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DROWNING

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Despite the great interest taken in drowning by various lay organizations in this country, there has been a truly astonishing neglect of this subject by physiologists and medical men. Another bathing season has begun, and the usual crop of drowning accidents may be expected. This article reviews the subject of drowning and its treatment, the aims being to stress the principles on which rational resuscitation must be based and to encourage the recording of clinical experience in this field.

In order to understand the problems of treatment of drowned persons it is necessary to describe the events in the body of the victim which may lead to death. The majority of important contributions in recent years have been by American workers, particularly H. G. Swann and his colleagues (1947, 1949, 1951a, 1951b, 1953). Of necessity, most of our information on what happens is derived from animal experiments. There is considerable variation in the reactions of these animals and still some doubt about certain aspects. These will only be mentioned when relevant to the problems of therapy.

Fresh-water Drowning

When an animal is totally immersed in fresh water there is an initial period of struggling and apnoea. From one to two minutes after submersion an involuntary inspiration occurs and water is drawn into the lungs, usually in large quantities. In some cases glottic spasm will prevent the lungs being immediately flooded. Banting *et al.* (1938) have shown that local anaesthetization of the glottis will prevent such spasm and allow even larger quantities of water to enter the lungs. They also described how many animals swallowed water violently as apnoea became intolerable. This was often immediately followed by vomiting and inhalation of water into the lungs in a series of gasps. In the experiments on dogs of Swann *et al.* (1947, 1949, 1951b) some flooding of the lungs occurred in all animals.

When the fresh water enters the lungs there is an immediate and enormous absorption of this fluid into the circulation across the alveolo-capillary membrane. The amount of drowning-fluid absorbed can be estimated by the degree of haemodilution or by measuring the levels of blood concentration of tracer substances placed in the drowning-fluid before the experiment. It has been shown that an amount of water equivalent to 60–150% of the blood volume can enter the circulation in a few minutes. The rate of dilution of blood is quite fantastic. Swann *et al.* (1951b) cite an experiment in which 72% of the circulating fluid was drowning-fluid three minutes after submersion and probably less than two minutes after the inhalation of water. It will be apparent that the blood entering the left side of the heart is greatly diluted and its electrolyte concentration proportionately reduced. Haemodilution is inevitably accompanied by haemolysis, and large quantities of free haemoglobin appear in the plasma. Since potassium is released when erythrocytes are lysed there is a considerable gain in plasma potassium from this source, together with a rise due to severe anoxaemia. Thus the plasma potassium concentration is not reduced to the same degree as that of sodium, with the result that the K/Na ratio is greatly increased. This disturbance of electrolyte ratios is more dangerous than overall changes in tonicity. The coronary circulation receives this highly abnormal blood and the myocardium is exposed to serious biochemical insult as well as extreme anoxia. In a few minutes ventricular fibrillation begins. The great overloading of the circulation probably contributes to this event. It is also possible that there is extreme pulmonary vasoconstriction when the lungs are flooded, as occurs in acute and severe anaphylactic shock. After death from drowning the right ventricle is usually found distended and the left ventricle contracted and almost empty.

Ventricular fibrillation usually occurs three to seven minutes after submersion (dogs) and there is an immediate and dramatic fall in blood pressure, and the absence of any effective cardiac output heralds inevitable death from absolute cerebral anoxia. The time of onset of ventricular fibrillation does not appear to be closely related to the degree of haemodilution. Respiratory failure, as judged by cessation of effective respiratory excursions, usually occurs almost simultaneously with the onset of ventricular fibrillation, but sometimes it occurs shortly before (10–20 seconds) and sometimes shortly after (10–20 seconds).

It is not so easy to obtain facts concerning the events in human drowning, but there is no reason to believe they are different. Previous literature would suggest that a number of human beings are drowned with dry lungs owing to glottic spasm, but little convincing evidence has been produced, particularly as it has been shown that animals with fatal haemodilution may show almost dry lungs. Lowson (1903), in a remarkable account of his experiences and sensations during near drowning, described how he "breathed in" when apnoea became intolerable and immediately swallowed the water in a large gulp. This occurred about ten times, and he experienced increasing relief, which he later attributed to the sedative effects of mounting carbon dioxide tensions in the body. He then became unconscious, but recovered on the surface and after a

number of breaths swam to shore, where he vomited copious quantities of water. He had no signs or symptoms of any water in the lungs. It would appear likely that if such a victim remained submerged the reflex closure of the glottis would cease and water would be aspirated.

Swann *et al.* (1951b) surmise from data reviewed that only one in twenty men drown in fresh water with no significant water in the lungs or haemodilution. There is also considerable species variation regarding the onset of ventricular fibrillation under these conditions. However, experiments (Gordon *et al.*, 1954) have shown that ventricular fibrillation occurs in one to three minutes after submersion in fresh water in the larger animals (horses, cows, pigs), and it would appear likely that the human is at the same risk. Electrolyte studies of human victims of drowning in fresh water (Moritz, 1944) showed changes capable of precipitating ventricular fibrillation in at least half of those studied (Swann *et al.*, 1951b).

Salt-Water Drowning

In salt-water drowning the electrolyte concentration of the inhaled fluid is greater than that of blood. Consequently there is considerable movement of water from the circulating blood into the lungs, and in dogs drowning in salt water there is approximately a 33% haemoconcentration. There is, however, no haemolysis or any disturbance of the K/Na ratio. Ventricular fibrillation does not occur, and the heart action fails gradually in five to eight minutes (in dogs) (Swann *et al.*, 1947, 1951b). There is evidence that the haemoconcentration is not so marked in humans who have drowned in salt water. In acute asphyxial episodes where ventricular fibrillation does not occur the systolic blood pressure often remains high for several minutes, although the diastolic pressure reaches very low levels. Swann (1951a) has shown that if the systolic blood pressure remains above a certain level (115 mm. Hg approximately) then resuscitation by artificial respiration is almost certain to be successful. If, however, it drops below a certain level (50 mm. Hg approximately) then resuscitation will certainly fail (dogs). The time between the "point of success" and the "point of failure" is as short as 20 seconds in these animals. The heart failure in salt-water drowning is almost certainly due to prolonged and severe myocardial anoxia. Very occasionally victims (dogs) of fresh-water drowning show this type of circulatory failure.

Apart from the movement of water into the lungs, there is also evidence of severe pulmonary oedema with plasma exudation into the alveoli in these animals in both fresh- and salt-water drowning. The resultant protein content of the lung fluids is partly responsible for the tenacious froth encountered in the air passages in drowning.

The all-important question of the time relationship between irreversible circulatory failure and respiratory failure must be considered. It is obvious that unless respiratory failure occurs before irreversible circulatory failure then the giving of artificial respiration will be useless in such cases. From Swann's data in a small series of dogs it would appear that respiratory failure occurs some time before irreversible circulatory failure in a high proportion of cases. In such instances artificial respiration will be life-saving. However, Swann states that respiratory failure occurs in only one-third of cases before circulatory failure begins, in one-third during the onset of failure, and in one-third after circulation has failed. More work needs to be done on this important aspect.

Pathological Appearances at Necropsy

At necropsy of drowned persons the obstruction of the airways by fine persistent froth is seen only in the smaller airways. The trachea and larger bronchi may also contain froth, but they are not obstructed by it. When the lungs are held up the froth and fluid do not drain out of these smaller bronchi. The lung parenchyma contains very varying amounts of fluid, tending to be greater in salt-water drowning for the reasons already mentioned. Swann opened the chest of dogs after drowning, cannulated the trachea down to the bifurcation, and drained for long periods. In fresh-water drowning, although moderate quantities of water were usually drained off, only a few millilitres of water was obtained in some cases despite great haemodilution. In salt-water drowning larger quantities of water were obtained. It is unfortunate that figures of immediate drainage of water from the lungs of intact drowned animals have not been reported. Many pathologists consider that it is most unlikely that any important drainage of the lungs or clearing of airways will be obtained under such conditions. If this is true then time spent in such manœuvres may only assure the death of the victim owing to the delay of artificial respiration.

The stomach may contain very large amounts of the drowning-fluid owing to reflex swallowing, and there may be evidence of stomach contents in the air-passages. This may be due to the agonal vomiting already described or to the transfer of stomach contents to the respiratory passages by increased abdominal pressure during artificial respiration. As already mentioned, the right ventricle is usually found greatly distended and the left heart contracted.

As in other forms of asphyxial death, small local haemorrhages may be found in different parts of the body, particularly in the central nervous system. These haemorrhages are usually far less marked in drowning, and, if they are conspicuous, asphyxia or violence before entry into the water must be considered. The most valuable test for drowning is the demonstration of difference in electrolyte concentration of the blood in the right and left ventricles. This test is now being adopted by all forensic laboratories. The significance of electrolyte changes in the blood after long periods of submersion is still in doubt, and studies continue on this problem. The examination of peripheral lung and stomach contents for water algae may be extremely useful in cases where the cause of death is uncertain.

Resuscitation from Drowning: Experimental Evidence

Schäfer (1908) reported that anaesthetized dogs could not be resuscitated after two minutes' submersion in fresh water. Cot (1932) confirmed this, but resuscitated three out of eight dogs after 60 to 90 seconds' submersion. Other work has been done on this subject, but unfortunately the time relationships are not stated in the reports, which are therefore of little value in this respect. Fainer *et al.* (1951) carried out a particularly interesting series of experiments which merit detailed description. He used one hundred and sixty dogs. Only the head was flooded with fresh water. Blood-pressure recordings were taken, and chest-wall movements were recorded by means of a band round the chest. Artificial respiration was given manually and, in some cases, by positive- and negative-pressure resuscitators. They described three stages of drowning. In the first stage, lasting 71 seconds (mean figure), the animal struggled and the blood pressure rose. Stage II was heralded by the cessation of struggling and ended with a precipitous fall of blood pressure. This stage lasted one minute (mean figure). Stage III began with this fall of blood pressure and ended when it reached zero (duration two minutes approximately, mean figure).

The first important finding was that no animal survived, with or without resuscitation, after the precipitous fall of blood pressure had occurred; that is, just over two minutes (mean) after submersion. It was presumed that this fall of blood pressure was due to the onset of ventricular fibrillation. This occurred in 70 to 160 seconds after submersion in 95% of 100 dogs. The findings were not so definite concerning the respiratory behaviour and inhalation of water. The published recordings show respiratory movements

throughout stage I, but it is well known that unanaesthetized animals hold their breath when first submerged and that rhythmic chest movements can continue with a closed glottis. For these reasons it is not possible to relate the resuscitation results to the time of inhalation of water.

In a large series of experiments the water was released 5 to 10 seconds after struggling ceased. At this stage the chest movements were usually irregular or had stopped altogether. The rate of survival (50 to 60%) was the same in dogs receiving manual resuscitation or mechanical resuscitation and also in those receiving no treatment at all. A series of experiments to determine the effect of drainage was also carried out 5 to 10 seconds after the cessation of struggling. Rapid tipping down of the head to 50 degrees followed by resuscitation with a 20-degree downward tilt, and a 20-degree tilt alone, made no significant difference to the survival rate. In these experiments excitement or exercise before submersion appeared to decrease the rate of survival.

Dogs were also studied one minute after the cessation of struggling. The survival rate (10%) was identical in those receiving no resuscitation, in those receiving manual artificial respiration, and in those receiving mechanical resuscitation.

Methylene blue was added to the drowning-water, and thus staining of the lungs, as determined after death or 10 minutes after resuscitation, gave a crude measure of the amount of water aspirated. Those with little or no staining showed a 95% survival, those with moderate staining 46% survival, those with marked staining 3% survival. It would appear that the amount of fluid aspirated in the lungs determined whether death occurred or not.

Free haemoglobin levels were also studied. In seven dogs which survived, the mean level of free haemoglobin in the serum was 0.3 g.%. In 28 dogs which died the blood serum showed a mean level of haemoglobin of 4.9 g.%. This confirmed that the degree of aspiration of water and its absorption was a vital factor affecting survival or otherwise.

Like Swann, these authors comment on the remarkable lack of free water that could be drained away in the lungs at necropsy. They also confirmed that larger quantities of water are recoverable by drainage in salt-water drowning.

In later experiments, H. E. Swann *et al.* (1953) drowned white rats in fresh water and determined the survival rate at the end of the struggling phase. They used these small animals because they do not develop ventricular fibrillation as usually occurs in larger animals. Fifteen were given artificial respiration and 15 were untreated; 31% survived, and artificial respiration had no effect on rate of survival. They also noted that, despite the failure to resuscitate these animals, the heart continued to beat slowly but strongly for six to eight minutes after recovery from the water. In all those surviving this procedure opening of the chest showed that the airways were clear of froth, whereas the fatal cases all had the trachea and bronchi blocked with foam and water. Again, these experiments would suggest that, even if irreversible circulatory failure has not occurred, respiration, natural or artificial, cannot give efficient pulmonary ventilation if considerable quantities of water have been aspirated. It must be remembered that these are very small animals and that the ventilation of larger animals is probably not so gravely impaired by froth and water, owing to the wider air passages.

Gray (1951), judging the chance of survival by the length of time after submersion before terminal gasping respiratory movements occurred, found, not surprisingly, that one of the main factors in delaying the onset of irreversible changes in rats was the initial period of apnoea when submerged. He also found that immersion in very cold water (1° C.) also prolonged the period of probable physiological survival. This has been attributed to reduced oxygen demands and extreme vasoconstriction in non-vital areas. However, submersion in water above body temperature (55° C.) also had the same effect.

Mueller and Malteur (1952) studied the efficacy of intramuscular injections in rats during various stages of freshwater drowning, by the ingenious use of fluorescein and hyaluronidase. Peripheral appearance of this substance in the cornea, pads, ears, and snout was easily appreciated. They used white rats, which it is now known do not develop ventricular fibrillation. They themselves noted that the heart action did not cease completely. They reported the effective circulation of substances injected during the struggling phase and during convulsive movements at the end of this phase. However, once anoxic apnoea had begun no peripheral fluorescein was seen after injection. In a number of cases the addition of leptazol to the injection given in the anoxic apnoeic phase caused the appearance of fluorescein at the periphery.

No detailed study of resuscitation from salt-water drowning in animals has yet been reported.

Clinical State after Resuscitation from Drowning

In a careful review of medical literature only five clinical reports (12 cases), none from British sources, on the after-effects of near drowning have been found. There are no doubt a few others, but, considering that drowning is about the third commonest cause of accidental death, this is a most surprising finding.

Trocmé and Lafarie (1947), Trocmé and Lebert (1949), Buchtala (1950), Romagosa *et al.* (1950), and Haddy and Disenhouse (1954) have all reported what appeared to be mild pulmonary oedema, as judged clinically and radiologically, in persons resuscitated after submersion in salt and fresh water. The clinical signs of pulmonary oedema were remarkably mild in most cases, and some of these authors considered that it may well have been caused by extreme anoxia, respiratory obstruction, or "central" causes rather than by the inhalation of water. In all cases the clinical and radiological picture became normal in a few days. There is no mention of renal damage or plethora, and no immediate electrolyte or free haemoglobin estimations were reported. Haddy and Disenhouse recommend the administration of oxygen by positive pressure and the trial of anti-foaming agents such as vaporized ethyl alcohol in the early stages.

If a victim has flooded his lungs with fresh water and yet has been successfully resuscitated then it would appear certain that he would be suffering from plethora, haemodilution, electrolyte disturbance, and pulmonary oedema. Further, he would be in danger of severe renal damage from the presence of freely circulating haemoglobin. Substitution bleeding with electrolyte correction, treatment for acute kidney damage, and oxygen therapy for pulmonary oedema and lung flooding would seem to be indicated. Hypotonic saline and oxygen therapy may be of help in those saved from salt-water drowning. The almost complete absence of any clinical reports of patients requiring and receiving such treatment in the vast medical literature of to-day can hardly be due to a deliberate silence on this subject, and it is difficult to avoid the sinister interpretation that such syndromes probably do not exist and that the aspiration of any marked quantity of water into the lungs is fatal.

Further Problems and Considerations

It will be apparent that there is still a great deal of work to be done on the problems of drowning, particularly in relation to resuscitation. Information is most needed concerning the state and resuscitability of larger animals after brief periods of submersion and immediately after the inhalation of water. The value or dangers of rapid drainage and the efficiency of pulmonary ventilation and blood oxygenation with early flooding of the lungs are not yet known. The dangers and degree of haemodilution, pulmonary oedema, and haemoglobin liberation at this early stage should also be investigated. The findings described would suggest that submersion in fresh water is far more lethal than in salt water, yet reliable figures are sadly lacking.

With regard to the instructions given to lay persons on life-saving and resuscitation, it is remarkable that no mention is made of the series of events in drowning, as briefly detailed in this article, in either of the commonly used handbooks. The emergency is described almost completely as an asphyxial one. Those who are willing to devote much time to life-saving practice and are ready to risk their lives to this end should be better briefed concerning the simple physiological events in drowning, and particularly the "awful draught" of fresh water down the trachea that may kill in seconds. The recommended treading of water while a violently struggling person "lessens his energy," and the suggested holding of the nose to make a drowning person "choke" and thus become more amenable, are procedures that appear to show little insight into the almost certain death that attends the inhalation of large quantities of water. An improvement of present techniques of life-saving to avoid water inhalation may not be possible, but its critical dangers must be emphasized more.

The great work carried out by these life-saving organizations in encouraging the general adoption of swimming and life-saving instruction in schools and institutes is the true prophylaxis of drowning.

Problems of Resuscitation

The condition of the victim will be very varied. Although different physiological states are mentioned here to emphasize important points regarding resuscitation, differential diagnosis is usually impossible and, in any case, should not be attempted before artificial respiration is begun. Let us first consider the victim who has not inhaled water and has suffered from severe asphyxia and is not breathing. If irreversible circulatory failure (blood pressure below "point of failure") has occurred then artificial respiration cannot succeed. However, if respiratory failure only has occurred then artificial respiration may save the life of such a person by preventing circulatory failure and reinstating spontaneous respiration. Only seconds may remain in such a case before irreversible circulatory failure occurs.

It is obvious that not a second, *literally not a second,* can be wasted. Artificial respiration of some sort must be started as the victim is landed, even at the risk of other injuries. No time should be wasted in clearing the airways, loosening clothes even at the neck, feeling the pulse, listening for the heart action, draining of the lungs, or selecting of suitable slope so that the head is down. If another person is available then he can clear any material from the mouth and check the tongue position, but such manœuvres must not interfere with unremitting artificial respiration. If an operator is alone he can adjust the head and check the airway between cycles. It is no good creating a perfect airway while the myocardium is failing completely from lack of oxygen. Even a small amount of air in the first few seconds may accomplish what pure oxygen and large pulmonary ventilation may fail to do 10 to 20 seconds later.

Detailed clinical examination before at least 15 minutes' efficient artificial respiration has been carried out may well be a lethal procedure, and it is possible that a person with medical knowledge may even constitute a threat in this respect. If the victim has irreversible circulatory failure then nothing has been lost by delaying the diagnosis. If he has only respiratory failure then prompt and continued artificial respiration may save his life.

Before proceeding to further details of treatment let us consider the state of victims who have inhaled water. Again there will be the victims with irreversible circulatory failure due either to ventricular fibrillation (fresh water) or to anoxic myocardial failure (salt water); these cannot be saved by artificial respiration. There will be those who are in respiratory failure without circulatory failure. Those from fresh water have probably less than a minute before the fatal event of ventricular fibrillation, and although such people may occasionally be snatched from the jaws of death it must be a very rare event. Nevertheless, it emphasizes that some chest movement should be initiated almost as the victim is brought out of the water. In salt-water drowning, where only respiratory failure has occurred, there may be a longer period before circulatory death occurs in some cases, but in animal experiments this period can be very short indeed.

The Holger Nielsen Method

The Holger Nielsen back-pressure–arm-lift method of artificial respiration is now officially recommended by all organizations. The victim is in the prone position with the head to one side, and this usually ensures a good airway. It is a "push-pull" method, and not only is pressure applied to the upper chest to induce expiration but the arms are also lifted to induce inspiration. The advantages of this method are not only the increased efficiency of pulmonary ventilation but also the lack of pressure on the abdomen with the risk of further inhalation of regurgitated water. There are even more vigorous and effective "push-pull" methods, such as the hip-lifting technique, which, although hard work, can be maintained for several minutes, during which the issue is usually decided one way or the other. Medical men and persons who may be called upon to carry out resuscitation should know these more vigorous methods of artificial respiration. Those interested in artificial respiration should consult the special issue of the *Journal of Applied Physiology* (December, 1951) devoted to research in this subject.

The efficacy of drainage is now regarded as most uncertain, although more studies are necessary in salt-water drowning, and, in any case, the overriding consideration is the need for immediate starting of artificial respiration. No time must therefore be wasted on this procedure. Artificial respiration itself may help to clear the passages of water more effectively than mere gravity. It is humbling to those who consider applied physiology to be in an advanced stage to know that, although a number of modern first-aid manuals still recommend drainage before artificial respiration, Dr. Jackson wrote in 1746: "The practice of hanging by the heels is not only useless but must also contribute to destroy those remains of life which may possibly be lost," and added, "a single moment often determines between a state of death and life."

The extreme urgency of the situation makes it unwise to think in terms of rocking stretchers, oxygen therapy, or "stimulating" injections. Manual ventilation is as efficient as any portable mechanical apparatus, and even though the apparatus may be brought to the subject it is distracting, and the issue is being decided already. Oxygen therapy is invaluable, but it is again rarely available in the first few minutes. However, it should always be given as soon as possible, provided the preparation of the apparatus and its application does not in any way interfere with unremitting artificial respiration. The firm and continued application of a mask to a person receiving artificial respiration by the Holger Nielsen method is difficult, as the head is slightly lifted with each pull manœuvre. The commonly used masks have been recently modified by the Wolverhampton and Birmingham Fire and Ambulance Services, and the general manufacture of such masks is now under way.

The covering of the victim and the use of hot-water bottles are procedures which are unlikely to have any effect on the issue of life and death. Extreme cold should theoretically allow a person to survive anoxia for longer periods, but in our present state of knowledge one can do no more than recommend sensible nursing practices under these conditions, provided they in no way interfere with continuous artificial respiration.

The medical man may arrive after a few minutes' artificial respiration has been carried out, and those present will look to him desperately for expert advice and treatment. He must not stop artificial respiration to examine the chest unless it has continued for at least fifteen minutes. If the heart has failed but is not fibrillating then the only tenuous

hope of resuscitation lies in further artificial respiration. If the heart action is still present then continued artificial respiration is a desperate necessity.

If ventricular fibrillation is present this can only be guessed at, and an injection of procaine would be useless without a functioning circulation. It could be given intravenously to a victim of fresh-water drowning to prevent its onset, but this is hardly feasible in practice. Again, if ventricular fibrillation is thought to have just begun it may be possible to save a person's life by cardiac massage, although the institution of successful cardiac massage in the few minutes before irreversible cerebral damage has occurred is unlikely ever to be seen in practice.

Other Problems

There is another danger that may, oddly enough, arise from a medical man's considerable experience in anaesthesia. He may be accustomed to dealing with patients who are anaesthetized and have been breathing high percentages of oxygen. Under these conditions, where the respiratory centre is often already depressed by the anaesthetic, respiration may cease and yet there is no risk of circulatory failure for a number of minutes. Further, such patients can be almost pulseless and yet continue to breathe over considerable periods and not be in great danger. As a result of this he may forget that in the case of drowned persons with gross anoxia both respiratory and irreversible circulatory failure ride together and only seconds may divide respiratory failure and death.

Lastly, the medical man will be consulted on how long artificial respiration should continue in a drowned person who is not responding in any way. It is known that small animals which are going to recover will almost always do so within 60 to 120 seconds of the initiation of artificial respiration, although recovery after five minutes has been reported. Let us suppose that the subject is examined after 15 minutes' artificial respiration and that the doctor can find no evidence of heart action or spontaneous respiration. He will consider it almost certain, provided his observations are correct, that the victim is dead, as the brain cannot survive more than five minutes' circulatory stasis. Even cardiac massage would be in vain. Yet the layman is suspicious of the medical man in these matters, and has a lore of his own concerning the "bringing back to life" after hours of patient labour. Experts consider that such cases, even if "substantiated," were almost certainly due to the return of consciousness and not of respiration, acute hysteria, or "playing possum" by the would-be suicide. Yet experts are fallible, and they themselves support the continuance of artificial respiration for at least half an hour, and usually much longer.

Knowledge is limited, and reliable clinical observations are not easy under these conditions. It would be too onerous a responsibility to place a time limit on the resuscitation of drowned persons in general terms, and the decision on how long artificial respiration should be continued must rest with those present on each occasion. As a general rule it should continue for at least an hour. There may not be such miracles, but it is wrong to deny the possibility of one and resist the perhaps hopeless but admirable desire to turn tragedy into triumph. It should, however, be emphasized again and again that it is in the first few seconds after the body has been recovered that prompt action may save life.

Care During Recovery

Reliable documentation of this stage is sparse, and personal experience inevitably very limited. The signs of recovery are usually self-evident; spontaneous respiration is often preceded by a gasp and the colour of the patient may suddenly improve. A slight movement of the patient may be noted. The operator must try to co-ordinate the timing of his movements with those of the patient's respiration, continuing for some time after respiration has been re-established. If respiration appears effective, then only the "pull" movement of the arms need be continued. Even after artificial respiration has been discontinued the patient should be carefully observed in case of a recurrence of respiratory failure. The giving of oxygen *after* artificial respiration has been discontinued is not without its dangers (Donald and Paton, 1955), and, although the patient may be a good colour, the respiratory excursions, degree of ventilation, and circulatory state should be watched most carefully.

All authorities emphasize the dangers of excessive stimulation (massage, heat) or change in posture during this early stage of recovery. If, however, the patient's condition is good then he should be placed on his side. The work of respiration is very great in the prone position, as the body weight is lifted with each inspiration.

The dangers of regurgitation and aspiration of swallowed water and stomach contents while unconscious are familiar to medically qualified persons, but this can occur very silently and should be carefully watched for. The turning of the patient on to his side and not his back is again of value in this respect.

Care must also be taken in the giving of stimulants by mouth, and it must be made certain that the patient has an efficient swallowing reflex.

All patients should be moved to a hospital as soon as possible. They should be handled with great care, as severe secondary shock has been described (reliable references not found) in a number of instances. It is difficult to suggest how this should be combated under first-aid conditions. In salt-water drowning the giving of "plenty of fluid," usually recommended, would appear to be rational, but in fresh-water drowning this may well be dangerous.

Conclusions

When a drowned person is rescued artificial respiration must be begun at the risk of other injuries as soon as he is taken from the water. The necessity of beginning artificial respiration at once and continuing it without interruption for at least 15 minutes cannot be overemphasized. All other procedures and considerations, such as postural drainage, examination of the patient, administration of oxygen or drugs, must be considered secondary and be implemented only if they in no way interfere with immediate, efficient, and unremitting artificial respiration. The airway must of course be watched, but this should be considered after artificial respiration has been started. Elaborate procedures of any sort that delay artificial respiration will gravely threaten the chance of survival. Usually a medical man arrives on the scene after the issue has been decided. He must never stop the administration of artificial respiration to examine the patient in the first 15 minutes unless obvious recovery has occurred.

The main contribution that the medical profession can make to this problem is the initiation of more active research into the physiology of drowning and the efficacy of various methods of resuscitation. The publication of any observations made on persons resuscitated from drowning who have been studied and treated by medical men would also be of great value.

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BEHAVIOUR OF CHIMPANZEE-AVIRULENT POLIOMYELITIS VIRUSES IN EXPERIMENTALLY INFECTED HUMAN VOLUNTEERS*

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Poliomyelitis is generally more severe in adults than in very young children. Any method of immunization must avoid the possibility of creating a large adult population without resistance to paralytic poliomyelitis. Natural inapparent infection in early childhood, the process by which the vast majority of the world population acquires immunity to poliomyelitis, is known to provide lifelong protection. The remarkable observations of Paul *et al.* (1951) on Eskimos indicate that even in the absence of reinfection the body continues to produce specific antibodies for as long as 40 years after exposure to the virus. Natural infection with poliomyelitis viruses thus seems to provide the body with a "built-in" booster mechanism.

Studies on experimentally produced and naturally occurring attenuated strains of poliomyelitis virus are designed to provide information which would indicate whether or not the natural process of immunization can be duplicated without incurring the varying risk of paralysis encountered in nature. The first exploratory qualitative experiments on human beings were performed by Koprowski *et al.* (1952, 1954, 1955) with a rodent-adapted Type 2 poliomyelitis virus of diminished virulence for monkeys. In 1953, utilizing tissue culture techniques, we succeeded in segregating from highly virulent strains of each of the three immunological types of poliomyelitis virus, variants which are avirulent for monkeys by the intracerebral, oral, and parenteral routes (Sabin *et al.*, 1954). Further studies showed that these strains possessed a varying and limited capacity for producing paralysis after direct injection into the spinal cord of monkeys, but similar inoculations into the spinal cord of chimpanzees produced neither paralysis nor lesions. Yet when these strains were fed to chimpanzees they produced clinically inapparent immunogenic alimentary infections, unassociated with the viraemia which is so regularly encountered in infections with virulent viruses (Sabin, 1954, 1955a, 1955b).

The purpose of the present study was to determine by quantitative methods the comparative behaviour in human beings of the same culture fluids which had been extensively studied for a period of 18 months in monkeys and chimpanzees. The volunteers, 21–30-year-old inmates of a Federal reformatory, were selected on the basis of previous tests which showed that they lacked demonstrable antibody for one or more types of poliomyelitis virus. The three types of virus in the form of monkey kidney tissue culture fluid were administered in doses of 0.001, 0.1, 0.5, or 1 ml. in a teaspoonful of milk to 26 men—8 men for the Type 1 virus, and 9 each for Types 2 and 3. Four men who on initial testing had no demonstrable antibody for any of the three types were inoculated intramuscularly with the Type 3 virus—two with 0.001 ml. and two with 0.1 ml. Quantitative tests for virus were performed at frequent intervals on the stools and blood and on swabs from the mouth and tongue, and from the posterior pharyngeal wall. Antibodies were determined quantitatively over a period of three months.

Results

The results of this study, which will be reported in detail elsewhere, may be summarized as follows:

1. Human beings are more susceptible than chimpanzees to infection by the oral route. The smallest dose used—that is, 0.001 ml. of culture fluid containing $10^{4.2}$ to $10^{4.5}$ TCD_{50} of virus—sufficed to produce an immunogenic alimentary infection in the volunteers (Table I).

TABLE I.—*Comparative Susceptibility of Human and Chimpanzee Alimentary Tracts to Infection with Chimpanzee-avirulent Poliomyelitis Viruses*

Type and Strain of Virus	Dose		No. Infected	
	ml.	TCD_{50}	Humans	Chimpanzees
1 "Mah." KP33	0·5	$10^{7.2}$	2/2	4/5
	0·1	$10^{6.5}$	3/3	—
	0·001	$10^{4.5}$	3/3	1/3
2 YSK, KP51	1·0	$10^{7.2}$	3/3	5/8
	0·1	$10^{6.2}$	3/3	1/3
	0·001	$10^{4.2}$	1/1*	0/2
3 Leon, KP34	1·0	$10^{7.4}$	3/3	5/5
	0·1	$10^{6.4}$	3/3	—
	0·001	$10^{4.4}$	3/3	0/3

* Two other volunteers who had irregularly demonstrable traces of Type 2 antibody showed no evidence of infection after ingestion of this dose of virus

2. There was no multiplication in the mouth and tongue, nor was virus present there except in traces on rare occasions, even when it was recovered from the throat in amounts as high as $10^{4.5}$ TCD_{50} per swab (Fig. 1).

3. Whether or not the virus localized and multiplied in the throat was determined by the dose that was swallowed.

TABLE II.—*Influence of Amount of Poliomyelitis Virus Swallowed on Localization of Infection in Throat of Human Volunteers*

Type of Virus Ingested	No. of Individuals with Virus in Throat or Stools 5 Days or Longer after Swallowing Indicated Amount (ml.) of Culture Fluid					
	Throat			Stools		
	0·5–1	0·1	0·001	0·5–1·0	0·1	0·001
1	2/2	2/3	0/3	2/2	3/3	2/3
2	3/3	3/3	0/1	3/3	3/3	1/1
3	3/3	2/3	0/3	3/3	3/3	3/3

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