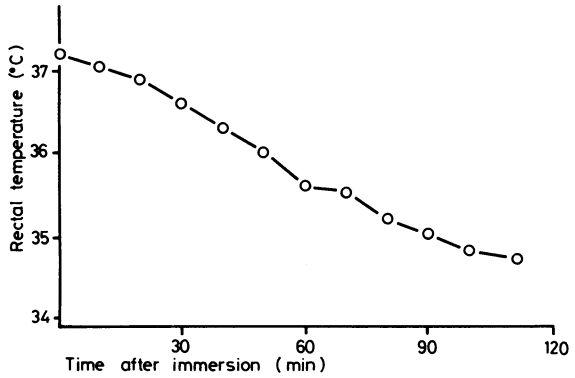
SHORT REPORTS

Progressive symptomless hypothermia in water: possible cause of diving accidents

Unexplained loss of consciousness, often followed by death, has been common during diving in British waters. Forty-two such deaths were noted by Childs and Norman[1] and no likely cause for them has been established. We describe here progressive symptomless hypothermia which developed during one of a series of fully monitored laboratory experiments designed to assess individual responses to mild surface cooling in water.

Case report

The subject was a physically fit, thin man aged 20. He was cox of a rowing eight and had been repeatedly exposed to cold air, close to or below 0°C, for about one and a half hours three times a week for one month before the experiment on 1 February 1979. His subcutaneous fat thickness measured by ultrasound averaged 3·8 mm over the upper chest and 4·1 mm over the lower abdomen at the level of the iliac crests. The figure shows that when he was immersed in water at 29°C wearing only bathing trunks his rectal temperature fell from an initial value of 37·20°C to 34·70°C after 112 minutes. This temperature was still falling, though at a reduced rate, at the end of this time.



Progressive fall of body temperature during immersion in water at 29°C.

Towards the end of the immersion the electrocardiograph showed ventricular and supraventricular ectopics.

The subject did not feel uncomfortably cold at any stage of the immersion. His metabolic rate during the last 10 minutes of the immersion, estimated from O_2 consumption, was 1·65 kcal/m²/min compared with 0·90 kcal/m²/min during an immersion in warm water at 37°C. His metabolic rate rose to 2·40 kcal/m²/min during another immersion in water at 26°C. Total body conductance in the last 10 minutes of the immersion at 29°C was 0·34 kcal/°C/m²/min compared with 1·36 kcal/°C/m²/min in water at 37°C. It fell to 0·27 kcal/°C/m²/min in water at 26°C. The subject therefore showed metabolic and vasoconstrictor responses to cold in water at 29°C which were less than he was capable of producing and were insufficient to stabilise body temperature during the immersion. The experiment was ended because of the continuing fall in temperature and the cardiac irregularities. The subject was then rapidly rewarmed in water at 42°C, when the ectopic beats ceased.

Comment

Two features about this subject are likely to have contributed to his failure to stabilise body temperature in water as warm as that of tropical seas. Firstly, thin people cool much more rapidly than fat people in cold water, because even when fully vasoconstricted they have less insulation from subcutaneous fat.[2] This alone would not account for his hypothermia, however, in which there was a failure of adequate metabolic response and of adequate vasoconstriction and appropriate sensation of cold, even at body temperatures below 35°C. Secondly, the subject's previous repeated exposure to cold probably contributed to hypothermia. Cold acclimatisation of different patterns may alter responses in complex ways but its commonest effect is to reduce reflex responses to cold. For example, repeated immersion in water at 15°C greatly reduces metabolic and respiratory responses to such immersion.[3]

There are no doubt several reasons for unexplained confusion and bad judgment leading to diving accidents, and for unexplained unconsciousness and death during dives, but "silent" hypothermia of the kind seen in this immersion could readily account for such cases. Such hypothermia would not be detected in a routine dive since standard practice during deep dives is to flood the suit continuously with warm water pumped down from the surface and to assess the adequacy of heating by the diver's own report of thermal comfort. Monitoring of body temperature seems highly desirable during deep dives in spite of the practical difficulties which it presents.

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[1] Childs, C M, and Norman, J N, *Médecine Aéronautique et Spatiale Médecine Subaquatique et Hyperbare*, 1978, **17**, 127.
[2] Keatinge, W R, *Survival in Cold Water. The Physiology and Treatment of Immersion Hypothermia and Drowning*. Oxford, Blackwell Scientific Publications, 1969.
[3] Keatinge, W R, and Evans, M, 1969. *Quarterly Journal of Experimental Physiology*, 1961, **46**, 83.

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Recurrent acute renal failure due to antibiotic-induced interstitial nephritis

Acute renal failure due to antibiotic-induced interstitial nephritis is uncommon but increasingly recognised. We believe this is the first report of recurrent episodes due to different antibiotics occuring in the same patient.

Case report

Between June 1974 and February 1978 a 55-year-old man with previously normal renal function had three attacks of non-catabolic, non-oliguric acute renal failure. On each occasion clinical examination was normal, but intravenous urography showed bilaterally enlarged kidneys with normal drainage tracts. Percutaneous renal biopsy specimens showed normal glomeruli but much interstitial oedema with an intense infiltrate of eosinophils, plasma cells, lymphocytes, and histiocytes in association with patchy tubular damage—changes typical of acute interstitial nephritis (figure).[1] The first attack was initially thought to be due to septicaemia after cystoscopy for recurrent urinary tract infection. During the previous four months, however, he had taken courses of co-trimoxazole, ampicillin, cephalexin, and nalidixic acid.

On admission he was taking co-trimoxazole. Investigations showed haemoglobin 13·2 g/dl; total WBC $8·6 \times 10^9$/l (8600/mm³), eosinophils 2%; blood urea 71 mmol/l (426 mg/100 ml); serum creatinine 1459 μmol/l (15·9 mg/100 ml); urine cultures sterile with excess erythrocytes and leucocytes; urine output 60 ml/h. Peritoneal dialysis was begun and methyl prednisolone 1 g intravenously given on two successive days after biopsy. After 72 hours he had a diuresis of 140 ml/h. Subsequent recovery was uneventful. One month later creatinine clearance was 68 ml/min. Nevertheless he became unwell with fever (38·9°C), and urine culture grew *Escherichia coli*. Intramuscular gentamicin was given, keeping serum concentrations within recommended limits. On the fourth day the serum creatinine concentration was 578 μmol/l (6·3 mg/100 ml) although urine output was 90 ml/h. After biopsy 1 g methyl prednisolone was given intravenously for four days. Recovery began on the second day and 16 days later creatinine clearance was 70 ml/min.