LESSONS FROM HISTORY

THE IMMERSION FOOT SYNDROME¹

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PATHOGENESIS

Since the review of Ungley and Blackwood (1942), additional evidence bearing upon the problem of pathogenesis has appeared in papers by Critchley (1943), White (1943, a and b), Ungley (1943, b), Blackwood and Russell (1943), and Blackwood (1944). Only three main causal factors require consideration:—

1. *The Direct Effect of Cold upon the Tissues.*—The effect of cold upon tissues *in vitro* was extensively studied by Lake (1917 and 1942), and more recently Sir Thomas Lewis has supplemented these researches by numerous clinical observations (Lewis and Love, 1926; Lewis, 1930, 1941, and 1942). For isolated tissues there is a critical temperature level in the neighbourhood of -6°C. , at which irreversible changes, probably of a physico-chemical nature, occur; after cooling to this level recovery is impossible. Intact tissues possess the property of super-cooling, and by virtue of this may be cooled to a lower level before actual freezing occurs. In frost-bite freezing has occurred; there is a central zone of devitalized tissue surrounded by a zone in which tissue damage is minimal. Immersion foot is due to chilling of the tissues without freezing. Opinions differ as to the amount of damage that may be done by cooling to temperatures within the range (-1.9°C. to $+15^{\circ}\text{C.}$) which causes immersion foot. Indeed, chilling of limbs to similar temperatures is now being practised as a therapeutic measure in the treatment of gangrene and peripheral sepsis (Allen, 1939). The argument which is advanced by those who believe that the effects of cold of this degree are *per se* harmless to the tissues, rests largely upon the results obtained by Lake (1917, 1942) in his tissue cultures. As the scale of temperature is descended, anabolism ceases before katabolism. In the zone between 25°C. and 10°C. the products of tissue katabolism accumulate. At temperatures about zero metabolism ceases and the tissues requiring neither oxygen nor nutriment

cannot therefore suffer from ischaemia. These observations suggest that damage to chilled limbs occurs not while the temperature of the tissues is low, but while the temperature is falling or rising, through the zone of imbalanced metabolism between 10°C. and 25°C. Metabolites which accumulate during this phase are responsible for the initial intense local vasodilatation which follows; exudation results and is responsible for the neurological and nutritional lesions. It is doubtful if this hypothesis based upon observations on isolated tissues is applicable to the human limb. The pathological studies of Smith, Ritchie, and Dawson (1915), Blackwood and Russell (1943), and Blackwood (1944) suggest that direct damage to tissues occurs during the period of low temperature.

The effect of cold upon the blood-vessels is of supreme importance. Both local and general cold cause peripheral vasoconstriction, and if these are operative together, as they are under the circumstances in which immersion foot occurs, the resultant vasospasm may be severe. In the Raynaud phenomenon vasoconstriction is sufficient to cause complete vascular stasis in a digit. It is therefore justifiable to assume that in immersion foot vasoconstriction is of itself capable of cutting off the blood-supply to the chilled extremity. Unless metabolism ceases during this phase, at least a partial ischaemia must occur.

Thrombosis need not be invoked. In experiments upon general hypothermia, Talbott (1941) found that although for many hours peripheral pulses might be impalpable and blood-pressure unrecordable, yet thrombosis did not occur. This is presumably true of immersion foot. There is no evidence that organic arterial obstruction is a factor in the causation of the syndrome. The work of Greene (1943) and certain Scandinavian workers (Bigelow, 1942) has shown that even in true frostbite, thrombosis does not occur, although the lumina of the vessels may become silted up with masses of red blood-cells. In pathological material obtained after chilling, there is no organic obstruction of main blood-vessels. The clinical and pathological evidence is therefore in favour of the hypothesis that during exposure and in the pre-hyperaemic stage intense vasoconstriction is the predominant feature.

¹ Excerpted from "The Immersion Foot Syndrome," by C. C. Ungley, G. D. Channell, and R. L. Richards, *Br J Surg.* 1946;33:17–31. Reproduced by permission of John Wiley & Sons Limited. Original style, spelling, and punctuation have been retained.

2. *The Effect of Ischæmia.*—The clinical results of ischæmia as seen in conditions such as traumatic arterial spasm and tourniquet paralyses are in many respects similar to those of immersion foot. The extremity which has its blood-supply cut off or seriously reduced for several hours by spasm of the main artery or the prolonged application of a tourniquet, is probably placed in a position somewhat analogous to that of an extremity exposed to cold. In the latter, the deprivation of blood-supply is less acute and an additional effect of cold will be to mitigate or delay changes in the tissues by reducing metabolism. The reactive hyperæmia which follows a period of circulatory arrest has its counterpart in the early hyperæmic stage of immersion foot. The sensory findings in cases of immersion foot are very similar to those recently described by Parkes (1944) in cases of ischæmia. Only the changes in the muscles (Blackwood, 1944) may be said to differ strikingly from the characteristic infarction which is seen in muscles deprived of their blood-supply.

3. *The Effect of Exudation.*—The hypothesis that the symptoms of immersion foot are the result of pressure from the excessive exudation which accompanies the hyperæmia lacks support. There is no other clinical condition in which unrestricted swelling of a limb causes vascular and neurological symptoms comparable to those of immersion foot.

We therefore advance the hypothesis that immersion foot is a peripheral vasoneuropathy resulting from a combination of chilling and partial ischæmia. The most prominent and interesting feature of the syndrome is the vascular disturbance, and it is therefore of importance to consider this further.

1. *Hyperæmic Stage.*—If it is assumed that vasoconstriction is present during exposure and the prehyperæmic stage, then the transition to the hyperæmic stage will occur when the warmth of the body and the proximal portions of the limbs overcome this vasoconstriction. The excessive vasodilatation which is present in the hyperæmic stage may be due to three factors: inflammation with the release of vasodilator metabolites, local damage to vessels, and vasoconstrictor paralysis.

Sir Thomas Lewis (1941) has demonstrated that an aseptic type of inflammation occurs in tissues exposed to cold. The vasodilatation which succeeds a brief period of exposure to cold has been shown by Lewis (1930) to be the result of an axon reflex producing a relatively stable vasodilator substance similar to histamine. A similar vasodilator substance is also produced in ischæmic tissues and is responsible for the phenomenon of reactive hyperæmia (Lewis, 1927). An accumulation of vasodilator metabolites may well boost the initial hyperæmia, but once an active circulation is established, these will

be rapidly washed away from the tissues. Unless substances continue to be released, they cannot be the cause of a vasodilatation, the duration of which is measured in weeks rather than days. Bacterial infection does not play a part in the early stages of the hyperæmic phase, but may be a factor in the presence of blisters, ulcers, and gangrene.

Blood-vessels may be damaged either directly by cold or from ischæmia. There is no histological evidence of gross damage to arteries and arterioles (Blackwood, 1944). The remarkable changes in skin colour on elevation and dependency of the feet and the presence of petechiæ and swelling indicate that the walls of the minute cutaneous vessels are damaged. Some of this damage probably occurs during exposure, and is accentuated by the sudden increase in blood-flow during the onset of the hyperæmia.

In both experimental and human biopsy material severe damage to peripheral nerves is present (Blackwood and Russell, 1943; Blackwood, 1944). In the main nerves of the foot the majority of the fibres are degenerated and any which escape are of large calibre. Sympathetic vasoconstrictor fibres are of small calibre, and although affected late in ischæmia, are relatively susceptible to cold (Bickford, 1939), and are therefore unlikely to escape. The blood-vessels of the foot are thus subjected to a form of post-ganglionic sympathectomy. A week after exposure skin-temperature gradients from affected limbs are very similar to those from sympathectomized limbs. The standard sympathectomy performed for the lower limb is predominantly preganglionic, and it is unwise to strain this comparison. Complete division of the sciatic nerve results in the interruption of all post-ganglionic sympathetic fibres to the foot (except for a few in the long saphenous nerve). Such lesions produce a complete vasomotor paralysis in the toes, resulting in a foot initially warm and pale, but later varying in temperature with that of the environment. The duration of the initial hyperæmia is of the order of 21 days, and there is not the wide fluctuation in the duration of the hyperæmia that is seen in cases of immersion foot. After the initial intense hyperæmia, moderately severe cases of immersion foot show a partial recovery of vasoconstrictor tone and a small rise or fall of temperature in response to immersion of the arms in hot or cold water. The vasomotor disturbances of this group are similar to those described by Wilkins and Kolb (1941) in cases of polyneuritis, and to those found by Richards (1944) in cases of incomplete division of the sciatic nerve.

These findings suggest that vasoconstrictor paralysis is an important factor in causing the continued hyperæmia of immersion foot. This hypothesis is further supported by the demonstration that the digital vessels

are abnormally sensitive to circulating adrenaline and to the effects of local cold; both these phenomena are known sequelæ of postganglionic sympathectomy (Grant, 1935–6; White and Smithwick, 1942).

2. *Post-hyperæmic Stage with Cold Sensitivity*.—The cold sensitivity of the late stages of immersion foot cannot be entirely explained by the recovery of tone in vessels remaining denervated. Contributing factors are disuse (the increased muscle blood-flow which accompanies even slight exercise and warms the overlying skin will not occur in denervated muscles), and the absence in denervated skin of the axon-reflex (Lewis, 1927 and 1930) which is responsible for the local vasodilator reaction to cold and normally comes into play at temperatures below 15–18° C.; these and other factors in the causation of coldness following peripheral nerve lesions are discussed by Richards (1943) and Doupe (1943).

There remains the difficulty that coldness and cold-sensitivity persist in some cases of immersion foot where observations on sensation and sweating indicate that a large measure of nerve regeneration has occurred. Here three further mechanisms may be involved:—

1. Exposure to severe cold, whether dry cold or wet cold, sensitizes the peripheral blood-vessels so that thereafter they are more susceptible to the effects of milder degrees of cold. The mechanism is obscure, but is presumably similar to that responsible for the Raynaud phenomenon. In the latter the digital arteries are sensitized either as the result of repeated exposure to mild degrees of cold, or because of some inherent 'local fault' in the arteries themselves.

2. Intense vasoconstriction of sympathetic origin might account for persistent coldness of the extremities and failure of reflex vasodilatation. The presence of excess sweating is suggestive of increased sympathetic activity. Reflex vasodilatation, however, fails to occur even when the initial temperature of the feet is fairly high (Gaylor, 1943).

Failure of reflex vasodilatation or a gradual rise in temperature might be the result of occlusion of the main arteries. This can be excluded in the majority of cases because the peripheral pulses are of good volume. Patency of the arteries of the foot in one case had been demonstrated by arteriography. In histological material, Blackwood (1944) has failed to find occlusion of arteries except in areas immediately proximal to gangrenous tissues. The possibility that smaller vessels of the order of arterioles are occluded requires further consideration. In biopsies from cases of immersion foot four months after exposure, White and Warren (1944) found extensive fibrosis of subcutaneous tissue and superficial muscle. They state: "The arterioles and venules show partial to almost complete occlusion as a result of a great increase

of the fibrous tissue in their walls. The arteries and veins of larger calibre show the same type of fibrous thickening of the wall, but with a lesser degree of occlusion of the lumen." If these findings are confirmed, they would be quite sufficient to account for a failure of reflex vasodilatation. It may further be assumed that the constriction of nerve-endings by interstitial tissue and collagen, which White and Warren suggest is the cause of the late pain in immersion foot, will affect vasoconstrictor nerves, thus interfering with normal vascular responses. On the other hand, after failure of reflex vasodilatation there may be an excellent vasodilatation in response to the local injection of histamine. According to Lewis (1927), this response is the result of an arteriolar dilatation.

3. An alternative explanation has been put forward by one of us (Ungley, 1943, b). Cold sensitivity is observed at a time when observations on sensation and sweating suggest that a certain amount of regeneration of damaged nerve-fibres has taken place. Partially re-innervated cutaneous blood-vessels may behave like partially denervated vessels, and react excessively to chemical vasoconstrictors (adrenaline, sympathin) circulating in the blood-stream or produced locally.

In conclusion, it may be stated that the available evidence favours the hypothesis that the initial hyperæmia of immersion foot is the result of the release in chilled and partially ischæmic tissues of relatively stable vasodilator substances, and that once the effect of these has subsided the hyperæmia is maintained because there is a paralysis of peripheral vasoconstrictor nerve-fibres. The late vascular phenomena are more difficult to explain, but it is assumed that they are the result of denervation and the subsequent re-innervation of peripheral blood-vessels, which may also acquire a sensitivity to cold analogous to that seen in the Raynaud phenomenon.

PREVENTION AND TREATMENT

Official instructions (*M.R.C. War Memorandum No. 8*) have been issued regarding the procedure to be adopted by men who abandon ship in latitudes where immersion foot is liable to occur. There is no need to repeat these here, but a word may be said regarding footwear and the advisability of rubbing the feet with oil. Footwear affords some protection during short exposure, but over long periods boots may constrict swelling feet and impair circulation. A booted foot often suffers more damage than a bare foot. Rubber boots are no better than leather ones; even if not filled with water, the feet soon become wet from condensed perspiration. If possible, wet socks should be changed for dry ones, and, as soon as boots begin to feel tight, they should be removed and

not replaced. Lewis and Love (1926) have shown that greasy skin supercools to a greater degree than dry skin. For this reason, and because of a traditional belief in the efficacy of fats as insulators, rubbing the exposed limb with any available oily substance has been advocated as a prophylactic measure. In the early stages of exposure this procedure is harmless and may help the peripheral circulation, but once the tissues become œdematous and friable, even gently rubbing may be harmful. The wearing of stockings impregnated with vaseline is probably advantageous.

In discussing the treatment of immersion foot, confusion has arisen because methods which are of value in one stage have been wrongly employed in another. Therefore it is advisable to consider the treatment for each stage separately.

Pre-hyperæmic Stage.—When rescued, the survivor must be carried; he must not be allowed to walk on damaged feet. Whether the extremities are chilled or actually frost-bitten may not be evident at once, but the principles of treatment are in any case the same at this stage. The first step is to warm the patient while protecting the affected extremities from direct heat. Stripped of wet clothing, his body is wrapped in blankets, leaving the affected limbs uncovered. He may be given hot drinks, but not left near a fire. Hot bottles may be placed near the trunk.

The feet are raised on pillows, exposed to the air, and kept dry. Massage is contra-indicated. Sulphanilamide powder may be applied to blisters and abrasions. Acriflavine is often used, but it stains the skin and obscures colour changes which should be observed.

Should the chilled limbs be warmed, cooled, or left exposed to room temperature? The fact that warmth greater than that of the human body is harmful to frozen or chilled limbs has been known for centuries. Clinical and animal experimental observations amply confirm this traditional belief.

According to some workers even gentle warmth is contra-indicated, because it increases exudation which may be in itself harmful (Smith, Ritchie, and Dawson, 1915; Lewis and Love, 1926; Lake, 1917 and 1942; Greene, 1942). In immersion foot individual variations have been too great to permit judgement of the end-results of gentle warming on rescue. In animal experiments Blackwood and Russell (1943) found that gentle warming (water at 28° C. and incubation at 37° C.) increased the initial tissue reaction but made no difference to histological findings a month later. Brahdy (1935) did not find that very slow thawing was important; many men with apparently severe frost-bite went into warm rooms without bad effect. Certain Russian workers (Girgolav at [sic] al., 1944) have discarded the old method

of gradual warming (rubbing with snow, etc.) of frozen tissues and recommend instead rapid warming without overheating.

Greene (1942) considers that the treatment of frost-bite and of “mere chilling”—trench foot and immersion foot—should be identical, and that from the beginning the parts should be kept cold, in the region of 2° C. to 5° C. He argues that temperatures in this region are optimal for the survival of isolated tissues in culture and of ischæmic limbs; and that even gentle warming dangerously increases the metabolism of ischæmic tissues, increases exudation, and encourages the growth of bacteria. He reasons that once the patient is warm and at rest in bed, vasoconstriction will be slight or absent. It is true that the change of environment brings about a reduction or cessation of vasoconstrictor stimuli initiated reflexly or through the circulation of adrenaline in response to cold and emotional stress. It must be remembered, however, that cold has a powerful *local* effect; arteries which are in spasm from the direct action of cold upon them are not likely to relax until they themselves are warmed. Thus keeping the extremities cold may have the unfortunate effect of prolonging the stage of vasospasm and ischæmia. Lewis (1942) has shown that temperatures below 15° C. are harmful to tissues.

The treatment advocated by Greene has been tried in high altitude frost-bite; Davis et al. (1943) treated a series of patients by keeping the parts at approximately 2° C. to 5° C. for 24 to 48 hours after the injury had been sustained. Although swelling and blistering were less, the end-results were rather worse than when the hands were allowed to warm up naturally to room temperature.

Until we have more evidence as to the relative merits of methods so at variance as cooling to 2° C.–5° C. on the one hand and rapid warming to 37° C. on the other, we advise an intermediate course. The degree of warmth applied should be no greater than that needed to permit relaxation of the arteries; exposure of the extremities to a cool atmosphere (18° to 22° C.) appears to be logical and, in our experience, a satisfactory method. The feet are protected by a cradle beneath which is placed a bath thermometer. The environmental temperature in relation to the feet may be adjusted by altering the room temperature, or, if this is too low, by covering the cradle with blankets and allowing warm air from beneath the bedclothes to enter at the proximal end.

Moderate warmth, in still air, will take a long time to penetrate severely chilled extremities. In cases treated by one of us (G. D. C.) aboard a destroyer, feet exposed to 12° to 15° C. became hyperæmic within five hours, but sometimes feet remain cold as long as two days.

Interruption of the sympathetic outflow by operation or infiltration has been recommended in the treatment of

trench foot and frost-bite by certain continental observers, but the findings of Lake (1917; 1942) and Greene (1942) suggest that the procedure is valueless and even harmful at this stage. Davis et al. (1943), however, claim that in high-altitude frost-bite blocking of the sympathetic trunk will effect dilatation of the peripheral capillary bed, provided that there has not been permanent anatomical injury to the capillary wall or thrombosis at the arteriolar capillary junctions. They found the following drugs did not release vasoconstriction: amyl nitrite, nitroglycerin, alcohol, aspirin, nicotinic acid, mecholyl. It must be emphasized that any efforts to hasten the release of vasoconstriction, whether by body warming, local warmth, sympathetic infiltration, or drugs, may do harm unless steps are taken to control the degree of vasodilatation when it occurs.

To sum up: In the pre-hyperæmic stage the patient's body should be warmed while the affected extremities are elevated and kept cool (18–22° C.). At present there is insufficient evidence to enable a categorical statement to be made regarding the therapeutic value of cold or the employment of measures destined to cause peripheral vasodilatation.

Hyperæmic Stage.—To limit the excessive exudation set up by a too rapid return of circulation, several measures have been suggested. In animals, Lake (1917; 1942) found that tying the femoral artery or injecting cocaine and adrenaline, diminished exudation and tissue damage dependent thereon. For frost-bite in man, Lewis (1941) recommends intermittent compression of arteries to allow blood to return little by little to the limb, which in the meantime is kept cool. White (1943, a) suggests that compression dressings might be useful. Treatment by cooling alone, as described by Webster et al. (1942) is often sufficient, and should begin as soon as vasodilatation develops.

Recent experimental evidence (Safford and Nathanson, 1944) indicates that the optimum cutaneous temperature for prolonged therapeutic cooling is 70° F. (21° C.), and that this temperature may be maintained for hours or even days if need be.

When hyperæmia is intense even ice-bags do not reduce the skin temperature below 80° F. (26.6° C.). Nevertheless, pain is relieved and within four hours the patients are comfortable; œdema usually subsides rapidly and blisters resorb. Too early removal of the ice-bags is followed by increasing œdema and even by extravasation of blood. Details of the method are given by White (1943, a). Instead of ice-bags, some form of cooling cabinet may be used (Webster et al., 1942; Greene, 1942; Bigelow and Lanyon, 1944; Eve, 1944).

In milder cases or when hyperæmia has become less intense, it will be sufficient to expose the feet to a cur-

rent of air from a fan in a cool room, 15° to 18° C. For fan cooling to be effective, the greater the degree of hyperæmia, the lower must be the room temperature. If slightly greater cooling is desired, water may be sprayed through the fan with an atomizer. The speed and distance of the fan can be adjusted to maintain skin temperatures of 23° to 26° C.; in practice further cooling is found to cause discomfort (Ungley, 1943, a). Where a fan is not available, the patient may be nursed on his face with feet and legs uncovered and elevated by an open window (Learmonth, 1943). In moderate cases when œdema has subsided, neither exudation nor necrosis follow interruption of cooling, and return to a higher skin temperature is not necessarily associated with an immediate return of pain and tingling. In these circumstances the fan may be used intermittently, when symptoms call for it; in the intervals the feet remain uncovered exposed to room air. The mode of action of treatment by cold requires further investigation. Webster et al. (1942) suggest that by reducing metabolic requirements, cooling overcomes a relative anoxia in the hot tissues. White (1943, a) believes that anoxia of nerve-endings is responsible for the early pain in immersion foot, and that the relief afforded by cooling is a direct result of a more balanced metabolism. It has yet to be determined whether prolonged cooling will influence the rate of recovery of the nerve lesions.

Pain, if not relieved by cooling, may call for codeine or morphine. Sedation may be needed for patients with anxiety states, insomnia, and nightmares.

The diet should, of course, be plentiful and contain an abundance of protective foods. Nutritional deficiencies should be treated if present, but in our experience intensive treatment with B₁ and other vitamins does not influence the course of the disease.

The feet must be kept scrupulously clean. After daily cleansing with a solution of weak dettol and liquid soap, they should be dried with spirit and dusted with sulphamylamide powder. Blisters are snipped and any loose epidermis removed. The toes are separated by small pieces of gauze and the feet wrapped in a sterile towel. Since gangrene is nearly always superficial, any question of amputation should be deferred until a definite line of demarcation has formed. Minor amputations of the 'trimming' variety are usually all that are required, but occasionally severe pain will necessitate a more radical amputation than would otherwise be necessary.

Post-hyperæmic Stage and Late Sequels.—Patients suffering from immersion foot must rest in bed until all swelling is gone and they are able to walk without pain. White (1943, a) suggests that patients should perform Buerger's exercises for a week before being allowed up. Early walking may cause excoriation, blistering, or increased swelling. Efforts should be made to maintain the

tone and bulk of denervated muscles and to prevent deformities such as flat-foot. Special boots and pads may be required. Learmonth (1943) suggests the use of arch supports until power has been restored to the small muscles of the foot.

Cold-sensitive states demand warm covering not only for the affected extremities, but for the rest of the limbs and for the trunk. Occupational therapy and exercises maintain muscular activity and lessen coldness from disuse. Smoking should be forbidden if there is a tendency to vasospasm. Hyperhidrosis can usually be countered by local measures such as foot-baths and dusting powder,* or by the administration of atropine.

It is in this stage that sympathectomy or sympathetic infiltration may prove of value in the treatment of immersion foot. Telford (1943) reports a case in which 7 months after rescue the feet were swollen, stiff, and hyperhidrotic, with chronic painful ulcers on the stumps of the toes. After a bilateral lumbar sympathectomy, pain was relieved and the ulcers healed rapidly.

Late amputation is occasionally necessary to trim awkward stumps, to remove stiff contracted toes, or as a preliminary to fitting artificial limbs. When pains are persistent and severe, White (1943, a) suggests crushing the peripheral nerves at ankle level; the pains are said not to recur when the nerves regenerate, a process which takes about three months. Some patients claim benefit from treatment with short-wave diathermy and other forms of heat, which at this stage is not contraindicated. Instructions to convalescents concern selection of footwear, frequent change of socks, use of dusting powder, and avoidance of excessive local heat and of cold and wet. Extremities once damaged by chilling are more than ordinarily susceptible to further damage by cold.

SUMMARY

1. Prolonged exposure of the extremities to cold insufficient to cause tissue freezing produces a well-defined syndrome. 'Immersion foot' is one of the descriptive but inaccurate terms applied to this syndrome. The clinical features, aetiology, pathology, prevention, and treatment of immersion foot are considered in detail. A discussion on pathogenesis is also included.

2. In the natural history of a typical case of immersion foot there are four stages: the period of exposure and the pre-hyperæmic, hyperæmic, and post-hyperæmic stages.

3. During exposure and immediately after rescue the

feet are cold, numb, swollen, and pulseless. Intense vasoconstriction sufficient to arrest blood-flow is believed to be the predominant factor during this phase.

4. This is followed by a period of intense hyperæmia, increased swelling, and severe pain. Hyperæmia is due to the release in chilled and ischæmic tissues of relatively stable vasodilator metabolites; pain may be the result of relative anoxia of sensory nerve-endings.

5. Within 7–10 days of rescue the intense hyperæmia and swelling subside and pain diminishes in intensity. A lesser degree of hyperæmia may persist for several weeks. Objective disturbances of sensation and sweating and muscular atrophy and paralysis now become apparent. These findings are correlated with damage to the peripheral nerves.

6. After several weeks the feet become cold-sensitive; when exposed to low temperature they cool abnormally and may remain cold for several hours. Hyperhidrosis frequently accompanies this cold-sensitivity. The factors responsible for these phenomena are incompletely understood; several possible explanations are considered.

7. Severe cases may develop blisters and gangrene. The latter is usually superficial and massive loss of tissue is rare.

8. The hands may be affected but seldom as severely as the feet. The essential features of immersion hand are the same as those of immersion foot.

9. Prognosis depends upon severity. The extent of anaesthesia at 7–10 days has been found a useful guide to the latter, and has formed a basis of a method of classification.

10. Rapid warming of chilled tissues is condemned. Cold therapy is of value for the relief of pain in the hyperæmic stage, but should not be used in the pre-hyperæmic stage. Sympathectomy and other measures designed to increase the peripheral circulation should not be employed immediately after rescue, but may have a place in the treatment of the later cold-sensitive state.

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* Sarkisov-Serasini (1941) recommends frequent washing with soap and water, followed by dusting with a powder consisting of salicylic acid 2, alum 8, lycopodium 5, and talc 20 parts.

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