LESSONS FROM HISTORY

C. C. Ungley's "The Immersion Foot Syndrome"— *A Commentary*

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I have chosen to review this article by Ungley et al on immersion foot syndrome because it is a ground-breaking comparison with trench foot. These nonfreezing cold injuries were often thought to be the same as frostbite. They have been of paramount importance to military campaigns throughout history. Ungley et al make a strong argument that the freezing injury of frostbite is different from that of both trench foot and immersion foot, but the latter two are essentially the same injury. Immersion injury occurs by direct immersion in water from sitting in a water-logged life raft or hanging in a life-jacket. Trench foot occurs on land from wet boots and socks.

The authors' eloquent description of the clinical picture of the stages of injury has never been improved upon. Each stage has clear clinical characteristics that define the sequence of cellular damage. The damage of both nerves and vasculature results in the symptomatology that is seen. In 16 short paragraphs, the entire injury is clearly described. The statement that "the most significant factor in prognosis is the amount of damage to peripheral nerves" is insight not previously noted by other authors. The authors liken the injury to long-term tourniquet release, which we now describe as reperfusion injury. Ungley and colleagues coined the phrase *peripheral neuropathy after chilling* to describe immersion foot.

Ungley et al outlined the etiological factors of malnutrition, exhaustion, seasickness, dehydration, and immobility as playing a role in producing this injury. They

also identified the "colored races" as being more prone to nonfreezing cold injuries and cited tight clothing and boots, along with dependent limbs, as contributing factors.

The authors list 2 key factors in the pathogenesis: the direct effect of cold and ischemia. Cold causes peripheral vasoconstriction, sludging, and an increase in metabolites. Ischemia deprives the tissues of oxygen. The detailed description of the impact both factors have on nerve and muscle function is well worth reviewing.

The description of treatment by stages begins with warming the patient but protecting the feet from direct heat. Patients need to be well hydrated as the feet are warmed at 18°C to 22°C. If too warm, greater damage with significant pain occurs; if too cool, return blood flow is slowed. Later, during the hyperemic phase, mild cooling of the feet with a fan relieves much of the pain. The feet must be kept clean, and topical antibiotics may be useful. Delayed surgical procedures and repairs along with intensive physiotherapy are required. Specialized footwear aids in the return of functional gait.

Although Ungley and coworkers suggested surgical sympathectomy to relieve hyperhydrosis and cold sensitivity, we would not opt for that approach today. The long-term problems of sympathectomy outweigh the short-term benefits.

It is testimony to the thoroughness and completeness of this article that 60 years later few improvements in management of nonfreezing cold injuries have been made. This work is still the most definitive description of these difficult injuries to date.