are small, however, and a larger study would be necessary to confirm this observation.

It has been suggested that a high intake of fibre can cause increased faecal bile acid excretion and hence decrease serum cholesterol concentration (Trowell, 1972). However, all the evidence does not support this hypothesis as is shown in the review by Cummings (1973). In this present study increased dietary fibre was not associated with any change in serum cholesterol concentration.

The association of dietary fibre and diverticular disease is relatively well established (Painter, 1968, 1969; Painter and Burkitt, 1971). It would appear from the results of the present study that the possible therapeutic effect of bran or cellulose in preventing or treating diverticular disease is largely due to the ability of these substances to increase stool bulk. From the data presented it is calculated that an 80-kg man passing 100 g stool per day would excrete faeces equivalent to his body weight about every two years. By the ingestion of 16 g of bran or cellulose daily this output would be achieved in only 12 months.

Significant alteration in colonic work load, intestinal function, and, possibly, in susceptibility of the bowel to pathological processes may be achieved by relatively minor alterations in dietary habit.

This study would not have been possible but for the co-operation of Messrs. Morgan, Karakoulis, McNicol, and Wilson. We are grateful to Dr. I. W. Percy-Robb in the clinical chemistry department for serum cholesterol estimations.

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MEDICAL MEMORANDA

Recovery from Profound Hypothermia with Cardiac Arrest after Immersion

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Hypothermia after prolonged immersion in cold water has been reported before (Kvittingen and Naess, 1963; Keatinge, 1965; Cahill, 1968). In the case we report here we believe that the low body temperature protected the brain against anoxic injury from a prolonged circulatory arrest.

Case Report

The patient, a boy aged 16 months, was brought to hospital at 5.40 p.m. on 6 February, 1973 after being found about eight minutes earlier, completely dressed, submerged face-down in a swimming pool at a nearby house. When rescued he had seemed dead and did not respond to mouth-to-mouth breathing carried out by his mother. She did not do cardiac massage. The air temperature at 6 a.m. had been 0·6°C and at 6 p.m. it was 11·4°C.

On admission the child was apnoeic, pulseless, and cyanotic. His pupils were dilated, his abdomen bloated, and his body very cold to touch. Mouth-to-mouth breathing together with closed chest massage were instituted immediately. During endotracheal intubation he vomited large amounts and inhalation of gastric contents was suspected. Sodium bicarbonate 80 mEq/l. was given intravenously and a drip infusion of 200 ml of 1/6 molar sodium bicarbonate was started. Asystole was replaced by ventricular fibrillation after intracardiac administration of epinephrine 1 mg. A D.C. shock of 200 watt seconds at 5.55 p.m. did not stop the fibrillation, but after a second shock five minutes later slow contractions of a supraventricular type were seen on the electrocardiogram. This was 30 minutes after the body had been taken from the water. No blood pressure could be recorded and norepinephrine 0·1 mg in 50 ml of 5% dextrose in water was administered. Positive-pressure ventilation with air and oxygen and external cardiac massage were maintained throughout resuscitation. No rales were heard in the lungs at any time. A blood sample obtained before intubation showed profound acidaemia (see table).

By 6.30 p.m. sinus rhythm was established with a pulse rate of 90/min and a systolic blood pressure of 90 mm Hg. The pupils were constricted and reacted to light. At 7.30 p.m. the rectal temperature was 27·8°C, the first accurate measurement obtained. Hypothermia persisted for seven hours. Twelve hours after admission the temperature was within the normal range (see chart). Spontaneous respiratory movements were noted at about 9.30 p.m., but the child remained comatose and unresponsive to painful stimuli. The pupils varied in size and responded sluggishly to light during the night. An *x*-ray film taken at 11.30 p.m. showed clear lung fields and a normal-sized heart. No free plasma haemoglobin was found. At 2 a.m. the serum sodium was 140 and serum chloride 107 mEq/l. Urine glucose was positive (+). The child moved spontaneously at about 4 a.m. The rectal temperature was then above 35°C. By 6 a.m. it had risen to 37·3°C, and he was able to breathe efficiently by himself.

No neurological deficit was found on examination on the day after admission and an electroencephalogram was within normal limits. Other findings were: haemoglobin 10·6 g/100 ml, serum sodium 138 mEq/l., serum potassium 4·1 mEq/l., serum chloride 99 mEq/l., blood urea 63 mg/100 ml, serum creatinine 0·9 mg/100 ml, urine osmolality 485 mOsm/l., urine sodium 49 mEq/l., and urine chloride 32 mEq/l. The urea concentration in urine was 8·8 mg/100 ml and the white cell count 16,600/mm³. Hyperthermia up to 38·8°C was observed over the next 72 hours. The chest *x*-ray films remained clear. Subsequent progress was uneventful and the child was discharged after 14 days.

Comment

The hypothermia in this case was probably due to prolonged

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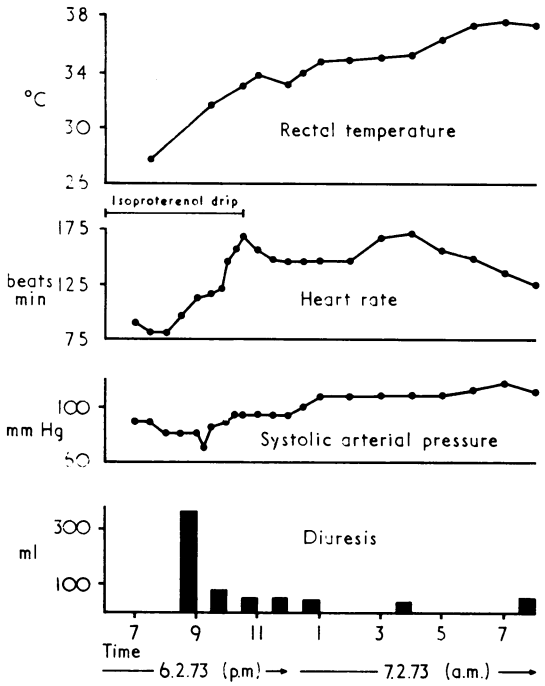
Blood Gases and Serum Potassium Concentration related to Rectal Temperature. Correction for Body Temperatures is needed for Accurate Evaluation of Acid-base Status at Low Temperature

Time:	5.55 p.m.	7.30 p.m.	9.30 p.m.	11.30 p.m.	2 a.m.	5 a.m.	6 a.m.
Sample	—	venous	arterial	arterial	arterial	arterial	arterial
Ventilation	manual*	controlled†	controlled	controlled	controlled	controlled	spontaneous‡
F_iO_2		0·6	0·6	0·45	0·45	0·45	½ l./min O_2
Po_2§ (mm Hg)	—	—	230	160	—	110	—
			(147)	(112)		(102)	
pH§ (units)	6·92	7·33	7·39	7·38	7·40	7·42	7·42
	(—)	(7·48)	(7·48)	(7·45)	(7·45)	(7·45)	(7·43)
Pco_2§ (mm Hg)	90	27	33	37	40	43	43
	(—)	(17)	(25)	(29)	(35)	(39)	(41)
Standard bicarbonate (mEq/l.)	16	17	22	22	24	26	27
Rectal temperature (°C)	—	27·8	31·8	33·0	34·8	36·2	37·3
Serum potassium (mEq/l.)	3·5	—	—	3·0	3·2	3·2	—

*Before intubation.
†Engstrom, model 200.
‡Spontaneous breathing before extubation.
§Figures in brackets are values at body temperature derived according to Severinghaus (1966).



Measurements of body temperature, heart rate, systolic blood pressure, and urinary output during period of resuscitation and recovery.

immersion in cold water. It takes 32·5 minutes to reduce the body temperature of a naked, anaesthetized infant to 24-25°C, and a further fall of 3-5°C after cessation of cooling has been observed (Mohri *et al.*, 1969). From the temperatures recorded during recovery we estimate that the lowest temperature in our patient was about 25°C at 6 p.m. and, allowing for a further fall after cessation of cooling, that it was about 28-30°C when he was removed from the pool. It would be reasonable to postulate an immersion time of at least 20 minutes to reduce the temperature of the child's body, fully dressed in winter clothing, from 37°C to 30-28°C.

Despite this prolonged time in the water there was no evidence of haemolysis (Kvittingen and Naess, 1963; Modell *et al.*, 1968), pulmonary oedema (Kvittingen and Naess, 1963), or electrolyte imbalance (Modell *et al.*, 1968). Though water was probably inhaled the amount must have been too small to induce haemolysis or radiographic lung changes. The arterial oxygenation was impaired, however, for the estimated alveoloarterial oxygen gradient at 9.30 p.m. was 137 mm Hg. Why so little water was inhaled by a child who was totally submerged must remain a matter for speculation.

The asystole cannot wholly be explained by the hypothermia, since asystole is expected to occur at temperatures of between 23 and 15°C (Gray and Nunn, 1971) and rhythmic heart activity has been reported at temperatures as low as 20°C (Mohri *et al.*, 1969). Slow sinus rhythm has been observed at 25°C (Edwards *et al.*, 1970). A combination of acidaemia, hypothermia, and hypoxaemia might therefore have been the cause.

Reductions in body temperature decrease brain oxygen consumption in animals (Rosomoff and Holaday, 1954; Bering, 1961), and a significant correlation between temperature and brain oxygen consumption was found in patients intoxicated with barbiturates (Malmlund, 1968). This may account for the lack of neurological damage in our patient.

We think this case shows that resuscitative measures should be initiated in hypothermic patients without regard to clinical appearances or evidence of prolonged circulatory arrest, and it should be maintained for longer than in normothermic patients.

Resuscitation of this patient was achieved through a joint effort and the participation of physicians, nurses, and technicians who assisted is gratefully acknowledged.

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