AETIOLOGY OF "IMMERSION FOOT"

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Shipwrecked men adrift on rafts, at times half covered with sea-water, are subject to the lesions of "immersion foot" and "immersion hand." The war has obviously caused a great increase in the number of these cases, yet the clinical descriptions available were scanty until Ungley and Blackwood (1942) published their careful observations. We have now seen four parties of men rescued after many days at sea in a half-soaked state. These cases showed features that surprised us; especially the fact that the lesions had occurred at a time when the temperature never reached freezing-point, and in one instance during the summer. This was contrary to all our preconceived ideas of the lesion, which was previously assumed to be similar to a slow frost-bite. Our purpose in this paper is (i) to confirm the clinical picture as established by Ungley and Blackwood (1942); (ii) to present a conception of its aetiology; (iii) to describe a remarkable complication.

There were four parties of rescued men. The numbers were respectively 9, 1, 1, and 10. Nearly all showed roughly the following syndrome, some having interesting complications which will be described afterwards.

Swollen Stage

This stage was present soon after they were taken from the water. The hands and feet were greatly swollen and the skin was shiny. The extremities showed muscular paresis and there was complete anaesthesia both of hands and of feet; in some cases the anaesthesia extended as high as the middle of the lower leg. Pain was not a marked feature at this stage. There was pyrexia for a few days.

Stage of Diminishing Oedema

After about a week the hands and feet shrank. The skin was no longer shiny, but wrinkled and dull; anaesthesia was passing off, though it was still present at the extreme distal parts—e.g., soles of feet. Paraesthesiae were present; these ranged from mere pins-and-needles to agonizing pains which prevented sleep for many nights. These pains often continued for several weeks with diminishing severity. Frequently there was so much tenderness that the patient could not bear the weight of the sheets upon his toes. Even when the lower leg bore no outward mark of any lesion, there was often agonizing pain on pressure at the lower part of the calf and tendo Achillis, and the ankle-jerks were diminished or absent.

It will be seen that the whole clinical picture bears a pronounced resemblance to a condition of peripheral neuritis. The muscular paresis, the anaesthesia, the agonizing pains, and the frequent absence of reflexes all help to build up this resemblance. Throughout this stage the feet are warm and the pulsation of the dorsalis pedis and posterior tibial arteries is good.

Complications

Gangrene.—In the first party of 9 cases gangrene did not occur. It was seen in the third party. In this one case the man had drifted for 15 days, in summer weather, with his feet partially immersed, and gangrene of the fore part of the feet and of the heels occurred. We saw this case only at a very late stage, by the kindness of Mr. McIndoe. There had certainly been gangrene; according to the patient's own statement, tingling followed by numbness began on the third or fourth day, and thereafter he noticed the gradual onset of blackness of the feet. There was no pain, but he was aware of what was happening. Gangrene probably became certain about the seventh or eighth day. The fourth party—10 men—had been waterlogged for 80 hours in very mild weather. Of these, one man had only slight superficial gangrene of the skin of the knuckles on one hand. This hand had been continually grasping a rope of the raft without letting go. It was quite obvious that the gangrenous patches corresponded to the whitening of the knuckles (and consequent ischaemia) that would result from the continual clutching. We feel that gangrene is not an essential or even a common feature of the typical lesion, and would appear to occur under exceptional conditions which would kill any normal man long before the onset of foot gangrene.

Salt-water Rash.—This occurred in two of the cases. It consisted of minute boils spread over the skin. It appeared only on the chest, and we have not yet seen it in relation to the typical lesion of immersion foot. Its interest lies in the manner in which the toxic

action of the salt water on the skin is shown in its fullest form—as apart from the action of cold.

Duodenitis.—We do not know how common this complication is, but as it certainly occurred in one, and possibly two, of our series, we shall describe it. The first patient, about a week after his rescue, began to pass altered blood and then bright blood per rectum. He rapidly became exsanguinated. A diagnosis of possible bleeding duodenal ulcer was made, although no previous history of ulcer could be obtained. His condition gradually became worse, and in spite of transfusions he died. The following is an extract of the necropsy report:

"The stomach contained a little mucoid fluid and had pale walls. The whole of the small intestine and much of the colon were filled with a dark tarry faecal material. There was no tumour or ulcer visible in the wall, but the whole of the duodenum was deeply engorged and oedematous—obviously the origin of the fatal haemorrhage. The liver weighed 70 oz. and had a smooth grey surface. The substance was soft, and showed bile staining around many of the intrahepatic channels. The kidneys weighed 11 oz. Their capsules stripped from a smooth pale surface. On section, differentiation of structure was poor and there was some cloudy swelling. . . . In my opinion the cause of death was: (a) small-intestinal haemorrhage; (b) acute toxic hepatitis and nephritis; (c) chilling of both legs from prolonged immersion."

The second patient, apart from any leading questions, complained of fairly severe epigastric pain $1\frac{1}{2}$ to 2 hours after meals. He had never before had dyspepsia, and the type of his pain is suggestive of duodenitis.

Discussion

Greene (1941) stated: "I gather that clinically it [immersion foot] is almost identical with trench foot." He also stated that "trench foot is almost identical with gradual frost-bite." We wish to draw a clear distinction between the two conditions (frost-bite and immersion foot) for the following reasons:

Lewis and Love (1926) have shown that very probably the essential feature in production of frost-bite is actual freezing of the skin. They demonstrated that if the freezing-point of the skin could be depressed (as by inunction with oil) it would be relatively protected from frost-bite. They showed, in fact, that it would need to fall to -2.2° C. at least for the skin to freeze. In the evolution of our cases the temperature never fell below zero, according to the cases at our disposal. We stress this point because a clear-cut phenomenon like freezing most probably changes the living cell immediately: its metabolism would cease, and its membrane must run the risk of bursting by reason of the sudden formation of ice within it. As Lewis and Love (1926) state, "The hardening and firm union of [ice] crystals, at first separate, is an event especially calculated to produce cellular damage, and it is to cellular damage that the after-effects must be ascribed, for they are in all respects similar to those produced by mechanical injuries delivered from outside—for example, the lash of a whip."

The aetiology of immersion foot is a gradual chilling, never frost. This chilling produces a vicious circle when the familiar protective reflex of local arteriolar spasm is superadded. Thus all tissues soon begin to suffer from a combined insult—cold and relative ischaemia. The weakest tissue would suffer most. It is logical to suppose that the biologically most delicate tissue would show the lesion first. Nerve tissue is probably the most delicate, hence the syndrome of peripheral neuritis is the predominant clinical feature. Support for this view is especially furnished by two of the cases. In both of these the tissue of the lower leg revealed no sign of skin damage, yet both showed signs of peripheral neuritis: one had anaesthesia of the lower calf, and the other great tenderness on deep pressure of the calf and an absent ankle-jerk. Since originally writing this we have read Ungley and Blackwood's (1942) paper. Their microscopical observations on post-mortem material confirm this point—namely, that Wallerian degeneration was the outstanding lesion.

It is obvious that if the chilling process is sufficiently prolonged the metabolic handicap would become intolerable to each tissue in turn. Thus each tissue, in the order of its strength, would successively fail in its function, then die. Finally, the lowest and most tolerant of all tissue (connective tissue and tendon) would fail to survive the more organized tissues. The whole part would then be gangrenous, as in the case of one of our patients who was 15 days on a waterlogged dinghy. Probably few men could survive this long test, and gangrene is therefore a rare occurrence in the disease of

immersion foot. Is cold, then, directly the major factor in the metabolic handicap, or is the relative ischaemia—an indirect result of the cold—principally responsible for the lesion? Both views are considered by Ungley and Blackwood (1942), who fully discuss the two theories. It seems more probable that ischaemia is the responsible agent.

The aetiology of frost-bite is quite different. In its purest and most dramatic form it occurs suddenly, as in the case of Greene's (1940) experience with a camera shutter. He states: "The contact of a momentarily ungloved finger with the button of a camera in a high wind at a temperature below zero Fahrenheit and at a height of about 25,000 ft. once cost me a finger-tip." Here we have a sudden mechanical accident. It produces the familiar appearance (like a localized infarct) of frost-bite. Within this area all tissues, irrespective of their biological resistance, are dead. In the mechanical explosion the strong have perished with the weak.

Of course there must be mentioned forms in which frost-bite becomes superadded to immersion foot. We have never seen trench foot, but in this there would be an opportunity for a combination of the two syndromes: a succession of rain, mud, and slush would give the prolonged chilling sufficient for the development of immersion foot; and, as Lewis and Love (1926) have shown, by the washing away of skin oils would, with the advent of frost, predispose the tissues to the development of frost-bite if the temperature suddenly fell below freezing-point.

Other Complications

Salt-water Boils.—It might be objected that the syndrome we describe could have been caused by either the chemical or the osmotic action of the sea-water and not by the chilling effect. However, the location of salt-water boils on the chest in two of our cases should answer this objection. The chest, by reason of its rich blood supply and high mass-surface ratio, is not liable to cooling, and would therefore show a pure result of toxic action as apart from chilling. With regard to osmotic action, sea-water is about $2\frac{1}{2}$ times as strong osmotically as human tissues. No osmotic action could have occurred through the skin, otherwise the tissues would have shrunk and not swollen.

Duodenitis.—Nearly all our patients had pyrexia in the early stages; they were not infected. This must have been non-specific traumatic fever, caused by the absorption of toxins from traumatized areas. Curling's ulcer of the duodenum occurs as a result of absorption of toxins from burnt areas. The similar aetiology of Curling's ulcer and our case of duodenitis is certainly striking, but it would be beyond the scope of this paper to speculate on the mechanism of this duodenitis.

Treatment

From time immemorial the danger of rapidly warming frost-bitten areas has been recognized. Greene (1942) has given a scientific basis to this idea by freezing a rat's tail and then thawing it in front of a fire. This caused the loss of nearly twice the tissue that would otherwise have occurred. Smith, Ritchie, and Dawson (quoted by Greene, 1940) suggest that it may be due to rupture of capillaries previously damaged by frost. Lewis and Love (1926) suggest that the presence of histamine-like substances causes an excessive and dangerous transudation.

It might be thought that these considerations were not specially applicable to cases of immersion foot. But Ungley and Blackwood (1942) sound a word of warning against the use of heat. They quote the experiment of Allen on chilled ischaemic rats' tails. These survived much longer than the unchilled controls. He suggests, therefore, that in a recent case of immersion foot it is dangerous to warm the part before circulation has returned: warmth raises its metabolism to a point beyond the capacity of the feeble circulation to supply it. Thus the tissues are rapidly brought to a state of metabolic bankruptcy, and necrosis occurs.

It should be noted that the theoretical objections to warmth in the treatment of frost-bite are based on the vasodilator effect of heat.

We refer again to Lewis and Love's experiment in which vesicle formation on warming a frost-bitten part was minimized

by compressing the main limb artery. Smith, Ritchie, and Dawson (quoted by Ungley and Blackwood, 1942) stated that actual rupture of frost-bitten capillaries occurred with too forceful vasodilatation. The objection to heat in cases of immersion foot, however, is somewhat different. The rise of temperature is considered undesirable because it raises the metabolic rate of the tissues out of proportion to their diminished blood supply. Therefore an increased vasodilatation, undesirable though it may be in frost-bite, is appropriate here. In this way the blood supply is adequately increased to keep pace with the enhanced metabolism.

There would here seem to be a field for the cautious simultaneous use of two separate elements in treatment: (1) local application of cool air to the damaged feet; (2) measures for active local vasodilatation. A mild type of local vasodilatation could be obtained by warmth applied to the whole body while the damaged feet were simultaneously kept cool to maintain their low metabolism. The French school have practised more intense measures for obtaining vasodilatation in the affected feet: the lumbar sympathetic chain is injected with a local anaesthetic. Stricker and Buck (1940) give a detailed report of two cases in which this treatment was carried out. They claim an immediate and remarkable improvement in the damaged limb; it becomes warmer, less dusky, and much less painful. Unfortunately, in their cases they make no clear distinction between trench or immersion foot and frost-bite. It should be noted that they started their treatment one day after rescue. English opinion is at present against measures for active vasodilatation; and, indeed, the average mild case will get better with the general treatment advocated. But with really severe cases it may be worth while cautiously to try the combination suggested by these theoretical considerations. We have not yet had the opportunity. After the first few days of illness most of our patients were allowed a heat-cradle to their feet. When the pain became severe they begged for the cradle; it relieved the pain.

In cases of older people with a weakened detoxicating action of the liver it would certainly seem worth while to prevent erosion of the duodenum by placing them on a peptic ulcer regime for about a week. The parietic condition of the feet should also receive consideration: with weakened neuromuscular apparatus in the foot, there is also the danger of acute flat-foot. Patients should not be allowed to bear weight too early, and severe cases should receive faradization and special exercises to the foot muscles as a preliminary to walking.

Summary

The syndrome of immersion foot is described. Commonly it bears a striking resemblance to peripheral neuritis.

It appears that the disease may be regarded as a massive chilling and ischaemia of all the various tissues in the foot or hand. These are successively affected in the inverse order of their biological fortitude. Thus the clinical picture may vary from a mere peripheral neuritis to the rare stage of gangrene. Most patients are rescued in the stage of skin and nerve damage. They would probably rarely survive to reach the stage of gangrene, but one case of gangrene is described.

The syndrome is in sharp contrast to that of frost-bite, in which by reason of a mechanical accident (ice-crystal formation) all tissues involved in the area suffer simultaneous death irrespective of their relative hardihood.

Treatment of the two diseases is not the same: measures for active vasodilatation are under certain circumstances indicated in immersion foot, but are dangerous in frost-bite. However, both types of lesion should be kept cool in their early stages.

Mixed forms of the two diseases are postulated.

The complication of duodenitis with duodenal haemorrhage is described. It is suggested that this may be due to the absorption of toxins from the damaged area. The similarity with Curling's duodenal ulcer in burnt patients is noted.

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