diuretic treatment is worth trying before resorting to potent drugs such as postganglionic adrenergic blockers or minoxidil in resistant hypertension. If false tolerance is present a postganglionic adrenergic blocker will cause further volume expansion and is unlikely to lower the blood pressure. Minoxidil will reduce the blood pressure in the face of expanded plasma and extracellular fluid volumes,[20] but concurrent use of high doses of diuretics is almost invariably needed. It would be sensible to observe first the effect of increased diuretic treatment alone.

The optimal method of using diuretics in resistant hypertension is not established. The effect of frusemide as a single daily dose seems satisfactory,[21] but it is not clear whether it should be added[5 9] or substituted,[7] given continuously[9 10] or intermittently,[5] or whether it is more[5] or less[11] effective than spironolactone. At present we suggest that compliant patients resistant to an adequate regimen of three drugs should have frusemide 80 mg or spironolactone 100 mg added to the thiazide. Frusemide should be used when there is renal impairment (serum creatinine >130 μmol/l). If the patient does not respond the dose of frusemide or spironolactone may be increased according to tolerance until weight loss of 1 kg is attained, before abandoning the manoeuvre as ineffective. Urea and electrolyte concentrations should be monitored whichever diuretic is added.

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(*Accepted 18 September 1980*)

Secondary drowning in children

JOHN H PEARN

Summary and conclusions

Secondary drowning (and near-drowning) is one of the post-immersion respiratory syndromes. It is defined as deterioration of pulmonary function that follows deficient gas exchange due to loss or inactivation of surfactant. A review of 94 consecutive cases of near-drowning in childhood showed that this syndrome occurred in five (5%) cases. Its onset was usually rapid and characterised by a latent period of one to 48 hours of relative respiratory well being. It occurred more rapidly after immersion in fresh water. The two children immersed in salt water died of secondary drowning, while the three immersed in fresh water recovered completely.

If it is anticipated, recognised, and treated vigorously prognosis of secondary drowning is good in fresh water cases but bad after salt water immersion.

Department of Child Health, Royal Children's Hospital, Brisbane, Queensland 4029, Australia
JOHN H PEARN, MD, FRACP, reader in child health

Introduction

In any series of drowned or near-drowned individuals, patients are described who initially respond well to resuscitation but whose respiratory function deteriorates over the next few hours. The phenomenon is well known from case reports,[1-4] and is thought to be due to loss of surfactant from chemical, anoxic, or osmotic damage to the pneumatocytes that line the alveoli. It may be fatal in both children[5] and adults[1] and is one of the causes of "delayed death subsequent to near-drowning."[6]

This phenomenon has been called "secondary drowning"[2 7-9] and is characterised by a latent period of several hours,[4] or even longer.[2 8 10] The syndrome may be defined as the occurrence of respiratory deterioration after successful resuscitation owing to primary alveolar membrane dysfunction. Estimates of its frequency have been unsatisfactory because of case selection, but the syndrome is thought to occur in at least 2% of sea water near-drownings.[11] The syndrome has occurred after both fresh water[1-7] and salt water immersions.[1 2]

As part of the Brisbane Drowning Study[12-14] we have encountered several examples of this phenomenon. Some children responded so well to rescue-site resuscitation that they were not initially admitted to hospital, only to be found in grave respiratory distress several hours later. This report describes five cases of

secondary drowning that occurred in a consecutive unselected series of 94 children who were not dead when taken from the water.

Patients and methods

The case records of 94 consecutive unselected children who had suffered drowning accidents but who had been alive when taken out of the water were reviewed. Fifty-nine of the accidents had occurred in fresh water and 35 in sea water. The epidemiological features of these cases have been described.[13 14] The clinical features of the children who suffered secondary drowning were reviewed.

There are two types of death after drowning, firstly, death in the water, and, secondly, "delayed death subsequent to near-drowning," defined by Modell as death after apparently successful rescue or resuscitation.[6] Secondary drowning was defined as death or serious clinical deterioration caused by inadequate respiratory ventilation, perfusion, and alveolar gas exchange which occurred after a period of relative respiratory well being and was not due to (*a*) neurological causes or (*b*) respiratory sequelae of inhaled foreign material or secondary infection.

Results and comments

Five cases of secondary drowning occurred among the 94 children who were unconscious and apnoeic when pulled from the water. Two had been immersed in salt water and three in fresh water. The table summarises the case details and the clinical syndrome. In each case the child was alive after being rescued and developed spontaneous respiration. The children were often fully conscious and appeared well. After one to 48 hours of respiratory well being they suffered a sudden and rapid deterioration in their pulmonary reserve and their arterial Po_2 fell. Dyspnoea, increasing cyanosis, and apnoea, which were not due to neurological damage, then developed. The prognosis seemed to be related to age, the severity of immersion, and the osmolality of the water.

In four of the five cases the inhaled water contained either chlorine or salt. Whereas most children who are going to survive make their first respiratory gasp in response to cardiopulmonary resuscitation within five minutes, the median time for initial response in this series was 15 minutes; this suggests that secondary drowning is more likely to occur in the more serious cases of near-drowning. The latent period before deterioration also varied between the two osmotic groups, lasting less than four hours in the fresh water cases, but lasting much longer in the salt water immersions.

Both the children who had been nearly drowned in salt water died, while the three immersed in fresh water recovered. The median intelligence quotient of the fresh water survivors was 104, which suggested that despite the clinical deterioration due to respiratory (as opposed to neurological) causes, the prognosis is still good for young children who suffer this respiratory complication of fresh water near-drowning.

Discussion

Excluding death, there are three defined syndromes which may follow near-drowning—neurological syndromes (either acute or chronic),[15] respiratory syndromes, and the salt water aspiration syndrome described by Edmonds.[2] The respiratory causes of post-rescue deterioration include true secondary drowning, bacterial pneumonia, pulmonary barotrauma, mechanical lung damage from resuscitation, foreign body or chemical pneumonitis (sand, mud, weeds, vomitus), inadequate ventilation or apnoea secondary to central neurological damage, and oxygen toxicity. These conditions constitute the differential diagnoses in victims of near-drowning accidents who show evidence of respiratory deterioration some time after the accident. It is important to realise that secondary apnoea (often sudden[16]) may be secondary to central anoxic damage.

Pulmonary dynamics during drowning have been studied by the ingenious single lung experiments of den Otter in Holland.[17] In both salt and fresh water inhalation dramatic water transfer occurs across the alveolar membrane in the direction alveolus-to-blood. Water-containing areas of lung are underperfused due to reflex vasoconstriction, but ventilation-perfusion ratios remain low. Alveolar-lining pneumatocytes are thus rendered hypoxic as well as suffering osmotic and chemical effects. The alveolar capillary membrane becomes oedematous or disrupted.[4] In Russian experiments with dogs using a bypass artificial circulation pulmonary oedema always followed fresh water inhalation and was a cause of death three to 72 hours after "rescue" in spite of adequate circulatory dynamics.[18] Besides physical damage to surfactant-producing cells, surfactant itself is inactivated by contact with hypo-osmotic water[19]; even if adequate treatment is given, surfactant may take 24 hours to regenerate.[20] The fact that salt water does not inactivate pulmonary surfactant[21] may explain the difference in latent period observed in this series between salt and fresh water cases. In fresh water cases the condition may result from direct inactivation of surfactant, whereas in salt water cases it may result from later diminished surfactant production due to damaged pneumatocytes. Hyaline material can be seen in alveoli, alveolar ducts, and respiratory bronchioles within 12 hours to three days after the immersion[1]; alveolar capillaries become disrupted and masses of agglutinated platelets can be identified.

The management of the near-drowned is governed by both respiratory and neurological considerations. Recent trends in the use of high-dose barbiturates (to reduce brain swelling[22]) to protect against hypoxic brain damage have necessitated greater recourse to elective artificial ventilation.

The management of respiratory problems after near-drowning is now fairly standard. If the patient is breathing spontaneously and the arterial Po_2 is improving with conservative treatment

Details of the five cases of secondary drowning

Case No	Age	Sex	Water	Estimated immersion time (minutes)	Resuscitation (expired air)	Time to first gasp (minutes)	Latent period (hours)	Progress	Outcome
1	9 mnth	F	Bath tub; pure fresh water	3-5	+	15	4	Rapid onset of wheezing with cyanosis and increasing dyspnoea. In hospital 2 days	Normal child IQ = 114
2	2 yr 7 mnth	M	Floating in chlorinated swimming pool	"Few minutes"	Nil	1-2	3	Onset of dyspnoea and cyanosis. At 5 hours $Po_2 = 28$, $CO_2 = 53$, $HCO_3 = 21{\cdot}4$ mmol/l. In hospital 16 days	Normal child IQ = 104
3	1 yr 7 mnth	M	Floating in chlorinated swimming pool	1-3	+	1	1	After latent period, rapid onset of dyspnoea with gradually weakening respiratory effort. Respirated for 18 hours. In hospital 12 days	Normal child IQ = 101
4	6 yr	M	Still salt water. Found on bottom	4-8	+	15-30	40-45	Still unconscious and hypotonic 36 h after rescue. Cyanosis developed; subsequent lessening of respiratory excursion	Died 56 hours after initial rescue
5	10 yr	M	Still salt water. Floating face down	Unknown	+	30-60	36	Gradual decline in effective tissue oxygenation. Signs of pulmonary oedema. Placed on ventilation 40 hours after rescue. Later developed pneumonic signs	Died 8 days after immersion

no special intervention is required. If ventilation is inadequate from the start or if arterial Po_2 deteriorates more aggressive treatment is indicated. Mechanical ventilation with positive-end-expiratory-pressure is required,[21 23] titrating the amount of pressure to produce the minimum amount of intrapulmonary shunt.[21] The use of corticosteroids is not routinely recommended, although their place in the patient with true secondary drowning has not been studied in any large series.

Recognising the possibility of this syndrome of secondary drowning is a major factor in management. An analysis of this and other series shows that a major high-risk group comprises young children who have almost drowned in salt or polluted water, in whom spontaneous respiration has not occurred for at least five to 10 minutes after rescue, but who appear to improve rapidly thereafter. Although it is not always done, all near-drowned victims must be admitted to hospital for observation, irrespective of their apparent relative wellbeing within several hours after rescue. Respiratory deterioration after apparent post-rescue wellbeing can occur rapidly.

With the recent increase in survival rates after near-drowning[24] more cases of this post-immersion respiratory distress syndrome are being encountered.[25] This review suggests that rescuers and clinicians should expect primary lung function to deteriorate within four hours of rescue in about one in 20 survivors of drowning accidents. Provided that the syndrome is anticipated, recognised, and treated vigorously the prognosis (generally excellent in childhood near-drowning[15]) should remain optimistic.

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(*Accepted 18 September 1980*)

SHORT REPORTS

Injection abscesses in a diabetic due to Mycobacterium chelonei var abscessus

Most diabetics do not sterilise their hypodermic syringes before each insulin injection. They use glass syringes, sterilise them at intervals, and keep them in disinfectant. We report the case of an insulin-dependent diabetic who stored her glass syringe in disinfectant which was inadvertently inactivated and who developed multiple injection abscesses caused by *Mycobacterium chelonei*, var *abscessus*.

Case report

The patient, a 24-year-old English woman whose diabetes was diagnosed in 1968, first noticed a lesion on the upper lateral aspect of her right thigh in October 1979. At this time she was using both thighs as insulin injection sites. The lesion was deeply subcutaneous, painful, indurated, roughly 2 cm × 3 cm in size, and the overlying skin was warm and discoloured, breaking down after two weeks with a central discharging sinus. Routine culture of sinus pus did not detect any recognised bacterial pathogens. A course of flucloxacillin was ineffectual. A similar lesion developed after two weeks on the opposite thigh, and culture of pus taken from it yielded an anaerobic coccus (*Peptococcus* spp). She was treated with metronidazole for five weeks with no perceptible effect upon the lesions. In November 1979 each of them had extended deep satellite lesions into the surrounding subcutaneous tissues, and one of these broke through to the surface, discharging buff-coloured creamy pus. In February 1980, after five weeks' incubation at 30°C, a Lowenstein Jensen culture of pus draining from the original abscess on the right thigh yielded a growth of an atypical mycobacterium. The organism was identified by Dulwich Regional Tuberculosis Laboratory and by Dr J L Stanford of the Middlesex Hospital as *M chelonei*, var *abscessus*, and was subsequently shown to be resistant in vitro to isoniazid, PAS, ethambutol, rifampicin, and co-trimoxazole but sensitive to erythromycin. At about the time the organism was isolated the patient developed two further inflamed lesions, one on each buttock close to the ischial tuberosities in areas remote from any insulin injection sites. Treatment was begun with a combination of oral erythromycin and co-trimoxazole and the lesions have steadily resolved since.

Comment

M chelonei is a rare human pathogen. When it is pathogenic it may cause injection abscesses[1] with a prolonged incubation period of up to several months or, less commonly, severe generalised infections in immunosuppressed patients.[2] Probably our patient's initial abscess resulted from organisms introduced by an insulin injection. We cannot be sure if the second abscess was caused in the same way, since infected lesions may develop at sites of trauma during a period of *M chelonei* septicaemia, and this could account for the third and fourth abscesses. Our patient, normally fastidious in her injection technique, used a non-disposable syringe and disposable needles. Each needle was used for two injections. The syringe was boiled weekly and stored, with the needle, in hypochlorite. Unfortunately, in her twice-weekly preparation of hypochlorite she often used boiling water, which would have driven off the chlorine and rendered the solution inactive. She now uses a new disposable needle and syringe for each injection.

One must accept that if a syringe and needle are reused without