the house should not go near the baby if they are suffering from a respiratory infection. Similarly, visits to waiting rooms at doctors' surgeries are to be avoided because of the risk of cross infection. Once again, we request the general practitioner to visit the patient at home in this situation.

Because of the potential danger of pertussis infection in these infants[52] we ensure that infants and their siblings complete their immunisations as soon as possible. There are few contraindications to pertussis immunisation (see Department of Health guidelines[53]) and yet such an infection could be fatal in a baby with bronchopulmonary dysplasia.

It is probably wise to cover respiratory infections with an oral antibiotic, such as amoxycillin, cefaclor, or erythromycin, particularly if

Table 3 Mobility using a size C oxygen cylinder

Flow rate (l/min)	Hours of oxygen provided (approx)
0·1	28
0·2	14
0·3	9
0·4	7
0·5	6
0·6	4·5

there is a fever. All parents are given instruction in the accurate measurement of axillary temperature and taught how to observe an *increase* in chest wall recession, a useful sign of respiratory distress (C J Morley *et al*, personal communication).

Often the baby will manifest low TcPO$_2$ values during an intercurrent infection or immediately after immunisation. These changes are due to either arterial hypoxaemia or reductions in skin blood flow, reflecting the presence of a non-specific illness. We suggest that whenever low TcPO$_2$ values are encountered, they are checked immediately against a pulse oximeter in order to be sure that the infant does not require additional inspired oxygen and/or admission to hospital. In the meantime parents are advised temporarily to increase the inspired oxygen through the nasal cannulas.

NUTRITION

Sometimes it is necessary for feeds to be partly or wholly given by nasogastric tube at home. After appropriate training many parents become adept at passing a nasogastric tube and most would be able to feed their baby using this method. It may also be necessary for milk to have carbohydrate and/or fat supplements (as described earlier). We continue folic acid, iron, and multivitamin preparations until the infant has been weaned onto a mixed diet.

RESPIRATORY SUPPORT

It has sometimes been possible to discharge the infant while still receiving negative extrathoracic pressure respiratory support and for this to be provided at home. In patients who are dependent on this only during the overnight period, we have found that the risks of remaining in hospital outweigh the difficulties of applying this at home. The application of CNEP by parents can be readily taught, but of course, parents need regular supervision with immediate access to the hospital.

EMOTIONAL SUPPORT OF THE PARENTS AND THE BABY

Infants who have severe chronic lung disease and have had prolonged intensive care will be a continual source of anxiety for both parents. It is important that the information they are given is honest, consistent, and regularly updated. This may be difficult when working on a busy unit. Thus the help of other counsellors and therapists may be particularly valuable in providing support. Lines of communication should always be available, and staff should be prepared to advise on more mundane needs such as financial and organisational problems.

PSYCHOMOTOR DEVELOPMENT

It is important that infants with bronchopulmonary dysplasia are given extra exercises and stimulation. Having had protracted neonatal intensive care, their development will more likely be hindered. Parents can be shown by physiotherapists how to stimulate their child appropriately for optimal development. In addition, the consultation of a speech therapist may help prevent the development of feeding difficulties. Once again it is important that any therapist or member of the Portage scheme who visits the infant at home does not do so if they have a respiratory infection.

TRAINING IN CARDIOPULMONARY RESUSCITATION

Provided that airway oxygenation is adequately maintained, cyanotic episodes should not be a problem in these patients. Nevertheless, all parents should be able to provide bag and mask ventilation with 100% oxygen and external cardiac massage. This is taught to our parents by clinical nurse specialists with the help of a Resusci Baby (Laerdal Medical), video, and resuscitation manual (the latter available on request). We ask British Telecom to provide for priority repair in the event of faults developing with the home telephone. We also alert the local casualty department and ambulance station concerning the possibility that such an infant will require emergency attention.

Conclusions

There are many new ways in which one can help the infant with bronchopulmonary dysplasia. Most important of these concern the adequate management of airway oxygenation, the monitoring of skin oxygen levels, the avoidance of infection, and the provision of an adequate diet.

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