Severe Pneumonia in Young Children

With the introduction of antibiotics and the general rise in nutritional standards there has been a steady decline in the number of children dying from pneumonia. But, as J. O. Forfar\(^1\) showed in his survey of child deaths in Scotland, the rate of fall has been subsiding and has now almost stopped.

Two groups of child deaths associated with respiratory tract infections are still difficult to prevent. One, which was the subject of a recent symposium held by the College of Pathologists,\(^2\) comprises very acute infections. These can present either as unexpected deaths, often termed “cot deaths,” or as deaths associated with a massive outpouring of mucus. Such children frequently die in the ambulance on the way to hospital or within minutes of admission to hospital, and may present with convulsions or even as intestinal obstruction.

The other group occurs in children who are clinically diagnosed as pneumonia and do not respond to antibiotics. The incidence of these cases varies from 1% to 12% in different series,\(^3\)\(^,\)\(^4\) and it appears to depend largely on the population studied. The former figure possibly applies to the general child population, while the latter occurred at Alder Hey in Liverpool, the largest children's hospital in this country. This hospital has an intensive care unit for such cases and R. S. Jones, J. B. Owen-Thomas, and M. J. Bouton\(^5\) have recently analysed a series of 70 cases admitted to that unit. The children, all between the ages of 7 days and 10 months, presented clinically in three groups: (a) 12 between the ages of 7 days and 6 months presenting with circulatory failure; (b) 45 between 10 days and 10 months with predominantly respiratory symptoms; and (c) 13 between 14 days and 10 months with staphylococcal infections of the lung.

The first group, presenting with circulatory failure, are probably those of greatest clinical importance, as these children did not present with overt pneumonia but with general lack of response, in stupor or light coma, and with pallor and patchy skin cyanosis. On admission they had raised blood ureas and in half of them the initial chest x-ray appeared normal. Only three out of nine of those admitted to hospital at Liverpool survived.

The primary lesion in such children is increasingly difficult to assess, as they can be maintained for many days by peritoneal dialysis and artificial ventilation, by which time classical bronchopneumonia can develop and alveolar exudate consolidate to form hyaline membrane. Such membranes can also apparently be produced by administration of too high a concentration of oxygen. It is probably best to consider these children in much the same category as the more familiar unexpected “cot deaths,” and they may be referred to as “missed cot deaths.” The pathological processes leading to their severely shocked state are possibly along the lines of those postulated by J. L. Emery\(^6\) as responsible for the unexpected “cot death” whereby a series of secondary vicious circles are set up as a result of either a virus infection or a phase of hypoxia. Such sequences are probably independent of any special causal organism.
The great importance of these cases lies in the need for earlier recognition and urgent treatment of incipient symptoms that indicate the onset of peripheral vascular failure or of cerebral hypoxia in a child during the course of what otherwise would be considered a normal upper respiratory tract infection. The present high mortality in hospital is usually due to the child's being irretrievably ill before resuscitation starts. When children arrive at hospital in such a severely shocked state, having frequently had periods of severe cerebral hypoxia, the decision to continue resuscitative measures must not be too lightly undertaken. Some children who have survived this state have sustained permanent cerebral damage; others kept "alive" for several days by resuscitative procedures have been found at necropsy to have actual putrescence of the brain.

Most young children with severe bronchopneumonia, constituting 60% of the Liverpool series,1 have respiratory symptoms and are usually diagnosed as pneumonic. Chest radiographs show areas of opacity. In the majority of these children the progressive pneumonia is a secondary disease and the failure of the child to respond is due to the persistence of the primary disease such as a congenital heart deformity or fibrolasticosis.

These children also frequently have prolonged hypoxia associated with obstruction to the respiratory tract by viscid mucus or pus. The bacteria associated with these lung infections vary considerably and are probably best considered as opportunistic. They usually show little sensitivity to the commonly used antibiotics. Among the organisms grown are those responsible for generalized Gram-negative infections, including Pseudomonas pyocyanea and Proteus species, particularly when the infection follows an operation. The post-mortem findings in these children depend largely on the technique of carrying out the necropsy and the amount of care taken to look for the primary disease. The lungs sometimes show evidence of inadequate maturatation, but there is often evidence of previous pulmonary vascular occlusion6 and septicaemia.

Staphylococcal infections of the lung7 make up the remaining group of these difficult cases. It suffices to note their low incidence—13 out of 70 cases in the Liverpool unit.

The severe pneumonias in children require highly skilled and intensive care, and it is to be hoped that further centres will develop on similar lines to that in Liverpool. The most important general clinical point is the early recognition of hypoxic and circulatory disturbances in children with minor infections of the respiratory tract.

7 Pryles, C. V., Pediatrics, 1958, 21, 609.

Change at the Medical Research Council

The retirement on 30 September of Sir Harold Himsworth from the secretaryship of the Medical Research Council affords an opportunity of surveying the evolution of the council's responsibilities during the past years and of contemplating what the future might or could hold in store. Sir Harold is the third secretary to hold office since the first charter was granted in 1920, and during his tenure of it the council's activities have expanded enormously. Though the constitutional position of the council has not changed since 1920, under the Science and Technology Act of 1965 the responsibilities of the former Committee of Privy Council for Medical Research were assumed by the Secretary of State for Education and Science, and it is to the Secretary of State that the council now reports. A new charter of the Medical Research Council received Royal Assent in October 1966, and this provided for the membership of the council to be increased from 12 to a maximum of 16 members, the members being appointed by the Secretary of State for Education and Science. The chairman and deputy chairman are appointed after consultation with the council itself, and the appointment of scientific members follows consultation both with the president of the Royal Society and with the council. The M.R.C. has three advisory boards—namely, the Biological Research Board, the Clinical Research Board, and the Tropical Medicine Research Board. In addition a large number of committees advise the council and its boards on special subjects.

The Medical Research Committee, the forerunner of the present council, originally received an annual grant of about £30,000. Just before the second world war the grant of the Medical Research Council was a little more than £200,000 a year, while in 1967–8 it spent nearly £15m.1 Though this annual sum may seem large by some standards and disbursing it to the greatest effect is a considerable administrative task, it is a small fraction of the cost of disease to the country each year. In fact, since the fruits of research in overcoming disease are so evident, the amount spent on it may well be criticized as inadequate.

The many fields of knowledge with which the Medical Research Council has concerned itself may be glimpsed from its annual reports. They range over the results of observations on patients, the classification of disease, the association of disease with environmental factors such as toxins, ionizing radiations, infection, and so on, the production of disease experimentally in animals, the examination of these processes in normal tissues which undergo change in disease, and the general biology of the cell, including its biochemistry and the mechanisms which maintain its functional normality. This is a wide range of knowledge for the council to foster, and its administrative system is certainly not unnecessarily elaborate for the task it undertakes. Moreover, the council has been particularly successful in attracting into its service workers who later win distinction in many aspects of biomedical investigation. The number of Nobel prizes for medicine—and indeed not only for medicine—awarded to its employees during the past 15 years or so is remarkably high.

This undoubtedly is good, though something good can be expanded to a point where balance is lost and the result may be harmful. The M.R.C. depends for recruitment to its

1 Medical Research Council Annual Report, April 1967 March 1968, H.M.S.O.