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Annotations

The urgency of immersions

Accidental child drowning continues to challenge many specialty disciplines in the broader field of child health. Despite resolute community based attacks on the problem of immersion accidents,1-4 some now going back more than 15 years,5-6 resuscitators, intensive care physicians, and paediatricians remain frequently involved. Epidemiologists, preventive medicine exponents, and legislators have had some success in reducing the numbers of fatal immersion incidents in childhood.4,7 Much remains to be done.

Clinical approaches to the management of the near drowned child have seen big changes in the past decade.8 The management pendulum has swung from that of basic life support in the late 1970s, through a phase best described as an aggressive but empirical interventionist approach,9 back to a current intermediate position of advanced life support10 for the child after immersion with a minimum of a residual heart beat. The years of the 1980s were controversial, not necessarily a bad thing in itself, but were wanting in the publication of sufficient control studies to lift treatment of the near drowned from the empirical to rational objectivity. Such studies are still awaited.

Epidemiological approaches to prevention have also been controversial, certainly at the community level of society. In the suburbs and on the farm, in a few enlightened societies, the reduction of the threat to children from water hazards has been bought at the expense of personal freedom, and considerable financial expense. In those countries (for example, Australia and New Zealand) that have grappled to introduce safety legislation for water hazards, and those countries in which it has been advocated, there has been bitter and prolonged public debate not only about the potential efficacy of such legislation but also its fundamental philosophy.

Epidemiology

The risks of drowning vary obviously with the density of water hazards in any child’s environment. Children living in water oriented societies, especially those with major recreational use of the sea, lakes, rivers, and swimming pools are at special risk. In the USA more than 2000 children drown each year,12 and residual neurological deficits occur in a percentage of another 8000 survivors.13 In the UK drowning has caused an average of 72 child deaths per year for the past 10 years.14 Another 150 cases per year survive, some 5% of whom have significant neurological abnormalities. There are particular problems in New Zealand.2 Some 800 toddlers have drowned in home swimming pools in Australia since the epidemic threat to children was first recognised,5-6 and more than 1000 survivors have been pulled pulseless from the water. Some 30% of survivors from fresh water near drowning manifest measurable residual deficits,15-16 a minimum of 3% existing in a permanent vegetative state.5,15

Accident syndromes

There are several distinct drowning 'syndromes' in childhood: bath tubs,17-18 pools and ponds,2-5 8 rivers and lakes,5 8 and sea,7 19 vehicle immersions with child occupants,20 spa pools,21 child abuse and homicide by immersion,8 22 23 and alcohol related water accidents involving older teenagers.8 24 25 All have different peak ages of risk, and all require different and highly specific approaches to primary prevention.5 8

Clinical management

From the clinical point of view, skilled rescue site resuscitation will convert some 30% of (otherwise) fatalities into survivors. If any suspicion of hypothermia8-16 or drug ingestion22 exists, resuscitation should be continued until mechanical ventilation can be instituted, and these potential confounding variables clarified. Mechanical ventilation and positive end expiratory pressure,8 11 and probable monitoring of intracranial pressure, remain the mainstay of treatment. One of the 'holy grails' of the resuscitation world is the search for a brain sparing drug to be given after the event. After the disappointment of the attempts using barbiturate rescue, there is nevertheless some hope that other drugs will hold promise. Appropriately controlled animal studies with blinded operators and observers are needed before further clinical trials are undertaken. Outcome predictors (once the child has reached the intensive care ward) such as persistent hypoxaemia, persistent acidosis (pH below 7·0),8 the 'time to the first spontaneous gasp,8 and the use of the Glasgow coma scale11 all enable reasonable predictions of significant neurological damage to be made. Provided hypothermia is not a feature of a near drowning episode, children who do not make their first spontaneous gasp within 40 minutes or so after extraction from the water, and in whom skilled resuscitation is being performed, virtually never escape serious neurological damage. Skilled and standard intensive care techniques are such that it is unlikely that a child with an intact heartbeat will die subsequently. Salvage rates, at least in terms of a
child with a beating heart and spontaneous intact respiration, exceed 80% of those cases who arrive alive in the intensive care unit. All the studies of childhood freshwater pond and pool immersions have found a minimum of a 50% survival rate, and although better resuscitation is condemned by some as only 'secondary prevention', it offers a potential short term solution to saving many children's lives.

The biggest challenge to clinical management therefore is improving prehospital resuscitation. Questions about the efficacy of out of hospital resuscitation are controversial and topical but our experience with child near drowning victims leads us to the uncompromising viewpoint that 'every parent should be a first aider'. Attempts to institute such a policy, at least for pool owning families, have so far failed. However, by continued societal advocacy, paediatricians can create a public arena where such must come to be regarded as one of the essential skills of parenting. May that time roll on.

Prevention and a medical model
The biggest impediment to the solution of childhood immersions is the failure of the medical profession to adopt a medical model to deal with the problem. Despite the fact that immersion continues to rank as one of the major killers of young children in many parts of the world, many remain reluctant to accept this as a medical or public health problem. The techniques of preventive medicine are well honed in the area of infectious and nutritional disease as these occur in the childhood years. Genetic diseases are currently vigorously approached by the portals of genetic counselling and prenatal diagnosis. Child trauma more generally has responded to aggressive intervention using the traditional themes of preventive medicine, but drowning remains the poor relation when judged by any audit of results revealed in contemporary surveys. One reason for this is the very high fatality:survivor ratios for immersion accidents. For every child who drowns, another (1:1) is pulled from the water pulseless but who subsequently responds to resuscitation. In the case of road trauma the ratio is 1:20 and 1:2000 in the case of accidental poison ingestion. Thus the size of the problem is partly masked as most of the immersion victims bypass the hospital and go straight to the mortuary, and are not seen in the ambience of the intensive care wards. Furthermore, in many countries society at large has been conditioned by newspaper reports of yet another toddler drowning fatality'. It is thus by continued advocacy, and the use of traditional methods that have conquered other high ranking problems in preventive medicine, that this problem will be effectively addressed.

Currently the urgency of childhood immersions is the responsibility of us all.

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Reactive amylloidosis
Reactive amylloidosis (also known as systemic amylloidosis or secondary amylloidosis) is a disorder which occurs as a complication of chronic inflammatory disorders such as juvenile chronic arthritis, chronic infective disorders such as bronchiectasis, osteomyelitis, tuberculosis, or leprosy in susceptible individuals. It is also responsible for renal failure in the hereditary form of amylloidosis associated with familial Mediterranean fever, particularly in North African Sephardic Jews.1 Recently it has been described to have been the cause of the severe and rapidly fatal nephropathy which occurs in adolescents with cystic fibrosis.2 The table is a summary of the different types of amylloidosis.

Fibrillar proteins are deposited together with glycosaminoglycans (GAG), serum amyloid P component, and fibrinectin, leading to cellular dysfunction. The spleen appears always to be involved in reactive amylloidosis. The kidneys, liver, adrenal gland, and gastrointestinal tract are frequently involved. By light microscopy, the amyloid deposit appears as a homogeneous eosinophilic material that stains with Congo Red. Under polarised light microscopy the congophilic material has a characteristic apple green birefringence.

Pathogenesis of reactive amylloidosis
The protein that forms the fibrils in amyloid deposits is...
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