

IMMERSION AND ACCIDENTAL HYPOTHERMIA *

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SUMMARY

The immersion or near-drowning syndrome is discussed and found to be closely related with accidental hypothermia. Pathophysiology and clinical picture of both are discussed, and particular emphasis is done to the treatment. The emergency care includes cardiopulmonary resuscitation, and control of the pulmonary oedema, initial acidosis, hyperpotassemia and hypocalcemia, and late hypopotassemia, but, most important of all, the rewarming. Hot water bath, hot moist air or oxygen breathing, extracorporeal circulation can be used, but peritoneal dialysis is showed to be technically simple and very efficient. Three clinical cases are described.

IMMERSION

The immersion, or submersion, or near-drowning syndrome has for some reasons long been considered a very complex delicate matter for treatment, often to a degree of impairing the very important immediate steps in the initial treatment.

Therefore, it might be of value to consider:

What happens when a human is totally immersed in water? According to Keatinge less than 10 per cent will suffer an immediate cardiac arrest, probably caused by a vasovagal reflex, and most often by immersion in very cold water.

But normally — which means in more than 90 per cent — the immersed victim will try to avoid water in the upper respiratory areas by holding the breath followed by a laryngeal spasm — often so long that the asphyxia will provoke loss of consciousness.

First after several minutes, this laryngeal spasm will relax, opening the passage to the respiratory organ. But even at this point, the exchange between water and air in the narrow tubules in the lungs will take place over some time, leaving some air in the lungs for a considerable amount of time.

How long, is impossible to declare. But as long as some air remains in the lungs, the victim will float head-up in the water. When the lungs are filled up with water, the victim will turn around in a head-down position. The first position, the head-up, can be seen up to hours after the immersion. And a rule of thumb in Denmark is, that in this situation, an attempt of resuscitation will be performed. Only when the victim is found in head-down position, even the paramedics are allowed to cancel any treatment.

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From several very interesting investigations on animals (dogs and cats) in the laboratory it is shown, that inhalation of fresh water will provoke haemodilution, haemolysis, hyperpotassemia and hypocalcemia. And inhalation of salt water will result in haemoconcentration and possibly lung edema.

But strangely enough these features are not very often seen in cases with humans — and almost never in a considerable degree. The reason for this has not been found. But this means, that the only treatment of interest initially will be artificial ventilation and external cardiac massage: simple first aid to restore cell oxygenation.

Until recently estimation of the body core temperature has only rarely been carried out in a correct manner. But of course cooling has often been a matter of fact by the immersion, leading to a condition of hypothermia. This is important to notice since the prognosis for total resuscitation from immersion *and* hypothermia is much better than normally considered at normothermia.

After hospitalization the person needs very close observation, best in an intensive care unit.

Sequelae after resuscitation can be the well known symptoms after brain anoxia or what is called the post-immersion-syndrome: pneumonia, lung-abscesses or atelectasis. And of course electrolyte disturbances must be corrected if they occur.

Lung edema is probably not as frequent as previously estimated, but it is essential to realize, that it can develop up to 72 hours after the incident and after a period of total well being. Therefore, observation should continue for at least 72 hours in a facility able to control and treat any possible disturbance in the functions of vital organs.

The treatment consist of *cardiopulmonary resuscitation immediately*: this is mouth to mouth first aid started on the scene of the accident, and continued during the ambulance transportation, and in the intensive care unit of the hospital by intermittent positive pressure breathing.

Steps to counteract the possible post-immersion syndrome can be taken according to the rules of the departments and may consist of use of antibiotics and steroids.

If the central temperature is found to be below 30°C and/or the victim is without active muscle movements including shivering, this state of profound hypothermia should be treated with active and rapid central rewarming (see later).

Any observation should be of at least 72 hours to exclude the risk of subsequent pulmonary edema.

It is of value to realize that water is rarely found in the lungs, therefore there should be no waste of time trying to aspirate the lungs.

Furthermore foam production during artificial ventilation is not necessarily a sign of lung edema.

As mentioned electrolyte-changes are rare in humans, but control and treatment of initial acidosis, hyperpotassemia and hypocalcemia is essential. In case of hypothermia the risk of hypopotassemia during the rewarming must be observed.

HEAT REGULATION

As an introduction to the study cold exposure the basic physiology concerning heat regulation will be considered.

First consideration should be given to factors increasing the heat production such as exercise or shivering, imperceptible tension of muscles and the chemical increase of the metabolic rate, this part better called the nonshivering thermogenesis.

And secondly some factors decrease the heat loss, and of those only the shift in blood distribution is of significance in humans.

To start with we shall consider the latter mechanisms, as it is the first thing which happens after cold exposure: the shift in blood distribution.

It is very important to realize that the central part of the body, *the body core* keeps the temperature almost constant at 37°C, whereas the peripheral part, the body shell by changing its blood distribution increases the insulation of the body core against the environment. The change is caused by opening or closing the arteriovenous anastomosis located in hands and feet. The closing is caused by application of cold to the skin surface, or to the heat regulation center in the hypothalamus. The opening of the anastomosis can easily be brought about by heating the body surface or by simple manipulations such as rubbing the skin on hands and feet. The mechanism is under control of noradrenalin acting via the alfa-adrenergic receptors in the vessels.

The protector effect of the arterio-venous anastomosis is tremendous, as body volume by cutting off the thermoregulatory circulation in the extremities is reduced by approximately one third and the surface almost halved. It is important to know, that the opening of the anastomosis is a reflex mechanism.

This means, that if the mechanism is triggered out by applying heat to one extremity, the result will be an opening of the circulation to all four extremities. Hereby relatively warm blood will circulate to the cold extremities, and return in a cooled condition to the body core, resulting in a decrease in body core temperature. This decrease can be up to 5°C within a few seconds and has been responsible for many fatalities during the years, as it seems to be even more harmful to the organism than the initial fall in core temperature. This effect is called the *after-drop*, and the harmful effect the *rewarming collapse*. So, any patient with a body temperature below 30°C, must be protected in order to avoid this to happen.

If this peripheral vasoconstriction is not enough to keep the body core temperature constant at an exposure to a cold environment, the heat production will increase. Depending on the degree of exposure, this will happen in different ways.

If you look at a typical temperature — metabolism — curve of a mouse, you will find its maximal oxygen consumption or total metabolic rate at an exposure to 5°C consisting mainly of a shivering component. The basic metabolic rate found at thermoneutrality at 34°C can be increased for a short period by 5 to 7 folds. But at milder exposures the heat production independent of active muscle movements seen at thermoneutrality, the so called nonshivering thermogenesis, can be expanded by a thermoregulative fraction of this component. It has been found in a study on non-acclimatized, adult humans, that the oxygen consumption can increase by 25 to 40%, that the mechanism probably is mediated by noradrenalin via the beta adrenergic receptors on the cells, that neither adrenalin nor the corticosteroids participate, that it is mainly the fat stores in adipose tissue which act as substrate for this increased energy demand, and that patients with a hypofunction of the thyroid gland are not able to meet the increased demand, as they are not able to lipolize their fat.

This regulatory nonshivering thermogenesis is of importance in the more profound stages of accidental hypothermia, since it is the only part of the heat production left. It can be improved by acclimatization to cold, probably by an improved sensitivity to noradrenalin caused by changes in thyroxin turnover, and it can be completely abolished by administration of beta-receptor blockers.

The more vigorous heat production known as shivering thermogenesis can be mediated by adrenalin, also via the beta-adrenergic receptors, but the fuel substrates

in this particular case are the carbohydrates, which can be depleted almost completely during a stage of pronounced shivering.

ACCIDENTAL HYPOTHERMIA

It is an unexpected decrease of the body core temperature due to exposure to the environment, and developed during a short period of time (some say within 12 hours). It can occur almost everywhere except for tropical areas by immersion into water, which is of particular danger because of the big capacity of water, by exposure to cold wind, especially in combination with rain or snow and by exposure to air in combination with alcohol and/or drug intoxication, a very common situation in the Scandinavian countries.

The typical signs and symptoms presented by the patient as the core temperature falls from normal to 20°C will now be dealt with but it must be born in mind that individual variations do happen such as cardiac arrest at 30°C, and a normal cardiac rhythm with preserved consciousness at 22 C. But *initially* the syndrome is characterized by a vigorous shivering, if excessive drug or alcohol intake has not diminished this possibility of heat production. Very soon the nerve conductivity is impaired, the exposed gets confused and disoriented, and starts tumbling because of a particular rapid cooling of the superficially located nerved on the legs.

Typically, one or two Danish boy scouts are lost every summer in Norway, surprised by cold rainstorms and not properly protected. They quickly start tumbling and falling, and remain sitting on the rocks. Not able to walk and not able to make a proper decision, they become an easy victim for further, fatal cooling. As the core temperature declines further the patient gets unconscious, cardiac arrhythmias can be seen below approximately 33 C and ventricular fibrillation and death can be expected at 26 C.

But as already mentioned, there are considerable individual variations in this picture.

As the shivering period gradually stops somewhere in the beginning of the thirties, the muscles first become stiff and tensioned. But later also this phenomenon is replaced by weakness of the muscles.

The tendon reflexes disappears at an early stage because of the decreased or even abolished nerve conduction, and it is important to know, that the pupils are dilated and without reaction to light stimulus as early in the development as at 32 — 33 C.

Initially the victim by obvious reasons has a sensation of cold, but it is now presumed, that it just before exitus converts this to a curious heat sensation. Many persons are found dead in the snow in Greenland and in the Scandinavian countries in an undressed condition as if they have had a huge feeling of heat. The reason for this is by no means known today, but you should pay attention to this picture, especially if you are involved in a case of forensic medicine, and, of course, especially if the victim is a female.

CLINICAL PICTURE

So, what is to be expected in a patient with accidental hypothermia?

The condition can be divided into several stages, where the first is called the excitation stage going down to 34 — 33°C. It is characterized by the intense peripheral vasoconstriction, if this — as afore mentioned — is not abolished by previous alcohol or drug intake, and by a vigorous shivering.

If this stage is passed, the adynamic — paralytic — stuporous stage will develop gradually, where the respiratory and heart function initially still is increased after the stage with increased metabolic rate and oxygen consumption. But the nerve conductivity is already from the beginning impaired and makes its manifestations in the reflexes, muscle strength, degree of consciousness, and cardiac function.

Ultimately, you will meet a patient with cold skin, low body core temperature (below the range of the thermometers normally used in clinical care), stiff muscles and joints (simulating rigor mortis), a very slow and shallow type of respiration, a similar peripheral pulse, often impossible to palpate, a sometimes unmeasurably blood pressure, inaudible heart sounds by stethoscopy, no reflexes including that of the dilated pupil, and an ECG with slow rhythm, broad complexes, multifocal extrasystoles, and atrial flutter, all together easily misinterpreted as electrical artefact.

If you enlarge the cardiogram a tittle bit, the patients often look dead — and according to the literature, many cold patients in this stage have been declared as dead. So it happened in Sweden in 1969, but as the police a couple of hours later arrived to the hospital to identify the victim, they saw, that she was now breathing and moving her extremities after this period of slow, passive rewarming. The physician was prosecuted by the Swedish National Health Service, but got free, as the jury composed by some medical professors from the university hospitals agreed, that the condition or disease was not too well known — and especially, that no education in the medical school had been given so far. It should be noted that this has not been changed since!

If you realize, that the oxygen consumption of the tissue cells including the brain is decreased to 50 % of normal at 30 C and to 20 % of normal at 20 C, a possible recovery of normal brain function from anoxia can consequently be prolonged to 10 minutes at 30 C and to *at least* 25 minutes at 20 C.

Therefore, the normal criteria of death are not valid for patients suffering from profound accidental hypothermia. They can not be declared as dead before resuscitation, including *rewarming*, has been attempted — and failed.

TREATMENT

Before any treatment is started, one should know, if the patient is hypothermic, and to what *extent*. This is of course an important matter, as it is very often forgotten. Physicians are still more concerned about the temperature of a child with pains in the right ear, than about a dripping-wet drowned person. And, measuring the temperature, please use a suitable thermometer. Should be used in Denmark the normally used thermometer has its lowest range at 34 C, and how many times is the temperature of drowned victims, not recorded to be 34 C, who then after 15 to 30 minutes attempt of cardio-pulmonary resuscitation are send to the morgue. The thermometer should be able to measure down to the freezing point, and it should be placed correctly. If you use the rectum (which is the most frequent method in Europe), you must exceed at least 15 cm or 6 inches up in the bowel to have a proper indication of the core temperature. The mouth temperature is correct if the thermocouple is placed under the tongue, and the mouth is kept closed. The localization of choice is in the esophagus back the heart *or* — and this is often more easy to apply — on the tympanic membrane, the ear drum.

If the patient is still in the exitation stage, it will suffice to apply nursing and an IV with glucose, as the blood sugar level and the glycogen depots in liver and muscles as already mentioned may be depleted after the period of severe shivering. If the core temperature rises above normal without obvious clinical reason, not too much attention

should be paid to that, as it is a normal physiological reaction from the temperature regulation center.

But if the patient already is in the more profound stage of hypothermia, the choice of treatment should be done very carefully. This estimation of the degree of hypothermia can be done by use of a thermometer, but as individual variations as already said are very common, the clinical picture of the patient is more valuable. So, if the patient has a temperature around or less than 30 C, or/and he is unconscious and without shivering or another form of muscle activity, you should consider the hypothermia to be profound.

Here, rapid rewarming is recommended as the treatment of choice. Many modalities have through the years been used to accomplish this, including immersion in hot water baths (this will be soon discussed), application of heated materials to the skin surface, and circulation of warm water through special garments fitted to the victim. However, these methods of peripheral surface rewarming are variable in their efficiency, and may harm the patient, as the decreased circulation in the skin may cause burns, when the patient is placed on e. g. a normal electrical heated blanket. This was the only sequelae at one of the patients in the Copenhagen area, centrally rewarmed from 26 C, but initially placed on a heated blanket.

But what is even more important — because it is the keypoints to the survival of the patients — is to remember the pathophysiologic changes which occur in accidental hypothermia. You remember that the body in cold environment tries to keep its core temperature constant by the change in core — shell relationship by obtaining a large difference in temperature between the two zones. And, that simple moving or manipulating the patient and local application of heat to the extremities will brake this peripheral vasoconstriction, resulting in the after-drop in core temperature.

This is one cause of death during treatment.

But furthermore it is also important to realize, that plasmaelectrolytes usually are normal when the treatment starts, but during the last phase of the rewarming, a severe hypopotassemia is often provoked. An explanation of this secondary hypopotassemia, which has been observed in almost all the cases of hypothermia in our department, can not so far be given. It *may* be a delayed stress-reaction of the pancreas with an increased outflow of insulin, which, in combination with the glucose-infusion, transport the potassium into the cells in an increased rate. But it can be so pronounced, that it may well be responsible for some of the deaths, which are reported after the active rewarming has stopped and the body core temperature has reached almost normal values.

So, considering the treatment of profound hypothermia, it should be a form of rapid rewarming, where opening of the arterio venous anastomosis and hypopotassemia is avoided.

To obviate these problems it is desirable that treatment fulfil the following criteria:

1. rapid rewarming using fluids because of the large heat capacity of water.
2. central rewarming to avoid after-drop and rewarming-collapse.
3. rapid correction of electrolytes during rewarming.
4. elimination of toxic agents as many patients are hypothermic because of drug overdose.
5. possibility for concurrent symptomatic treatment as artificial ventilation, external cardiac massage, defibrillation etc., and
6. the treatment must not per se pose severe risk to the patient or to the personel.

One of the methods used is by no means central, but as it recently has been adopted as the treatment of choice at the Navy STANAG it will be discussed here:

The hot water bath has been recommended long ago in order to avoid the after-drip, the extremities should previously be kept out of the water. But studies performed on normo- and slightly hypothermic persons have shown, that if the temperature of the water is high enough (40 — 42°C), the superficial veins of the extremities will act as a heat exchanger from the ambient, hot water to the relatively colder body core. So, today the Navy STANAG recommends immersion of the body *and* the extremities, just leaving the mouth and nose out of water. But as this theory requires a minimum of circulation, we are not too convinced about its efficacy, a statement, we will later come back to. Furthermore, it can be assured, that it is difficult to carry out, in practice.

Back to the methods of proper central rewarming:

One is to make the patient breath *hot moist air or oxygen*. The investigations behind this theory are all carried out on normal persons who after immersion dropped to 35°C in central temperature, and this is not a hypothermia asking for active treatment. The methods can be used e. g. under transportation, but it is not effective for rapid rewarming of profound hypothermia. Furthermore, it does not fulfil the demands concerning correction of electrolytes and elimination of toxic agents during the rewarming.

Another — and ideal way — is to use *extracorporeal circulation*. This has been done successfully, but it is a treatment which demands time for preparation and which is only available in relatively few centers.

The *peritoneal dialysis* is technically simple, using the abdominal cavity with its splanchnic blood area as exchange field as it is known from the nephrology. Normally only one catheter is placed distal to the umbilicus, but using a technique with two catheters and with a slight suction at the outflow, it is possible to obtain a flow of more than 12 liters per hour, which is the optimum for a high urea clearance. This means that also the clearance of calories or transfer of heat to the body core is estimated to be possible at this high flow rate. For a child of 15 kg, a suitable flow is about 1500 cc per hour.

Fig. 1 shows pure calculations on the relationship between the rewarming time and various combinations of fluid-temperature and flow. If we consider a person with a body-weight of 70 kg, his body core to be 60 % of this as he presumed to be cold, — and his mean specific heat factor to be 0.83, you will see, that with 10 liters of dialysis fluid warmed to 42 C it is possible to increase the body temperature from 20 C to more than 26 C in one hour.

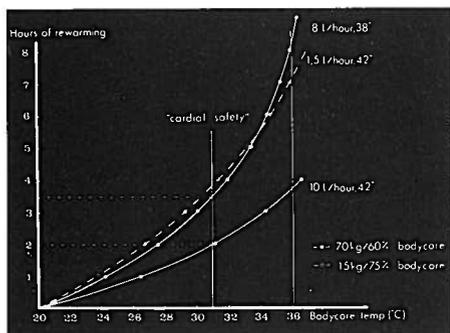


Fig. 1 — Calculations on the relationships between the rewarming time and various combinations of fluid temperature and flow

The figure also indicates that the flow rate is most important up to a core temperature of 31°C (*cardiac safety* because at this temperature cardiac problems are rarely seen). And above 31 C the temperature of the fluid is more important.

These calculations — which do not include neither a heat loss from the body nor an increased heat production during rewarming — show, that individual of all ages can be warmed to a body core temperature above 31 C within a few hours. As it is a central rewarming, collapse, and the risk of hypopotassemia can be prevented so to speak automatically by the use of a potassium concentration in the dialysite fluid corresponding to the normal serum values (4 meq per liter). Furthermore, variations in the content of glucose can be used, as pulmonary edema is a well known risk in cases of hypothermia caused by submersion or drowning within the first 72 hours. This risk can be counteracted by increasing the content of glucose from its normal 1.5 vol%.

It should also be mentioned here, that the protein content of the out-flow fluid is normally 0.5 to 1 gram per liter, mainly consisting of albumin. Therefore, a plasma-expander is estimated to be useful during the treatment, human plasma albumin should be the choice. And finally, since accidental hypothermia in many cases is caused by overdose, it should be mentioned briefly, that peritoneal dialyses for several years has been used to treat especially drug overdose with barbiturates, salicylates and glutethimides.

Now, to prove that these thoughts and calculations not only are a matter of speculation, some examples of cases, treated in the Copenhagen area will be next presented.

Case report 1 (Fig. 2)

A 30 year old female was found in the harbour of Copenhagen, immersed in 4-5°C cold water. She was declared death at the scene and no attempts of resuscitation were done during the transport. The rectal temperature was 34°C (measured by a thermometer with the lowest point of its range at 34°C!). However, at admission to the hospital it was suspected that the core temperature was lower than that and measurements made with an electrothermometer in the esophagus and on the tympanic membrane showed that the core temperature was 23,4°C. ECG showed asystole. External cardiac massage and artificial ventilation were commenced, and the patient was at the same time placed in a bath tub with 40°C hot water. 1 mg adrenalin i.v. had no effect on the asystole.

After 2 1/4 hours treatment had only increased the esophagus temperature to 24°C; it was therefore decided to start peritoneal dialyses, and with two catheters and a fluidflow of five liters per hour at 40°C, it was possible to raise the temperature of the body core to 30°C, within another two hours and 20 minutes.

At approximately 28°C insufficient spontaneous respiratory movements were observed, the pupils reacted to light and the patient began to move head and extremities. Occasional ventricular complexes were seen on the oscilloscope following a second dose of adrenalin. Despite repeated attempts at remedial therapy extending over one hour the cardiac action alternated between ventricular fibrillation and asystole, and further attempts at resuscitation were abandoned.

Lessons learned from this particular case are, that during immersion into a hot bath, the rectum is not a suitable place for temperature measurement, as you have a sphincter paralysis with water in the bowel and faeces in the water.

Further, it seems as if the whole body immersion is insufficient when the circulation is impaired, and that peritoneal dialysis in this case a better means of rewarming. And again:

The first physician did not think of the possible hypothermic condition, and used a wrong thermometer!

Although this patient did not survive, the apparent efficiency at rewarming by peritoneal dialysis gave rise to an optimism, which was confirmed in subsequent cases in the department. In other hospitals in the Copenhagen area the recommendations were followed successfully, and two remarkable cases, demonstrate the results which can be obtained using of peritoneal dialysis in profound accidental hypothermia:

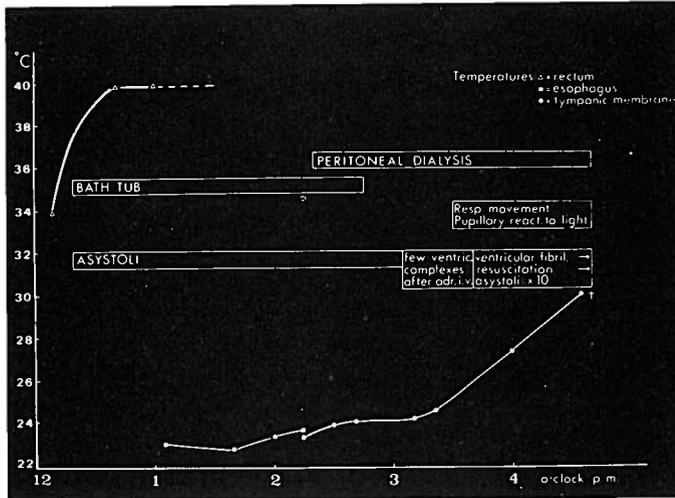


Fig. 2 — Case 1. Course of events during treatment

Case II (Fig. 3)

A 4 3/4 year old boy was admitted to hospital after a submersion of approximate 10 minutes in a lake (water temperature: 2-3°C). In spite of CPR done by a police officer and later by the ambulance people during the transport, he was without respiration on arrival at hospital. The pupils were dilated and without reaction to light. ECG showed asystole.

The rectal temperature was measured to 24°C.

Resuscitation was commenced with external cardiac massage and artificial respiration, and intracardiac adrenalin was administered converting the asystole to atrial fibrillation. After 15 minutes a blood pressure of 45 mmHg was recorded. Peritoneal dialysis was initiated 70 minutes after admission. 9,2 liters of fluid at 38-40°C was passed through a single catheter during a period of 3 1/2 hours. The temperature measured in the esophagus increased to 30°C, the atrial fibrillation converted spontaneously to a regular sinus rhythm and the blood pressure rose to 90 mmHg. 4 hours after admission the pupils reacted to light and after further 11 hours spontaneous movements of the extremities were observed. There were no signs of pulmonary edema or hemolysis, and the serumpotassium remained normal during the rewarming period. The patient was kept in a ventilator for 72 hours at a core temperature of 32°C (regulated by the peritoneal dialysis) in an attempt to counteract any sequelae after the brain anoxia.

After stopping the treatment, he woke up, but had difficulties in talking and walking during the first week. After four weeks all sequelae had disappeared and there was a complete recovery. A physical and psychological check up one year after the accident verified this total recovery.

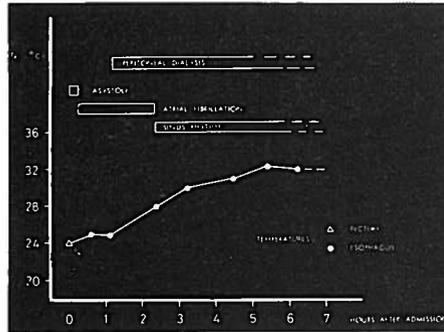


Fig. 3 — Case II. Course of events during treatment

Case III (Fig. 4)

In the same hospital, one week after the incident described in case II, a 6 year old boy was admitted with asystole, dilated pupils and without respiration after a 15-20 minutes submersion in fresh water at 2-3°C.

His rectal temperature was 21°C.

Peritoneal dialysis was started at once accompanied by normal procedures for resuscitation at a cardiac arrest. One hour after the administration of 4,4 liters fluid at 40°C the esophagus temperature had increased to 29°C. When the core temperature reached 27°C, a regular sinus rhythm appeared spontaneously on the cardioscope, and the blood pressure was 120 mmHg. The pupils reacted to light after 8-10 hours. X-ray of the chest showed initially signs of pulmonary edema. The serum-potassium remained normal during the rewarming.

The patient was, like case II, ventilated for 72 hours at a core temperature of 32°C, and after extubation he was at once fully awake without any neurological abnormalities — and remembered details of the incident!

Subsequent controls up to one year later confirmed that in this case also the recovery was complete.

These cases are chosen between several similar successfully examples of a complete recovery from profound accidental hypothermia.

The peritoneal dialysis is a routine procedure in most modern hospitals, it has few contraindications, complications are few and exceptional, it can be started rapidly and easily, and it fulfils all the previously mentioned criteria for the treatment. It is now the treatment of choice in the Copenhagen area, incl. the Poisoning Control Center, and can hereby strongly be recommended.

ADJUNCT TREATMENT

Now, how should the patient be handled during the transport until he reaches the hospital:

First of all, remember, that when the core temperature is already low, the patient

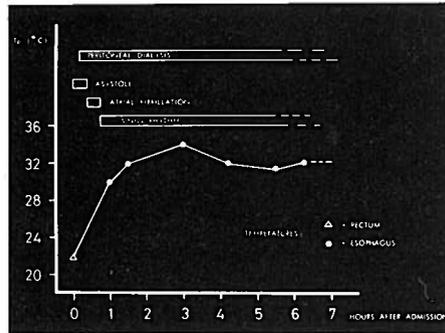


Fig. 4—Case III. Course of events during treatment

is somehow protected. So, he should be kept at this low temperature, but a further drop should be prevented. Therefore, be gentle, cutting his wet clothes off and wrapping the patient in NON HEATED blankets to insulate him against heat of the surroundings (cut off the heater in the ambulance or helicopter).

Never the technique of the eskimoes by rubbing the extremities should be used. This will promptly open the anastomoses and contribute to an after drop in core temperature. This has as mentioned killed many unnecessarily.

Make sure, that the patient is well oxygenated. Ventilate him, best with pure oxygen, and even better with hot, moist air or oxygen. If necessary, external cardiac massage, must be performed but this is a matter of discussion. Our friends from the Royal British Navy are a little afraid of this, and have a study going on.

But as anesthesiologists we still recommend it.

But, there is no discussion about the fact, that defibrillation on a cold heart has no effect and therefore no meaning. Furthermore, most often the cardiac function will regulate itself automatically when the temperature of the heart-tissue comes up.

No beta blocking treatment should be given as an antiarrhythmicum. It will block the only heat production eventually still going on in the profound stage. Also this has been a clinical lesson learned.

As the patient has depleted his oxygen reserves by the shivering, iv-glucose should be given initially, eventually followed by human albumin. All iv's should be centrally lacated, and the fluids prewarmed.

PROPHYLAXIS

A very few words about the prophylaxis:

Education is here the key word and include knowledge. Teach about the syndrome, and explanation of what happens by acute immersion, so that victims can avoid the very dangerous state of panic. They should be taught to stay calm in water, not to swim, as movements in the water will increase the heat loss more, than it increases the heat production. That they must keep their cloths on, because even wet cloths give some insulation between the skin and the water.

They ought to know teach them how to be prepared when walking in the rural areas. They must carry wind-and waterproof garment, which they rapidly and easily can use. And to avoid to be wet in the snow, either by the snow itself or by sweat in water. Combined Intelligence Objectives Subcommittee, Item 24. Washington DC. Office of the Publication Board, Department of Commerce. Report N.º 250. training.

RESUMO

Depois de rever os mecanismos de morte por submersão ou *afogamento*, o A. realça que a inalação de água não é nunca o factor importante no homem, e que a vida se mantém por muitas horas graças à hipotermia verificada. Deste modo os sinais aparentes de morte não têm significado e devem ser reanimados todos os afogados encontrados ainda de cabeça para cima. A faceta fundamental desta reanimação, depois das manobras de socorrismo imediato, consiste exactamente nos cuidados em restabelecer temperatura central do corpo em condições, evitando a abertura das anastomoses arteriovenosas periféricas e a hipocaliémia secundária.

A diálise peritoneal, nas condições que se descrevem, é o meio mais eficiente para obter tais resultados.

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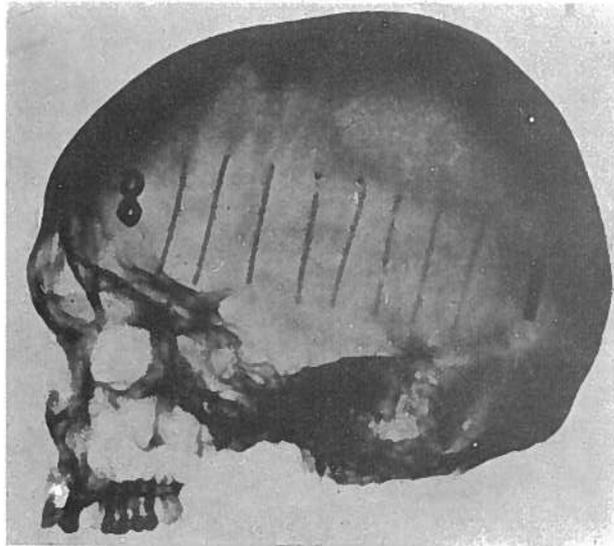


Fig. 1. — Opacité du bromure de strontium de 10 % (1) à 80 % (8).

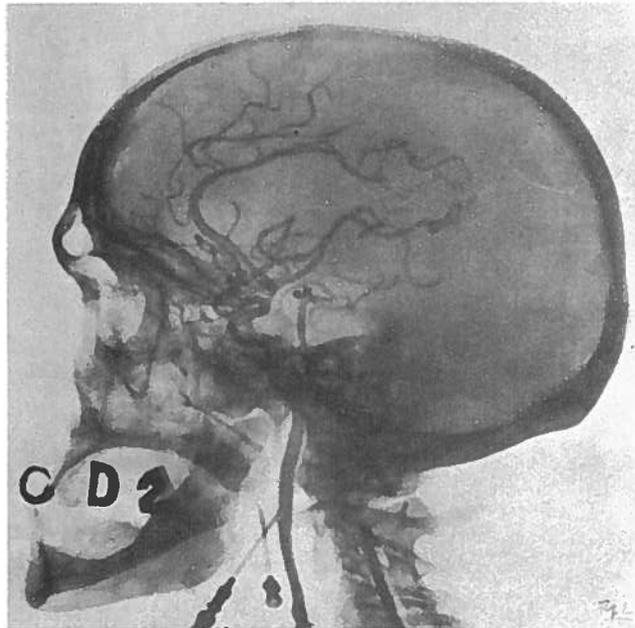


Fig. 2. — Réseau artériel dérivé de la carotide interne. Injection de NaI à 30 %.