

¹² Poswillo, D E, *et al*, *Teratology*, 1974, 8, 339.¹³ Clarke, C A, McKendrick, O M, and Sheppard, P M, *British Medical Journal*, 1973, 3, 251.¹⁴ Nevin, N C, and Merrett, J D, *British Journal of Preventive and Social Medicine*, 1975, 29, 111.¹⁵ Renwick, J H, Possamai, A M, and Munday, M R, *Proceedings of the Royal Society of Medicine*, 1974, 67, 360.¹⁶ Lorber, J, Stewart, C R, and Ward, A M, *Lancet*, 1973, 1, 1187.

Cow's milk intolerance

Interest in gastrointestinal allergy (a subject thought by some to be beyond the fringe of clinical credibility) has been encouraged by recent growth in the use of the biopsy capsule and the electron microscope and advances in immunology. Coeliac disease is now known not to be alone in causing malabsorption changes in the small intestinal mucosa in infancy; among other conditions responsible, intolerance to cow's milk demands consideration. A recent review¹ by a group of Finnish workers who have been studying the problem for a decade or more showed that in their series of 54 children gastrointestinal symptoms—vomiting and prolonged diarrhoea leading to failure to thrive and occasionally to severe dehydration—appeared on average at 2 months of age, or about 1 month after cow's milk feeding had begun. About 20% also had atopic eczema and recurrent respiratory infections. This clinical picture accords well with that described by others.^{2,3} Malabsorption was a criterion for admission to the Finnish study, and was proved in all cases. The jejunal mucosa was always abnormal, and in about half the children it had the flat appearance indistinguishable from coeliac disease. French workers,⁴ on the other hand, have found partial villous atrophy most commonly, and reported that occasionally the changes were minor.

Strict criteria including milk challenges have been suggested before a diagnosis of cow's milk intolerance can be made.⁵ Kuitunen and colleagues,¹ though not following these to the letter, found their patients all did well after substitution of human milk and reacted adversely to cow's milk challenge either within a few hours or gradually over 3–4 weeks, some showing a quick, others a less serious reaction. After an initial period of 2–4 months on human milk (apparently readily available) the infants were given a gluten-free diet in which soya milk or a dried milk with hydrolysed protein replaced human milk. They were challenged again with cow's milk after 6–14 months, returning to it permanently whenever possible, though gluten-containing food was not introduced for another 5–6 months. Clinical symptoms disappeared by about 1 year of age, though a fifth of the infants were still found to have malabsorption and two-thirds abnormalities of the proximal jejunal mucosa.

In most of the children studied the mucosal abnormalities had disappeared after two years on a normal gluten-containing diet, and the condition may therefore be regarded as transient, with full recovery possible eventually. Nevertheless, symptoms, laboratory tests, and biopsy findings may mimic other conditions leading to mucosal damage and malabsorption. Gastroenteritis is sometimes followed by a period of intolerance to both lactose and cow's milk protein. By substituting human milk initially with good clinical effect, the Helsinki group showed they were not dealing with lactose intolerance. Transient gluten intolerance and even intolerance to other foodstuffs may accompany cow's milk allergy. However, the differentiation from true coeliac disease must be made as soon as possible.

If gluten has never been given, and biopsy changes are minor, coeliac disease can be excluded; but when this is not so only very careful observations with serial biopsies over several years may distinguish the two.¹

Provocation tests with milk protein fractions (there are 20 or so in cow's milk) suggest that β -lactoglobulin, not present in human milk, is the most important.^{1,3} Possibly there may be a common response of the local immunoglobulin-producing system of the gut to various noxious agents⁶; during relapse of cow's milk intolerance, as in untreated coeliac disease, there is a definite increase in the jejunal mucosa of cells containing IgA and IgM. Further study of the altered immune mechanism associated with allergic tissue reactions will doubtless soon provide us with more information. At present there are varying opinions concerning IgE mediation^{6,7} and complement consumption^{6,8} in cow's milk intolerance. In practical terms, however, while it seems likely that paediatricians will see severe effects of this condition, relatively rarely milder cases may go unrecognised, and they would do well to ponder the view⁹ that one of the important functions of breast feeding is to ensure a smooth transition from immunological dependence to independence.

¹ Kuitunen, P, *et al*, *Archives of Disease in Childhood*, 1975, 50, 351.² Gerrard, J W, *et al*, *Acta Paediatrica Scandinavica*, 1973, Suppl 234.³ Freier, S, in *Clinical Immunology—Allergy—in Paediatric Medicine*, ed J Brostoff, p 107, Blackwell, Oxford, 1973.⁴ Fontaine, J L, and Navarro, J, *Archives of Disease in Childhood*, 1975, 50, 357.⁵ Goldman, A S, *et al*, *Pediatrics*, 1963, 32, 425.⁶ Savilahti, E, *Gut*, 1973, 14, 491.⁷ Shiner, M, Ballard, J, and Smith, M E, *Lancet*, 1975, 1, 136.⁸ Matthews, T S, and Soothill, J F, *Lancet*, 1970, 2, 893.⁹ Gerrard, J W, *Pediatrics*, 1974, 54, 757.

Hospitals for the developing world

In the western world hospitals have evolved over the past 100 years or more from very small premises, often a single house with a few rooms to serve as wards, to our modern vast structures with hundreds of beds and almost as many doctors. To these have been attracted the cream of the medical graduates from developing countries, who have basked in the facilities that such hospitals offer and—not surprisingly—have persuaded their governments that similar structures are needed in their own countries. Is this really what is needed? With limited finances it is probably better for a nation to have several small units than one large one. Clearly large units are necessary in major conurbations, but surely not so large and so well equipped that there is no money left for anything else?

A new report¹ from the Missionary Societies of Great Britain and Ireland has brought out the value for developing countries of simple health centres and their potential links with district and regional hospitals. The report describes in detail the planning of such a model health centre to serve a defined community, capable of future expansion into a small hospital of 25 or 125 beds should the need arise. The centre includes outpatients, a clinic for under-5s, family planning and antenatal clinics, and both general and maternity beds. Refreshingly, the greater portion of the population—children—are well catered for, so that, in addition to outpatient provision for the under-5s, a plot for a garden and keeping small animals is included where mothers can be taught enough to alleviate

the ever-present threat of malnutrition of their children. It would have been an advantage if the plans had included a separate health and nutrition rehabilitation unit, where there should be a model, local kitchen to demonstrate the importance of keeping the cooking fire off the ground to prevent burns to toddlers; shelves for dangerous substances such as kerosene; and covered containers suitable for drinking water. Another useful addition might be a cheap model latrine for the home with details of its method of construction. Nothing is said about security—an ever-present threat to microscopes and even far less valuable materials: theft of simple equipment such as scales or the paraffin stove used for sterilising syringes for immunisation may be disastrous when there are no funds to replace them.

There is useful advice on simple laboratory construction and contents and on reception, clerical, sterilisation and stores areas. Fifty-two appendices include costing and construction plans for roads, water supply, and waste disposal. The cheap teaching aids described would prove immensely useful.

The value of such units is beginning to be more widely appreciated, and more and more countries will be planning them in the near future. Every doctor, nurse, or administrator concerned with the construction of a health centre or a small hospital will find this book a mine of practical information; while government offices with a responsibility for planning will find it will save them an enormous amount of effort and time.

¹ *A Model Health Centre*. Obtainable from the Conference of Missionary Societies in Great Britain and Ireland, 2 Eaton Gate, London SW1, price £3.

Pneumothorax in the newborn

Recognition of pneumothorax in the newborn is important, since it may sometimes need urgent treatment. In other instances, however, no active treatment is required at all; hence, proper management demands a knowledge of the natural history of the condition.

In normal vigorous newborn infants full aeration of the lungs is rapid and smooth during the first few breaths. Nevertheless, negative intrathoracic pressures transiently reaching 100 cm of water have been recorded during these breaths,¹ and not surprisingly from time to time there are alveolar leaks leading to pneumothorax and related complications such as pneumomediastinum and interstitial emphysema. These complications are particularly likely to occur if alveolar ventilation is uneven as a result of intrapartum inhalation of meconium or blood.

Radiological surveys in the 1930's when medical intervention was at a minimum, showed that pneumothorax was present in 1-2% of all newborn infants,^{2,3} but very few required any treatment or showed any symptoms. An account of the problem in contemporary neonatal practice was given in a recent survey by Yu and his colleagues⁴ from Oxford, where pneumothorax was detected clinically in 0.3% of live births. One-third of the cases occurred in term infants without evidence of underlying lung disease and, though some had required resuscitation at birth, in most the condition was associated with the aspiration of meconium or blood. Symptoms of respiratory difficulty from pneumothorax occur in this

group of infants soon after birth, and usually within minutes, with tachypnoea, intercostal recession, displacement of the apex beat, and poor air entry on the affected side. Diagnosis should always be confirmed by radiography if possible, but if facilities are not immediately available and the infant's condition is deteriorating the diagnosis may be confirmed by needling the intrapleural space and aspirating air by a 20 ml syringe and three-way tap. The needle should be inserted into the second intercostal space in the mid-clavicular line and withdrawn as soon as the aspiration is completed. This procedure carries the slight risk of perforating the lung, and any infant in whom it has been performed must be watched very closely for the development of further pneumothorax.

If a pneumothorax is confirmed radiologically the decision to aspirate depends on the severity of the symptoms and whether or not they are progressing. Radiological evidence of tension pneumothorax with a completely collapsed lung and evidence of mediastinal shift or increasing respiratory distress are indications for needle aspiration or the insertion of an intrapleural drain connected to an underwater seal or one-way valve. In most cases, however, spontaneous resolution will take place, and this may be accelerated by the administration of oxygen.⁵

Two-thirds of the cases in Yu's study⁴ occurred in premature infants with hyaline membrane disease, especially those requiring ventilatory assistance. Indeed, the risk of pneumothorax developing in infants requiring continuous distending pressures for the management of their ventilatory problem may be more than 20%.^{4,6,7} Again the aetiological factor is increased transpulmonary pressure, whether due to intermittent positive pressure ventilation, constant positive airways pressure, or vigorous respiratory effort in association with stiff lungs. In these conditions pneumothorax usually occurs on the second or third day or even later, and the signs of diminished air entry or mediastinal shift may be difficult to detect in an infant with severe underlying pulmonary disease. The essential feature is a sudden or progressive deterioration in general condition, and since such infants are normally nursed in units with continuously available x-ray facilities the diagnosis can usually be confirmed before treatment is instituted. There is no place for needle aspiration except as an emergency under these conditions, since the major causative factor is a continuing one; an intrapleural drain should be inserted and left in place until the underlying pulmonary problem is resolved.

When this complication develops in the course of hyaline membrane disease the mortality is high. In the Oxford study it was 31%, whereas in the same unit the overall mortality from hyaline membrane disease is less than 10%.⁸ This increased mortality may reflect the severity of the underlying disorder—and, indeed, in one-third of the cases the pneumothorax developed while the infants were breathing spontaneously—but the association with various techniques of ventilatory support suggests that, as with the administration of oxygen, the therapeutic margins are narrow. Their successful application requires painstaking control and monitoring, and of course the necessary skilled staff to supply this.

¹ Karlberg, P, *et al*, *Acta Paediatrica*, 1962, 51, 121.

² Davis, C H, and Stevens, G W, *American Journal of Obstetrics and Gynecology*, 1930, 20, 73.

³ Solis-Cohen, L, and Bruck, S, *Radiology*, 1934, 23, 173.

⁴ Yu, V Y H, Liew, S W, and Robertson, N R C, *Archives of Disease in Childhood*, 1975, 50, 449.

⁵ Chernick, V, and Avery, M E, *Pediatrics*, 1963, 32, 816.

⁶ Hall, R T, and Rhodes, P G, *Pediatrics*, 1975, 55, 493.

⁷ Blake, A M, *et al*, *Lancet*, 1973, 2, 1176.

⁸ Robertson, N R C, and Tizard, J P M, *British Medical Journal*, 1975, 3, 271.