whose cerebral symptoms necessitated lumbar puncture the cerebrospinal fluid pressure was from 60 to 210 mm of water higher than the level after recovery. One patient, thought to have a cerebral tumour, proved on biopsy to have cerebral oedema.

A rapid increase in intracranial pressure may explain these ocular changes. Muller and Deck[4] concluded from necropsy studies that effusion of cerebrospinal fluid into the optic nerve sheath in cases of sudden intracranial hypertension results in compression of the central retinal vein and dilatation of the nerve sheath, swelling of which reduces the venous drainage of the eye by compressing the retinochoroidal anastomosis, producing retinal venous hypertension and haemorrhage. Cerebral oedema raises intracranial pressure and may be due to a number of mechanisms. Increased ventilation causes hypocarbia,[5] and this by decreasing cerebral blood flow may cause cerebral oedema. Sutton[6] found increased serum cortisol and postulated other hormone changes. Oliguria is an early symptom of mountain sickness and often a precursor of pulmonary or cerebral oedema. Both frusemide and spironolactone have been advocated as prophylactics of mountain sickness, while diuresis at altitude is a sign of likely freedom from this complaint. However it is produced, cerebral oedema can cause increased pressure in the retinal veins; the hypoxic retinal capillaries may then be unable to withstand the increased back pressure, and the typical splinter and flame shaped haemorrhages appear.

Acute mountain sickness, pulmonary oedema, cerebral oedema, and retinal haemorrhage are different facets of failure to acclimatize at high altitude. Rapid ascent over 8000 ft (2500 m) is hazardous. Travellers in mountains must guard against two temptations especially—going too high too quickly by aeroplane and car and the inclination to "press on regardless."

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Risk of Cot Deaths

The unexpected death of a child at home remains the major cause of death in children between the ages of 1 week and 2 years. Much of the recent work is recorded in the proceedings of a meeting held in Toronto in 1974[1] that was sponsored by the Canadian and British Foundations for the Study of Infant Deaths.[2]

No matter how complete the necropsy, about a quarter of cot deaths remain unexplained, though the same is true of many children of this age dying in hospital. Much of the basic work on unexpected death, such as Wealthall's study of apnoea,[3] applies at least as much to hospital as to home deaths.

The concept that the deaths are due to a cessation of respiration during a period of prolonged apnoea,[4] nasal obstruction,[5] or laryngeal spasm[6] has recently been extended by Tonkin[7] in Auckland. She has shown that some infants are vulnerable at the oropharyngeal level and that airway obstruction can occur during the muscular relaxation of R.E.M. (rapid eye movement) sleep. Meanwhile the chase after specific infections continues. Gardner and his Newcastle group[8] back the respiratory syncytial virus as chief culprit, while Nelson and colleagues[9] in Chicago prefer influenza A virus. There is no shortage of new hypotheses such as that the deaths are due to preleukaemia[10] or hypoimmunity.[11]

The supposition that all unexpected, unexplained infant deaths have a single cause is now yielding to the idea that they have a variety of causes. The examination of vitreous humour at necropsy[12–13] and the discovery that some infants presenting as cot deaths had severe hypernatraemia and uraemia[14] have concentrated awareness on the dangers of feeding babies overconcentrated milk and milk with increased salts,[15] and this has been a stimulus to encourage breast feeding.

Meanwhile, in some centres such as Philadelphia,[16] where there has been a longstanding interest in cot deaths, there is an indication that the numbers are diminishing, and though the figures are not yet published the same probably holds for at least one centre in Britain. In northern Europe the cot death rate seems to parallel the general infant mortality rate. But it is odd that a country such as Holland, which has a similar genetic, climatic, and infection background to Britain, has a cot death rate of about one-third,[17] and the same seems to be true of Sweden.[18]

The fact that at necropsy most babies found unexpectedly dead show the presence of some treatable disease[19] may indicate that the different death rates between Holland and Britain could lie in the home care services for children. Simply by improving these could we halve our total and unexpected infant death rate? The study by McWeeny and Emery[20] of the backgrounds of some cot deaths due to recognizable disease disclosed the existence of incompetence (social, parental, and medical) that was hinted at by a survey in Glasgow in 1970.[21] Meanwhile, as Brimblecombe points out,[22] it is possible to recognize families at risk of cot deaths[23] and to try to improve their home care.

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