SHORT REPORTS

Hypothermia during saturation diving in the North Sea

We recently suggested after laboratory experiments[1] that undetected hypothermia might account for unexplained casualties during working dives in the North Sea. At depths greater than 50 m in the North Sea water temperature is below 10°C. Heat is usually supplied by flooding the diving suit continuously with warm water pumped from the surface via the diving bell; its temperature is monitored at the bell, but not at the diver, and is regulated mainly on the basis of the diver's report of feeling hot or cold. We found that simulation of this with warm water at 29°C around a thin man could produce progressive hypothermia with cardiac irregularities but without any serious sensation of cold.

We are now reporting the body temperatures of divers during saturation diving operations using the conventional heating system at a depth of 130-145 m in the North Sea in August-November 1979.

Subjects, methods, and results

The divers breathed helium and oxygen throughout. Their urine temperatures[2] were measured within eight minutes of their return to the bell after working dives with a maximum-reading Digitron thermistor with a response time of under 10 seconds in at least 50 ml of urine flowing through the outlet of a perforated funnel. This volume had been found with this apparatus to yield readings accurate to within 0·2°C. Skinfold thicknesses were measured with Harpenden callipers.[3]

The table shows that the thinnest diver in the group developed hypothermia at 34·7°C during a dive lasting only 55 minutes. He had felt a little cold and shivered at one point during the dive, but when the temperature of the warm-water supply, measured at the bell, was increased by 4°C he had stopped feeling cold. Two of the other divers cooled to near hypothermia with temperatures below 35·5°C at the end of 4-4½ hour dives, in one of which the warm-water system had failed for the last hour. That diver felt cold and shivered, but the other did not. In contrast, one relatively fat diver had a normal body temperature even after a one-hour failure of the warm-water supply; he reported considerable shivering and sensation of cold at that time. Body temperature after other dives was usually normal; in one instance it was a little high at 38·3°C.

Physical characteristics and body temperatures of divers, temperatures of warm water, and duration of dives

Diver	Height (m)	Body weight (kg)	Skinfold thickness* (mm)	Approximate temperature of warm water at bell (°C)	Duration of dive (min)	Body temperature at end of dive (°C)
1	1·80	74·8	9	40-44	55	34·7
2	1·73	68·9	13	41	207	36·6
				38	76	38·3
3	1·83	79·4	27	44	189	37·2
				42	216	35·6
				42	118	36·6
				44	235	37·1
4	1·83	82·6	23	42†	265	36·4
5	1·70	78·0	19	44	210	36·4
6	1·91	85·7	11	42	240	36·0
7	1·85	87·0	28	42	240	35·2
				42	182	37·6
8	1·73	79·4	19	42†	278	35·3

*On abdomen 50 mm below and lateral to umbilicus.
†Supply failed for last hour.

Comment

Even in dives in which the warm-water supply functioned normally, one diver cooled to near the point of hypothermia and another thin diver cooled below this without reporting any serious discomfort. The second, like the thin subject in our experiments,[1] cooled to 34·7°C, a temperature at which our subject developed an atrial arrhythmia of the heart—and at which mental impairment can be expected to present an increasing hazard. Any failure of the warm-water supply at that time, or continuation of such dives without raising the diver's temperature, could have rapidly caused confusion and unconsciousness as body temperature fell further.

Such changes produced by hypothermia could readily account for unexplained incidents in working dives. On average in 1974-9 over six divers died yearly in the North Sea, mainly in the British sector. At least half of these deaths were partly or completely unexplained. Many other divers survived periods of unexplained confusion and loss of consciousness.[4] A heating system that kept skin temperature reasonably uniform and mean skin temperature and inspired gas temperature at an optimal level near 35°C could make hypothermia impossible. A control system on the outside of the diving suit supplying a closed circulation of warm water under the suit, with hard-wired sensors to monitor temperature in the suit and circulating system, could probably achieve this most simply.

We are greatly indebted to the divers for their co-operation and to Mr T L Gosling and Mr T A Cardale, of Star Offshore Ltd, for arranging for the measurements to be taken.

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[3] Edwards DAW, Hammond WH, Healey MJR, Tanner JM, Whitehouse RH. Design and accuracy of calipers for measuring subcutaneous tissue thickness. *Br J Nutr* 1955;9:133-43.
[4] Childs CM, Norman JN. Unexplained loss of consciousness in divers. *Médecine Subaquatique et Hyperbare* 1978;17:127-8.

(*Accepted 2 January 1980*)

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Immunological studies of minimal-change nephropathy

The pathogenesis of minimal-change nephropathy (MCN) is unknown, partly because examination of affected kidneys has yielded no important information. Clinical data, however, have led to the postulation that lymphocytes of patients with minimal-change nephropathy can release a factor (lymphokine) that increases vascular permeability, particularly in high-pressure glomerular capillaries, thus causing proteinuria.[1] Supportive results have been reported[2] but have proved difficult to repeat.[3] We have attempted to determine whether lymphocytes in minimal-change nephropathy do release such a factor.

Subjects, methods, and results

Lymphokine production—Peripheral blood lymphocytes were separated on a Ficoll Hypaque gradient, washed three times in mixture 199, and resuspended to a final concentration of 1×10^6 cells/ml in mixture 199 and 10% heat-inactivated normal human AB serum. Phytohaemagglutinin (PHA) 5 μg was added to test cultures and 50 μl physiological saline to control cultures, all of which were performed in duplicate or triplicate and maintained at 37°C for 48 hours (reduced to 24 hours in later experiments). At the end of the culture period the cells were removed and the supernatant stored at −80°C after PHA 5 μg had been added to control ("reconstituted") supernatants.

Vascular permeability assay—The backs and flanks of Hartly-strain guinea-pigs (weighing 250-300 g) were shaved 24 hours before assay. Supernatant (0·1 ml) was injected intradermally at eight sites. Evan's blue 1 ml (1% in 0·15 mol sodium chloride/l) was injected through an ear vein and the animal killed 30 minutes later. Each supernatant was assayed in duplicate. The diameters of areas of blueing around each injection site were measured on the reflected skin surface and areas of blueing calculated.

The table shows the results of the assay. Seven assays were done in five patients whose cells were taken during relapse of minimal-change nephropathy before steroid treatment began; 10 were done in controls. The difference in mean areas of blueing for reconstituted supernatants between controls (R_1) and patients (R_2) was significant ($p < 0.001$) as was