Current Practice

Drowning. Its Clinical Sequelae and Management

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Drowning is responsible for between 1,000 and 1,500 deaths annually. About 20% of these deaths are due to dry drowning, the so-called asphyxial deaths. The remainder are classical wet drowning cases from either fresh or salt water. Secondary drowning—that is, delayed death from drowning between a quarter of an hour and as long as four days after the drowning incident—has received far less attention. In the vast majority of cases the patient will either survive as a result of immediate resuscitative measures beside the swimming-pool or on the beach or die. It is usually thought that once the patient has been admitted to hospital the crisis is over. In one large series of 77 cases of near drowning reported on, however, 25% succumbed to secondary drowning (Fuller, 1963). A good deal of research has already been carried out into the physiopathological consequences of drowning in fresh and salt water (Swann *et al.*, 1947; Swann and Spafford, 1951; Modell, 1968).

The purpose of this paper is to review the clinical consequences of near drowning and to discuss their management. Little attention has been directed towards the mechanisms and management of secondary drowning. Indeed, very few standard medical textbooks even mention drowning and its sequelae and those that do still place undue emphasis on defining the type of drowning as a prerequisite of treatment. The immediate consequences of drowning are either death or successful resuscitation. The secondary effects, which make up the clinical situation met with in patients eventually admitted to hospital, may result in disaster in those who appear to have recovered from the acute episode unless the utmost vigilance is maintained. We describe four cases of near drowning, two in salt water and two in fresh water, which illustrate all the facets of the clinical presentation and management of such cases.

Cases 1 and 2

The first two cases are considered together as they were involved in the same drowning incident.

A 19-year-old non-swimmer (Case 1) slipped into a fresh-water pool on 8 September 1968 and went under the surface immediately. He managed to struggle to the surface twice and then remained submerged. His colleague (Case 2), also a non-swimmer, jumped into the rescue but immediately submerged and failed to resurface. It is estimated that both patients were on the pool bottom for something like two minutes, though obviously Case 1 was there longer. Both patients were dragged out unconscious from the pool blue, cold, but still breathing. At 7.38 p.m. (eight minutes after slipping into the pool) both patients were coughing up frothy pink sputum. Case 2 apparently recovered quickly, but Case 1, who had been submerged longer, regained consciousness more slowly, about 10 minutes after immersion. Neither patient required artificial respiration or closed chest cardiac massage. It is not known whether they received oxygen on the way to hospital. On arrival at hospital, however, both patients were given oxygen, particularly Case 1, who had vomited during the journey and was severely cyanosed. He improved rapidly when given oxygen at 8.05 p.m. Both patients were then transferred to St. Mary's General Hospital, where they arrived at 11 p.m.

Case 1

The patient was conscious, with a temperature of 101°F. (38·3°C.), pulse 126 per minute, regular; blood pressure 116/80 mm. Hg. He was still cyanosed but again improved on oxygen through a Polymask. On examination of the chest there were widespread coarse crepitations over both lung fields. Urine analysis showed acetonuria and haemoglobinuria. Haematological, biochemical, and electrocardiogram findings on admission are shown in Table I. The chest *x*-ray appearances are shown in Fig. 1. At 12.30 a.m. (five hours after the incident) he was pale and sweating; his blood pressure had dropped to under 100 mm. Hg systolic, with a rapid thready pulse. He was still coughing up frothy pink sputum, which was also exuding from both nostrils, and his respiratory rate had risen to 40 per minute. He became cyanosed when the oxygen therapy was stopped. He vomited on several occasions. There were still widespread crepitations over both lung fields, but bronchial breathing was now heard at the right base.

Treatment.—Treatment with intravenous plasma was started and its effect on blood pressure and pulse rate is shown in Fig. 2. At 2 a.m. he was transferred to the resuscitation unit because of continued deterioration and in case intermittent positive-pressure ventilation should be required. Ampicillin 500 mg. six-hourly was started. At 3 a.m. repeat urine analysis showed continued haemoglobinuria, diminishing acetonuria, and albuminuria. At 9.30 a.m., though he was much improved, continuous oxygen was still necessary to prevent central cyanosis. His respiratory rate had dropped to 34 per minute and he was still somewhat sweaty and looked pale. At 1 p.m. he was much better but became cyanosed when taken off oxygen for more than four minutes. A chest *x*-ray

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TABLE I.—*Urine Analysis and Biochemical, Haematological, and E.C.G. Findings in Four Cases of Near Drowning on Admission to Hospital*

Case No.	Water	E.C.G.	Hb (g./100 ml.)	P.C.V.	Plasma Hb (mg./100 ml.)	W.B.C. per cu. mm.	Urea (mg./100 ml.)	Na+ (mEq./l.)	K+ (mEq./l.)	Cl (mEq./l.)	HCO_3 (mEq./l.)	Total Protein (g./100 ml.)	S.G.	Albuminuria	Hb in Urine	Acetonuria	Deposit
1	Fresh	S.R.	17·4	—	Present	16,000	37	132	3·8	99	22	6·5	1022	++	Yes	Yes	W.B.C. R.B.C. Casts
2	Fresh	S.R.	16·8	—	9·6	17,800	34	136	4·4	101	26	6·6	1023	++	Nil	Nil	Nil
3	Sea	A.F.	13·1	42	0	12,800	25	163	3·4	106	17	7·2	—	Trace	Nil	—	—
4	Sea	S.R.	13·2	37	0	—	24	150	3·7	111	26	7·2	—	—	Nil	—	—

A.F. = Atrial fibrillation. S.R. = Sinus rhythm.

picture at this time is shown in Fig 3. Investigations were as follows; serum haemoglobin 9·6 mg./100 ml. (upper limit of normal 5·0 mg./100 ml.,) Astrup figures: *p*H 7·35, P_{CO_2} 57·5 (off oxygen for five minutes), bicarbonate 25 mEq./l., base excess +1. The serum electrolyte findings were not appreciably different from those on admission. By the following day (10 September) he was obviously very much better and off oxygen therapy. The pulse was still a little rapid at 120 per minute and the blood pressure was 130/70. Crepitations were present only at the right base and he had stopped expectorating frothy pink sputum. He was discharged from hospital eight days after admission.

Case 2

The patient, aged 19, was admitted with a temperature of 100°F. (37·8°C.), pulse 120 per minute, regular, was conscious, and had a blood pressure of 140/80. He was cyanosed peripherally on oxygen, expectorating frothy pink sputum, and he felt nauseated. On examination of the chest there were widespread crepitations over both lung fields. A chest *x*-ray picture showed multiple soft opacities over both lung fields, more on the right than on the left. Haematological, biochemical, and electrocardiogram findings are shown in Table I. Urine analysis showed a specific gravity of 1023 and albuminuria only. By 12.30 a.m. on 9 September (five hours after the drowning incident) he was very much improved. His respiratory rate was 30 per minute and his pulse had settled at 72 per minute and was regular. He no longer became cyanosed on stopping oxygen and had stopped expectorating frothy sputum. There was complete resolution of the radiological findings after three days.

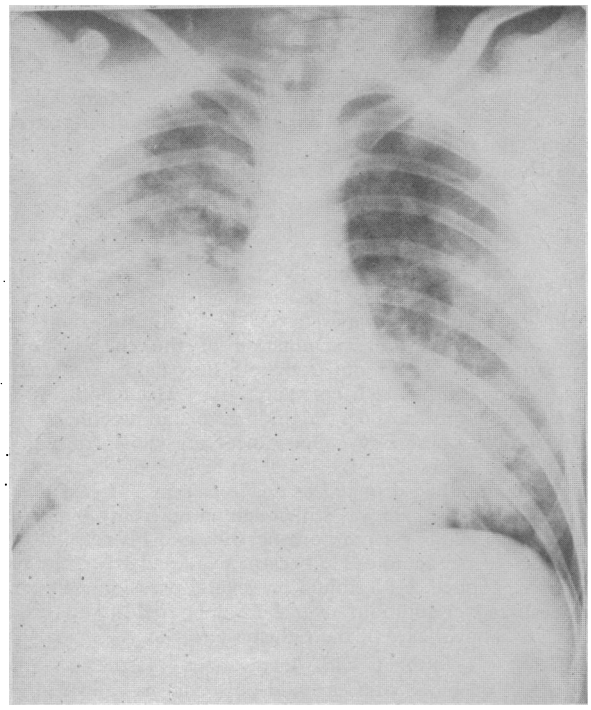

FIG. 1.—Case 1. Chest *x*-ray appearances on admission.

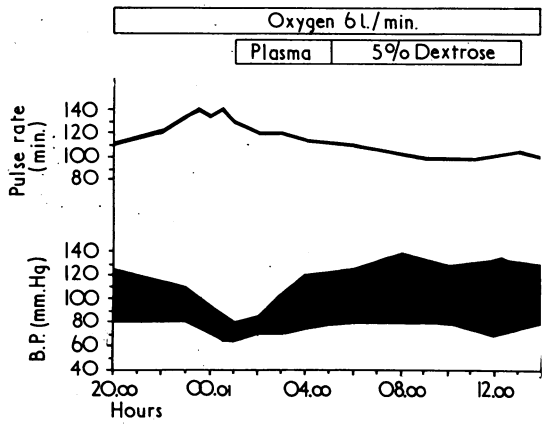

FIG. 2.—Case 1. Effect of intravenous plasma on pulse rate and blood pressure.

Case 3

The patient, a woman aged 60, was a non-swimmer. At 1.15 p.m. on 21 August 1967 a dinghy in which she was sailing capsized. Initially she was supported in the sea-water for an unspecified time but eventually broke loose and when rescued was found unconscious and face down in the water. After mouth-to-mouth resuscitation she quickly recovered consciousness and developed a good colour. Physical examination carried out half an hour later at another hospital disclosed nothing abnormal. At 3.05 p.m., when she was admitted, she was found to be conscious but cyanosed and in atrial fibrillation, with a blood pressure of 90/70. The cyanosis disappeared and her blood pressure rose to 130/80 when she was given oxygen via a Polymask. By 3.45 p.m. she had deteriorated

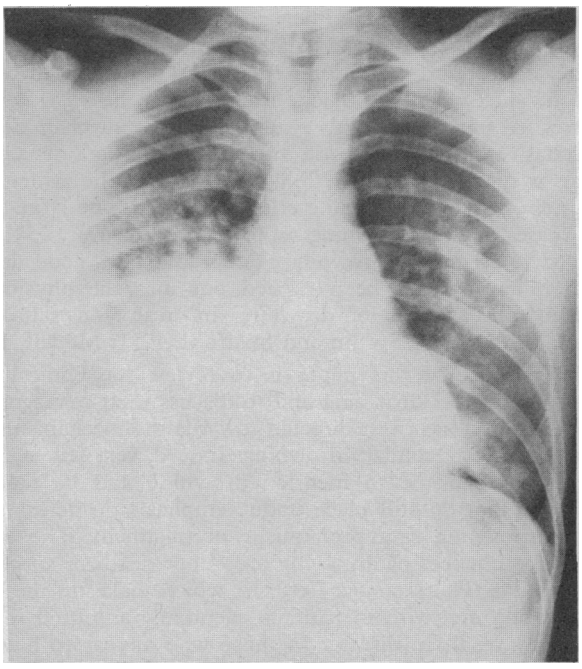

FIG. 3.—Case 1. Chest *x*-ray appearances 17½ hours after admission.

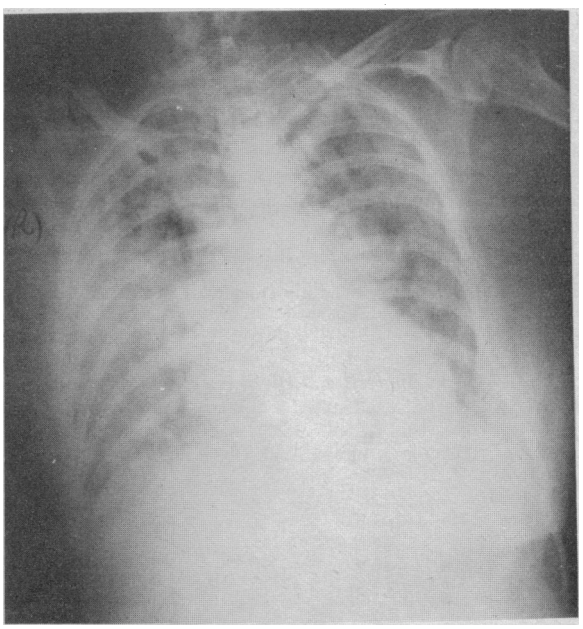

FIG. 4.—Case 3. Chest *x*-ray appearances 2½ hours after near-drowning incident.

and become drowsy and disorientated. Atrial fibrillation persisted, there were crepitations at both lung bases, and she was hyperpnoeic. The *x*-ray appearances at this time are shown in Fig. 4. The biochemical, haematological, and electrocardiogram findings on admission are shown in Table I. Her temperature was found to be 96°F. (35·6°C.).

By 4.30 p.m. the cyanosis had deepened in spite of oxygen and it became necessary to ventilate the patient manually. She was given intravenous injections of hydrocortisone 100 mg. and aminophylline 250 mg. and an intravenous drip giving 5% dextrose was set up. Treatment with ampicillin was begun. She remained adequately oxygenated and semi-conscious until 6.30 p.m., when she suddenly lost consciousness and became deeply cyanosed and required intubation and intermittent positive-pressure respiration (I.P.P.R.). The patient soon recovered consciousness and subsequently began to resist the I.P.P.R. Accordingly, phenoperidine 2 mg. was given slowly over two minutes. At 6.40 p.m. she collapsed and became pulseless. Immediately before this it had been noted that the I.P.P.R. required 36 cm. of water pressure. She required external cardiac massage and intravenous methylamphetamine hydrochloride 2·5 mg. was given. Soon after, an electrocardiogram showed her to be in sinus rhythm at 120 per minute. The blood pressure fell slowly over the next four hours and intravenous Rheomacrodex and dextran were given. Her response to treatment is shown in Fig. 5. She was eventually taken off I.P.P.R. at 8.15

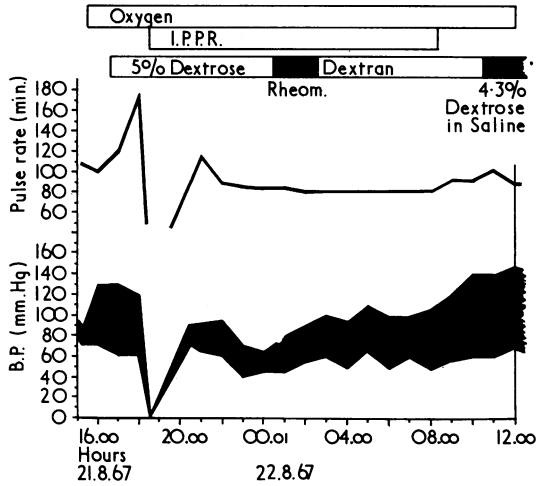

FIG. 5.—Case 3. Negative effect of conventional therapy for salt-water drowning followed by a favourable response to plasma volume expanders.

the following morning and spontaneous respiration recurred with satisfactory clinical oxygenation. The tracheal tube was removed at 9.20 a.m. Bilateral basal crepitations were noted and the *x*-ray appearances at this time are shown in Fig. 6. Oxygen therapy was eventually discontinued after three days. On the evening of the fourth day her temperature rose to 103°F. (39·4°C.) and a sputum culture grew *Klebsiella aerogenes*, and accordingly her antibiotic therapy was changed to cephaloridine and polymyxin E, to which she responded well. She was discharged on the 16th day.

Case 4

The patient, a boy aged 5, was able to swim. At 4.40 p.m. on 23 August 1967 while he and his brother were swimming in the sea he was missed and noticed face downwards in the water. He was brought to land and mouth-to-mouth resuscitation was started immediately. This was followed by Ambu-bag respiration on 100% oxygen. He was admitted to hospital at 5 p.m., when he was noted to be stuporose, cyanosed, and shivering. The rectal temperature was 96·4°F. (35·8°C.). His pulse was 100 per minute, regular, and his blood pressure 130/70. He had a grunting respiration and crepitations at the left base. A chest *x*-ray picture taken at this time showed multiple soft opacities over both lung

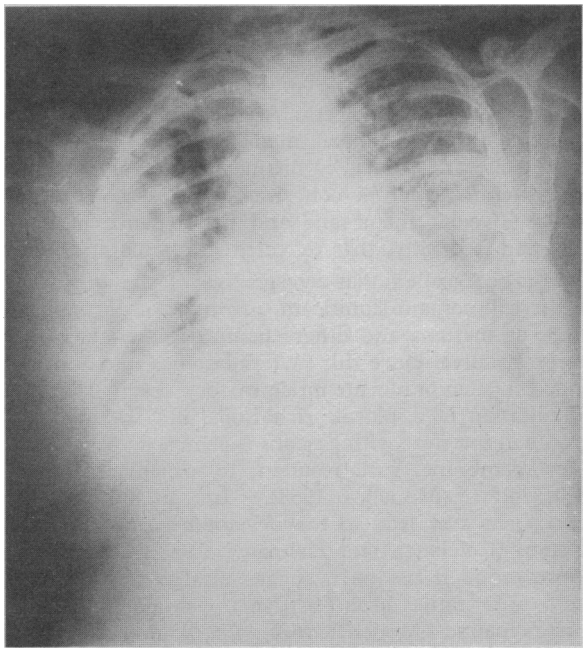

FIG. 6.—Case 3. Chest *x*-ray appearances 20 hours after admission.

fields. Treatment with 100% oxygen via a Polymask was started. By 6 p.m. he was fully conscious and at 10.20 p.m. his only complaint was of some coldness and shivering. A chest *x*-ray picture taken the morning after admission was completely normal and he was discharged home 48 hours later.

Discussion

Though much emphasis has been placed on the differentiation between salt-water and freshwater drowning, with special reference to serum biochemical and haematological values in animals (Swann *et al.*, 1947; Swann and Spafford, 1951; Modell, *et al.*, 1966), it appears that these bear little relation to the type of drowning incident in humans (Fainer, 1963; Fuller, 1963; Modell, *et al.*, 1968; *Lancet*, 1962) (Table II). The results of such determinations are therefore unlikely to help in the diagnosis of the type of near drowning and may even be misleading in the therapeutic management. Probably the most valuable investigations in these situations are those concerned with acid-base balance. Accordingly, treatment should not be delayed while awaiting the results of investigations.

TABLE II.—*Collected Data on Laboratory Findings in Cases of Secondary Drowning (Fainer, 1963; Fuller, 1963; Modell, et al., 1968; Present Series)*

	Sea-water Drowning			Freshwater Drowning		
	No.	Mean	Range	No.	Mean	Range
Sodium (mEq/l.)	15	149	132—163	15	136	127—146
Potassium (mEq/l.)	15	4·1	3·4—4·9	14	4·0	3·0—6·2
Bicarbonate (mEq/l.)	8	19	11·5—26	9	22	17—26
Chloride (mEq/l.)	17	112	96—120	18	101	94—116
Total Protein (g./100 ml.)	4	7·2	6·7—7·4	3	6·6	6·4—6·8
Urea (mg./100 ml.)	2	25	—	2	33	—
Haemoglobin (g./100 ml.)	32	14·1	12—19·8	23	14·8	11·4—20
P.C.V.	33	43	36—56	19	47	36—65
Plasma Haemoglobin (mg./100 ml.)	6	1	0—4	7	141	8—500

Haemoconcentration effects seen in both freshwater and sea-water near drowning related to shock and loss of plasma into pulmonary alveoli.

It appears that after the immediate resuscitative period the subsequent clinical course is very nearly identical in both types of near drowning. It must be emphasized that no matter how good the apparent results of immediate resuscitation are, the patient may still die from the effects of secondary drowning (Miles, 1968). These patients should therefore be admitted to hospital for observations for at least 24 hours. The most valuable indices of progress are the clinical observations rather than the laboratory investigations (*Lancet*, 1962). The time of onset of secondary drowning is variable. Secondary drowning following sea-water near-drowning is twice as lethal as freshwater near-drowning and the time of onset of the secondary drowning can be within three minutes (Modell, 1968). The clinical management of these patients resolves itself into two fairly distinct areas. Firstly, that of a purely respiratory nature concerned with gaseous exchanges across the alveolar membrane and maintenance of acid-base balance, and, secondly, that reflected in circulatory failure.

It must be recalled that irrespective of the type of water inhaled it will contain significant quantities of particulate matter, such as diatoms and other chemical material, some inert and some reactive. Once this material comes into contact with the alveolar membrane an inflammatory reaction will occur, with exudation of a plasma-rich fluid into the alveoli. This situation will interfere with gaseous exchange across the alveolar capillary membrane, with resultant hypoxaemia, hypercapnia, and acidosis (Modell, 1968). In addition, irritative bronchospasm may lead to the development of acute airways obstruction (Colebatch and Halmagyi, 1963). On the basis of this observation it has been suggested that those cases complicated by irritative bronchospasm may benefit from nebulized isoprenaline (Modell, 1968). We would agree with the recommendation that all near-drowned patients be given oxygen therapy en route to hospital (Modell, 1968) but that further administration should be determined by their clinical progress. Oxygen must always be given at the least sign of cyanosis. Oxygen via a Polymask is often sufficient, but should this prove inadequate, then, intermittent positive-pressure ventilation is indicated.

There is evidence that a degree of metabolic acidosis may accompany the changes seen in secondary drowning (Modell *et al.* 1968; Modell, 1968) (Table II) and thus justifies the routine use of sodium bicarbonate (*Lancet*, 1962; Modell, 1968). Continued use of bicarbonate therapy must, however, be titrated against acid-base measurements.

Whenever secondary drowning occurs it seems reasonable to assume that an infective or chemical pneumonitis is part of the picture. The effects of secondary drowning have been likened to those occurring after the aspiration of vomitus during anaesthesia, the so-called Mendelson's syndrome (Bannister and Sattilaro, 1962). This has led to the suggestion that steroid therapy might be of benefit to this type of patient. There is little evidence to support this contention and indeed there is even dispute about the definite value of steroids in the treatment of Mendelson's syndrome itself. There is also an important distinguishing feature between the post-anaesthetic problem and that of secondary drowning, in the former the aspirated contents are highly acid, whereas in the latter this is not necessarily so. From a purely pathological standpoint, however, it is difficult to envisage that the reactive pneumonitis that follows either situation will be very different, since in both situations an inflammatory response is evoked by the presence of foreign material. In many patients adequate oxygenation, bicarbonate therapy, and timely antibiotics will result in a satisfactory outcome.

In other patients more clinically ill, which is not necessarily related to the type or severity of the near drowning incident, the features of cardiovascular failure will appear, as evidenced by a fall in blood pressure, a rise in pulse rate, sweating, and pallor. Hitherto, many authorities have suggested that the treatment of hypovolaemic shock should be geared according to the the type of near-drowning incident (*Lancet*, 1962). This has led to the concept that if fresh water has been inhaled a hypertonic intravenous solution should be given and, conversely, that if sea-water has been inhaled a hypotonic solution should be given. This approach, however, seems unjustified in the light of the present clinical evidence and fails to take into account the basic physiopathological lesion of secondary drowning—that is, the exudation of plasma-rich fluids (inflammatory process) into the alveoli (Fig. 7). Though both mannitol and Rheomacrodex have been recommended in

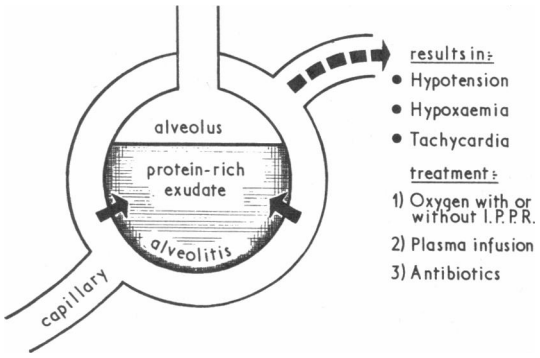

FIG. 7.—Diagrammatic representation of the physiopathology of near drowning.

this situation, their value can be only transient, since they have a short life-span in the actual circulating blood volume. The use of diuretics has been suggested in the treatment of secondary drowning (Miles, 1968), but the fact is ignored that the fluid in the pulmonary alveoli in this situation is not simply water but a plasma-rich exudate. Thus to give diuretics in this situation would be to further reduce the already depleted circulating blood volume.

There can be little doubt that the most important intravenous requirement in these cases is plasma. In two cases described above, both of whom suffered from cardiovascular collapse, it can be clearly seen that their clinical improvement coincided with giving intravenous plasma or other similar plasma expanders. It would seem reasonable from the available clinical evidence to give any patient developing signs of circulatory failure in secondary drowning one litre of plasma.

Experimental animal work has given rise to the belief that haemoglobinaemia and haemoglobinuria with possible acute renal failure is a grave risk in patients who have had a freshwater near-drowning incident. In a review of 77 cases of near drowning (31 cases in freshwater, 46 cases in salt-water) (Fuller, 1963) there were only two examples of haemolysis with ensuing haemoglobinaemia and haemoglobinuria, one in freshwater and one in salt-water. One of our two cases of freshwater near-drowning showed similar changes though not very pronounced. Nevertheless, where haemoglobinaemia and haemoglobinuria are found in patients after near-drowning it is reasonable to encourage a high fluid intake to maintain an adequate urine output and thus prevent possible acute renal failure.

The renal sequelae of near-drowning are usually mild, transient, and insignificant and only very rarely of serious import. Albuminuria was recorded in 12 of the 77 cases described by Fuller (1963). One case had a transient rise in blood urea together with the presence of albuminuria and another case died of anuria when the blood urea nitrogen reached 192 mg./100ml. This latter case, however, was complicated by severe pneumonia and it seems unlikely that this was purely a renal death of secondary drowning. Two of Fuller's cases and one of our own developed acetonuria, but the mechanism of this is not understood.

The clinical features of secondary drowning are very varied, but the most comprehensive review has been given by Fuller

(1963). From his data and those of Modell (1968) and Fainer (1963) and ourselves the following observations would seem to be correct. About half the patients involved in such incidents will develop a pyrexia, at times 105-106°F. (40·6-41·1°C.), and will usually have a leucocytosis anywhere up to 40,000/cu.mm. Many patients will have transient neurological symptoms or signs, such as trismus, motor hyperactivity, convulsions, fear, and headaches. The single most important sign, and of very serious prognosis, is the return of coma. In 14 out of 77 cases (Fuller, 1963) in whom coma returned there was a uniformly fatal outcome.

All near-drowned patients who get the complications of secondary drowning will by implication have respiratory signs and symptoms. These vary considerably in type and severity. They range from a rapid shallow breathing to the inability to take a deep breath, to laryngospasm, burning sensation underneath the sternum, pleuritic pain, hoarse rasping cough, expectoration of frothy pink sputum, and signs such as rales, rhonchi, and dullness over both lung fields. Not all cases necessarily show radiological changes. Sixteen of 57 cases who had clinical evidence of pulmonary involvement failed to show any radiological change (Fuller, 1963). In the remaining 41 cases the radiological changes were those of hazy or multiple opacification over both lung fields. The duration of the radiological findings would, of course, depend on the presence or absence of secondary infection.

In animal experimental work using mainly dogs (Swann *et al.*, 1947; Swann and Spafford, 1951; Modell *et al.*, 1966) the most feared complication of freshwater near drowning is ventricular fibrillation. This is thought to result from the haemolysis of red blood cells with the liberation of potassium, which, in the presence of hypoxaemia, leads to ventricular fibrillation. In the dog erythrocyte, however, the main intracellular cation is not potassium but sodium, and hence the release of intracellular potassium cannot be the full explanation. Ventricular fibrillation or any cardiac arrhythmia is rare in near drowning.

The commonest arrhythmias are tachycardia, gallop rhythm, and occasional extrasystoles. In only one of Fuller's series and in one of our own cases was atrial fibrillation seen. Such abnormalities of cardiac rhythm as do occur are usually relieved by correction of hypoxaemia.

As can be seen from the data analysed in Table II there are not any significant differences in the clinical pathology that can be attributed to differences in the salinity of the drowning fluid. Little attention has been directed to serum magnesium levels after sea-water drowning, but these could be anticipated to rise rapidly in view of the known high magnesium content of sea-water.

It would appear from both our own experience and that of others that the clinical and laboratory findings are best ascribed to the effects of secondary drowning, such as hypoxaemia, aspiration pneumonitis, and pulmonary oedema, rather than to the type of water aspirated.

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TODAY'S DRUGS

With the help of expert contributors we print in this section notes on drugs in current use.

Pancreatic Extracts

The pancreas is both an endocrine and an exocrine gland. The endocrine secretions (insulin and glucagon) have been extensively investigated and their therapeutic uses are well established; but the exocrine secretion, which contains a variety of digestive enzymes together with bicarbonate, has not been so thoroughly studied. At first sight oral administration of extracts of whole-animal pancreas would appear to be a logical and simple remedy for patients with pancreatic insufficiency, even though such therapy does not include bicarbonate and does not resemble the co-ordinated secretory response of the normal pancreas.

Preparations available

Pancreatin *B.P.* is an alcoholic extract of pig pancreas in the form of a powder with a meaty odour, which is standardized by its ability to digest starch and casein in vitro. The powder also has lipolytic activity, but this has not been standardized in Britain since agreement has still to be reached on a satisfactory method (though it has[1] in the U.S.A.). Pancreatin may be dispensed as powder, capsules, granules, and tablets, and the last two preparations are also available in enteric-coated forms. Capsules may be broken and sprinkled on food. Strong pancreatin *B.P.* (Pancrex V) contains five times the concentration of pancreatin and is useful in reducing the mass of preparation to be ingested in order to obtain a therapeutic effect. This is specially valuable in infants. Various proprietary preparations are also available in which the strength of pancreatin has been increased by factors of 1½ (Panar), 2 (Enzypan), 3 (Panteric; Pancreatin, C+A.; Trypsinogen; Combizym), and 15 (Pancrex V Forte).

Cotazym, another proprietary preparation, is the only lipase-enriched pancreatic extract that is available in Britain. An attempt has been made to standardize its lipase activity, but only scanty details of the method used have been published, and the potency of each 170 mg. capsule is expressed as the ability to digest 17 g. of fat under unstated physical conditions.