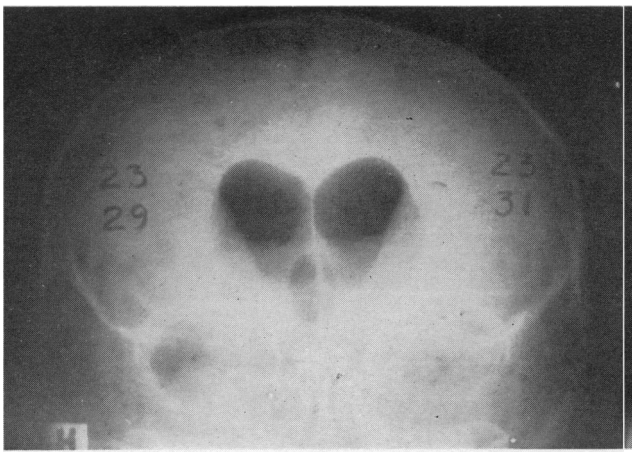


FIG. 1.—Air-encephalogram, frontal view (six weeks after drowning), showing general dilatation of both lateral ventricles and considerable widening of the third ventricle. (Dr. H. Eie.)

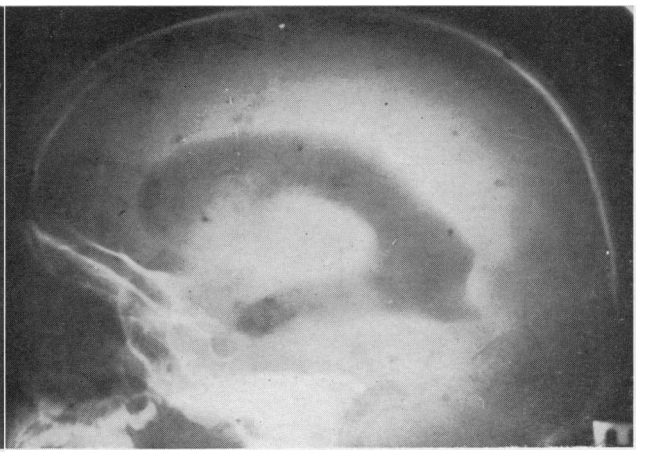


FIG. 2.—Air-encephalogram, side view (six weeks after drowning), showing general enlargements of all portions of the lateral ventricle. (Dr. H. Eie.)

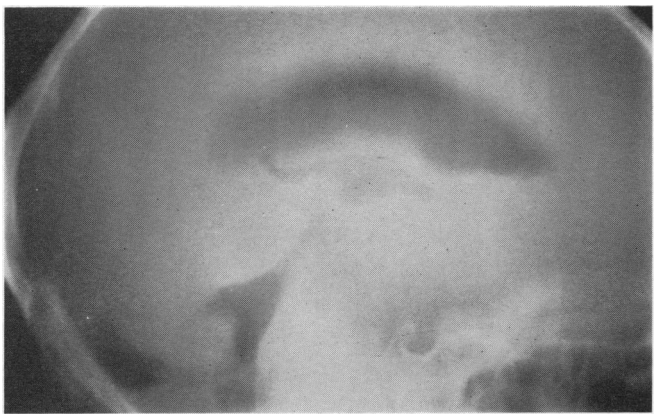


FIG. 3.—Tomogram of infratentorial structures (six weeks after drowning) showing enlargement of cisterna magna and the fourth ventricle. (Dr. H. Eie.)

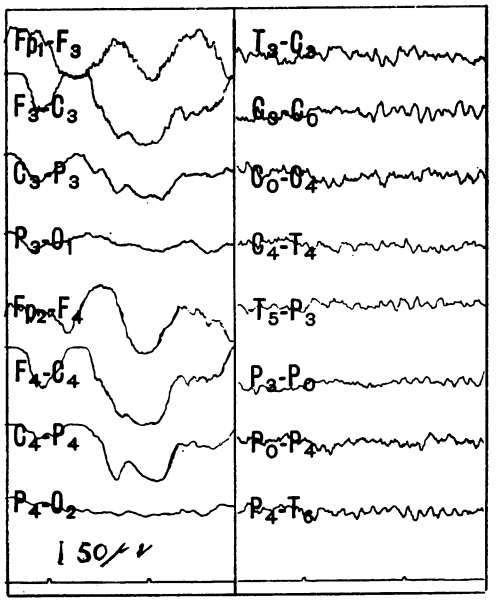
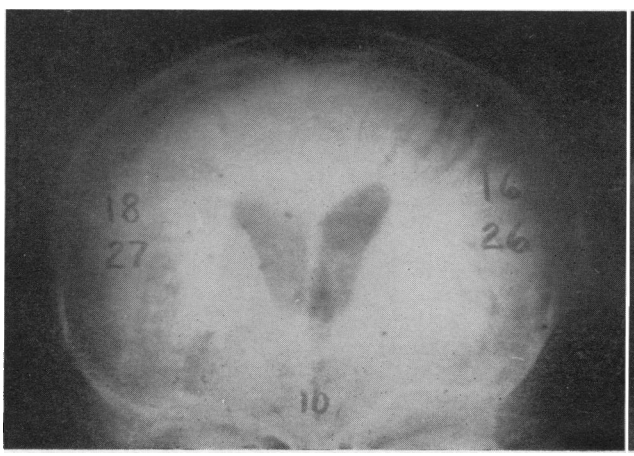


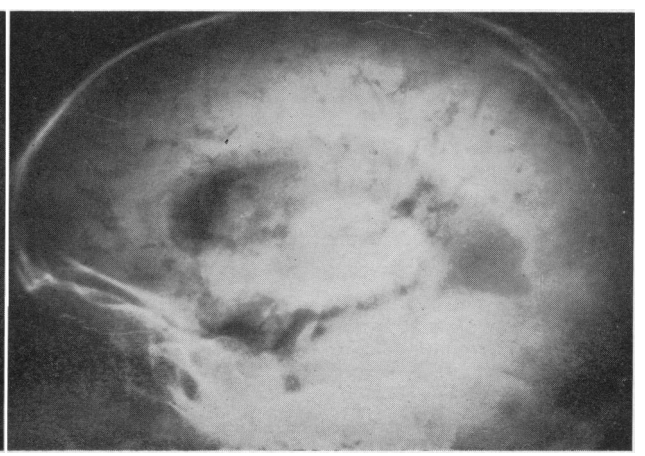
FIG. 4 FIG. 5

FIG. 4.—Electroencephalogram (seven days after drowning) showing severe dysrhythmia with uneven slow waves, most pronounced over the hemispheres. FIG. 5.—Electroencephalogram (six months later) showing normal electrical activity. (Dr. S. Østensjø.)

FIG. 6.—Air-encephalogram, frontal view (six months after drowning), showing reduction of the general enlargement of both ventricles and especially of the distance between the septum and the nucleus caudatus and the width of the third ventricle. (Dr. H. Eie.)

FIG. 7.—Air-encephalogram, side view (six months after drowning), showing reduction of the width of the temporal horn.





RECOVERY FROM DROWNING IN FRESH WATER

BY

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[WITH SPECIAL PLATE]

It is apparently extremely rare for a person to survive after being submerged for so long that the heart has stopped beating.

After inhalation of fresh water it appears that the heart usually goes into ventricular fibrillation both in the dog and man (Donald, 1955; Haukebø, 1960; *Lancet*, 1962; Redding, Voigt, and Safar, 1960a and b; Redding, Cozine, Voigt, and Safar, 1961; Swann and Spafford, 1951). In a leading article in the *Lancet* (1962) it is stated: "The heart is then submitted to hypoxia, over-filling, potassium excess and sodium deficit. It would be hard to think of a more certain way of producing ventricular fibrillation, and it is this that causes death."

The case to be presented shows the following unusual features: (1) The time the patient was under water was estimated to be 22 minutes. (2) The heart did not fibrillate in spite of the fact that fresh water was inhaled with subsequent haemolysis of the blood. (3) The heart resumed normal contractions after approximately two hours' external cardiac compression. (4) The electrolyte changes and haemolysis were successfully treated with exchange-transfusion. (5) The patient recovered with small neurological and mental sequelae in spite of atrophy of the brain as shown by dilatation of the cerebral ventricles demonstrated by air-encephalography.

Case History

The patient was a 5-year-old boy who was admitted to the Central Hospital, Trondheim, on March 6, 1962, after drowning in the river. It was a cold day with a temperature of about −10° C. and deep snow. The river was partially frozen.

The call for help came to the police-station at 14.55. One of us (T. D. K.) arrived at the river bank while the rescuer was still partially in the water, doing mouth-to-mouth respiration. The boy was then apparently dead, the pupils were widely dilated, and the skin was blue-white. On attempting to intubate the trachea it was seen that the entire mouth and pharynx were filled with vomitus. The patient had to be turned head down and the stomach contents in the mouth were partially removed with the fingers. Even so, it was only possible to see the tip of the epiglottis. Intubation succeeded and by deliberately pushing the contents in the tube into the tracheal tree it was possible to move the chest. As no pulse could be felt external heart compression was started immediately. Artificial ventilation and heart compression were continued on the journey to the hospital.

The patient arrived at the hospital at 15.40. His pupils were still widely dilated and the lips were deeply cyanotic, but the ear lobes had regained a pink colour and a certain degree of peripheral circulation had returned.

No pulse could be felt in the central arteries when external compression was discontinued. A needle which was put through the chest wall into the heart failed to demonstrate any beating of the heart. Three ml. of a mixture of 0.5 ml. 1‰ adrenaline and 9.5 ml. 1% procaine was injected, but apparently with no result. An electrocardiogram taken half an hour later showed complete block with approximately 30 contractions a minute, but still no pulse could be felt. As soon as the cardiac compression was stopped the patient's colour deteriorated and the peripheral circulation disappeared. A sample of blood had been taken immediately on arrival to test for haemolysis and to determine the patient's blood group, and a blood transfusion was started. At 17.40 (two and a half hours after submersion) the heart started to contract spontaneously; the pulse could be felt in the radial arteries, the colour improved, the pupils contracted, and the blood-pressure, which had not been measured up to this time, was found to be 90 systolic. The lips were still deeply cyanotic and the hands and feet were blue. In an attempt to improve the peripheral circulation he was given chlorpromazine 2 mg. at a time, to a total of 8 mg. in all. This improved the colour and the pulse, which could be counted to 100 beats a minute, but the blood-pressure became unmeasurable.

A short time after the heart had regained an effective output, inasmuch as a spontaneous pulse could be felt, the patient began to take deep, gasping breaths. These gasps were increasingly interspersed with normal respiratory movements, until normal respiration was resumed.

At 20.30 the body-temperature was 24° C. The report from the laboratory department showed haemolysis (177 mg. % haemoglobin) in the serum of the blood sample withdrawn on arrival. Through a catheter in the bladder 150 ml. of burgundy-red urine was obtained which reacted positively to benzidine. No red cells were found on microscopic examination, but many casts. The catheter was left in place.

One hour after the boy had started to breathe normally the rales in the lungs increased and he had a sudden attack of lung oedema with bloody froth expelled forcefully through the endotracheal tube. With the aid of lanatoside C (0.4 mg.) and theophyllamine (0.12 g.) and by increasing the pressure of the assisted ventilation the attack was controlled. Later on 2 mg. of morphine was given and the respiration was assisted.

As it was believed that he would have no chance of surviving the haemolysis, the haemoglobinuria, and the electrolyte changes following inhalation of fresh water, it was decided to carry out an exchange-transfusion. The temperature was slowly increased to 30° C. A cut down in both femoral veins was performed and 20 ml. of blood was withdrawn from one side as 20 ml. of fresh blood was given on the other. The exchange-transfusion of 3,000 ml. of blood lasted for three hours in the course of which 500 mg. calcium gluconate was given twice in the exchanged blood. (Fig. 1 shows a radiograph of the chest taken during this period.) During this time he became rigid and had convulsions which were stopped with 0.5 ml. of "hypnophen" (a mixture of 10% allypropymalum, 11% barbitone, 25% urethane) in the exchange blood.

A fresh sample of blood taken after 2,500 ml. had been exchanged showed serum of normal colour, and the laboratory test showed 40 mg.% haemoglobin in serum. When the exchange-transfusion had been performed a tracheostomy was done. A considerable amount of stomach contents was removed from the lungs by means of suction through the tracheostoma, and the colour of the lips improved. Hydrocortisone and antibiotics were given.

In the following eight hours he had three attacks of pulmonary oedema, but these were controlled by "cedilanid" (lanatoside C) plus theophyllamine and increased ventilatory pressure.

The following morning the blood-pressure was stabilized at 60 systolic and the pulse at 120 beats a minute. There was no excretion of urine. An unsuccessful attempt was made to raise the boy's blood-pressure by means of intra-

venous injections of " aramine " bitartrate (metaraminol acid tartrate) 1 mg.+2 mg.+5 mg. A low-molecular plasma expander (" rheomacrodex ") was then given in doses of 50 ml. per hour. The blood-pressure remained low, but he began excreting urine of normal colour. The results of laboratory tests on March 7, the day after admission, were: haemoglobin, 12.3 g./100 ml.; haematocrit, 37%; total CO_2 34 mM/1,000 ml.; chloride, 102 mEq/1,000 ml.; non-protein nitrogen, 30 mg./100 ml. The serum potassium level could not be determined because of a temporary technical fault.

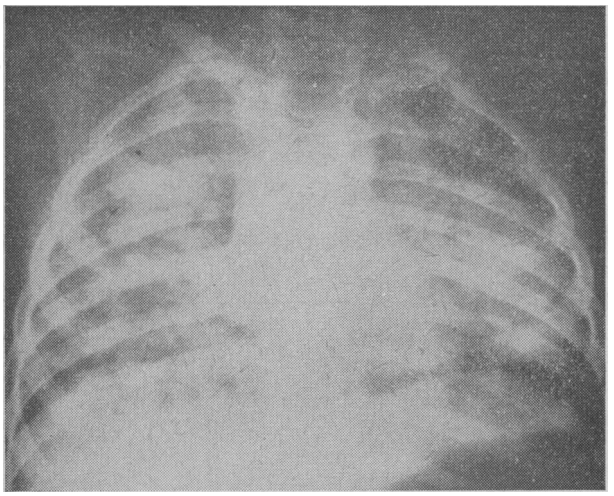

FIG. 1.—Radiograph of chest during exchange-transfusion, showing severe infiltration, aspiration pneumonia, and pulmonary oedema.

During the succeeding 24 hours he stopped breathing five times and the pulse became difficult to palpate, but he improved again as soon as artificial ventilation was started. He had several attacks of convulsions, which were stopped by means of hypnophen. The administration of hydrocortisone, antibiotics, and chlorpromazine was continued, and the temperature was kept between 30 and 33° C.

He was also given heparin, and gradually his condition improved. The output of urine was satisfactory.

On March 8 the laboratory findings were: haemoglobin 9.2 g./100 ml.; haematocrit 25%; total CO_2 23 mM/1,000 ml.; *p*H 7.26; non-protein nitrogen 150 mg./100 ml.

In the days that followed the laboratory findings continued to be satisfactory, except that haemoglobin fell from 12.3 g./100 ml. on March 7 to 9.2 g./100 ml., and he was given 500 ml. of blood. Non-protein nitrogen increased to 190 mg./100 ml., but in a few days fell to a normal value. The output of urine increased when the amount of intravenous fluid was increased, and the ability to concentrate and dilute the urine was soon resumed. A radiograph of the chest showed almost normal lungs (Fig. 2). The temperature was kept below normal values for four to five days, after which it was allowed to reach 37° C. When the boy was examined neurologically two days after the accident pupillary and corneal reflexes were absent and there was no reaction to painful stimuli. On the fifth day pupillary and corneal reflexes had returned and he reacted to painful stimuli. The results of an E.E.G. carried out on the seventh day after the accident are shown in Special Plate, Fig. 4.

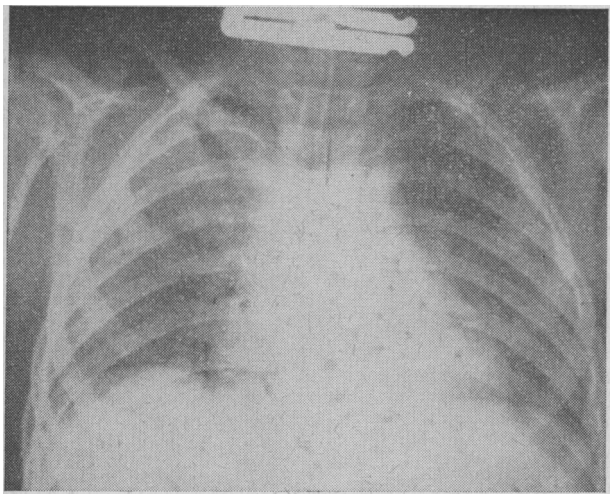

FIG. 2.—Radiograph of chest three days after the accident showing almost normal lungs.

At the end of a week he began to swallow, and intravenous fluids were stopped. He coughed satisfactorily, and the tracheostomy tube was removed on the ninth day.

Gradually his mental condition improved. On the tenth day after the accident he could obey simple commands, could see and recognize his mother, and could answer " yes " and " no."

However, eleven days after the accident his mental condition deteriorated. He became unconscious and very restless, and began to utter sudden, meaningless shrieks; the uncoordinated movements of arms and legs increased to such an extent that he had to be sedated with chlorpromazine. This agitated period lasted for about 14 days, gradually decreasing.

There now followed a period when he appeared to be completely decerebrated. He would open his mouth when the lips were touched and his appetite became enormous. His physical condition steadily improved and gradually he began to sit up. He had difficulty in keeping his balance, and appeared to be blind—for instance, he fell on his face against the bedpost when his eyes were open.

Six weeks after the accident his mental condition improved. (Special Plate Figs. 1, 2, 3 show air-encephalograms and a tomogram taken at this time.) From that time onwards he improved every day, and he began to speak. Seven weeks after the accident his vision returned; at first he could only see objects near to him, but gradually he could also see at a distance.

As far as clinical observation went he gradually became a normal child. When he was discharged two and a half months after the accident he was still unsteady on his feet, and the finer movements of the fingers were a little clumsy.

When he was examined six months after the accident psychological tests showed him to be mentally almost of normal age. The finer movements of his fingers were clumsy and the peripheral vision was reduced or absent. Neurological examination and an electroencephalogram gave normal findings (Special Plate, Figs. 5, 6, 7). By ordinary clinical standards he behaved as a normal child.

Discussion

The police estimated that 22 minutes must have elapsed from the call to the police-station reporting the accident until the patient was out of the water. It appears that he was seen hanging on to the edge of the ice by some boys on the other side of the river, which is approximately 40 yards wide and has a strong current. How long he had been able to hang on to the ice is not known. Presumably he had had time to be cooled and exhausted before he let go. The day was cold and the river partially frozen. The policeman who rescued him started mouth-to-mouth breathing as soon as the patient was ashore, even before the rescuer himself was out of the water. It was carried on for another 10 minutes before one of us reached the shore. At that time it was obvious that mouth-to-mouth breathing could not be successfully performed because of the stomach contents

in the mouth and pharynx. The time during which the patient had been submerged is therefore uncertain. The boy had drifted across to the other side of the river where he was found, and the rescuer had to swim twice across the river against the strong current and this must have taken a considerable time. That our patient suffered damage to the brain is shown by the air-encephalograms and the E.E.G. In spite of this he has recovered to become an apparently normal child. It seems fair to assume that exhaustion prevented him fighting for breath after submersion, and the cooling of the body before he drowned probably delayed the damage to the brain that is caused by anoxia.

Jude (1962) states that electrocardiographic activity can be present without cardiac output. The first E.C.G. taken approximately half an hour after admission demonstrated ventricular contractions, but there was no satisfactory output, no palpable pulses in the greater arteries, and the peripheral circulation disappeared when the cardiac compression was stopped for a moment. We were unable to resuscitate him effectively enough to contract the pupils, and the lips continued to be blue. We believed this failure was due to the aspiration of vomitus, which would reduce the uptake of oxygen in the lungs. When the heart resumed efficient contractions, however, the colour improved, the pupils became small, and he began to breathe spontaneously.

Jude, Kouwenhoven, and Knickerbocker (1961) in their report on 112 cases of cardiac arrest treated by external cardiac massage state: "Successful return to the prearrest central nervous system and cardiac status occurred up to 90 minutes." Our patient required 120 minutes of cardiac compression before the heart resumed efficient contractions. It is possible that he, being a child with normal myocardium and being cooled to subnormal temperature, was able to tolerate hypoxia better than an adult with normal temperature.

Our patient developed pulmonary oedema as expected. The pulmonary oedema was successfully treated with lanatoside C and theophyllamine, morphine, and increased positive-pressure ventilation. Chlorpromazine may have played an important part in the treatment as it produces peripheral vasodilatation, thereby helping to combat the overfilling of the lungs. In fact, it was our impression that the pulmonary oedema, as long as it did not kill the patient, helped to clear the lungs, because pieces of stomach contents were expelled with the froth.

The pneumonia, due to aspiration of vomitus and inhalation of the dirty river water, was treated with hydrocortisone and antibiotics, beside frequent suction in the trachea. Bannister, Satillaro, and Otis (1961) have shown that this is the most effective way of treating aspiration pneumonitis.

He did not develop ventricular fibrillation.

The treatment of the electrolyte changes and haemolysis with exchange-transfusion has been suggested by others (Donald, 1955; Haukebø, 1960; Swann and Spafford, 1951). In our case it was suggested by Dr. Hans Fortun. We have not found any reports where exchange-transfusion had been performed in a case of fresh-water drowning in which the patient survived.

The kidneys showed impairment of function during the first few days as demonstrated by the increase of non-protein nitrogen. Microscopically the urine contained many casts, red blood corpuscles, and protein. He also had anuria during the first 12 hours. The kidney damage and the haemoglobinuria could well be due to the severe shock. The kidneys soon recovered normal function and many examinations later on showed completely normal findings.

Our patient has recovered to a gratifying extent. Air-encephalograms performed six months after the accident suggest reduction of the dilatation of the ventricles. We intend to re-examine him, however, by air-encephalography in a year's time. Psychological examination shows that his mental age is almost normal. The tests reveal that the finer movements of the fingers are still a little clumsy, and that the peripheral vision is reduced or absent. Routine neurological examination and E.E.G. show normal findings. So far as ordinary clinical impressions are concerned he talks, moves, and behaves as a normal child. There has been gradual improvement all the time and further improvement is expected. Only the years to come will demonstrate whether his mental and neurological functions are adequate to deal with the stresses of life.

Summary

Survival after drowning in fresh water is rare when submersion has lasted so long that aspiration of water has led to haemolysis and haemoglobinuria, and cardiac arrest has also occurred.

A case of a 5-year-old boy who drowned in ice-cold water is presented. He was successfully resuscitated by external cardiac compression, which was carried out for two hours before spontaneous cardiac contractions were restored. The haemolysis and haemoglobinuria were treated with an exchange-transfusion of 3,000 ml. of freshly drawn blood.

Apart from a brief return to consciousness on the tenth day he was unconscious for about six weeks. Air-encephalograms taken six weeks after the accident showed severe dilatation of all cerebral ventricles.

In spite of this he has recovered with little if any neurological and intellectual damage. Air-encephalograms taken six months after the accident showed that the dilatation of the cerebral ventricles had decreased.

It is suggested that the cooling of the child's body as he hung on to the ice and later during treatment may have played an important part in the successful outcome.

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On April 6 and 7 the Medical Research Committee of the National Deaf Children's Society held a conference at Pembroke College, Oxford, of workers undertaking research on the problems of deafness in children. Otologists, paediatricians, neurologists, neurophysiologists, psychologists, psychiatrists, and other specialists met to discuss the possibilities of further research into the prevention and cure of deafness in children.