THE SUDDEN INFANT DEATH SYNDROME

In 1950, when Keith Bowden published in this journal an article entitled "Sudden Death or Alleged Accidental Suffocation in Babies", he reflected in this title the prevailing medical and lay opinion of the likely cause of the sudden unexpected death of a previously well infant. Bowden went on to describe 40 cases of sudden infant deaths. In most of these there had been a story from the parents that the child had been unwell shortly before death, and the post-mortem findings in most instances supported a diagnosis of infection. These findings, together with observations made on healthy sleeping infants, who were easily able to push aside covering bed clothes and to adjust their posture if their face should become obstructed by a mattress or a pillow, led him to the conclusion that infection was the likely cause of death in these babies.

The First International Conference on the Causes of Sudden Death in Infancy, held in 1963, recognized the need for identification of this problem (which has sometimes been colloquially called "cot death") and suggested the term "sudden death syndrome" (SDS). During the next few years, the publication of many epidemiological studies has enabled a "profile" of the child at risk to be built up. This profile applies to any of the affluent "westernized" societies. The child is usually a male infant, aged between one and seven months, living in overcrowded substandard housing in a city centre; he is likely to have been born prematurely to a young mother, who has bottle fed him. The risk is greater in the winter months, especially if he has a mild respiratory tract infection. There are many other factors potentiating this risk—being a twin, belonging to a minority racial group and the presence of a pillow or another child in the cot, to mention a few.

The story of sudden infant deaths cannot be entirely explained on the basis of poor housing, poverty and bottle feeding, as a sudden infant death may occur amongst households where none of the above-mentioned risk factors are found to apply. Death may occur amongst children in affluent rural and suburban households and amongst the children of professional and managerial classes, although the incidence is many times less than in communities where the risk factors are highest. Houstek\(^2\) considered that the incidence of the sudden infant death syndrome paralleled that of total infant mortality—that is to say, when the infant mortality was highest, the occurrence of the sudden infant death syndrome was highest. Teare and Knight\(^3\) however, point out that in a longitudinal study of the comparative incidence of total infant deaths and cot deaths, the incidence of cot deaths has remained relatively unchanged since 1940, whereas the total infant mortality rate has fallen dramatically. The overall incidence of sudden infant deaths is from 1.5 to 3 per 1,000 live births.

These children appear to die in their sleep, without a struggle, often in the early hours of the morning. The only pathological abnormalities which are found at all frequently are petechiae, particularly of the lungs and thymus, a degree of pulmonary oedema and evidence of mild upper airways disease.

As Susan Beal (this issue, page 1223) points out, theories abound, but to date there is no convincing proof to support any one of them. The Second International Conference on the Causes of Sudden Death in Infancy (1969)\(^4\) suggested that the pathophysiology is multifactorial, consisting of a chain reaction of potentially reversible stages culminating in either complete or functionally complete airways obstruction. Respiratory infections were thought to play a significant part. Among more recent theories put forward is that of allergy to the house-dust mite (*Dermatophagoides pteronyssinus*), and this is argued by P. M. Mulvey on page 1240 of this issue. A review of the Proceedings of a recent symposium on SDS appears on page 1261.

\(^1\)Bowden, K., Med. J. Aust., 1950, 1: 46.

\(^2\)Houstek, J., in "Sudden Infant Death Syndrome", edited by A. B. Bergman, University of Washington Press, 1970: 55.

\(^3\)Teare, D., and Knight, B., Med. J., 1971: 71.

\(^4\)Bergman, A. B., in "Sudden Infant Death Syndrome", edited by Bergman, A. B., University of Washington Press, 1970: 209.
Faced with a wealth of epidemiological knowledge, but a paucity of etiological certainties, there is not much that can be done at present to prevent what (if we extrapolate the South Australian figures in Dr Beal’s paper) may amount to hundreds of deaths that are occurring every year in Australia.

Where then does the clinician’s duty lie? Arrangements to see the parents should be made as soon as possible, the prime aim of this interview being to reassure the parents that this unexpected tragedy was in no way their fault and that accidental suffocation could not possibly have caused their baby’s death, nor could they have foreseen or prevented this tragedy. The parents’ cooperation should then be sought in searching for any possible cause of death. This will include taking a history from the parents, a post-mortem examination, and virological and bacteriological investigations. A definitive diagnosis in this way should be sought in every case for two reasons. First, it will relieve the parents’ own natural guilt feelings that a definitive cause of death has been found and, secondly, it may help to increase our understanding of this little-understood condition. An interview with the parents should be arranged at a later date when the results of all investigations initiated are completed. The parents can then be informed of the cause of death if this has been found, but, more importantly, the parents having recovered from the immediate shock of their unexpected loss will have many questions to ask and many anxieties that need to be relieved. If the cause of death has been found, this should be explained fully, and, depending on the cause of death, queries about further pregnancies and other siblings can be answered. Explanation will not be so easy in those cases in which no cause of death has been defined. Parents can, however, be reassured that the same tragedy is not significantly more likely to happen to subsequent babies, and that, with our present knowledge, these deaths may be due to an unusual reaction to a relatively minor infection.

*Williams, H., Med. J. Aust., 1963, 2: 643.

**COMMENTS**

**YOUNGER CHRONIC SICK**

In recent years, concern for the welfare of dependent people has increased. Many children afflicted in early infancy with permanently damaging conditions now survive to adult life. Enthusiastic treatment of babies suffering from meningomyelocele or brain damage from birth anoxia, hyperbilirubinemia or intracranial hemorrhage leaves a heritage of disabled survivors. These children are in general well cared for by their parents through childhood. The modern trend, with its emphasis on individual independence, prohibits the former custom of able siblings being responsible for the care of the handicapped one. Thus the time comes sooner or later when the devoted parents are unable, because of increasing age, infirmity or death, to provide the necessary care for their disabled son or daughter. The community, both as the extended personal family and at large, rejects the disabled person as its responsibility, and permanent institutional care becomes inevitable.

The term “younger chronic sick” includes those who are permanently disabled and handicapped by incurable conditions. In general they are healthy people who require partial or total care in ordinary daily living. “Younger” is interpreted as being non-geriatric—that is, under 60 years.

E. J. Miller and G. V. Gwynne, two sociologists working from the Centre for Applied Social Research of the Tavistock Institute of Human Relations in London, have recently published the results of a pilot study of residential institutions for cripples in Great Britain. The investigation was originally requested by an inmate of Le Court Cheshire Home, one of the institutions included in the study. The authors were at first reluctant to become involved, and admit that this reluctance was coloured by their own inexperience in communications with disabled people. It was only through persistence that the resident ultimately persuaded the authors to visit the home. After some four years the Ministry of Health sponsored the project, and the study was carried out in the period 1966-1968.

In common with other consumer groups subject to authoritative rule, such as may be seen in universities or prison communities, the residents of Le Court protested that institutional life increased their dependence unnecessarily, and stifled any remaining potential for running their own lives. All this was precipitated by a threatened replacement of the matron and the secretary, which indicated how dependent such inmates are on the staff in command, even in an institution enlightened enough to have a Residents’ Welfare Committee.

Miller and Gwynne set out to determine what was involved in providing residential care for incurables, and to do so they visited many institutions in and around London. However, they concentrated their attention on five: two voluntary homes, one Local Authority home and two Regional Hospital Board Units (one an annexe to a geriatric hospital). All had been set up since 1948 and were regarded as modern and progressive.