1954

The Management of frostbite

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Submitted in Partial Fulfillment for the Degree of
Doctor of Medicine

University of Nebraska, College of Medicine

March 24, 1954

Omaha, Nebraska

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The helpful suggestions and criticism of Stanley Potter M. D. are gratefully acknowledged.

Frostbite has plagued man ever since he first ventured from the tropical Garden of Eden. It has been a particular menace to armies because of their undue exposure to the elements. Most scientific investigation of this entity has been stimulated by an effort to reduce wartime casualties. With World War II, the widespread high altitude flying presented a new problem when heating was damaged or windows broken in flight.

Frostbite is a condition wherein the tissues of the body are actually frozen solid. This may occur after a prolonged exposure to cold as with the foot soldier or instantaneously in the case of an airman with a window broken while flying at high altitude. In either case the result is the same.

In order to investigate methods for treatment of frostbite one must first understand the physiological pathology of the condition and attempt to reverse or medify the process.

First one must consider the fact that there is a tremendous variability in the susceptability of different individuals to frostbite and that the severity of injury is roughly proportional to the temperature of the frozen part and the duration of the frozen state. Therefore one can see that injury may vary from mild, in which there are no sequellae to the extreme in which there is gangrene and loss of the entire frozen part, in spite of all therapy.

The following sequence of events takes place in living tissue when exposed to cold sufficient to freeze and later to produce gangrene. On first exposure to cold there is a direct and persistent local vasoconstriction. This is seen grossly as a blanching of the skin, then follows a transient generalized reflex vasoconstriction. As exposure progresses in time or temperature, the blood temperature is lowered sufficiently to effect the medulla which in turn stimulates a more prolonged generalized vasoconstriction. If exposure is sufficient to maintain a tissue temperature of 3-4° C. for about thirty minutes, an alternate local vasoconstriction and dilatation occurs. There is progressive numbness, weakness and paralysis of the exposed part. Blood flow becomes sluggish.

These components of vasoconstriction appear to be a protective mechanism for the body. Decrease of circulation to an exposed part limits the heat loss from the total body; hence, nature has provided for sacrifice of a limb to preserve the body. The alternate vasoconstriction and dilatation just prior to freezing appears to be a last resort for protection of the limb.

Freezing occurs at a variable tissue temperature due to the phenomenon of supercooling. There is no explanation of this. Tissue temperature may become as low as -15° C. to -20° C. before freezing occurs whereas one would expect freezing at about -1° C. from calculation of the molar concentration of the body fluids. Hence, whereas one person may freeze a finger at a tissue temperature of -2° C. another may be able to withstand -20° C .

At the freezing point all circulation stops and the part becomes solid, brittle and white. The victim experiences a sharp stinging sensation as this happens.

When warmth is applied, thawing begins. With this there is a marked hyperemic phase, due to vasodilatation. The blood flows first in the larger channels and in the next few minutes through the capillaries. The capillaries become distended and there is extravasation of plasma through the capillary walls. The limb is red, painful and there is a bounding pulse in the large arteries. Slowing of capillary blood flow (secondary to vasodilatation) coupled with extravasation of plasma causes a sludging or silting out of the red blood cells. Thus, the capillaries become occluded and blood flow ceases about fifteen minutes after thawing. Grossly the part remains hyperemic but blood flows only through the arterio-venous fistulas, not the capillaries. It must be noted that there is no fibrin in these clumps of red cells, hence, they do not represent true thrombi.

Vessels proximal to the frozen part become dilated for from twenty to fifty minutes after thawing and then constrict, especially at the juncture of the frozen area with unfrozen tissue.

The extravasation of plasma causes gross edema, wheals, or bullae depending on the severity of the injury. The fluid in the bullae is viscous with chemical content almost identical with plasma. A day after injury the fluid becomes less viscous. This edema formation is accompanied with severe pain.

Within the first few hours after thawing, muscle tissue is seen to fragment sometimes and become vacuolated. Sarcoplasm becomes homogenous with loss of myofibrillar structure. Nuclei may fragment and small extravasations of red cells can be noted.

About six hours after thawing, edema has reached a maximum and begins to subside. Eight to ten hours after injury there is an infiltration of neutrophiles and mononuclear cells. Connective tissue cells begin to proliferate. At about this time cyanosis becomes evident. The cyanotic area is a fair indication of the extent of impending gangrene.

Forty-eight to seventy-two hours after thawing, the red cell clumps have become homogenous and true thrombi with a fibrin network forms around them. Bacterial invaders find easy access through ruptured bullae and infection is an almost constant complication of untreated frostbite.

The temperature of the part becomes lower, finally to reach that of the environment. At eight to ten days a wet gangrene becomes apparant which soon drys. The tissue becomes leather hard and black. If these gangrenous eschars surround fingers or toes, compression ischemia of the deeper tissue may occur.

Pus forms between the gangrenous and surviving tissue. New vessels begin to penetrate the injured tissue after seven days.

The gangrenous eschars may slough at any time after eight days but this may not occur until as much as three months have passed. Circulation is poor and vascular tone erratic in the area proximal to the slough. Healing of the stump is very slow because of the poor blood supply. In the following months or years any of the frozen part that recovered is subject to hypersensitivity to subsequent cold. It exhibits the typical Raynaud's phenomenon of increased vascular tone leading to repeated frostbite and pain.

Less severe injury may simply result in ulcers instead of gangrene, or in no loss of tissue at all.

Let us now examine the mechanisms of injury. It has been shown by many (1,2,3,4,) that the first indication of injury is loss of plasma through the capillary walls and concurrent clumping of the red cells within the lumen of the capillary. Poor circulation and tissue anoxia appear to follow from this sequence. Reasons for this increased capillary permeability are not clear. Sir Thomas Lewis (5) suggested that this was due to a histamine-like substance produced in the tissues by ice crystals, or cold without actual freezing. One reason for this suggestion was the similarity of the gross appearance of the "triple response" (i.e. blanching, erythema and wheal formation) to that seen in mechanical, chemical or heat injury, which is due to histamine release. Also, the alternate constriction and dilation of the capillaries in a slow rythmnic manner just preceding freezing suggests the presence of a humoral control of capillary tone at this temperature. When the capillaries

are constricted, a buildup of this substance is allowed which in turn dilated the capillaries allowing it to be washed away. However, edema fluid from a frostbitten area was injected into a rabbit and no histamine-like response was observed (6). Also an assay using atropinized guinea pig gut showed a histamine concentration of less than 0.2 mgm/cc. which is about the amount to be expected from a small extravasation of blood (6).

It has been suggested that there is a direct rupture of the capillary and tissue cell walls due to the formation of ice crystals, increase in osmotic pressure due to the high salt concentration in the unfrozen fractions or some obscure disturbance in gel-sol relationships (7).

Greene(2) noted swelling of the endothelium five minutes after thawing the tail of a mouse. The significance of this is not known.

Lange(4) feels that the clumping of red cells is independent of plasma loss, perhaps being due to some unknown cold agglutinins. He observed clumping of red cells before actual freezing took place although there was no extravasation of plasma until after freezing and thawing were complete.

The question of whether freezing per se is harmful to tissue is debateable, and the final answer is yet to be determined. Pirozynski and Webster(8) published photomicrographs showing fragmentation of muscle cells within two hours after freezing. Greene(2) noted that connective tissue nuclei stained poorly within thirty

minutes after thawing, thus indicating necrosis. Various tissues show different degrees of susceptability to frostbite. Muscle is the most sensitive followed by skin, connective tissue and nerves in that order. Differences in vascularity would not explain this so it seems that the cold per se must be the injuring factor.

On the other hand, Greene(2) noted in frozen mouse tails that the distal portion which was destined to slough contained the clumped red cells whereas the proximal part which was also frozen but did not slough did not contain these clumps. Lange(4) saw no evidence of tissue damage until after thrombosis had occurred (second to third day after injury.) These two items indicate that there was no apparant injury to the tissue until after the vascular change.

It must be remembered in evaluating the foregoing points of view that the individual variation of susceptability or resistance to frostbite is tremendous and that small differences in temperature or duration of exposure profoundly effect the final outcome. It seems reasonable that severe freezing may produce immediate histological changes whereas less severe freezing but still sufficient to produce gangrene may result in injury only secondary to the vascular occlusion.

The thrombosis observed two to three days after injury unquestionably contributes to the gangrenous result.

Treatment has been difficult to evaluate in human patients because of the lack of control of the injury and the tremendous

variability in the susceptability of individuals. For this reason much work has been done with experimental animals under strictly controlled conditions. In all of these experiments, prevention of or reduction in the extent of gangrene has been the goal. It is well to repeat at this time that some frostbite is so mild that complete recovery will occur whether treated or not and that some is so severe that all known treatments cannot prevent gangrene.

Since vascular thrombosis is such a prominent part of the pathological picture there has been much experimental work, trying to prevent gangrene by prevention of thrombosis. In 1945 Lange and coworkers(9) stimulated hope by announcing that they had prevented gangrene in experimental rabbits by the use of heparin to prolong clotting time. They froze the hind legs of twenty-two rabbits in alcohol baths at temperatures of -12°C. to -20° C for forty-five to ninety minutes. Freezing was noted in from five to eight minutes. Eleven animals were heparinized with 30mgm. heparin every twelve hours, begun within three hours after freezing and continued for five days. At all times the clotting time remained above thirty minutes by the Lee-White method. Nine animals recovered completely, two had superficial ulcers and two died on the fourth and fifth days. Eleven control animals developed gangrene with spontaneous amputation of the extremities. This experiment has been criticised repeatedly for the rather loose technique employed with variable freezing times and temperatures.

In 1947 Lange(10) reported a series of forty-one rabbits with legs frozen at -30°C. for thirty minutes. Twenty-one rabbits were heparinized with 30mgm. each, every twelve hours, which maintained the clotting time above thirty minutes. Of these, eighteen escaped gangrene and three showed superficial ulcers. Twenty control animals all suffered gangrene and spontaneous amputation. In four human volunteers, frozen areas were produced on the skin of the forearms. Each was frozen in four different places at four different times. On each was an untreated control, all of which developed gangrene. For the second exposure, each was heparinized immediately with continuance for six days. No gangrene developed. On the third exposure, heparin was delayed twenty-four hours. There was some ulceration but no gangrene. The fourth exposures were maintained at a low temperature for twenty-four hours and then warmed. Blisters did not form so quickly but after warming they were larger and the gangrene more extensive than seen in the control areas.

Many experimenters have tried to verify or refute Lange's excellent results (11,12,13,14). There has been little agreement in the results of further experiments. Quintanilla and coworkers(11) got poor results with rabbit legs but they continued their heparinization for only thirty-six hours. Pichotka and Lewis(12) carried out elaborate experiments with variable amounts of heparin. In their results, it was seen that the rabbits which received heparin every four hours, there was less gangrene than the controls; whereas in those which received heparin every six or twelve hours, no benefit

was seen. These experimenters were unable to maintain sufficiently high clotting times with doses every twelve hours as Lange had done. This exemplifies an important point. Adequate heparinization is definitely beneficial while only slightly less has no value whatever. At this point it may well be interjected that the most accurate method of measuring clotting time is that of Lee and White wherein venous blood is placed in an immaculately clean test tube. Adequate heparinization is that which maintains the clotting time above thirty minutes at all times regardless of dosage or frequency of administration. Others (13,14) have shown inconclusive results with heparin. Many used the capillary tube method of measuring clotting time. This is not sufficiently accurate. Difference in strains of rabbits or state of nutrition may have caused poor results.

Immediate sympathectomy has been considered for the treatment of frostbite on the assumption that this would increase the flow of blood to the part. Here, as with the use of heparin, the reports are conflicting. Crissmon and Fuhrman(1) noted that with sympathetic block in rabbit ears that capillary stasis was delayed about thirty minutes. Finneran and Shumacker(15) froze the feet of forty dogs and treated half by lumbar sympathectomy . Of the twenty dogs treated, nine showed no or only slight loss of tissue and eleven developed severe gangrene. Of twenty controls, only four had little or no gangrene while sixteen developed severe gangrene. The same authors also treated ten rabbits by stellate ganglionectomy after freezing their ears. All of these developed gangrene as did their controls.

Other workers(1,16) have noted failure of sympathectomy but their experiments do not necessarily indicate that sympathectomy has no value because if all the animals of a series develop complete gangrene it seems reasonable to assume that their injury was so severe that slight beneficial effects were overshadowed.

Medical sympathectomy has been attempted in experiments with the potent vasodilators such as hexamethonium, hydergine and tetraethyl-ammonium chloride. Hurley and coworkers(17) froze rat tails and compared the loss of tissue among treated and control animals. The treated animals received 0.2mgm/kg., 0.4mgm/kg. and 0.8mgm/kg. of hydergine (trade name for the combination of dihydroergocormine, dihydroergocrustine and dihydroergokryptine) intramuscularly every six hours. In the untreated control animals the mean tail loss was 4.81 cm. The rats receiving 0.4mgm/kg. showed the most benefit, losing an average of only 1.33 cm. This appears to be very significant because the injury was in the proper range of severity and definite benefit was demonstrated. In addition, these experimenters combined the use of 0.2mgm/kg hydergine with heparin of an unspecified amount. The results of this were better than with the identical dose of hydergine alone but not so good as with 0.4mgm/kg. hydergine alone. This is of little significance because the clotting times attained with heparin were not specified but it indicates that the effects of hydergine and heparin are probably additive and in no way antagonistic. In later experiments the same men showed that the drug could be given orally. Prophylactic doses gave equally

good results. In contrast to this were the results of Lewis and Moen (18) which indicated that there was no value to be had from hydergine. These authors question whether systemic drugs effect the vasomotor tone in frostbitten tissue. Other authors(14,15) have shown that tetraethylammonium chloride has beneficial effect similar to those cited for hydergine.

Experiments with rutin have been attempted thinking that this agent might reduce the capillary permeability and thus prevent the clumping of red blood cells. Fuhrman and Crissmon(19) injected rabbits with rutin, 50mgm/Kg/day. Ears were not protected from gangrene in the treated animals. In rabbits, however, in which the feet were frozen, only toes were lost while in the control group, there was almost complete gangrene of the leg. These men noted that capillary stasis was delayed up to eighty minutes which was similar to their observations on the effect of sympathectomy. Also, diffusion of trypan blue dye into the frozen area was delayed to sixty-eight seconds compared to a control of thirty-two seconds. This is evidence that the drug definitely reduces capillary permeability. Shumacker (20) using doses of 100mgm/Kg/day rutin reduced the mean tail loss of mice from67% to 49.3% while in his series of rat tails the mean tail loss was increased from 58% to 73%. Only slight benefit was seen with rabbit ears. Lewis and Moen (18) had inconclusive results.

The advent of antihistaminics allowed experimentation to prove or disprove Sir Thomas Lewis's thought that the hyperemic phase of

the "triple response" of frostbite was due to histamine or a histamine-like substance. As with other drugs, the results of the antihistaminics have been i ndefinite for the prevention of gangrene. Benadryl was given intraperitoneally 0.43mgm/Kg. every six hours to mice, rats and rabbits by Shumacker(20). The gangrenous loss of tissue was slightly greater than in controls in the experimental mice and rats. In rabbits, however, mean tissue loss was reduced from 99% to 94%. Frommel and Piquet(21) treated guinea pigs with antergan 10mgm/Kg daily for twelve days with some decrease in gangrene as compared with controls but freezing times and temperatures were not specified. Macht and coworkers(22) froze areas on human volunteers and demonstrated that benadryl and pyribenzamine iontophoresis given locally one hour before freezing greatly reduced the vesicle formation below that of control areas.

Since edema is a prominent part of the pathology of frostbite it was felt by some that pressure dressings applied to the part in an effort to reduce the edema might have therapeutic value. Crissmon and Fuhrman(23) froze rabbit legs for various specified times and at various temperatures. Plaster casts were applied immediately after thawing. Four animals treated this way showed less gangrene than controls. Nine others with vinyl plastic casts applied at the time showed no benefit. They also tried simple elastic dressings after edema had reached a maximum. This showed no beneficial effect compared with controls. Lange (9) and Boyd had poor results, as did Shumacker(20)

The most startling development in frostbite management is that rapid thawing by immersion of the frozen part in water at 42° C. or 107° F. is the best single therapeutic method discovered to date. This is in direct contradiction to all of the early thought. An interesting item is recorded by Barron Larrey (24) who was with Napoleon's army in Russia. With regard to the frostbitten soldier,"If the projecting parts of the body were brought too near the bivouac fire, he was attacked with gangrene so suddenly as to be seen while watching." In the past, as well as among uninformed lay people today, the accepted treatment of frostbite has been to keep the part cold by rubbing snow on it or by other means. Sir Thomas Lewis commented that this was good because the formation of wheals was delayed and immediate pain reduced. This and other early writing was mere armchair speculation not backed up by real evidence. Numerous recent references have shown that prolonged cooling is harmful (3,14,25).

Lemple and Shumacker(14) froze the tails of forty mice and treated half with air cooling at 3° C. for seventy-two hours. Gangrene was more extensive than in those left at room temperature. They also treated the frozen feet of mice by immersion in ice water for one hour. This gave very poor results.

Experimental proof that rapid thawing is the treatment of choice is now well established. Fuhrman and Crissmon (26)

froze the ears of rabbits for various times at -55° C. These were treated by thawing in water at 42° C. for one to two minutes. In all cases the incidence of gangrene was less than half that seen when the part was allowed to thaw at room temperature. Similar results are shown by others.(14,27)

Typical of these are the results shown by Shumacker and coworkers (14). Tails and feet of mice, treated by thawing in water at 42° C. for two minutes showed a mean tissue loss of 18.7% of the exposed tissue. Controls lost 67.7% of tissue. Rat tails and feet treated in the same way lost a mean of 35% while loss in the controls was 58.5%. More dramatic were their results with rabbits. Mean tissue loss in the treated ones was 28.2% with loss in controls of 99.6%.

The conditions of the rapid thaw are quite important. Experimental evidence indicates that thawing should ideally be in water at 42° C. just long enough to thaw and to warm the part. This usually takes four to five minutes; then the part should be dried and allowed to remain at room temperature. Shumacker and Finneran (15) showed that thawing in water at 38° C. is not so effective as at 42° C. and that thawing at 50° C. is harmful. Also, thawing with maintenance at 40° C for seventy-two hours was more harmful than no treatment at all(14).

ACTH and Cortisone have been tried in an effort to prevent gangrene without good results (27,28) but it was noted that the

necrotic zone demarcated more quickly; this does not seem to be a sufficient indication for its use.

As shown above several individual methods can reduce the incidence or extent of gangrene. There has been little experimentation with combinations of these. Finneran and Shumacker(15) combined rapid thawing with heparinization. They found little significant improvement over rapid thaw alone, but considerable improvement over heparinization alone. Lemple and Shumacker(14) combined rapid thawing with tetraethyl ammonium chloride for seventy-two hours. 95% of the animals had no tissue loss. This was better than either treatment separately in their series.

Many other measures are useful in the management of frostbite cases. The high incidence of secondary infection (25-66% of cases) (28) has been noted and steps should be taken to prevent this. Finneran employed soaking the part in zephiran 1/1000 once daily for a week or more(29). In addition, 300,000 units penicillin intramuscularly daily was recommended. In the tissue destined to necrosis, it is doubtful that parenteral penicillin would prevent or stop infection, however it is indicated for prevention of spread of organisms into the viable tissues. Most of the invaders are Staphyloccus which usually responds well to penicillin.

Lewis (30) had considerable difficulty with Pseudomonas aureugenosa infection in experimental rabbits. This was prevented

in all cases by application of sulfa ointments to the skin of the part. This organism, however has not been mentioned in reports on humans.

There is frequently a collection of pus immediately beneath the gangrenous eschar. If this is noted, surgical drainage is indicated. This relieves pain which is common at this stage of the pathology.

In spite of measures to prevent gangrene, it still occurs in many cases. After the first ten days, the physician is faced with the problem of treating gangrenous stumps, poorly healing ulcers and vasomotor instability. Extreme conservatism is paramount when considering debridement for there is no way of anticipating the depth of the necrosis. One should wait two to three months before surgery is contemplated, except in cases where the necrotic eschars completely surround a digit. These eschars may shrink causing a compression ischemia. They frequently should be cut along both sides of the digit to allow free contraction. The eschar usually sloughs spontaneously, leaving a delicate skin or an unhealed ulcer beneath. However, if sloughing has not occurred at three months, debridement should probably be done with caution. Care should be exercised to prevent removing more tissue than is necessary. It will be noted that amputation stumps will be relatively bloodless. This is due to the severe damage to the blood vessels.

Sympathectomy is indicated in selected cases two weeks after injury, if this has not been done during the acute phase. Individual judgement must be exercised. Results to be expected include quicker healing of ulcers, quicker slough of necrotic tissues (because of quicker healing beneath them) and prevention of the cold hypersensitivity "post-frostbite" syndrome. If the effected area is small and the surface appears to be healing well, there is little need for subjecting a patient to sympathectomy, but if the frozen areas were large or suspected to be deep it would certainly be beneficial. A sympathetic block by injection of procaine may be tried if it is questionable whether or not much response can be expected.

A recent study (28) indicates that few patients experience the described pain and vasomotor instability, and that most of those who do, have no difficulty after five to six months. It seems then that the chief value of sympathectomy is to shorten the convalescent period. Buerger's exercises and whirlpool baths may aid circulatory recovery.

SUMMARY AND CONCLUSIONS:

The physiopathology of frostbite has been reviewed. Prominent features include, increased capillary permeability with edema formation and clumping of red blood corpuscles within the capillaries, capillary stasis and later thrombosis. The end result is loss of tissue by gangrene, with vasomotor instability in the surviving parts. Possible mechanisms of injury have been discussed with the

conclusion that freezing per se injures tissue and that vascular occlusion secondary to thrombosis adds to the tissue insult.

Many experimental attempts have been made to prevent gangrene following frostbite. It has been shown definitely that the single most effective treatment is rapid thawing of the part by immersion in water at a temperature of 42° C. or 107° F. This obviously is a first aid measure since it is unlikely that a victime could make his way to a doctor's office before the part would thaw. Unfortunately, under the conditions that frostbite occurs there is no hot water available. The next best thing is putting frozen fingers into the mouth or between the thighs, clapping a hand over a frozen nose, etc.

It has been shown that definite value is to be had in the use of anticoagulants. Care must be taken to use them correctly. The dosage of heparin must be adjusted according to the clotting time. At all times this clotting time should be maintained above thirty minutes (by the Lee-White method). Heparinization must be begun as soon as possible. It has little value if started after the second or third day. The initial dose should be in the range of 50-75 mgm. This may be given subcutaneously or by slow intravenous drip. Depot or repository heparin is available. With this a single dose of 300-400 mgm. should maintain sufficient effect for twenty-four hours, compared to the four hours obtainable with other forms. There has been no experimental work with tromexan or dicumarol

used in frostbite. It seems reasonable that these would be effective but certainly the first forty-eight hours should be covered with heparin while the other agents begin to work. Therapeutic levels of dicumarol are attained when the prothrombin times are between 20-30% of normal. The initial dose of dicumarol is 250-300 mgm. with later adjustment downward to an average maintenance dose of 50-75 mgm per day. Anticoagulant therapy should be continued at least six days and preferably eight to ten days. One should be aware of contraindications. These include open wounds, blood dyscrasias bleeding tendency, peptic ulcer and liver disease. The patient should be watched for urinary bleeding, blood in the stool and retinal hemorrhage. Hemoglobin should be checked daily to detect a slow internal blood loss.

The efficacy of immediate sympathectomy is debateable but in light of the present knowledge I feel that it is helpful. However since anticoagulant therapy is of more value, I think that the use of hydergine or hexamethonium for a "medical sympathectomy" is better because a surgical proceedure would be contraindicated in the face of therapeutic levels of anticoagulant.

Antihistaminics, rutin, pressure dressings and prolonged cold do not seem to be of any definite value or may be damaging and should not be used.

Immediate treatment should also include daily soaking of the part in zephiran 1/1000, followed by application of a dry dressing. Penicillin 300,000 units daily is indicated. Topical antibiotics may be used.

If pus forms under the gangrenous eschar it should be drained surgically. If eschars surround a digit they should be bivalved. Surgical sympathectomy is indicated in severe cases after about two weeks. At this time the vasodilator may be discontinued. Amputation or debridement should be delayed as long as possible. One should preferably wait for a spontaneous slough.

Bed rest is helpful with the extremity either level or slightly elevated, for the first two or three weeks. After that, Buerger's exercises and whirlpool baths will stimulate circulation. Adequate nutrition should not be overlooked.

NOTES ON PREVENTION OF FROSTBITE

As with any disease or affliction, prevention is far more desirable than the best treatment. It has been noted by arctic explorers and others, that if the skin is not washed, it is less likely to freeze. It is felt that this is due to an alteration in the supercooling mechanism by washing away the natural skin oils. Wet clothing is a "kiss of death" to soldiers and others exposed to severe cold. This is due to the fact that moisture carries heat away from the body rapidly. Therefore, clothing should be changed and dried as frequently as possible to allow drying of sweat or other moisture. Clothing should be loose so that there is a layer of dead air next to the skin, and to allow unrestricted circulation. The armed forces have recently developed a new type of insulated clothing which has proven to be miraculous

in Korea. This clothing is constructed of a double layer of waterproof material with an insulating material between. With this arrangement, the skin may become wet but the insulating material remains perfectly dry and effective. Fingers, toes, cheeks, nose and ears are the most susceptable parts of the body so their protection should be emphasized.

Whiskey may save a limb from frostbite by allowing a vasodilatation but in the same way it may cost a life, so it should be avoided during severe cold exposure.

Previously frozen parts of the body are more susceptable than normal parts so they should be given special attention.

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