ACCIDENTAL PROFOUND HYPOTHERMIA

Case Report

BY

SIV ANDERSON, BENGT G. HERRBRING AND BERTIL WIDMAN

SUMMARY

A 3-year-old boy who had been exposed for at least 15 hours in an air temperature of approximately 0°C, was found to have a rectal temperature of 17°C about 20 minutes after he arrived at the hospital. He was rewarmed to 34°C in a bath of water at a temperature of 37-38°C during a period of 2 hours 15 minutes. He was also given large amounts of intravenous bicarbonate at the same time. Complications during rewarming consisted of transient arrhythmias, and the insignificant neurological changes disappeared after approximately 2 months. At examination 6 months after the exposure no residual defects could be demonstrated.

Hypothermia is defined as a condition in which the body temperature is lower than 35°C. Controlled hypothermia has been used for many years in certain branches of surgery. It has been shown that it is possible to lower the central body temperature as far as 4.2°C (Kugelberg et al., 1967) without injuring the patient. However, in accidental hypothermia the patient is not prepared for the lowering of body temperature and is thus exposed to greater risks. In most of the cases described in the literature accidental hypothermia was associated with another illness or intoxication. Accidental hypothermia also commonly occurs in association with shipwreck and drowning. Patients who, due to exhaustion or injury, have been exposed to low ambient temperatures and found in time to resuscitate, are rare. The case which is presented here had one of the lowest body temperatures which have been recorded. In spite of this the patient survived without any residual defects.

CASE REPORT

A previously completely healthy 3-year-old boy disappeared from his home at 11 a.m. on December 17, 1968. He was found about 5 km from his home just before 10 a.m. the following day.

At this time of the year sunset is about 2-45 p.m. One can assume that he must have fallen asleep from exhaustion only a couple of hours after dusk and therefore was exposed to a cold atmosphere for at least 15 hours. The air temperature during the night was about 0°C. There was hardly any wind but a slight snow fall. The boy wore warm clothes, a hat and gloves.

When he was brought into Karlskoga Hospital there was no apparent sign of life. The skin was pale grey, ice cold, and there were no heart sounds. There was no respiration and the pupils were maximally dilated. The electrocardiogram showed very irregular, slow activity with a frequency of approximately 8-10 complexes per minute. The rectal temperature recorded about 20 minutes after arrival at the hospital was 17°C measured by a laboratory thermometer. The patient was immediately intubated, the lungs ventilated with 100 per cent oxygen and external heart massage was performed. A venous cut-down was done and 0.6 molar bicarbonate was given rapidly intravenously. Due to the extreme peripheral vasoconstriction blood-gas analysis was not taken at that time.

After about half an hour following arrival at the hospital the patient was placed in a bath of warm water with a temperature of 37-38°C. The ECG was continuously monitored during the warming-up period. After 1 hour’s warming the temperature was 24°C and the ECG showed atrial fibrillation with a ventricular frequency of about 40 beats/min. By this time he had received 140 m.equiv of sodium bicarbonate and blood-gas analysis showed: arterial pH 7.425; Pco2, 54 mm Hg; standard bicarbonate 30 m.equiv/l., whereupon the bicarbonate infusion was changed to 5.5 per cent glucose. The patient now began to move and to show signs of wakefulness and in order to prevent shivering, he was anaesthetized with nitrous oxide-oxygen 3:2 together with halothane 1-2 per cent. It was not necessary to use muscle relaxants.

After 2 hours 10 minutes of warming the temperature was 33°C and blood-gas analysis showed: pH 7.345; Pco2, 54 mm Hg; standard bicarbonate 25 m.equiv/l. The ECG now showed sinus rhythm with a frequency of approximate 100 beats/min. At this point warming and anaesthesia were discontinued. During the

SIV ANDERSON, M.D., Dept. of Int. Med., Karlskoga Hospital; BENGT G. HERRBRING, M.D., Dept. of Anaesthesia, Karlskoga Hospital; BERTIL WIDMAN, M.D., Dept. of Anaesthesia, Central Hospital, Örebro, Sweden.
warming period there were several episodes of arrhythmia which responded to short periods of external heart massage. Soon afterwards the patient began to react to painful stimuli but was not otherwise rousable. About 15 minutes later the systolic blood pressure was 90 mm Hg but his skin-colour was still pale. At that time he was transferred to the regional hospital in Örebro for continued treatment.

In the next few days he developed bronchitis with a temperature rising to 38.5°C, and he was treated with penicillin. He was tired and slept for most of the day. There was some swelling of the hands and feet, and an area of pressure necrosis about 3 cm in diameter developed on the inside of his right knee. He refused to walk and to grip the hand but his condition was such that he was discharged home after 5 days. During the next 2 months he had some difficulties in walking, was abnormally tired and had difficulty with hand-grip. His condition progressively improved with physiotherapy. A neurological examination 6 months after the accident showed no changes and the e.e.g. was normal. There were no detectable mental changes. Twelve months after the accident the boy is quite healthy.

**DISCUSSION**

In the cat it has been shown that spontaneous recovery can take place following a drop of body temperature to 19°C but not from 16°C. Dogs under hypothermia die when the body temperature reaches approximately 17°C (Zingg, 1966). In man accidental hypothermia with a temperature falling to 34–35°C is associated with a mortality of 30 per cent (Leading article, 1966). Newborn babies and animals tolerate a lower body temperature better than adults, but it is not known for how long this protection remains in man (Zingg, 1967). According to Kugelberg and associates (1967) the mortality is 100 per cent in older patients if the body temperature is lower than 28°C. There are a few cases described following accidental hypothermia with a temperature of 18–23°C (Lash, Burdette and Ozdil, 1967; Kugelberg et al., 1967; Fell et al., 1968). Most of these patients were intoxicated and presumably the prognosis is more favourable (Lash, Burdette and Ozdil, 1967). The lowest temperature so far recorded on a surviving patient is 18°C (Laufman, 1951). The prognosis following accidental hypothermia is worse if the patient is exposed to damp together with the low temperature (Hunter, 1968; Pugh, 1966).

Warming of the hypothermic patient presents a major problem. In hypothermia there is a large temperature difference between the body core and the peripheral parts where there is marked vasoconstriction and reduced blood flow. With external warming the peripheral vessels are dilated thus leading to a risk for rewarming shock (Davies, Millar and Miller, 1967; Exton-Smith, 1968; Kugelberg et al., 1967; Zingg, 1966). Previously only external warming has been used. This could be effected rapidly by placing the patient in a warm bath or slowly by allowing the body temperature to rise spontaneously. Recently good results have been reported using extracorporeal circulation (Davies, Millar and Miller, 1967; Kugelberg et al., 1967) or peritoneal dialysis (Lash, Burdette and Ozdil, 1967). Warming of the heart and mediastinum with warm solutions following thoracotomy has also been described. The longer the patient has been exposed to a low temperature, the slower should be the rewarming process (Fell et al., 1968; Gjemdal, 1966; Mathews, 1967). Rapid rewarming is considerably more dangerous in older patients than in the young (Exton-Smith, 1968; Mathews, 1967). In children, with their small body mass in relation to a relatively large body area, the temperature difference between the periphery and the centre of the body is smaller than in an adult, thus reducing the risk of rewarming shock. The difference in temperature is more quickly evened out. The most physiological method of rewarming is from the inside (Lash, Burdette and Ozdil, 1967; Zingg, 1966), e.g. extracorporeal circulation, but this is only possible in a few hospitals. As our case shows, rapid external rewarming is possible in children, whereas it would seem that peritoneal dialysis should be the normal method with adults. If shock occurs during the rewarming this should be treated as hypovolemic shock.

The hypothermic patient develops a marked metabolic acidosis, due partly to the tissue hypoxia and partly to depletion of the patient's glycogen reserve (Kugelberg et al., 1967; McNicol, 1967; Phillipson and Herbert, 1967). This can be treated by intravenous injection of glucose and bicarbonate or THAM-buffer solution. The hypothermic patient suffers also a reduction in lung function, due to the known metabolic changes, together with disturbances of the central respiratory centres, changes in the solubility of gases and their transport in the blood, together with changes in the mechanics of breath-
ing. Presumably the successful result in our case is due in no small part to the immediate massive bicarbonate transfusion, together with controlled ventilation with 100 per cent oxygen.

With a reduced temperature the heart rate falls and the so-called J-waves can sometimes be observed. With rewarming of the hypothermic patients, arrhythmias and ventricular fibrillation are feared complications. The e.c.g. is characterized by different types of heart block, increase in the PR interval and lengthening of the intraventricular conduction time. Before our patient had been rewarmed to about 33 °C several types of arrhythmias occurred which were treated by external cardiac massage. If ventricular fibrillation occurs defibrillation cannot be effective until the patient’s temperature has reached at least 28 °C (Kugelberg et al., 1967). There were no residual e.c.g. changes in our case.

Complications in the form of renal damage (Fell et al., 1968; Gjemdal, 1966; Kugelberg et al., 1967), electrolyte changes (Gjemdal, 1966), frostbite (Kugelberg et al., 1967), together with mental changes and nerve damage (Gjemdal, 1966; Fell et al., 1968; Phillipson and Herbert, 1967) are described in the literature following accidental hypothermia. In our case only minor frostbite, together with transient difficulties in handgrip strength and walking, were observed.

REFERENCES


HYPOTHERMIE PROFONE ACCIDENTELLE: DESCRIPTION D'UN CAS

SOMMAIRE
On observe chez un garçon de trois ans, exposé durant au moins 15 heures à une température d'air d'approximatively 0 °C, une température rectale de 17 °C environ vingt minutes après son arrivée à l'hôpital. Il a été réchauffé à 34 °C dans un bain de 37-38 °C durant une période de 2 heures et 15 minutes. Il reçut simultanément de grandes quantités de bicarbonate par voie intraveineuse. Le réchauffement se compliqua par des arrhythmies passagères et les troubles neurologiques insignifiants disparurent après environ deux mois. On ne trouva aucun trouble résiduel à l'examen six mois après l'accident.

AKZIDENTELLE STARKE HYPOTHERMIE: EIN FALLBERICHT

ZUSAMMENFASSUNG
Sir,—The complication of sustained generalized contraction of skeletal muscles occurring in patients following induction of anaesthesia and the subsequent administration of suxamethonium has been well documented. The case to be described is that of a patient thought to be suffering from polymyositis who developed a hypertonic response following suxamethonium injection.

Case report.

A woman aged 34 years had, over a period of 3 months, developed marked and progressive muscular weakness associated with muscle tenderness. During the last 3 weeks of this period she had been totally bedridden and had developed some difficulty in speech.

On examination she was found to have myopathic facies and the skin of the face had a slight lilac tinge. She had weakness of both lungs with oxygen on the right side than on the left. Myotonia was not present clinically. She had a slight degree of exophthalmos. The thyroid gland was not palpable and there were no hand or finger tremors. X-ray of the chest showed an opacity in the right lower lobe. The blood pressure was 125/80 mm Hg. The cerebrospinal fluid was at a pressure of 160 mm H₂O; its protein content was raised, being 150 mg per cent; its cell content was normal.

The patient was scheduled for left carotid angiography in order to exclude intracranial pathology. Anaesthesia was induced with thiopentone 250 mg followed by suxamethonium 100 mg. The patient then developed a sustained generalized contraction of skeletal muscles; the arms became rigid (the elbows and wrists were partially flexed and the fingers hyperextended) and the muscles of the neck were markedly contracted. A pronounced brawny swelling appeared beneath the mandible. A striking patchy cyanotic mottling affected the face and neck. The radial and carotid pulses were not palpable. It was possible, however, to intubate the patient and to ventilate the lungs with oxygen; thereafter there was a gradual return of the radial pulses. The cyanotic mottling persisted. The muscular rigidity lasted approximately 4 minutes, after which it diminished considerably and voluntary breathing commenced. Following the return of voluntary breathing the pulse volume returned to normal, the brawny swelling beneath the mandible disappeared and the cyanotic mottling, although still present, gradually became less obvious.

Anaesthesia was continued for 40 minutes using 50 per cent nitrous oxide in oxygen supplemented with halothane 1–2 per cent, and left carotid angiography was carried out. Towards the end of the procedure the patient's skin became very hot and flushed and the pulse rate rose to over 160 beats/min. The hypertonic state of the patient's circulation was confirmed by the appearance of the cerebral angiograms. Following extubation the patient made an uneventful recovery from the anaesthetic.

The angiographic studies did not reveal any intracranial pathology and routine biochemical studies were normal. The administration of suxamethonium had been followed by a marked creatinuria of 600 mg/24 hours and a slight increase in the serum level of protein-bound iodine.

Five days after the administration of the anaesthetic the patient developed some difficulty in coughing and in taking deep breaths. The following day, immediately after having had an X-ray of the chest carried out in the ward, the patient suddenly collapsed and died.

HYPERTONIC SYNDROME ASSOCIATED WITH SUXAMETHONIUM ADMINISTRATION

It was ascertained that on two previous occasions (9 years and 4 years before) suxamethonium had been given to the patient without any untoward effects having been noted.

Comments.

The patient was undoubtedly suffering from a disease process affecting the skeletal muscles but as muscle biopsy and postmortem examination were not carried out, the exact nature of this disease process is not absolutely certain. A diagnosis of polymyositis is, however, strongly suggested by the history of recent progressive muscular weakness with some muscle tenderness, the lilac erythema affecting the face and the marked creatinuria.

Muscle rigidity during anaesthesia in a patient suffering from polymyositis has been reported previously (Saidman et al., 1964). The patient in question developed an increasing "stiffness" of the abdominal and jaw muscles, and later developed a progressive hyperthermia, death occurring on the 5th postoperative day. In this case no suxamethonium had been administered. The general anaesthetic agents used had been nitrous oxide, halothane and ether.

In the case described the generalized contraction of skeletal muscles which occurred after induction of anaesthesia and which diminished after approximately 4 minutes, is likely to have been causally related to the administration of suxamethonium. The brawny swelling appearing beneath the mandible was undoubtedly due to contraction of the muscles of the neck (the elbows and wrists were partially flexed and the fingers hyperextended) and the muscles of the neck were markedly contracted. A pronounced brawny swelling appeared beneath the mandible. A striking patchy cyanotic mottling affected the face and neck. The radial and carotid pulses were not palpable. It was possible, however, to intubate the patient and to ventilate the lungs with oxygen; thereupon there was a gradual return of the radial pulses. The cyanotic mottling persisted. The muscular rigidity lasted approximately 4 minutes, after which it diminished considerably and voluntary breathing commenced. Following the return of voluntary breathing the pulse volume returned to normal, the brawny swelling beneath the mandible disappeared and the cyanotic mottling, although still present, gradually became less obvious.

Anaesthesia was continued for 40 minutes using 50 per cent nitrous oxide in oxygen supplemented with halothane 1–2 per cent, and left carotid angiography was carried out. Towards the end of the procedure the patient's skin became very hot and flushed and the pulse rate rose to over 160 beats/min. The hypertonic state of the patient's circulation was confirmed by the appearance of the cerebral angiograms. Following extubation the patient made an uneventful recovery from the anaesthetic.

The angiographic studies did not reveal any intracranial pathology and routine biochemical studies were normal. The administration of suxamethonium had been followed by a marked creatinuria of 600 mg/24 hours and a slight increase in the serum level of protein-bound iodine.

Five days after the administration of the anaesthetic the patient developed some difficulty in coughing and in taking deep breaths. The following day, immediately after having had an X-ray of the chest carried out in the ward, the patient suddenly collapsed and died.