only one putative father is named, or divorce, which usually provides the opportunity of testing more than one possible father for the child. A blood group investigation is also occasionally requested in criminal cases such as alleged incest, rape resulting in pregnancy, or, rarely, kidnapping. Ironically, pari passu with the ever-increasing efficiency of blood-group studies in solving paternity problems has gone a considerable decline in the number of affiliation cases brought before the courts. These reached a peak of 9000 in 1968 and fell to 3000 in 1975. No doubt the greater availability of the pill must have had some role in this.

[1] Race, R R, and Sanger, R, *Blood Groups in Man*, 6th edn, p 506. Oxford, Blackwell Scientific Publications, 1975.
[2] Stevenson, A C, and Kerr, C B, *Mutation Research*, 1967, **4**, 339.
[3] Cook, P L, *et al*, *Annals of Human Genetics*, 1969/1970, **33**, 125.
[4] *Family Law Reform Act 1969*, London, HMSO.

Immersion and drowning in children

In the ten years to 1974 the Registrar General's records show 700 deaths per year in Britain from immersion in water.[1] The number, which excludes suicides or open verdicts, will probably increase because of the growth of aquatic sports. In the United States drowning is the third most common cause of death in children between 1 and 4 years (after road traffic accidents and burns) and second only to road traffic accidents in those aged 4 to 14. The popularity of swimming pools in California makes drowning the second most common cause of death there in all children.

The main problems presented by the near-drowned child are anoxia, inhalation, and hypothermia.[2-4] Anoxia may be caused by obstruction of the airways and other mechanisms. Deliberate overventilation before underwater swimming—often in an attempt to break some record—is highly dangerous.[5] The hyperventilation causes mild respiratory alkalosis, and, with the lowered partial pressure of CO_2, the arterial oxygen saturation may decrease dangerously before the blood CO_2 rises sufficiently to make the child surface to take a breath—so that he may become unconscious under water. Overventilation may also occur involuntarily when a child (or adult) is suddenly immersed in cold water and may continue even under water, so that he may drown.

Inhalation is dangerous, though not all children inhale water when they fall in. The result depends partly on the nature of the water inhaled—fresh or salt water, clean or polluted. Inhaling fresh water into the lung causes haemodilution from absorption into the blood stream, with a consequent fall in the blood sodium and chloride and occasionally some haemolysis. Salt water has a higher osmotic pressure than the blood, and so withdraws water from capillaries, causing hypernatraemia and haemoconcentration but no haemolysis. Inhaled vomit is another serious risk.

The importance of hypothermia is being recognised only slowly.[1 3 4 6 7] As the core temperature falls below 35°C, the pulse and respiration rates both decrease.[4] Below 33°C shivering may stop and consciousness become clouded. Below 30°C consciousness is usually lost and cardiac arrhythmias may develop, especially below 28°C. If a hypothermic child is unconscious and not reacting to pain the core temperature may be assumed to be under 30°C. Below 27°C life is difficult to detect—the patient may be motionless and "pulseless" with flaccid limbs and fixed dilated pupils. Failure to respond to resuscitation is the only definite criterion for diagnosing death from hypothermia: a flat ECG is not proof of death. Hence, except in tropical waters, the main threat to life of immersion is cold, or a combination of cold and drowning.[3] A child's relatively large ratio of surface area to body mass leads to more rapid chilling than in an adult: cerebral metabolism falls and there is a longer interval between cardiac arrest and brain damage.

If a child is conscious he is unlikely to have severe hypothermia, but he may lose consciousness after rescue if his core temperature falls further by loss of heat to the cold peripheral tissues. His wet clothes should be removed and further heat loss minimised by blankets, a sleeping bag, or a polyethylene "exposure" bag. If he is conscious he will rapidly recover in a hot bath. If he is unconscious and not breathing, debris or vomit should be cleaned from the mouth, and if he has been in sea-water he should be given a moment's postural drainage before starting mouth-to-mouth resuscitation. The difficulty is to distinguish hypothermia from cardiac arrest. In severe hypothermia there may appear to be cardiac arrest when there is not; the pulse cannot be felt and an incorrect assessment can be made. Golden[4] suggested that hypothermia is likely if the body is in a life jacket and recovered apparently dead; treatment for hypothermia is then the first essential. If there were signs of life just before rescue, hypothermia is less likely to be the primary problem.

The distinction is important. If the basic problem is one of severe hypothermia it is essential to avoid cardiac massage, rubbing or massaging the arms or legs, intubation or other instrumentation—all of which carry a grave risk of precipitating ventricular fibrillation, the most common cause of death in hypothermia.[6 7] Handling of any sort should be reduced to the absolute minimum. The urgent need is to rewarm the child, preferably by heating the core of the body first, since this is less likely to cause hypotension and arrhythmias than peripheral rewarming, as in a hot bath.

The best method of central rewarming is to make the patient breathe hot moist air or oxygen; a portable device[10] is now available which can be used at the site of the accident. If such an apparatus is not available the child should be placed in a hot bath at 40-44°C (as hot as the hand can bear), if possible keeping his arms and legs out of the water. Deterioration in the bath may be due to ventricular fibrillation, and in that case external cardiac massage may be necessary, both cardiac massage and ventilation being applied at half the usual rate. Metabolic acidosis often decreases spontaneously as the temperature rises, but if necessary it can be corrected by intravenous 4·2% sodium bicarbonate heated to 37°C.

If the problem is not one primarily of hypothermia the child should be ventilated and external cardiac massage applied, once debris and vomit have been cleared from the mouth. Antiarrhythmic drugs should not be given. Most arrhythmias (except ventricular fibrillation) disappear spontaneously as the child improves, and the drugs are ineffective and possibly dangerous.[3]

Complications of immersion may include pneumonia; aspiration pneumonia; collapse of the lung; acute pulmonary oedema (developing any time between 15 minutes and 72 hours after the accident); pneumothorax or pneumomediastinum (both possibly due to resuscitative measures); and a later respiratory-distress-like syndrome with loss of surfactant.

The prognosis depends on several factors: the temperature of the water; the degree of pollution; the duration and degree

of hypothermia; the duration of cardiac arrest; and the promptness and correctness of treatment. Peterson's pessimistic report from California,[8] in which all 15 near-drowned children who had fits, fixed dilated pupils, flaccidity, and loss of pain sensation suffered severe anoxic encephalopathy, may be explained by the high water temperatures there. In warm water the protective effect of hypothermia against brain damage would be missing. In contrast was the case described in Trondheim, Norway,[9] in which a 5-year-old fell through ice in fresh water with an outside temperature of 10° below zero centigrade. He was in the water for 22 minutes and was brought out apparently dead, with widely dilated pupils and bluish white skin. He was warmed up (the method was not described) and—despite the risks noted above—was given external cardiac massage without suffering ventricular fibrillation, the heart beat returning in 2½ hours. Subsequently the child had an exchange transfusion to cope with haemolysis and haemoglobinuria. He was unconscious for six weeks but recovered, becoming a normal active boy with only trivial clumsiness of the hands and an almost normal mental age. The moral is that persistence with intensive measures is usually justified.

[1] Golden, F St C, and Rivers, J F, *Anaesthesia*, 1975, **30,** 364.
[2] Imburg, J, and Hartney, T C, *Pediatrics*, 1966, **37,** 684.
[3] Keatinge, W R, *Survival in Cold Water*. Oxford, Blackwell, 1969.
[4] Golden, F St C, *Proceedings of the Royal Society of Medicine*, 1973, **66,** 1058.
[5] Craig, A, *American Journal of Diseases of Children*, 1973, **125,** 643.
[6] Lloyd, E L, *British Journal of Anaesthesia*, 1973, **45,** 41.
[7] Lloyd, E L, and Mitchell, B, *Lancet*, 1974, **2,** 1294.
[8] Peterson, B, *Pediatrics*, 1977, **59,** 364.
[9] Kvittingen, T D, and Naess, A, *British Medical Journal*, 1963, **1,** 1315.
[10] The Reviva, made by Peter Bell Engineering, the Slack, Ambleside, Cumbria.

Localisation of visual hallucinations

Hallucinations are perceptions in the absence of external stimuli; illusions are misinterpretations of external stimuli. As it is impossible to establish how much insight an individual has into phenomena of this kind it is better to work with broad definitions.[1] Imagery forms part of everyone's mental process, providing thought-vehicles for reasoning, for memory, and for taking in sensory data, as in rapid reading. The quality of visual (or eidetic) imagery varies from person to person, with every inflexion from achromatic silhouettes to the vivid recollection of colours and hues. Hallucinations and illusions impinge on the consciousness of normal people in dreams; in hypnagogic states; in wish-fulfilment hallucinations of the bereaved; under stress or sensory deprivation; or as organic symptoms such as tinnitus, thalamic pain, and sensations from a phantom limb.

Most observers have been unhappy to attribute all hallucinations to disturbances in the psychological mechanisms of thought: thus Hughlings Jackson[2] claimed that hallucinations originated in instability of cortical cells. Duke-Elder[3] argued that these phenomena had a physical basis, for a non-physiological (inadequate) stimulus applied to any part of the neural pathway may produce visual hallucinations. Many hallucinations result from impaired cerebration in dementias, arteriosclerosis, toxic states, or hallucinogenic drugs—or from dissociation of the reticular activating system, as in disturbances of sleep or consciousness.[4] Hallucinations may be evoked by sensory deprivation, but other factors are usually present; thus the "black-patch" delirium that may follow cataract extraction in the elderly probably results from sensory deprivation and mild senile brain changes and is more frequent when hearing is also impaired.[5]

Hallucinations may be due to focal lesions. Past experiences, the quality of eidetic imagery, and psychodynamic factors influence the content of organic hallucinosis,[6] and it would be unwise to lean too heavily on the occurrence and nature of hallucinosis in localising intracranial lesions. Visual hallucinations are a relatively infrequent accompaniment of lesions of the calcarine cortex (Brodmann's area 17), which has few thalamocortical connections; yet they may occur as a false-localising symptom of frontal or subtentorial lesions. Ritchie Russell and Whitty,[7] tabulating the degree of complexity of visual hallucinations found with wounds of the calcarine cortex, optic radiation, and higher centres, have shown considerable overlap in the type of recorded hallucination.

Formed and unformed hallucinations have been reported with subcortical and neural lesions. Lesions of the optic tracts, chiasm, optic radiation, and lateral geniculate bodies can evoke simple hallucinations, usually in the form of brief flashes of light. Although more complex hallucinations have been described, the circumscribed localisation of the lesions cannot be accepted uncritically.[8] Of historic interest is Lhermitte's peduncular hallucinosis,[9] where a lesion of the cerebral peduncles results in mild confusion with hallucinations of brightly coloured, kaleidoscopic, Lilliputian people.

Reports of tumour localisation cannot match the physiological exactitude of the stimulation experiments of Penfield[6] or the studies of traumatic epilepsy of Ritchie Russell and Whitty[7] and of cerebral infarction recently presented by Lance.[8] Such data provide a basis for a hypothetical scheme of cerebral localisation, but its value is theoretical rather than clinical.

Hallucinations from the occipital cortex and association areas usually project on the contralateral visual field, which more often than not is a blind one. Lesions of the occipital pole of the calcarine cortex produce static lights and stars. With more anterior lesions the lights appear at the periphery and move towards the centre. From the parastriate area 18 luminous sensations may be obtained of coloured flashes and rings; alternatively, stimulation of these areas may produce negative phenomena as a grey or black fog—phenomena similar to the scotomata of migraine.

Hallucinations from the parieto-occipital cortex, including parastriate area 19, are stereotyped, with an emphasis on objects, people, and animals. These bizarre apparitions are one degree more complex than the flashes, zigzags, and whorls of colour obtained from the primary receptive cortex, but they fall short of an integrated visual memory.[8] From the visual association areas other phenomena may occur along with visual hallucinations.[10 11] These include perseveration of a visual image in time (palinopsia) or in space (visual illusory spread)[12]; impaired visual recognition (visual agnosia or prosopagnosia); defective visual localisation[13]; errors in naming colours (anomia)[14]; and defective perception of colours (achromatopsia).[15]

Hallucinations from the temporal cortex are more complex. Scenes may be recalled from experience after stimulation of the posterior part of the temporal lobe. Visual hallucinations alone may result from stimulating a large area of the lateral surface of the non-dominant temporal lobe. Elsewhere they may be combined with auditory hallucinations—and there may