MEDICAL PRACTICE

Occasional Survey

Frostbite

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Definition

Injury due to cold may be general or local. In general cold injury, or hypothermia, the individual is said to be suffering from exposure. Local cold injury may occur at temperatures above freezing (wet-cold conditions), as in immersion or trench foot. At temperatures below freezing (dry-cold conditions) frostbite occurs; the tissues freeze and ice crystals form in between the cells. Local cold injury may or may not be associated with hypothermia.

People at Risk

In civilian life frostbite is uncommon despite populations of about 100 million at risk in areas where sub-zero temperatures occur at some period of the year. During war at sub-zero temperatures frostbite is more common, often as a complication of wounds or disease. In the winter of 1943 frostbite injuries among U.S. heavy bomber crews were greater than all their other casualties combined.[1]

Polar travellers of the pre-1920 era suffered severely and frostbite seems to have been common. More recently, increased knowledge and better equipment have lowered the incidence in polar regions, though among mountaineers at high altitude cases still occur regularly. Nevertheless, frostbite is not inevitable even at high altitude and the three highest of the world's peaks have been successfully climbed without cold injury.

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Temperature Regulation

A useful but oversimplified concept is to imagine the body as consisting of a central core with a relatively uniform temperature of 37°C, and an insulating shell, with a temperature of 33°C. Heat transfer within the body occurs by convection through the circulation and by conduction through the peripheral tissue to the skin surface. As the maintenance of the central core temperature is essential to life this may be carried out at the expense of the peripheral expendable structures such as the toes and fingers.

The control of body temperature depends on the interaction between heat production and heat loss. The former depends on normal body metabolism, which at rest corresponds to the production of about 1,700 kcal per day with an oxygen consumption of 250 ml per minute. During heavy exercise and shivering the heat output may rise considerably, and maximal oxygen consumption in highly trained athletes may approach 4-5,000 ml per minute—though this level cannot be maintained.

Normally mountaineers operate at about 60% of working capacity—thus at sea level, with a normal maximum oxygen intake of 3 l./min, they operate at 2 l./min. If for any reason this working capacity is lowered, they will have to work nearer maximum capacity to keep warm. If they are unable to keep up heat production, the periphery will cool to the ambient temperature (which may be below freezing point) and then the central core temperature will fall. At progressively higher altitudes the maximal oxygen consumption falls until at 24,5000 ft (7,460 m) it is about 1·5 l./min.[2] At extreme altitudes the margin between maximum oxygen consumption and that necessary for work and heat production narrows, and cases of mild frostbite of the fingers and toes have been recorded in healthy well-clad people while climbing.

Heat loss occurs by convection, conduction, radiation, and evaporation. In dry-cold conditions, the most important is loss by convection, or transfer of heat by bulk movement, when the air adjacent to the body is heated. If the air so warmed remains trapped it will serve as insulation. If it is disturbed by external wind considerable heat loss occurs, and this is prevented by windproof clothing. Loss of heat by conduction occurrs when clothes become wet due either to rain or sweat or by direct contact with

snow. As air is a poor conductor of heat, and water a good one—wet clothes result in heat loss.

Heat loss by radiation, or the emission of discrete packets of electromagnetic energy, is independent of air movement. If the body heat is greater than that of the surroundings, heat is lost—if less, the body gains heat. Heat loss by evaporation may occur through perspiration or in the expired air. At the high ventilation rates of altitude, the latter may be of great importance.

Heat loss from the body is prevented by the insulation of the tissues, air, and clothes. Of these, that provided by the clothes is the most important. Because of the greater relative surface area, the heat loss from the curved surface of a small cylinder is greater than that of a larger curved cylinder, and increasing the thickness of insulating material will not increase the total insulation greatly until a cylinder is more than about 1 in in diameter. As the normal finger is about 0·5-0·75 in (1·25-2·0 cm) in diameter, the fingers are difficult to insulate effectively even when a mitt glove is used.

Pathophysiology

Skin freezes when fingers have been immersed in brine at —1·9°C. The true freezing point, however, is about —0·53°C. Freezing in brine at —1·9°C for seven minutes caused no lasting damage. The fingers were painful when thawed but became normal after a few minutes. After freezing for 11½ minutes, the affected fingers were red and tender for several days. Blistering occurred after repeated exposure for 20 minutes or more.[3] Nevertheless, the skin of the fingers can be cooled to temperatures below its true freezing point without freezing. Such "super cooling" is common if the skin is dry. On further cooling, freezing spreads rapidly in the super-cooled tissues, with tissue hardening.

Two main reactions take place when tissues come into contact with a very cold object. Firstly, a vascular reaction occurs under the frozen superficial tissues consisting of damage to the wall of the blood vessels, leakage of plasma into the tissues (forming blisters), and an increased viscosity of the remaining intravascular blood, with local haemoconcentration or "sludging." The small vessels may thus become blocked.[4] If the blood flow is then stopped by the action of the precapillary sphincters, the arteriovenous shunts will open up and blood bypasses the frozen area, which becomes avascular: in other words, the diseased part is sacrificed for survival of the whole organism. The second reaction is the formation of intercellular ice crystals. The intracellular osmotic pressure rises and enzyme mechanisms are disturbed with subsequent cell death.

Tissues vary in their resistance to frostbite. Skin appears to freeze at —0·53°C, while muscles, blood vessels, and nerves are also highly susceptible. Connective tissue, tendons, and bone are relatively resistant. Thus the blackened extremities of a frostbitten hand or foot can be moved since the tendons under the gangrenous skin remain intact, and the muscles to which these tendons belong are far removed from the area of severe cold injury.

High Altitude and Cold Injury

Frostbite at high altitude seems to be commoner for comparable conditions of cold than at lower levels. Both cold and high altitude raise the blood packed cell volume and viscosity and slow the peripheral blood flow. Cold injury to capillary walls leads to plasma leakage and intravascular sludging. This local haemoconcentration will be increased at altitude, and impaired tissue nutrition and necrosis may occur more rapidly.

Dehydration, the result of abnormal water loss from the lungs due to increased respiration, will also increase the viscosity of the blood, and thrombosis may be encouraged by relative inactivity over 21,000 ft. (6,400 m). Even at normal temperatures, the blood flow in the skin is reduced at high altitude, the result of arteriolar vasoconstriction.[5] Cardiac output is also decreased,[6] as may be basal metabolic rate. Moreover, the maximal oxygen uptake is progressively lowered with altitude, and with it the ability to increase heat production through exercise, and so is the ability to shiver. Hypoxia also blunts mental function and precautions normally taken against cold injury may be inadequate. Poor appetite—a cardinal feature of high altitude deterioration—may mean that calorie intake is inadequate with resulting diminution of the insulating layer of subcutaneous fat.

The only factor where high altitude does not appear to potentiate liability to cold injury is the wind-chill factor. Because of the relatively less dense atmosphere, this is diminished by comparison with sea-level values.

Clinical Features

FROSTNIP

Frostbite damages the tissues, whereas frostnip produces reversible changes. The skin blanches, and becomes numb with a sudden and complete cessation of cold and discomfort in the affected part. A tingling sensation may occur on rewarming. With immediate treatment frostnip will not progress to frostbite.

SUPERFICIAL FROSTBITE

Only the skin and subcutaneous tissues immediately adjacent are concerned in superficial frostbite. The frozen part, though white and frozen on the surface, is soft and pliable when pressed gently before thawing. After rewarming, it becomes numb, mottled, blue or purple, and it will then sting, burn, or swell for a period.

Blisters may occur within 24-48 hours, depending on the site of the injury. Thus blistering is more common on the dorsum of the fingers and hand, where the tissues are lax, than on the palm. The blister fluid is slowly absorbed; the skin hardens and becomes black, producing a thick insensitive carapace of tissue. In certain sites the black carapace may occur without preceding blister formation.

There is associated oedema, and within weeks a very definite line of demarcation occurs. Throbbing or aching may persist for weeks. If the contour of the blackened carapace corresponds to that of the original part, then loss of tissue is unlikely. If, however, the contour of the pulp of the finger disappears, and the carapace has a tendency to wrinkle, then loss of tissue is likely.

Unlike that in arteriosclerosis, gangrene occurring after frostbite is essentially superficial, and the necrotic tissue may not extend more than a few millimetres in depth. The black carapace fits like a glove around the tissues and peels off bit by bit over the months. After the carapace has been shed, the underlying shiny red babyskin will be abnormally tender and unduly sensitive to heat and cold. Abnormal sweating may occur. In two to three months it will gradually take on the appearance of normal skin. Generally the subcutaneous tissue feels rather wooden for the same period, but gradually becomes more pliable. The nail may be lost but is likely to grow again either normally or with a wrinkled appearance.

DEEP FROSTBITE

Deep frostbite involves not only the skin and subcutaneous tissue but also the deeper structures, including muscle, bone, and tendons. The affected part becomes cold, mottled, and blue or grey, and may remain swollen for months. Blistering may take weeks to develop but is not inevitable. Initially the part may be painless, but shooting and throbbing pains may occur and frequently abnormal sensations are encountered for up to two months.

As tendons are resistant to cold injury, the patient will be able to move his fingers and toes for long periods despite their gangrenous appearance. Thus even with severe frostbite patients can walk and use their fingers and hands for crude movements such as gripping. Eventually a carapace forms and sloughs off. Often a complete cast of the finger or toe with nail attached may separate.

Permanent loss of tissue is almost inevitable with deep frostbite, and this may be surmised from the shrivelled appearance of the affected finger or toe. Even with a diagnosis of deep frostbite, however, a limb may return almost to normal over some months, and amputation should never be carried out until a considerable period (probably at least six to nine months) has elapsed.

Treatment

PREVENTION

It is extremely important to dress for the temperature with which the part will be in contact. In deep powder snow, the feet can be in snow many degrees below freezing point, while at the same time the ambient temperature may be many degrees above freezing. Even the ambient temperature may vary enormously, with a 50°C drop at sunset.

PRINCIPLES

Frostnip

Frostnip is the only form of frostbite that should be treated on the spot. As this commonly occurs on the exposed portions of the body, such as the cheek or nose, each person should keep a watch for these signs in other members of the party. As soon as whitening of the skin is observed it is treated immediately. A place sheltered from the wind is found, or the back is turned to the wind, and the affected part is warmed by the hand, or the glove. Once the normal colour and consistency of the area is obtained normal working is resumed.

Frostbite

For many years rubbing the affected part, either with snow or with the normal hand, has been advocated for treating frostbite. This method neither melts the intercellular ice crystals by raising the temperature nor increases the blood supply to the injured area. It also has the disadvantage of breaking the skin and allowing infection. Other forms of violent therapy are open to the same objection. Vasodilator agents[7] do not improve tissue survival, nor are the results of sympathectomy striking.[8 9] The use of dextran sufficiently early to prevent sludging will rarely be practicable, and there is some possibility of danger in its use.[10 11] Possibly at sea level the use of hyperbaric oxygen or at altitude that of supplementary oxygen [12 13] may increase tissue tension of oxygen and just tip the balance in the favour of a cell partially damaged by cold injury.

Warming seems to be the most effective treatment, with rapid warming showing less loss of tissue than slow warming,[14 15 16] possibly because the area of circulatory arrest is smaller after rapid warming,[17] damage to the blood vessels is less, and there is less sludging.[18] Warming has the additional advantage that it can relatively easily be done on the spot—so shortening the time that the blood vessels and cells remain frozen and exposed to temperatures at which high electrolyte concentrations are dangerous. Once active rewarming has been started it seems doubtful whether any other active treatment is of benefit.

All cases diagnosed as frostbite should be treated either at a well-equipped camp, from which evacuation by air or some other means is easy, or in a hospital. Attempts to treat frostbite at high camps or anywhere with inadequate facilities are ill-advised. No attempt should be made to treat the frostbitten part except under ideal conditions, as once rewarming has started the tissues are liable to infection and may die. No patient should be allowed to walk on thawed or partly thawed feet. Walking on frostbitten feet does not appear to increase the liability to tissue loss, and may be the best method of reaching safety.

CODE OF PRACTICE

Treatment should be directed at both the whole individual and the affected part. The most important factor in treating the whole individual is to keep morale high. Generalized rewarming may be necessary, as some hypothermia is almost inevitable, especially at high altitude. This is best carried out by giving hot liquids or even oxygen. The patient must be put in a sleeping bag and an extra source of heat may be provided by his companions lying alongside him in a sleeping bag, or in the same sleeping bag.

Food should be given, as in itself it causes some peripheral vasodilation. Alcohol may also be given, but only if some time is to be spent in shelter. It will cause vasodilation—but more important will boost morale and diminish any pain caused by the rewarming. A broad-spectrum antibiotic should be started as a prophylactic against infection, while mild analgesics such as aspirin may be used to alleviate pain.

The affected part should be warmed, preferably using a container with water at 44°C. To measure the temperature accurately a thermometer should be available, as water that is too hot will damage the limb. If a thermometer is not available the temperature should be tested with a normal, unfrostbitten finger. If too hot for comfort more cold water should be added. The temperature of the water should never be tested with a frostbitten finger, because this will be partially anaesthetic owing to nerve injury. If no container is available, hot water poured over towels, or a cloth wrapped around the part may be used.

Rewarming should last about 20 minutes at a time; the temperature of the water should be checked frequently to see that it does not fall below 42°C. Additional hot water should not be poured over the affected part.

If rewarming by fluid is impossible, the part should be placed against a warm abdomen, armpit, or held in warm air. It should never be placed by an open fire as, again, it is partially anaesthetized and can be burnt without pain.

After rewarming, the part should be cleaned. But, because the tissues are extremely friable and liable to infection if broken, dirt maust be removed gradually and gently. Blisters should be left, and should not be pricked or removed as they form a covering. Nevertheless, some blisters may be broken, and soft dry absorbent dressing should be used as cover. Similar dressings between the fingers or toes will prevent friction and prevent further damage to the skin. Even light pressure can cause pressure sores, infection, and loss of tissue.

So long as the affected part is warm and does not get rubbed it may be kept exposed. Swelling may occur, and this can be countered by raising the part. Local antibiotics may be used but it is unwise to rely on this method alone for combating infection. No tissue should be removed surgically for three main reasons: it is impossible to assess the depth of frostbitten tissue; the black carapace acts as a protective covering for regenerating tissue; and premature surgery appears to have been the most potent cause of the poor results of treatment. Active movements should be carried out to prevent joint stiffening, and if these are not possible, passive movement should be employed.

PROGRESS

Surgical intervention must be minimal. The blackened carapace will gradually separate by itself without interference. Efforts to hasten separation are usually ill-advised and are more likely to lead to infection, loss of tissue, and delay in healing rather than the reverse.

Because of the disturbance of sensation that accompanies frostbite, pockets of infection may appear either under the nail or under the carapace, and an abscess may occur without the accompaniment of pain. These are extremely difficult to diagnose in the early stages, but may necessitate removal of the nail or drainage.

Too few cases are seen by any individual and there is a tendency to try more than one treatment in the hope that more rapid healing will occur. In general, however, provided there is no surgical intervention, most cases of frostbite seem to heal in six to twelve months.

Prognosis

The prognosis should be guarded, but optimistic, as the gangrenous tissue is essentially superficial. Thus in one patient with bilateral gangrene of the feet extending to the ankles, conservative treatment resulted in complete recovery without tissue loss in nine months. In another patient, both of whose legs were essentially blocks of frozen tissue to above the knees when first seen, conservative treatment for about a year resulted in recovery except for patches of persistent gangrene on the heels. Bilateral amputation was carried out for this reason alone.

Probably many unnecessary amputations have been carried out because of impatience at the very slow recovery. The gangrenous carapace may persist for months, and the failure to appreciate that it is a "superficial" rather than a "deep" gangrene may lead to precipitate surgery.

Once a part is frostbitten, it is more liable to cold injury on subsequent occasions. The skin may crack when dry, even at normal temperature, causing painful fissures in the pulps of the thumb and fingers. The use of a hand cream at regular intervals will soften the skin.

It is important to differentiate the vasospastic and neuropathic sequelae of frostbite. Sympathectomy produces good results in the former, though it aggravates paraesthesiae. In mixed cases a peripheral nerve block with xylocaine may differentiate the two: if the skin temperature rises after this sympathectomy is useful.[19] Decalcification of bone, possibly due to local ischaemic necrosis, has also been reported.

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Contemporary Themes

Misuse of Drugs Act 1971

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Summary

An attempt is made to explain the provisions of the Misuse of Drugs Act 1971 stressing, where possible, those portions of the Act which affect members of the medical profession.

Introduction

The events leading to the drafting of the Misuse of Drugs Bill have been reviewed elsewhere.[1] This Bill became the Misuse of Drugs Act 1971.[2] The Act repealed the whole of the Drugs (Prevention of Misuse) Act 1964, and Dangerous Drugs Acts 1965 and 1967.

Legislation on drugs liable to abuse, like international agreements on the same subject, had grown up piecemeal. The resulting laws were fragmentary and inflexible and ill-designed to cope with a problem characterized by its constantly changing nature. Fashions in drug abuse are fickle. A drug which has never been abused before may become popular among abusers in a very short time, yet it was impossible in this country to bring such a drug under the restrictions of the old Dangerous Drugs Acts unless this was recommended or appeared to be about to be recommended by the United Nations Narcotics Commission.

The Misuse of Drugs Act 1971 is therefore more than a consolidating Act. It gives powers to Ministers to act swiftly to combat the misuse of drugs and makes much less rigid the law concerning drugs which are deemed to require control at any given time. The Act makes certain things unlawful. The Misuse of Drugs Regulations 1973 set out circumstances in which things which would otherwise be unlawful become permissible.

Advisory Council on the Misuse of Drugs

Though the Misuse of Drugs Act received the Royal Assent in 1971 it did not become operative, and then only in part, until

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