TRENCH FOOT

Introduction

Trench Foot has caused havoc on occasions throughout military history. Prevention is possible if medical and other officers understand the factors involved and see that precautions are taken before trouble arises. I make no apology for reporting the present incident. Few medical officers today have seen Trench Foot; many imagine it to be a First World War curiosity unlikely to be repeated (an outlook encouraged by vague comments in several text books); and others (including myself before the present experience) may not appreciate the importance and urgency of preventive action at unit level.

History

Early in January 1978 men engaged in advanced training on the Brecon Beacons dug slit trenches in which they slept fully clothed for two successive nights. The weather was inclement with rain, snow, high winds, low temperatures and occasional frost. Overhead cover was added to the trenches on the second night.

Symptoms

Nineteen men — over 25 per cent of those at risk — developed painful feet. When questioned two weeks later many said they had suffered from cold or numb feet during the exercise. The significance of these answers to leading questions is doubtful. Within days however, definite symptoms developed — “pain in the feet”, “shooting pains”, “burning and feeling of cramp”, “like electric shocks”, “throbbing, could barely walk”. They were in discomfort and limping badly. Some were bedded down for a few days but the majority managed to drag along.

Examination

Between the 13th and 17th day after exposure, all these cases (ages between 18 and 24) reported to the Regimental Medical Officer on account of persisting pain. They were hobbling and most were unable to wear boots. Removal and putting on footwear was a slow and painful business. Pain was mainly in the distal half of the feet, only one had pain around the heels in addition.

Morale and motivation were exceptionally high. Many commented they had “enjoyed the experience” or had “learnt a lot”. The medical officer had the unusual problem of guarding against underestimation of symptoms.

Visual examination of the feet was negative. The colour was good, the skin dry, no maceration, no blisters, no relics of healed lesions, no fungus or other infections. On palpation however the distal half of the foot was exceedingly tender to light touch, stroking, pressure and movement of the joints. “Numbness” was commonly mentioned but only hyperaesthesia was found at this examination. Likewise there were no clinical signs of oedema despite complaints of “swollen feet”. The arterial pulse in the foot was only felt in a few cases but there were no suggestions that circulation was inadequate.

Treatment

Some cases were excused all duties (bed down), others were employed on light sitting duties. An analgesic was given to relieve pain particularly at night and
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instructions were given to devise means of keeping the weight of bed clothes off painful feet. Tetanus immunization was checked.

Progress

At this stage it was difficult to assess how long disability might continue. On the 16th and 17th days after exposure, many cases had an unexpected increase in symptoms. This was associated with a relatively sudden local hyperaemia and for the first time the feet looked red and felt hot, as opposed to warm. Some cases attended the local military hospital during the evening on account of severe pain. This was most troublesome at night and I voluntarily placed myself on call. Fortunately this increase in pain subsided in 24 to 48 hours and the addition of Mogadon at night ensured reasonable rest. The following is a typical case:

A private age 18 years, reported at 10.00 hours sixteen days after exposure. The feet were cool and tender to touch and movement, his hands were warm. He was excused duties (bed down) for 24 hours and given analgesic tablets. He again reported the same day at 15.45 hours on account of increasing pain. He was distressed. Both feet were now red and felt hot. The only additional treatment given was Mogadon at night. The following day (17 days after exposure) he said he had "slept like a log" and his feet were less tender. On examination the feet were still hot but less red in colour. He did not report again.

Although concerned at the time, on looking back I regard these symptoms as due to improving circulation and a herald of clinical recovery. Within five days all cases were back on duty and none have reported since.

The unit concerned was warned about the need for vigilance since cases may remain susceptible for a long time and further exposure could cause irreversible changes necessitating downgrading. I recommended that field exposure to wet and cold elements should be avoided until after the next winter.

Addendum

Since this incident I have seen a report in which 150 Gurkha soldiers early in November 1977, on an escape and evasion exercise in Wales involving immobility in ambush, suffered 25 cases (16.7 per cent) similar to those reported above. There was a "50 per cent recovery after two weeks". Changing socks frequently was thought to be an important factor in prevention but the figures given are not statistically significant.

Discussion

The cases described were mild but unit activity was disorganised for some three weeks. As in the first World War, many were labelled "Frost bite" which is not in accordance with present day nomenclature and the two conditions require dissimilar treatment.

The aetiology is complex. Cold conditions — not necessarily freezing — in which heat is constantly drained from the lower limbs, are dangerous. Lowering of temperature by wind on damp surfaces (wind chill index) or by immersion in water (immersion foot) are important factors usually associated with exposed terrain and poorly draining subsoil. Most injuries occur at night.

There is a normal variation in susceptibility to cold which is increased by ill
health, circulatory disease, skin conditions (especially those causing hyperidrosis), some drugs and alcohol (cold injuries are found in young alcoholics sleeping "rough" in the London area during the winter). Important factors are fatigue and immobility for long periods in cramped positions — crouching by day and not properly recumbent by night. Wet and ill fitting footwear with pressure over the toes and heel is a major cause of trouble. Susceptibility after a previous attack must be kept in mind because further exposure may lead to permanent disability.

**Signs and symptoms**

Signs and symptoms are confined to the feet. In mild cases there are no abnormal appearances but there is severe pain and tenderness. More severe cases may have some oedema of the feet and legs and occasional blistering. On rare occasions there may be discoloration of the feet but this does not generally indicate gangrene and the majority recover. During the First World War a minority proceeded to gangrene, tetanus was frequent and complications included thrombophlebitis and dermatitis. Debility may persist for months and is demoralising.

**Pathology**

Under wet and cold conditions indirect reflex arteriolar vasoconstriction takes place to conserve heat. If prolonged by cold, immobility, pressure from footwear and other factors, the linings of blood vessels are damaged and decreased blood supply leads to Wallerian degeneration of nerves and stocking type anaesthesia. Recovery is preceded by hyperaemia and hyperaesthesia. Oedema if present is not usually severe. Mild cases may take months to recover and residual sensory and vascular changes may persist for a year or longer. Further exposure during this period may cause permanent changes. A milder form of a similar condition is seen in children after snowballing and is commonly called the "hot-aches". An hour or so after returning home they develop red and painful hands and a feeling of swelling which may last until the following day.

**Treatment**

Patients need protected surroundings but admission to hospital is rarely indicated. The feet should be washed and abrasions cleaned and dressed (dry dressing). The tetanus immunization state should be checked. If bed is indicated a cradle should be used to prevent pressure from bed clothes and the foot raised if there are signs of oedema. Every three hours grouped patients should be instructed and supervised in exercises to all joints and self massage of all areas from the hip to the toes, except where tenderness is severe. Simple analgesic and hypnotic drugs are given as necessary.

**Prevention**

Anticipatory action is all important. Unit officers should be instructed regarding this hazard and the importance of cold weather vigilance; medical officers should carry out frequent health and hygiene inspections including night examinations of sleeping conditions.

Suitable clothing must be worn and men protected from mud, wet and wind as far as possible and not be over-fatigued or immobilized for long periods. If confined, regular exercises must be devised including removal of boots, friction, foot exercises and change of socks.
Footwear is important. Protection against wet and cold is desired, on the other hand footwear that increases sweating is a hazard (rubber soled sneakers are the worst offenders). The substitution of rubber for leather soles in present day army footwear is a matter which should be watched. Constriction or pressure on the toes or around the heel must be avoided. British army boots proved superior to American military boots in the Second World War. The latter were smart but too well fitted and tended to cause constriction. Trench Foot was rife in the American army but practically unknown in the British.

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HYPOKALAEMIC PERIODIC PARALYSIS COMPLICATING THYROTOXICOSIS IN A NEPALI

Periodic paralysis has been reported to occur in 8.9 per cent of Japanese males and 13 per cent of Chinese males with thyrotoxicosis. The corresponding figures for females were 0.4 per cent and 0.17 per cent. McFadzean and Yeung commented that the high incidence of periodic paralysis among thyrotoxic mongoloids suggests that the basic defect may be genetically determined but that if this is so it is strange that the complication had not been reported among other Mongoloids. This is thought to be the first reported case in a Nepali who was ethnically of a mongoloid tribe (Limbu).

Case report

The patient was a 28 year old Nepali male who had ten days previously been diagnosed as a thyrotoxic and started on treatment with thyroxine 0.05 mg daily and carbachol 15 mg to be taken three times a day (tds). One day prior to his admission on this occasion he complained of weakness in his legs, and a sore throat for which he received penicillin. On the morning of admission he awoke unable to get out of bed and had to be helped to the toilet; that afternoon he was admitted by stretcher to hospital.

Examination revealed a patient so moulded to the bed that his ear on the dependant side was bent forwards the patient being unable to raise his head in order to be more comfortable. He had normal facial movements, phonation and respiration, and was able to flutter his hands from the wrist as well as wriggle his feet and toes; the cranial nerves apart from both spinal accessaries were intact. The triceps jerks were present on both sides but all other tendon reflexes were absent. The abdominal and plantar reflexes were normal. He complained of tingling in his hands and feet as well as ankles and knees. Whilst clinical evidence of thyrotoxi-