talking to the subject did not have so much effect and the response to mental arithmetic became less pronounced. Similarly, it can be shown that the response to local cooling becomes less marked as central vasomotor tone is released by raising body temperature.

With these techniques to study the interactions between the two factors influencing the neurogenic component and the effects of local temperature on hand blood flow, the selection of cases for sympathectomy might be improved.

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Frostbite: General Observations and Report of Cases Treated by Hyperbaric Oxygen

Frostbite occurs when the tissues freeze; individuals at risk are those exposed to below zero temperatures (0° F; −18° C) in polar or mountainous regions and in countries where seasonal freezing occurs, and air crew.

In the pre-1920 era of polar exploration associated with the names of Scott and Shackleton, frostbite appears to have been relatively common. However, it is now an uncommon condition except in the case of accidents.

In the Himalayas where temperatures (−50° F; −46° C) and wind velocities (100 miles/h; 45 m/s) comparable with those of polar regions are encountered, there is the added complication of altitude. At normal altitudes, frostbite rarely occurs except when the patient is inadequately clothed due to low intelligence, alcoholism, illness or accident. Though frostbite can occur without general body cooling it may be associated with hypothermia.

Normally mountaineers operate at about 60% of maximum work capacity or 2 litres/min if maximum oxygen intake is 3 litres/min. If for any reason their physical working capacity is lowered they will have to work nearer to their maximal capacity to keep warm (Pugh 1966). If they are unable to keep up heat production, eventually the central core body temperature will fall and they may become gravely ill and die. Cases are recorded in which death has occurred within two hours of the onset of symptoms. At high altitudes, a steady work rate of 2 litres/min is more difficult to maintain and the individual is working nearer to capacity. Oxygen lack also causes forgetfulness and elementary preventive measures may be neglected. Heat loss increases also with overbreathing, as maximal breathing capacity is approached during physical work. Heat production will fall in cases of illness, injury or poor physical condition and the extremities will cool, despite clothing, to the ambient temperature, which, as it is often below freezing, will result in frostbite.

Frostbite, however, is not an inevitable sequel of sub-zero temperatures, at high altitude, in inadequately clad subjects. On the Himalayan Scientific Expedition 1960–1, a Nepalese pilgrim aged 35 spent a number of nights under observation without shelter at altitudes between 15,300 and 17,500 ft (4,600–5,200 m); he was wearing thin cotton clothing and both hands and feet were bare; minimum night temperature was −15° C, yet no frostbite occurred. During this period his diet consisted mainly of tea with sugar. This man normally lived in a village at 6,000 ft (1,830 m) where the night temperature in winter was below freezing and the windows were unglazed; he was therefore well acclimatized to cold. Overnight and daytime investigations suggested that his general metabolism was raised and he underwent a continuous light shivering rather than intermittent violent attacks (Pugh 1963). He also seemed relatively insensitive to afferent cutaneous cold stimuli as he never complained of

painful hands or feet. Skin temperature recordings of the hands never fell below 10–12° C so that episodes of vasoconstriction and frostbite never occurred.

Clinical Features

In superficial frostbite the skin loses sensation and becomes white, and blister formation may follow. Deep frostbite involves necrosis of muscles and bone in addition to the skin and subcutaneous tissue. It may be associated with hypothermia.

Anomalous features occur. Exposure of the ear in the slipstream of an aeroplane for a short period in Arctic conditions at sea level has resulted in complete loss; by contrast, an airman who fell from 56,000 ft (10·5 miles; 17,000 m) at an estimated speed of 180 miles/h (80 m/s) in an ambient temperature of −56° C before his parachute opened automatically, and who was not wearing gloves, sustained only mild frostbite of the fingers.

Following frostbite the affected part may show evidence of vascular occlusion, vasomotor irritability, hyperhidrosis and partial nerve injury as manifested by numbness and tingling. Osteoporosis may also occur. It is necessary to differentiate between the vasospastic and neuropathic sequelæ of cold injury as sympathectomy may help the former. Disorders of sensation and excessive sweating may persist for months and are important features in air crew. A part once frostbitten is rendered more liable to recurrent cold injury.

Management

Preventive measures, in addition to indoctrination and an understanding of the environment, include maintenance of body warmth, the avoidance of sweating, the 'buddy system'[1] and wearing of correct clothing.

A variety of methods have been used in treatment: vasodilator drugs, sympathectomy, anticoagulants, ultra-sound, pressure dressings, oxygen, low-molecular-weight dextran, packing the part in ice, rapid rewarming, antibiotics, cortisone and combinations of one or more (Greene 1941, Mills & Whalley 1960, Washburn 1962, Goodhead 1966).

In the acute phase the most convenient, quickest and best treatment is rapid rewarming in a vessel with water at 100–112°F (38–44·5°C); the water should never be warmer than 112°F (44·5°C). Immersion should be for up to 20 minutes. As thawing is painful, salicylates may be needed. In addition to local warming, general body warming is important. If rewarming by liquid is not possible, the part should be placed against a warm abdomen or wrapped loosely in warm blankets after a sterile dressing is applied. It should never be rubbed. For local treatment after the initial rewarming it is preferable to expose the part in a dry atmosphere. It should be elevated to a moderate degree and active movements should be encouraged. Surgery should never be contemplated in the early stages but should be reserved for reconstruction later; the blackened areas should be allowed to undergo auto-amputation. Infection must be prevented at all costs and parenteral antibiotics started as soon as the injury occurs. Treatment should always be carried out in a hospital. If necessary the patient must be allowed to walk on frostbitten feet if this diminishes the time between the onset of injury and the start of treatment.

Oxygen was first used in the treatment of frostbite by Greene (Ruttledge 1934) at 24,500 ft (7,500 m) on Mount Everest, with beneficial results in one of two cases. It has also been used by one of us (M P W) at high altitude in the spring of 1961; the results were difficult to evaluate.

Four cases were treated with hyperbaric oxygen in the Intensive Care Unit at the London Hospital. They were fit young men aged between 24 and 32. All had been frostbitten whilst mountaineering in the European Alps. Treatment with hyperbaric oxygen started 5–10 days after the initial injury. In all cases 'pinking' occurred in the affected part after immersion in the tank; in addition there was an immediate increase in warmth and movement of the limbs. Hyperbaric oxygen at two atmospheres for two hours a day for up to 19 days was given; treatment was stopped when no obvious further improvement occurred.

All cases had had antibiotics prior to admission; in one an attempt at an intra-arterial injection had been made and in another nicotinyl alcohol had been given. Whilst in hospital, reflex vasodilatation was used between tank treatments in one case.

Ledingham (1963) first reported the use of hyperbaric oxygen in the treatment of frostbite and a further case was reported from Montreal (Perrin & Bissonnette 1965). Our 4 patients differ from those described by Ledingham and Perrin &

[1]Continuous observation of companions and immediate rewarming, usually with the back of a glove, when the clinical features of early frostbite occur on exposed skin

Bissonnette in that they were all fit young men and used to mountaineering. Cade (1944) described a group of similar age in whom cold injury was due to sudden exposure when flying at high altitudes; he classified the condition into three groups, mild, moderate and severe. Other authors (Mills *et al.* 1960, Washburn 1962), with considerably more field experience, consider that a tentative classification into superficial and deep lesions, with or without body cooling, may be more appropriate. By comparison with Cade's cases, 3 of ours were in the moderate group and one, who already had some gangrene, on the border line of the moderate and severe groups.

The main features at the site of contact are that the superficial tissue freezes to a depth dependent on the length of contact and degree of cold. Intercellular ice crystal formation occurs and cellular dehydration and death may result. In addition it is believed that arteriolar constriction may occur and arteriolar-venular shunts open.

Immediately adjacent to the affected areas, the blood becomes viscous and capillary circulation slows, due to vessel damage leading to fluid extravasation and blister formation. Thrombosis may occur in the terminal arterioles (Davis *et al.* 1943, Merryman 1957, Washburn 1962).

Vascular stasis and œdema may effectively damage the local circulation. It has been shown that skin frozen and thawed on the surface of the body underwent necrosis for this reason and not because of individual cell death (Kreyberg 1949). It has also been shown that skin taken from a rabbit's frostbitten ear would take when grafted on to a normal ear but normal skin would not when grafted on to a frostbitten ear, suggesting that local vascular insufficiency was important (Weatherley-White *et al.* 1964). Whether tissue viability is decreased mainly by cellular damage or by local circulatory effects, it appears that with an increase in local tissue oxygen tension, using hyperbaric oxygen, the viability of the tissue may be improved and maintained whilst vascular and cellular regeneration occurs.

Assessment of tissue loss in the initial stages is notoriously difficult and serious further loss may occur as a result of infection. In 2 cases it was considered that some loss would occur: in fact this was not so and loss was minimal in one case. Nor was there tissue loss in the one case with infection due to previous deroofing of the blisters.

Hyperbaric oxygen has been shown to have a bacteriostatic action on surface infection (Ross & McAllister 1965) and that it did not prevent infection in this case may have been partly due to inadequate rest of the injured part.

In human volunteers hyperbaric oxygen has been observed to cause vasoconstriction and a reduction in blood flow (Bird & Telfer 1965). In the light of this observation it is interesting that one patient felt an increase in warmth in the affected part soon after he was placed in the tank and skin temperature recordings during the first two exposures confirmed that a rise had occurred. The rate of healing appears to be enhanced, for the blisters went within four days of treatment even when they were still prominent eleven days after the injury, thus removing a potential portal of entry for infection. The two cases quoted by Cade in the moderate group took five and twelve weeks respectively to heal. One mountaineer had had frostbite before, 'which took six weeks to heal and I did not recover the full feeling for several months, whereas following this treatment I have had the full feeling back in my toes since leaving hospital'.

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