The diagnosis of 'congenital laryngeal stridor' in laryngology means no more than 'anaemia' or 'congestive cardiac failure' in internal medicine. Many causes of congenital stridor exist and the diagnosis can be clarified by endoscopy.

An understanding of the anatomical features of the neonatal upper airway clarifies the causes and problems of paediatric airway obstruction. The smallest diameter of the larynx is at the subglottic region, not at the vocal cords. The subglottic region is surrounded by cartilage, and progressive oedema causes tissue enlargement at the expense of the airway. The paediatric larynx is higher, softer, and more easily irritated during endoscopy than adult larynx.

Some important specific conditions are as follows.

1. **Laryngomalacia**
   - This condition was wrongly and loosely termed 'congenital laryngeal stridor' in the past. It is probably secondary to inadequate rigidity of supraglottic structures. On endoscopy the flaccid epiglottis and aryepiglottic folds are seen to be sucked into the larynx in a fluttering fashion on inspiration, whereas expiration is unimpeded. Once this condition is diagnosed a confident prediction of ultimate improvement can be made.

2. **Congenital Subglottic Stenosis**
   - This condition is becoming recognized more frequently and may present in many ways including persistent or recurrent croup. Post-extubation stridor in anaesthesia may also be a mode of presentation, and steroids may be helpful in treating this situation. This may also be the underlying lesion in patients in whom there is difficulty in removing a tracheostomy tube after a poorly performed tracheotomy, or an endotracheal tube after ill-managed prolonged intubation.

3. **Laryngeal Papillomata**
   - These are the most common benign neoplasms of the larynx in children. Their features include frequency of recurrence and resistance to treatment, yet spontaneous regression and disappearance after some years may occur. The advent of microlaryngeal instruments in conjunction with the operating microscope has revolutionised the ability of the surgeon to remove these lesions with precision.

4. **Choanal Atresia**
   - In the infant, choanal atresia or nasal obstruction resulting from a fractured nose or oedema can cause severe and sometimes fatal airway obstruction. It is a curious fact that the newborn will not normally breathe through his mouth, so that a baby with bilateral atresia has no difficulty in breathing when he cries but becomes obstructed and cyanosed as soon as his mouth closes. An emergency per-oral endotracheal tube or McGovern nipple may be necessary.

5. **Laryngeal Foreign Bodies**
   - Foreign bodies in the larynx or subglottic region must be treated with great respect. Often tracheotomy under inhalation anaesthesia may be necessary for removal.

6. **Subglottic Haemangioma**
   - These lesions are often associated with other cutaneous haemangiomata. Irradiation following diagnosis has proved satisfactory.

7. **Tracheobronchial Compression by Cardiovascular Anomalies**
   - Compression by an anomalous innominate artery can be accurately diagnosed by bronchoscopy. Double aortic arch is best diagnosed by barium swallow. The symptoms may vary from mild stridor (both inspiratory and expiratory) to severe dyspnoea with cyanosis. A serious feature is 'reflex apnoea'.
Various degrees of innominate artery compression are now being recognised, thus laryngoscopy without bronchoscopy is an incomplete examination for children with obstructive airway problems.

(8) Gross Enlargement of Tonsils and Adenoids

This is a recently recognised but now well documented cause of airway obstruction and may present with noisy stertorous breathing and somnolence.

Anaesthetic Considerations

The patients are often seriously ill and teamwork between surgeon and anaesthetist is mandatory.

The anaesthetic principles of management are:

1. Adequate oxygenation and carbon dioxide elimination.
2. Relaxation of mandible and pharynx.
3. Depression of vagal reflex activity.
4. Retention of vocal cord movement to allow assessment.

A common technique in the past has been to use muscle relaxants during endoscopy, along with intermittent hyperventilation. The only complication encountered has been that, after the use of a depolarizing relaxant, the removal of the bronchoscope and the return of spontaneous ventilation have been accompanied by laryngeal spasm. The solution to this problem lies in the use of topical analgesia. More recently the preferred technique of anaesthesia has been halothane and spontaneous respiration throughout, with topical analgesia applied to the respiratory tract. Spontaneous respiration allows unhurried observation of vocal cord movement. The local analgesia ensures that laryngoscopy will not be complicated by unwanted reflex activity and has virtually abolished laryngeal spasm following withdrawal of the bronchoscope.

In some children, post-operative stridor may have been produced or accentuated by instrumentation, dry anaesthetic gases and atropine. Nursing in a moist air atmosphere for a few hours is wise. Steroids may be indicated, and rehydration by intravenous fluids may help.

Comments

The authors, both very experienced in their fields, have removed the ‘blanket’ diagnosis of stridor in favour of a lucid differential diagnosis.

They discuss the common causes of stridor from the point of view of presentation, endoscopic findings, prognosis and therapy. Their paper indicates the importance of endoscopy in any small infant with significant stridor. The selection for endoscopy of children with milder degrees of stridor is not within the scope of this article.

The techniques of endoscopy and associated anaesthesia make teamwork between surgeon and anaesthetist essential. Many diagnoses can only be made by an unhurried view of the moving larynx and bronchial tree. The anaesthetic technique of applying topical analgesia to the respiratory tree, using spontaneous respiration with halothane and oxygen, is valuable in regard to both patient safety and accurate diagnosis.

As prognosis and therapy are dependent on diagnosis, adequate endoscopic examination is mandatory when significant stridor is present.

J. H. Overton

CONTINUOUS INTRAGASTRIC FEEDING IN SMALL INFANTS OF LOW BIRTH-WEIGHT*

In this feeding trial 66 babies with birth weights between 1000-2100 gm were given continuous intragastric milk feeds from the fourth hour of life. Of these, 10 were withdrawn from the trial leaving 56 to complete the study.

Each infant received only full strength milk, which was either expressed human breast milk, or SMA-S26 (a proprietary low-protein adapted cow’s milk) or half-cream Regal milk (partly-skimmed evaporated cow’s milk). Each milk had the same energy value (65 cals/100 ml) although the protein content varied, human milk 1.2 gm/100 ml, SMA-S26 1.5 gm/100 ml and half-cream Regal 4.1 gm/100 ml.

*Brit. Med. J., (1972). 3: 547-550.

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The milk was given through a gavage tube (diam. 3.5-4.5 F.G.) passed through the nose into the stomach. This was connected to a paediatric intravenous set, attached to a sterile infusion bottle containing the milk. After an initial dextrose feed, milk was given at 60 ml/kg/24 hours, and then increased daily by 30 ml/kg/24 hours until the maximum of 300 ml/kg/24 hours was achieved on the ninth day, that is a caloric loading of 195 cals/kg/24 hours. Each infant was nursed prone, usually with the head higher than the feet, in an incubator at an appropriate neutral thermal environment. The bottle was suspended about 1 meter above the infant and was changed every 8 hours, intravenous tubing every 24 hours, and gavage tube every 3 days.

Of the 10 infants withdrawn, 3 died. In none was milk aspiration thought to occur clinically, nor was it seen at post mortem in the 2 submitted to autopsy. Of the other 7 infants, 2 were withdrawn because of vomiting and abdominal distension, while 2 others developed respiratory distress which was not thought to be related to aspiration of milk.

Despite the large volumes of milk given, milk aspiration was not detected in the 56 cases who completed the trial. Two infants had episodes of vomiting, but later settled and completed the trial. Hypoglycaemia was looked for at 24 hours of age and not detected, while only one infant had a serum calcium level below 7 mg/100 ml when screened on the seventh day despite the high phosphate loading from half-cream Regal milk.

In all groups, whether pre-term or light for dates, the mean weight gain was rapid, being greater than 200 gm in the third week in all groups. In infants on half-cream Regal milk, a good weight gain was achieved in the first week. However the protein load with this milk, in the volumes given was 12.3 gm/kg/24 hours, and it was not surprising to find high plasma amino acid levels in these infants. This milk was not recommended for this type of feeding by the authors.

Comment

It is now accepted that the prevention of starvation by early feeding in low birth weight infants is of great importance for survival and normal development (Davies et al., 1968; Dobbing, 1970). It is difficult to achieve an early adequate intake of calories in these infants, and the trend in many centres throughout the world has been towards early intravenous nutrition with specially prepared high caloric feeds (Sinclair et al., 1970). This presents considerable problems in cost, personnel and time.

This paper is therefore of considerable significance for it describes a method of giving early adequate calories to the low birth weight infant, using a technique, which is claimed to be safe, effective, undemanding on nurses' time, and can be performed by pupil midwives. If these claims can be justified, this method is a considerable contribution to the medical care of these babies.

The lack of complications, especially aspiration of milk, I find most surprising. Clinically, aspiration of feeds can be difficult to diagnose as periods of apnoea may be brief. How aspiration was excluded in this trial is not clear, for there is no mention of the observations recorded or whether routine radiological examinations of the lungs were performed.

The large milk load given requires critical assessment, for the water load of 300 ml/kg/24 hours and the caloric load of 195 cals/kg/24 hours, is well in excess of the accepted requirements of 120 cals/kg/24 hours for low birth weight infants (Sinclair et al., 1970). Nevertheless, the mean weight increase in these infants when compared with the increase in utero at this period, is not as great. This suggests that some of the feeds were not absorbed or not metabolised. Unfortunately no data on excretory losses were available. The high plasma amino acid levels found in infants given Regal milk was initially picked up with the routine Guthrie test. Abnormal levels of other constituents may have also been found if a wider metabolic screening programme had been used.

Clearly this method requires further evaluation, preferably in a unit where metabolic trials can be performed. Ideally, a controlled trial is required to compare the outcome of infants treated by this technique with those given intermittent gavage feeds using smaller daily volumes of milk, or those receiving prolonged intravenous nutrition.
It is unlikely that continuous gastric feeding will ever completely replace intravenous nutrition, because many low birth weight infants are unable to tolerate oral feeds. However, if other workers can substantiate the claims of these authors, a considerable advance has been made in the care of low birth weight infants.

Michael Adamson

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Author's Comment

COMMENT BY DR. VALMAN

The details of the paper have been summarised accurately. However, I do not agree that many low birth weight infants are unable to tolerate oral feeds. At the Hackney Hospital, all infants weighing less than 2.1 kg at birth have been fed by continuous intragastric milk feeds for the past 10 years. All autopsies have been performed by Dr. N. E. France, a paediatric pathologist, who has constantly sought evidence of milk aspiration. None has been found; this suggests that the danger has been exaggerated and is less than the well-documented hazards of intravenous feeding.

H. B. Valman

A CONTROLLED TRIAL OF ANTEPARTUM GLUCOCORTICOID TREATMENT FOR PREVENTION OF THE RESPIRATORY DISTRESS SYNDROME IN PREMATURE INFANTS*

SUMMARY

This study is the first reported on the place of antepartum glucocorticoid in the prevention of the idiopathic respiratory distress syndrome (IRDS) in premature infants.

In a controlled blind trial performed over 22 months, 287 women were studied, comprising those admitted in premature labour at 24 - 36 weeks, or in whom premature delivery before 37 weeks was planned because of obstetrical indications. Of the 287 women, 5 were withdrawn because of procedural errors, the remainder being allocated at random to 2 groups. In the treated group, a mixture of betamethasone phosphate (6mg) and betamethasone acetate (6mg) was given IM on admission and repeated after 24 hours. The control group were given IM cortisone acetate (6mg), equivalent to one seventh of the glucocorticoid activity of the treated group. If delivery threatened, ethanol or salbutamol infusions were given in an attempt to delay delivery for 48 to 72 hours. In those women in whom premature delivery had been planned, the first injection was given 3 days before elective induction.

Following delivery each infant had an Apgar score performed at 1 and 5 minutes, and gestational age was assessed. Evaluation of the IRDS was based on clinical features using the Silverman score, radiological signs, and serial arterial samples for blood gas analysis. Treatment included infusions of bicarbonate, positive-end-expired pressure, and mechanical ventilation where indicated. Hypoglycaemia and hyperbilirubinaemia were looked for at regular intervals. In infants who died a necropsy was obtained in all but one.

The women were divided into 5 divisions, only 3 of which were discussed: (i) Rh isoimmunization, (ii) hypertension-oedema-proteinuria syndrome and (iii) unplanned premature labour. In the first 2 divisions the numbers were small and results did not reach significance, although in both divisions the incidence of IRDS was less in the treated group. However, the foetal death rate was much higher in the treated group of the mothers with hypertension-oedema-proteinuria, as compared with the control group, and the reasons are unknown.

In the group with unplanned premature labour, 213 mothers were studied, 117 in the treated group and 96 in the control. In both groups the foetal death rate was similar (3%) but the neonatal mortality in the treated group was less (3.2%) than in the control (15.0%) (p<0.01). Of the infants dying in the neonatal...
period, pulmonary hyaline membranes were seen at necropsy only in the control group. The incidence of the IRDS was less in treated infants who survived (9.0%) than in controls (25.8%) (p<0.003), but on further analysis the difference was confined to babies of less than 32 weeks gestation who had been treated for at least 24 hours before delivery, 11.8% of the treated babies compared with 69.9% of the controls (p<0.002). In infants whose gestation was more than 32 weeks, a positive benefit from treatment was suggested, but the numbers were too small to reach statistical significance. Apart from the incidence of the IRDS no other details were given except mortality figures and autopsy findings.

No complication of pregnancy, labour, delivery or the puerperium was detected, nor any affect on neonatal morbidity (as monitored by Apgar scores, or the presence of hypoglycaemia, jaundice or diarrhoea).

DISCUSSION

IRDS is still one of the major causes of death in the premature infant (Butler and Alderman, 1969). Although the role of pulmonary surfactant in its pathophysiology is accepted, its aetiology is unknown and treatment is supportive. Up to now, prophylactic treatment has not been available apart from efforts to avoid foetal asphyxia and premature labour. This paper by Liggins and Howie is therefore of great importance to clinicians, for it is the first report of a technique aimed at preventing IRDS in premature infants.

The background to this study was a chance observation by Liggins in 1969, when he found that maturation of the foetal lung in the lamb was accelerated by stimulation of the foetal adrenal cortex or by intra-foetal administration of glucocorticoids. These observations have been confirmed by Platzker et al. (1972) and De Lemos et al. (1970) in lambs, and by Kotas and Avery (1971) in rabbits. The mechanism whereby the steroids induce pulmonary surfactant is unknown, but specific receptor sites for compounds with glucocorticoid activity have been demonstrated in the rabbit's lung and, recently, in the human foetal lung (Ballard and Ballard, 1972).

Clearly, from animal studies the antepartum administration of glucocorticoids to the foetus is a practical way of preventing the IRDS, but what of the human?

The results so far published are promising, although not as convincing as the background animal studies, for the only group to reach statistical significance was infants of 32 weeks or less in whom labour was delayed at least 24 hours. This group comprised 17 treated infants and 23 controls, i.e. only 40 infants out of the total 226 studied. In this light the results are not as impressive as on initial reading. However, in all other groups except those infants 37 weeks or greater, the trend was the same as above, although statistical significance was not achieved.

In this study the glucocorticoids were administered to the mother, in contrast to previous animal studies in which it was administered directly to the foetus. However, it is accepted that the human placenta, unlike the ovine placenta, is relatively permeable to glucocorticoids, although in this study no level in cord blood was given to support this fact.

This work will undoubtedly be the first of many on the premature induction of pulmonary surfactant, for the potential gain in the welfare of the premature infant is enormous if the technique can be perfected. Complications of the method appear to be negligible in the postnatal period, although the effect on long-term development of survivors is as yet unknown. The increased evidence of foetal deaths in mothers with hypertension, oedema and proteinuria, has not been explained and clearly more evaluation of the method is required before it can be introduced in the management of these high-risk pregnancies.

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