in Canada. The main source of supply is Mexico.

The agent is available in both tablet and injectable form. When taken orally the drug seems to have much greater toxic potential, presumably owing to increased conversion to hydrocyanic acid by intestinal glucosidase.

Previous cases of Laetrile poisoning have been so rapidly fatal that serum cyanide levels could not be documented. Graham and associates[7] suggested that levels as low as 112 μmol/l could be lethal, but our patient survived a substantially higher level when given prompt therapy for cyanide poisoning.

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Frostbite arthritis

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Delayed damage to bones and joints is a recognized sequela of frostbite. Harsh Canadian winter conditions can cause frostbite, and the increasing popularity of winter sports has increased the risk of this injury. In approximately half of those affected by frostbite of the hands or feet frostbite arthritis will develop many months or years after the original injury.[1] The characteristic clinical and radiologic features of frostbite arthritis have been well described by others.[1-4] These features, which resemble those of osteoarthritis, are particularly well illustrated by the following case.

Case report

A 29-year-old man had suffered a frostbite injury to his hands at the age of 16 years; on a night when the temperature was −27°C he had consumed an undetermined amount of whisky and acetylsalicylic acid at a party and then lost consciousness while he was walking home. He had been outside for 4½ hours when he was found, semiconscious, and taken to a local hospital. His frozen clothes were cut off and he was immersed in a bath of water at body temperature. Because the frostbite injury to his hands was so severe and extensive (Fig. 1) he underwent skin grafting. The injury to his knees and feet was less severe and healed without skin grafting or sequelae.

The patient's hands were essentially free of symptoms for 7 years, until, over 3 months, swelling and stiffness developed in the proximal interphalangeal (PIP) joint of the right second finger, followed by similar symptoms in the PIP joint of the left index finger. Six years after these symptoms began his hands appeared osteoarthritic, with an unusual degree of flexion of the terminal phalanges (Fig. 2). Xeroradiographs taken at this time showed flexion of most of the distal interphalangeal joints, loss of bone in some distal phalanges and subchondral cysts, but relatively little narrowing of the joint spaces or evidence of adjacent osteosclerosis (Fig. 3).

At present, 14 years after his injury, the patient has very little joint pain, but describes interphalangeal joint stiffness and some loss of dexterity; these make it difficult for him to play a guitar. His hands are unusually sensitive to cold: exposure produces a purple discolouration and a feeling of discomfort. Laboratory findings, including the complete blood count and the erythrocyte sedimentation rate, were normal; tests for rheumatoid factor gave negative results.

Discussion

The initial phase of frostbite arthritis can be categorized according to four degrees of severity:[5]

- Erythema, edema and desquamation.
- Vesiculation and loss of several layers of skin.
- Loss of the full thickness of skin, with the digits icy white.
- Immediate bone involvement, with necrosis and subsequent loss of the affected part.

Bone and joint changes may occur weeks, months or years later. The radiologic changes are most pronounced at the distal ends of the digits and may be asymetrically distributed. Initially these changes involve demineralization; they are sometimes associated with small areas of increased density in the distal tufts or small punched-out subarticular cysts or both. Later, marginal spurs and flexion contractures of the distal interphalangeal joints may occur, sometimes with resorption of the distal phalangeal tufts.

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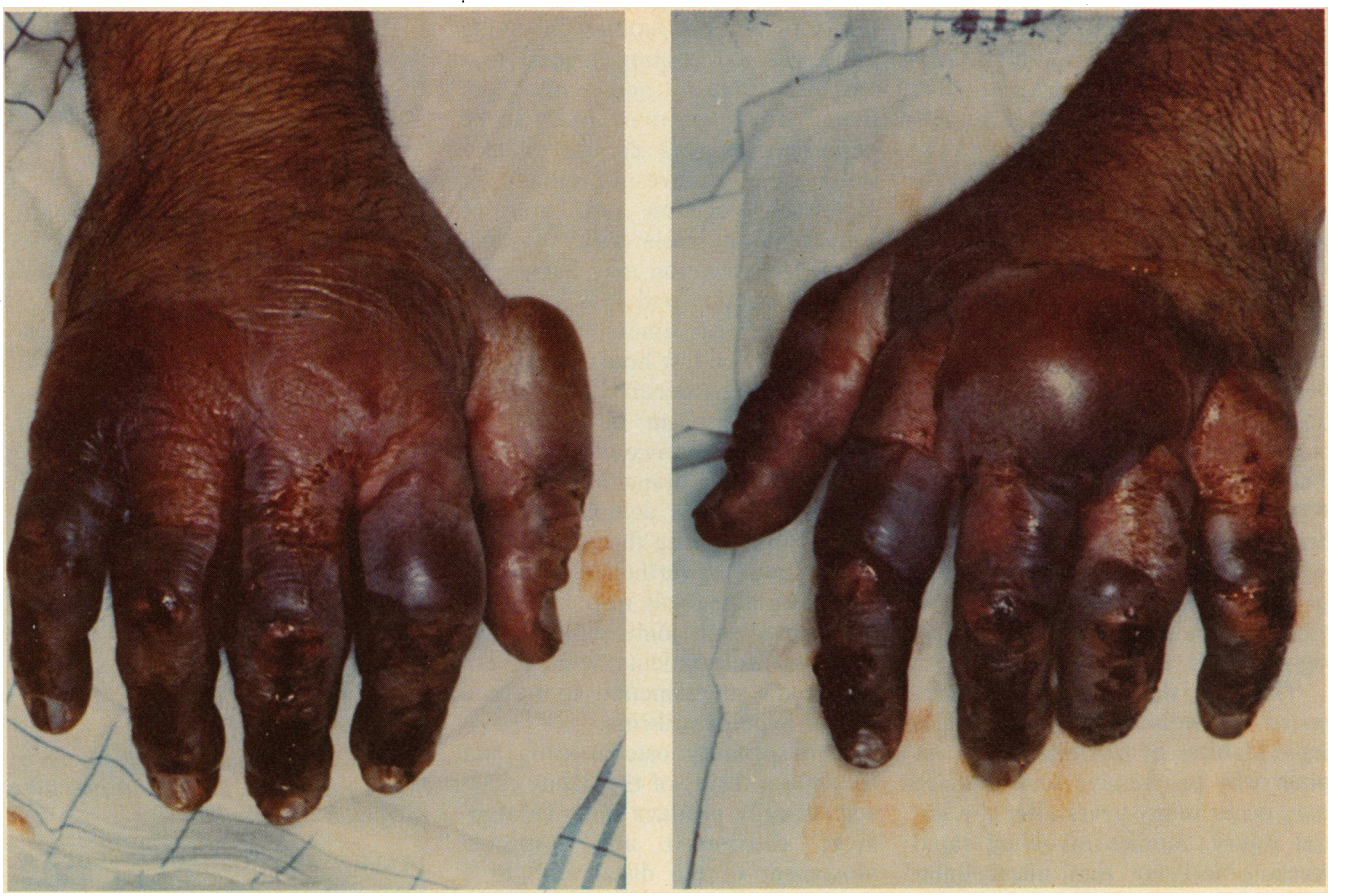

FIG. 1—Hands immediately after frostbite injury.

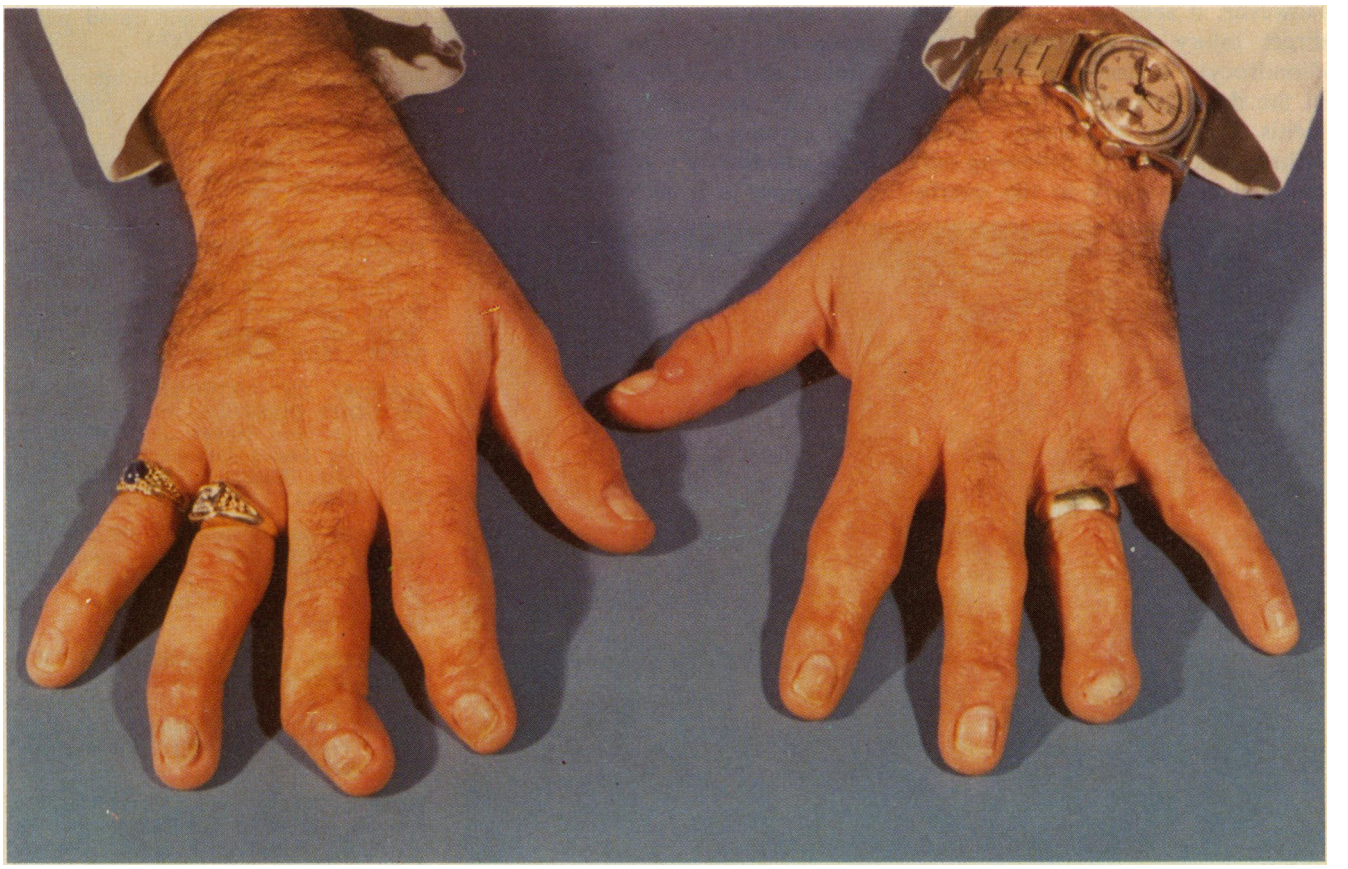

FIG. 2—Hands 13 years after initial injury.

The patient presented in this case report demonstrated all these features. Frostbite in children may cause epiphyseal fusion or abnormal epiphyseal growth; either may result in clinodactyly.[6]

Symptoms of arthritis secondary to frostbite seem to vary from episodic attacks of quite severe joint pain to very mild pain and stiffness, as in the case reported here. Other chronic cold-induced sequelae have been described, including hyperhidrosis, dysesthesia, persistent nail changes and vasomotor instability.[7] This patient reported that his hands were so sensitive to cold that they became purple after brief exposure. Although he did not suffer from hyperhidrosis or dysesthesia, transverse nail ridging was noted on his right middle finger.

Theories that have been proposed to explain the pathophysiologic features of frostbite fail to explain why progressive joint damage may occur many years after the initial injury. Studies involving rapid freezing indicate that intracellular ice crystals may produce cell dehydration and damage by ionic shock. However, it seems improbable that direct injury of this type to the chondrocyte is necessarily involved in the development of frostbite arthritis because moderately severe arthritis may develop following only minimal cold injury to the overlying soft tissues. Studies involving more gradual cooling suggest that vascular damage and consequent tissue anoxia are more likely to be important. During cooling, hemostasis occurs as a result, initially, of platelet aggregation and, later, of endothelial necrosis with swelling. Platelet aggregation may have been inhibited in this patient because he ingested a large amount of acetylsalicylic acid a few hours before the frostbite injury occurred. However, a reversible spasm in digital arteries and the absence of blood flow through arterial branches in the fingertips have been demonstrated by angiography during the acute phase of frostbite.[8] Nevertheless, if vascular spasm alone was responsible for the subsequent joint damage, we might expect patients with severe Raynaud's phenomenon to have a similar type of arthritis.

Both primary osteoarthritis and frostbite arthritis of the hands characteristically produce bony enlargement, discomfort and restricted movement of the distal and, to a lesser extent, the proximal interphalangeal joints. Although there are substantial differences between these two conditions, the similar clinical and radiologic features suggest the possibility of common pathophysiologic mechanisms. Perhaps chondrocyte damage, due to cold injury in frostbite arthritis and to biochemical or mechanical factors or both in primary osteoarthritis, is responsible for the similar clinical and radiologic features.

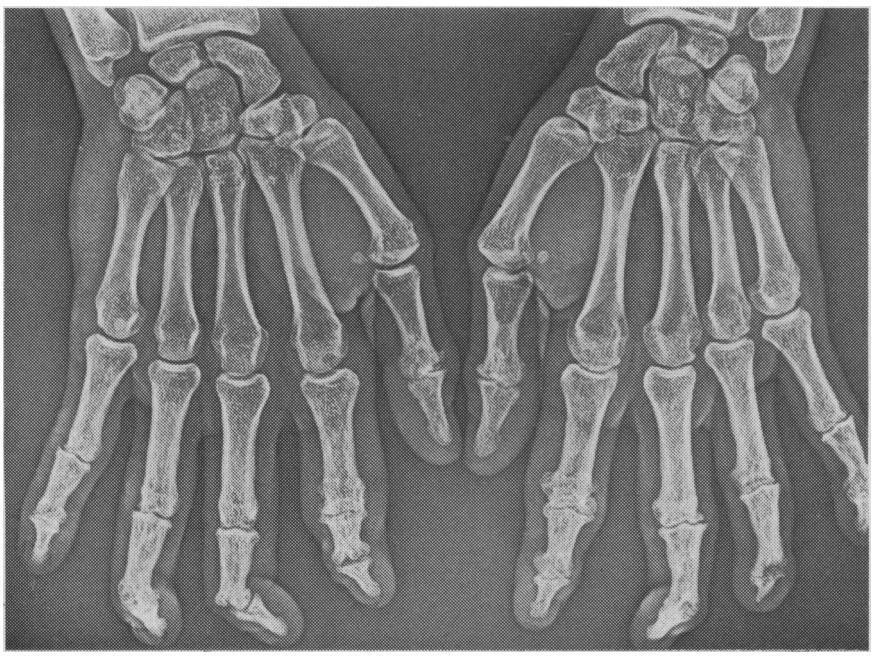

FIG. 3—Xeroradiographs made 13 years after initial injury.

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This list is an acknowledgement of books received. It does not preclude review at a later date.

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