ing the efficacy of these vaccines for any of these diseases are not yet available. Studies which would yield such data have been planned or are already under way.

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GENERAL PRACTICE

Drowning: Its Mechanism and Treatment

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ABSTRACT

Pertinent experimental work and literature relative to drowning are reviewed. The different concepts of the mechanism of drowning are dealt with and the view is emphasized that asphyxia is complicated by hemodilution in fresh water and by hemoconcentration in salt water, with resulting electrolyte imbalance. A short description is given of the sequence of events in drowning. Lethal heart failure has occurred as early as two minutes after total submersion. Treatment consists of artificial respiration, cardiac massage, correction of the electrolyte imbalance and continued observation for complications. The danger from hyperventilation in underwater swimming, of cases of so-called "secondary" drowning and the use of diatoms as a proof of drowning are mentioned.

DEATH by drowning is common; in 1960, the last year for which official figures are available, 224 accidental drownings and submersions occurred in Ontario. Eighteen people drowned in Ontario over the 1962 Victoria Day weekend. In the United States 7000 bodies were recovered from water in 1961. In England, 400 to 600 people commit suicide by drowning each year and there are as well 800 to 1000 cases of accidental drowning annually.

Knowledge of the mechanism of drowning has altered considerably in the course of time. Early in the eighteenth century the prevailing view was that it was caused by the swallowing of water and overfilling of the stomach. When the role of the lungs was discovered, it became generally accepted that drowning was caused by inhalation of water into the air passages and lungs, causing asphyxia. Then, as now, victims were found with no water in the lungs and this was explained by the concept that shock and reflex inhibition of the heart (from chilling or from water entering the upper air passages) prevented the entry of water into the lungs. By 1862 workers in England conducting experiments in this field concluded that some cause other than asphyxia alone must operate to render drowning so speedily fatal. It was suggested that death was likely due to water aspirated into minute bronchioles and alveoli. By 1880 it had been established that water also entered the circulation but this attracted little attention. In 1938, Banting[1] reported that the terminal event in fresh-water drowning was ventricular fibrillation. Although other workers disagreed at the time, he was later proved to be correct. From 1942 to 1951, the experimental work carried out in this field reflected a new interest in cardiac failure, rather than asphyxia, as the mechanism of death in many cases of drowning.

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Investigations showed that in at least 90% of cases of fresh-water drowning a large amount of water entered the lungs and was absorbed into the circulation, giving rise in a very few minutes to ventricular fibrillation with rapidly fatal heart failure. Three minutes after immersion the blood volume was increased by dilution with as much as an equal amount of water.

In salt-water drowning, water was rapidly withdrawn from the blood into the lungs, concentrating the blood and giving rise to a more gradual onset of heart failure, usually without ventricular fibrillation. After three minutes' immersion, the blood has lost 40% of its water and large amounts of electrolytes have passed from the sea-water into the blood. Therefore, in fresh-water drowning, hemodilution occurs and in salt-water drowning, hemoconcentration.

Despite the hemodilution which occurs in fresh-water drowning, blood potassium levels are increased rather than decreased; this is due to hemolysis of the red cells which releases potassium into the plasma. The concentration of calcium is low because of hemodilution. A high potassium level and low calcium results in an increased cardiac irritability and is probably an important factor in causing ventricular fibrillation.

In salt-water drowning, the increase in the sodium-potassium ratio does not reach a level at which ventricular fibrillation is precipitated except in exceptional cases.

In waters other than fresh or salt, the mechanism of drowning will depend on asphyxia and on the saline content of the fluid. There may be complications from the presence of algae, sewage or waste in the inspired water.

The sequence of events in drowning is now relatively clear. First there is a sense of panic expressed in violent struggles and automatic swimming movements. Usually a period of voluntary apnea of one or two minutes follows. Eventually an attempt is made at taking a breath. Water may be freely inhaled, or by its impingement cause glottic spasm against which violent attempts at inspiration will be made. In 10 to 15% of victims (in the older literature, this figure is given as 10 to 40%) glottic spasm becomes severe enough to cause death by asphyxia. Water may not enter the lungs unless unconsciousness results in relaxation of the spasm later.

In the remaining 85 to 90% of victims, water is swallowed, inducing vomiting, gasping and aspiration of water into the lungs; in the expiratory effort, fine froth, sometimes blood-stained, appears. After a few breaths or attempts at breathing, conscious struggling ceases and is followed by convulsive spasms, twitching, dilatation of the pupils and clinical death.

TREATMENT OF DROWNING

From a review of the literature it appears that resuscitation is usually successful if heart failure has not occurred. Once heart failure and falling blood pressure supervene, survival is most unlikely. Lethal heart failure often occurs as early as two minutes, is usual within six minutes and invariable within 10 minutes of total submersion. Many individuals removed from the water while still alive are doomed to die within a few minutes as a result of devastating changes. A small number have been rescued before large amounts of water have been inhaled; this fortunate circumstance is due either to the rapidity of the rescue, to glottic spasm, shock or reflex inhibition—all of which prevent much inhalation of water. Artificial respiration offers a chance for good recovery in these individuals because they do not have the complications of gross disturbance of fluid and electrolyte balance.

Reports exist which attest to successful resuscitation after longer periods of immersion than those recorded above. However, in the opinion of Smith and Simpson[2] "it would seem the earlier or more loosely recorded cases of recovery after submersion for more than seven to eight minutes are wholly unreliable. It is possible that some air may be drawn down in the clothing or in the hull or sail of an overturned boat." The submersion may have been intermittent or incomplete. Some harbour rescue-workers consider it a miracle to achieve successful resuscitation after four minutes' submersion. Survival will depend on the fitness of the person, the duration of the immersion and the amount of water inhaled. When survival occurs it is usually complete but observation is necessary for several days to detect complications.

If the mechanism of drowning is known, what measures should be undertaken to ensure the best chance of survival? From a survey of the literature the consensus is as follows:

1. Nothing in the modern view is against the use of artificial respiration; it is still necessary, its use is imperative and it should be undertaken by some technique as soon as the individual is removed from the water. The prevailing view is that one should not waste time clearing the airways, but this is subject to debate. The argument against clearing the airway immediately is that no useful purpose is served by creating a perfect airway while the myocardium is failing from lack of oxygen. Even a small amount of the oxygen from air reaching the vital centres in the first few seconds may achieve what pure oxygen and generous ventilation may fail to do one or two minutes later because even a small volume of oxygen will increase hemoglobin saturation quickly. However, no time should be lost in loosening the victim's clothing, feeling the pulse, examining the pupils, listening to the heart or finding sloping ground where he can be placed in a head-down position. Some of these measures can be carried out later by an assistant during artificial respiration.

2. However, at a recent Coroners' Course held under the sponsorship of the Attorney-General of Ontario, a participant recalled that the performance

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of a tracheotomy was necessary in the resuscitation of a drowned child at Toronto Island in 1914. Persistent glottic spasm of this degree is rare.

3. Knowing that cardiac failure is imminent, cardiac arrest must be prevented or, if present, treated at once. The development of techniques for closed cardiac massage has rendered thoracotomy unnecessary. While one person carries out artificial respiration (the technique of mouth-to-mouth breathing would leave more room for the second operator), another performs cardiac massage by applying pressure over the lower sternum. Pressure is applied using the heel of one hand with the other hand on top, to depress the sternum 1 in. to 1¼ in. with each stroke in adults, followed by release of pressure, at the rate of about 60 per minute. Kouwenhaven, Jude and Knickerbocker[3] report 70% recovery in 20 drownings in which the victims had developed cardiac arrest. Damage has been reported only in those instances where the application of pressure was not confined to the sternum.

The application of artificial respiration and cardiac massage should be continued for at least 15 minutes before any detailed clinical examination is made. If the victim has irreversible circulatory failure, nothing has been lost by delaying such examination. If only respiratory failure is present, prompt and continuously applied artificial respiration may save the individual's life. If at the end of 15 minutes the heart beat has not returned, the person is probably dead. If spontaneous respiration and restoration of the circulation do not occur within 15 minutes, further efforts are probably fruitless. If there is discernible evidence of active circulation, artificial respiration should be continued until spontaneous respiration is restored. On the other hand, marked body cooling or evidence of early *rigor mortis* indicates the futility of continued efforts in this direction. The presence of fixed dilated pupils persisting for 15 minutes is suggestive of clinical death. Members of the general public do not believe that death supervenes so early. Some bystander will recall that in a very different condition, electric shock, artificial respiration has been continued for up to four hours.

In order to avoid hysteria among bystanders and to allow treatment of the important circulatory disturbances by intravenous therapy, the patient should be sent to hospital as quickly as possible. If a truck is available or if an ambulance has already arrived, the patient should be placed in it as soon as the return of adequate respiration and circulation warrants removal from the scene. Artificial respiration and cardiac massage might be necessary again *en route* to the hospital.

On arrival at the hospital, appropriate solutions for intravenous therapy are ordered. In fresh-water drowning, this consists of 1000 c.c. of 3% saline, which is repeated in three to six hours if the serum sodium level is below 110 mEq./l. The deficit in serum calcium may need to be corrected by an infusion of calcium gluconate. Transfusion with whole blood, alternating with bleeding, may be needed later. In salt-water drowning, intravenous therapy is carried out with 5% dextrose—never saline, whole blood or plasma. Venesection may be needed later.

Biochemical tests must be repeated for a considerable period to avoid overcorrection of the electrolytic imbalance which may do great harm.

In the interval while intravenous solutions are being obtained and the apparatus set up, the respirations may still be inadequate. Redding *et al.*[4] recommend switching to an anesthetic machine using intermittent pressure and pure oxygen. Donald and Paton[5] warn against including carbon dioxide in the anesthetic mixture because it may be harmful to the victims of drowning. If the cardiac status is unsatisfactory, injections of epinephrine have been employed by some workers. If ventricular fibrillation has occurred, the use of external stimuli such as a quick blow over the heart, the application of electrodes with one or more shocks using 480 volts for .25 second, are recommended by some authorities. Defibrillators are now generally available. Open-chest massage is still used also.

In victims of fresh-water drowning the urinary output must be carefully observed; the hemolysis of red cells may cause renal tubular nephrosis.

The unexpected deaths of even well-trained underwater swimmers has attracted much attention in the recent medical literature. Loss of consciousness due to anoxia occurs with little or no warning and is the result of hyperventilation induced by deep breathing before diving, muscular exercise, increased oxygen consumption, and the excitement invoked by a competitive goal. Athletes should be warned about the effects of induced hyperventilation, and swimming events should be closely supervised.

There is little doubt that under circumstances which induce emotions such as fear or surprise, the swimmer may be in such a state that an ordinarily innocuous stimulus will cause vagal inhibition and immediate cardiac arrest. The shock of unexpected immersion or the stimulus of water impinging on the pharynx or larynx and causing spasm may account for many of these fatalities. These considerations may explain incidents such as that of a young man, a good swimmer, who, fleeing from the police and falling into water in the dark, was later found dead.

Occasions will always arise when doubt exists whether an individual whose body is found in the water died by drowning. The question should be left to the pathologist to answer. His examination may disclose a cause such as ruptured congenital vascular aneurysm. An individual with heart disease may die as a result of the shock of a sudden fall into water, or his heart may fail while he is swimming or struggling in water. Non-specific myocarditis may be more common than is supposed. Murderers often throw the bodies of their victims into water to confuse the authorities. Often the

pathologist may be unable to decide whether drowning occurred or not; for example, decomposition may render the decision impossible in many cases. The finding of microscopic silaceous plants, the diatoms, in organs that they could reach only through the circulation may be useful. This finding is especially useful where only portions of the body are recovered.

SUMMARY

The different views concerning the mechanism of drowning are reviewed. The present concept is that asphyxia is complicated by hemodilution in fresh water and by hemoconcentration in salt water. The experimental work bearing on these problems is summarized. The treatment of drowning obtained from a review of the literature is described, with emphasis on cardiac massage, intravenous therapy and continued observation. Some comment is made on the cause of deaths in underwater swimming and "secondary" drowning.

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CASE REPORTS

Hypertension Following Closure of a Postnephrectomy Arteriovenous Fistula: A Case Report and Hypothesis

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TO DATE only five* instances of postnephrectomy renal arteriovenous (A-V) fistula have been reported.[1-4] It is the purpose of the present communication to add a sixth case, unique in that the patient developed sustained hypertension following corrective surgery. The possible significance of the findings is discussed in terms of the role the fistula may have played in the clinical course.

S.R., a 38-year-old white male of Polish extraction, was admitted to the Jewish General Hospital, Montreal, for the first time on March 30, 1961, with a diagnosis of bronchopneumonia. This was subsequently confirmed by the usual physical findings, and radiographic, bacteriological and hematological studies. The patient was treated with penicillin.

Between 1945 and 1948 the patient passed a number of renal stones and had numerous attacks of bilateral renal colic. He had undergone numerous genitourinary operations at various Toronto hospitals. In 1945 he underwent a *left* pyelolithotomy and calycectomy with removal of the lower pole of the *left* kidney. Microscopic sections of this tissue showed evidence of severe chronic pyelonephritis. In 1948 a total *right* nephrectomy was performed because of calculous pyonephrosis. Repeated analyses showed the stones to be composed of triple phosphates. Between 1945 and 1948 blood pressure was recorded as normal. In 1951 he was again admitted to hospital in Toronto for left renal colic and apparently passed a stone spontaneously. Blood pressure was 134/80 mm. Hg at that time. In 1955 the patient developed tuberculous orchitis (left). An orchidectomy was performed. The blood pressure was 120/84 mm. Hg at that time. There is no family history of hypertension.

During the first week of admission to this hospital in April 1961, the pertinent physical and laboratory findings related to the cardiovascular-renal systems were as follows: blood pressure 120/80 mm. Hg, pulse 96 per min., apical impulse diffuse and forceful with an ejection systolic murmur heard best along the left sternal border. There were well-healed flank scars. The lungs had the typical physical findings of bronchopneumonia. Blood urea nitrogen (BUN) was 84 mg. %; the BUN three days later was 57 mg. % and the creatinine was 2.5 mg. %. Numerous urinalyses showed the specific gravity to range between 1.008 and 1.012; 3+ proteinuria and a few white cells were present in the sediment. No casts or red cells were seen. The hemoglobin was 13.1 g., calcium 9.4 mg. %, phosphorus 3.6 mg. %. Urine cultures were sterile. Chest radiograph showed pneumonic infiltration; the cardiothoracic ratio was 14.2/29.5. Intravenous urography showed no dye on the *right* side (absent kidney). Two curvilinear calcific densities were noted in the region of the right renal pedicle. The left kidney had dilated calyces, but no evidence of obstruction was seen.

On the fifth hospital day the patient developed an area of ecchymosis over the flanks. Coagulation studies were normal except for a slightly increased prothrombin consumption time. The bleeding appeared to be subcutaneous, although retroperitoneal bleeding could not be ruled out. The patient had no complaints; he voided well, without gross or microscopic blood. At this time

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*An additional case, without details, was mentioned by Vest[5] in 1954.