

What the report does not do to any great extent is to interpret the pattern of change. One important conclusion to emerge is that in the provision of psychiatric facilities it is impossible to legislate on a national basis. Psychiatric morbidity in a region is as important as the size of the population, if not more so, and could well account for the differences between Oxford and Liverpool. Again, the reduction by half in the inpatient population in the 25-44 age group could be accounted for in part at any rate by the increase between 1961 and 1969 in the number of offenders remanded in custody for psychiatric report from 6,366 to 13,452. A considerable percentage of these are known to have been discharged from psychiatric hospitals within the previous year. How many of the same group are at present in reception centres or in doss houses or sleeping rough? And of those patients who have been discharged or redistributed in society how many absconded, or discharged themselves against medical advice, only to be swept back via the police or as social emergencies? Some must be contributing to the staggering increase in the admission or, rather, readmission rate to a point where the revolving door is beginning to spin. Answers to these questions must be sought before we applaud too vociferously the so-called run down of our mental hospitals.

¹ Department of Health and Social Security, *Psychiatric Hospitals and Units in England and Wales: Inpatient Statistics from the Mental Health Inquiry for the year 1969, Statistical Report Series No. 12.* London H.M.S.O., 1971 (92p net).

Cot Deaths

Slowly and through much skilful work information is growing on "cot deaths," though there is no certainty about their cause or causes and no recipe for their prevention. A notable contribution has now come in a further report by P. Froggatt and colleagues on a survey made in 1965-7 in Northern Ireland,¹ and it will help considerably in sorting out the more probable from the less probable hypotheses.

The incidence of sudden unexplained deaths was 2.5-3.0 per 1,000 live births. It was comparable to that in other surveys, a finding curiously consistent among all Europeanized communities in temperate zones. As in other surveys, the incidence was low in the first three weeks of life, rose, and then declined rapidly after three months. This pattern is fundamental, for it appears that, though known factors such as small birth weight and high room occupancy may slightly alter the risk, the main one is bound up with a change which takes time to develop after birth. Once again more deaths occurred in the six months November to April than in the other half-year. This distribution and the fact that these deaths increased at the times of two recorded outbreaks of influenza suggest strongly that infection may at least sometimes contribute to their cause. Yet attempts at isolating viruses and bacteria were unrewarding.² C. G. Ray and his colleagues³ showed in a similar study, a significantly high rate of virus isolations from respiratory and enteric sites, and they now report⁴ that no viraemia was detected in any of 20 cases examined and circulating interferon, which might be taken as an indicator of recent infection, was found in only 4 of 119 cases. In the Northern Ireland survey 79 of 148 cases had recently been discharged from hospital or had been under medical care or on recollection were thought to have been unwell in the last week compared

with 15 among a similar number of controls. The 69 who gave no history of indisposition are in conformity with R. G. Carpenter's findings in a study of twins:⁵ in 44 out of 112 cases of cot death neither the affected twin nor the surviving one was reported to be unwell. If infection plays a part it appears as a trivial affliction rather than as a frank one.

The survey included a greater number of families who had already lost one or even two children by cot death than seems likely by chance alone or than could be accounted for by inaccurate attribution. The team in Northern Ireland paid particular attention to possible genetic causes and found no evidence for a Mendelian interpretation. In their cases there was no parental consanguinity; the mothers had not suffered an excess number of miscarriages; and there was no evidence of amino-acid metabolic defects as judged by urinary chromatograms. Among the four, and possibly six, cases in which there had been unexplained deaths of sibs there was a history of previous "fainting," cyanotic or apoeic attacks, or periodic breathing. The authors suggest that such cases may have a different aetiology.

Despite the difficulty in determining the time of death there is no doubt that in the Northern Ireland survey as in others the time of highest risk is the night. Diurnal variation in metabolism may ultimately be found to influence the outcome. The more likely interpretation is that sleep somehow hinders the proper response to respiratory difficulty. Much research is now concerned with failing reflexes or inhibiting reflexes affecting respiration in infants, but sleep and inappropriate reflexes do not appear to explain the initial respiratory difficulty or collapse nor the characteristic age at death. One hypothesis is that the remarkable changes in heart tissue taking place with growth in infancy may take a false step and lead to cardiac failure.⁵ Anaphylaxis to milk or other, possibly microbial, antigens, perhaps because of functional deficiency of IgA in the baby, is still a likely mechanism.⁷ The immunological techniques now being used for its investigation were not available at the time of the Northern Ireland survey. They at least show that the conditions necessary for its action are present.⁸

From the practical point of view one important fact emerges from all studies: parents should not blame themselves for the deaths, and the public at large should learn neither to show nor to feel the cruel primitive rejection that some do. Though breast feeding does not guarantee safety—two babies in the Northern Ireland series were breast-fed throughout their lives—it must be regarded as the most effective prophylaxis, if continued for months, that a mother can take. IgA and other components in human milk (and not just in colostrum) are now known to have positive antiviral and antibacterial activity in the baby, and human milk is believed to be non-allergenic to man. If death comes from failure to cope with respiratory difficulty, it seems sensible to put a baby to sleep on his side or back rather than face down, when the weight of the trunk, and not merely that of the ribs and abdominal wall, have to be lifted at every breath.

The special group of babies who have apnoeic or cyanotic attacks are a worry to their doctors and their parents. How far resuscitation methods should be in the minds of mothers with normal babies or babies subject to such attacks is hard to say. Disproportionate fear could bring disproportionate use. It is doubtful whether a monitoring mattress (price about £50) would help, but there are near-misses in cot

deaths and there must be a place for resuscitation. The doctor faced with a frightened mother who has just read an article on cot deaths has to explain that all living is dangerous and that at least her baby has got past the more dangerous stage of birth. Fear will not help a mother to feed her baby.

- ¹ Froggatt, P., Lynas, M. A., and MacKenzie, G., *British Journal of Preventive and Social Medicine*, 1971, 25, 119.
- ² Froggatt, P., in *Proceedings of the Second International Conference on Causes of Sudden Death in Infants*, p. 32. Seattle, University of Washington Press, 1970.
- ³ Ray, C. G., Beckwith, J. B., Hebestreit, N. M., and Berghan, A. B., *Journal of the American Medical Association*, 1970, 211, 619.
- ⁴ Ray, C. G., and Hebestreit, N. M., *Pediatrics*, 1971, 48, 79.
- ⁵ Carpenter, R. G., in *Ministry of Health Reports on Public Health and Medical Subjects*, No. 113, p. 51. London, H.M.S.O., 1965.
- ⁶ James, T. N., *American Journal of Cardiology*, 1968, 22, 479.
- ⁷ Matthews, T. S., and Soothill, J. F., *Lancet*, 1970, 2, 893.
- ⁸ Coombs, R. R. A., *Report on the Samuel Bedson Seminar*, 1970, in press.

Sharman and his colleagues at Loughborough,² in a controlled trial with 400 mg vitamin E daily, examined the performance of adolescent schoolboy swimmers. They found no significant differences at the 5% confidence level in the effects of the vitamin as such, and in particular no evidence was obtained of its suggested ergogenic properties.

- ¹ *British Medical Journal*, 1970, 3, 361.
- ² Sharman, I. M., Down, M. G., and Sen, R. N., *British Journal of Nutrition*, 1971, 26, 265.
- ³ Blaxter, K. L., Brown, F., and MacDonald, A. M., *British Journal of Nutrition*, 1953, 7, 105.
- ⁴ *Report of Medical Research Project into Effects of Altitude in Mexico City, 1965*. London, British Olympic Association, 1966.
- ⁵ Cureton, T. K., *American Journal of Physiology*, 1954, 179, 628.
- ⁶ Cureton, T. K., *The Research Quarterly*, 1955, 26, 391.
- ⁷ Cureton, T. K., *Journal of Physical Education*, 1959-60, 57, 27 and 59.
- ⁸ Cureton, T. K., *Science of Sport*, Tokyo, 1964, 481.
- ⁹ Cureton, T. K., *British Journal of Sports Medicine*, 1971, 5, in press.
- ¹⁰ Thomas, P., *The effects of vitamin E on some aspects of athletic efficiency*, 1957. Ph.D. Thesis, University of Southern California, Los Angeles.
- ¹¹ Prokop, L., *Sportärztliche Praxis*, 1960, 1, 19.

Vitamin E in Athletics

The provision for athletes of a diet rich in nutrients known to have either a direct or indirect effect on muscular performance has been tried,¹ and there are several reasons why vitamin E has attracted interest in this respect.

Recently I. M. Sharman and colleagues² have studied the question whether α -tocopheryl acetate can increase the output of energy and whether it can help sportsmen to perform better and so win their events. Thus in some animals deficiency of the vitamin causes muscular dystrophy,³ and it might therefore be reasonable to assume that when the muscles of competitors are subjected to strain, as in athletic contests, their requirements might be increased and not met by the amounts provided by an ordinary diet. A further reason for expecting that vitamin E might benefit athletic performance is based on the evidence that in experimental animals resistance to both hypoxia and hyperoxia can be influenced by their vitamin E status. The possible protective effect of vitamin E in relation to hypoxia gained importance from the venue of the Olympic Games at Mexico City in 1968. In an investigation organized by the British Olympic Association strong evidence was obtained that the performance of athletes, particularly long-distance runners, is adversely affected by competition at high altitudes.⁴

There have been several claims that vitamin E can improve performance. T. K. Cureton, in a series of papers,⁵⁻⁹ has found beneficial effects when giving athletes doses of either vitamin E or wheat germ oil. On the other hand P. Thomas¹⁰ could find no significant differences between dosed and undosed people in cardiorespiratory and motor-fitness tests. L. Prokop,¹¹ who subsequently investigated the short-term effects of dosing people with the vitamin, found that when they performed a standard exercise task those who had been dosed recovered quicker after exercise. These claims and attempts by other workers to improve performance, mainly swimming, by vitamin E therapy have been made, and while the conclusions have usually been favourable to the value of the vitamin the results have generally been unconvincing on scientific and statistical grounds. Furthermore the effects of training during the trials have not always been taken into consideration, nor has sufficient care been taken to eliminate any psychological influences which might motivate a bias in the results.

"The Loose Back"

Lumbago continues to be a major cause of disability and consequent loss of work. Thus in the year ending June 1967 there were 9.63 million certified days of incapacity from this cause in men and 0.31 million in women.¹ Nevertheless, the word "lumbago" merely signifies low back pain, and its causes range from ligamentous injury and protrusion of an intervertebral disc to secondary neoplasm and osteomyelitis, or psychological upsets.

One little discussed cause of backache is generalized hypermobility of the joints. Well described in 1967 by J. A. Kirk and his colleagues,² this condition is characterized by hypermobility in the hands and wrists, and to a lesser extent in the legs. The hypermobility of the joints is an isolated phenomenon, and is thought to be the result of generalized familial laxity of the ligaments. The symptoms vary in duration and severity, but usually patients complain of pain in the muscles and joints. Often, however, the symptoms are so mild that sufferers do not seek medical attention. Nevertheless, out of 19 of the 24 patients studied by Kirk and his colleagues in whom the sites of joint pain were recorded, four had experienced pain in the back.

R. G. Howes and I. C. Isdale³ have now applied for concept that hypermobility and ligamentous laxity may produce skeletal pain to a study of backache. They have analysed their findings in a study of 102 consecutive cases of "problem" backache. In most of the 59 men the authors were able to satisfy themselves about the cause of the backache, and this group contained no more patients with joint hypermobility than would have been expected from a control series also studied. Nevertheless, the group of 43 women with problem backache contained a subgroup of 20 in whom no definite cause for backache could be found, and in them there were many more patients with hypermobile joints than would have been expected by chance; 17 had increased ranges of movement by two of the criteria used and nearly all of these had hypermobility of the spine.

Lumbago is a symptom, and to be certain of its cause there must be a characteristic history, specific and objective physical signs, or a diagnostic radiological or other abnormality. In most soft tissue lesions only the history and signs are relevant, and it is logical to assume that while both ligamentous strain and a disc protrusion will produce pain and