Orthotics and Prosthetics, Vol. 33, No. 3, pp. 32-39, September 1979

The Diabetic Patient: Orthotic Considerations

EDWARD P. VANHANSWYK, C.O., B.S.[1]

The orthotist plays an important role in the care of the diabetic patient, especially in the treatment of the major complications presented by the insufficient vascularity and neural function associated with diabetic neuropathy. These diabetic complications, including muscle weakness of the extremities and decreased protective sensation of the skin and joints of the foot, require expert orthotic management.

History of Diabetes

That diabetes is caused by the inability of the pancreas to produce the natural insulin needed for proper body metabolism is perhaps the one fact agreed upon by most authorities in the literature.

Controversy remains on most all other areas, including etiology and treatment, and, although diabetes has been recognized for centuries, these controversies still exist.

The name "diabetes" was first introduced by a Roman physician, and accounts of the disease were recorded as early as 1500 B.C. While references to diabetes mellitus in early medical writing are few, it is not surprising, in view of the late-in-life onset of the disease and the short life expectancy in early times.

Clinical interest in the complications of diabetes began in the late 19th century, with the increasing age of the population and the development of clinical biochemistry.

The discovery of insulin in 1921 allowed the successful treatment of acute manifestation of the disease, and although this development offered hope to those affected, it also presented a greater survival rate of the diabetic population and a greater preponderance of diabetic complications.

Prior to 1921, the average duration of life after onset of diabetes, was 1.3 years for those with onset under age 10; 2.7 years for those with onset between ages 10 and 19; and 4.3 years for those with onset between ages 20-29. In contrast, comparison figures in 1968 were 29.8, 26.7, and 27.2 years, respectively (1).

However, almost 60 years after the development of insulin, the exact cause of the disease is still unknown, even though, in the last two or three decades, interest in diabetes and recognition of related physical and social problems have been greatly intensified.

It is estimated that in the U.S. at least 2 percent of the population, or 4,000,000 people, have diabetes. It is estimated further that in the U.S. probably 50,000,000 persons now living, either have diabetes, will develop diabetes, or have a diabetic relative.

The impact of the disease as a public health problem becomes more apparent

when one considers the medical and social complications of long term diabetes. These complications lead to marked disability due to involvements in the brain, eyes, heart, kidneys, and the limbs.

Classifications of Diabetes

Diabetes can be classified into two distinct types, I, Hereditary and II, Non-Hereditary, with the hereditary classification the most predominant. The non-hereditary type can be broken down further into juvenile and adult types.

Juvenile Onset

The growth or juvenile onset type is unstable from the beginning. The onset is often abrupt, usually with diabetic coma as the first indication of the disease. This type of diabetes is prone to ketoacidosis (the inability of the body to overcome the disruption in metabolism), and is dependent on insulin for control.

Adult Onset

The maturity or adult onset type is by contrast stable and more difficult to diagnose immediately, or even to establish a date of onset, which usually occurs after age 40. Coma and keto-acidosis is usually achieved by dietary restriction, with or without insulin.

The slow onset of the disease must be recognized and treated, and satisfactory control is dependent upon patient understanding and cooperation. The relative mildness of onset is at times the forewarning to development of other major characteristic complications.

Diabetic Complications

There are three major characteristic complications, a) diabetic retinopathy, b) nephropathy, and c) neuropathy.

Diabetic retinopathy, or vascular disease of the retina, is one of the more serious complications of diabetes. It is estimated that 2 percent of the diabetic population suffers blindness due to retinopathy. There is no known cure, and the pathogenises of the condition remains obscure.

Diabetic nephropathy, or renal vascular disease, is also a serious complication, and the resultant kidney dysfunction is a cause of death in diabetics.

Diabetic neuropathy, or vascular insufficiency and derangement of metabolism in the peripheral nerves, can lead to catastrophic complications of diabetes. These are the complications which especially lend themselves to Orthotic/Prosthetic management.

Diabetic neuropathy is a common name for several disorders of the peripheral nervous system. The conditions involving the lower limbs include: 1) polyneuropathy, 2) mononeuropathy, 3) arthropathy, 4) amyotrophy, and 6) diabetic cold feet.

1. Polyneuropathy, the most common variant of diabetic neuropathy, begins with sensory impairment, in a stocking effect (beginning at the feet and moving upward), followed by numbness and stiffness of the limb. Motor symptoms, weakness, poor proprioception, and trophic changes including Charcot joints, also occur.

2. In mononeuropathy, the nerve most frequently involved is the common peroneal.

3. In arthropathy, both sensory and autonomic nerves of the joint are involved. A variety of changes occur in the joint, initially painful but later with painless destructive lesions.

4. Anhidrosis, a specific syndrome involving the autonomic nerves, causes excessive sweating over the upper body, but a total absence of sweating below the waist resulting in dry, cracking feet and legs.

5. Amyotrophy, with the occurrence of proximal muscle weakness of the lower

limbs associated with extensor-plantar response. Pain, weakness, and weight loss, but without objective sensory loss occurs in the iliopsoas and quadriceps group.

6. Diabetic cold feet, an early manifestation of an alteration of symphatic control of the blood vessels, marked by bluish lower limbs.

In general, the outlook for recovery from the various forms of diabetic neuropathy varies. Although mild motor weakness and slight insensitivity persists, complete recovery from polyneuropathy is expected. On the whole, recovery from mononeuropathy is also expected, although when severe, collateral circulation may not be adequate. The outlook is less than satisfactory for amyotrophy. While recovery of clinical function may take a few years, the neuropathic foot remains insensitive and the Charcot joint suffers recurrent trauma, and the outlook is poor.

Treatment

Treatment of diabetic neuropathy is based mainly on relief of symptoms, with measures taken to prevent increased complications. Continued control of the diabetes by diet and appropriate doses of insulin are necessary, and during the acute and painful phases of mononeuropathy and amyotrophy, bed rest and passive exercises are helpful.

It is in the postacute stages that orthotic management is necessary. Insensitive feet, Charcot joints, and muscle weakness, are all complications of diabetic neuropathy requiring orthotic management and treatment.

Insensitive Feet

The major complication, and perhaps the most significant orthotic consideration in diabetic neuropathy is decreased protective sensation of the foot.

Hemodynamically poorly placed and requiring an accurate monitoring of blood flow by the sympathetic and parasympathetic nervous systems, the foot also requires the assistance of a normally functioning muscular system to insure return of the blood to the body. The foot is exposed repeatedly to trauma by frequently being brought into contact with the ground, and therefore requires an adequate sensory system for protection. Finally, it is farthest away from the central nervous system and, therefore, has to maintain the longest supply lines for these functions. These motor, sensory and autonomic systems are all at a disadvantage in the foot, and nerve fibre damage can easily combine with circulatory disturbances to produce complicated lesions.

A clinical classification of foot lesions has been described by Dr. F. William Wagner, Jr., Co-Chief of the Diabetic Surgical Services at Rancho Los Amigos Hospital (2). The classification from potential breakdown to total foot destruction is defined in six grades (Fig. 1). Starting at grade 0, a condition of bony prominence and pressure points, but without open skin lesion, through grade 5, a condition of whole foot gangrene.

Considerable success in treating grades 0, 1, and 2 conservatively, without amputation, is described by Dr. Wagner. Partial success in treating grades 3 and 4 was achieved by healing the lesion and thereby preserving the foot for orthotic management. Of a total 151 patients evenly distributed through grades 0-5, 107 were healed short of Symes amputation.

Healing Cast

The use of protective plaster casts is one conservative method of treatment (Fig. 2).

In the diabetic foot, tissue breakdown is caused by unappreciated trauma. Excessive pressure or intolerable shear forces traumatize insensitive skin and

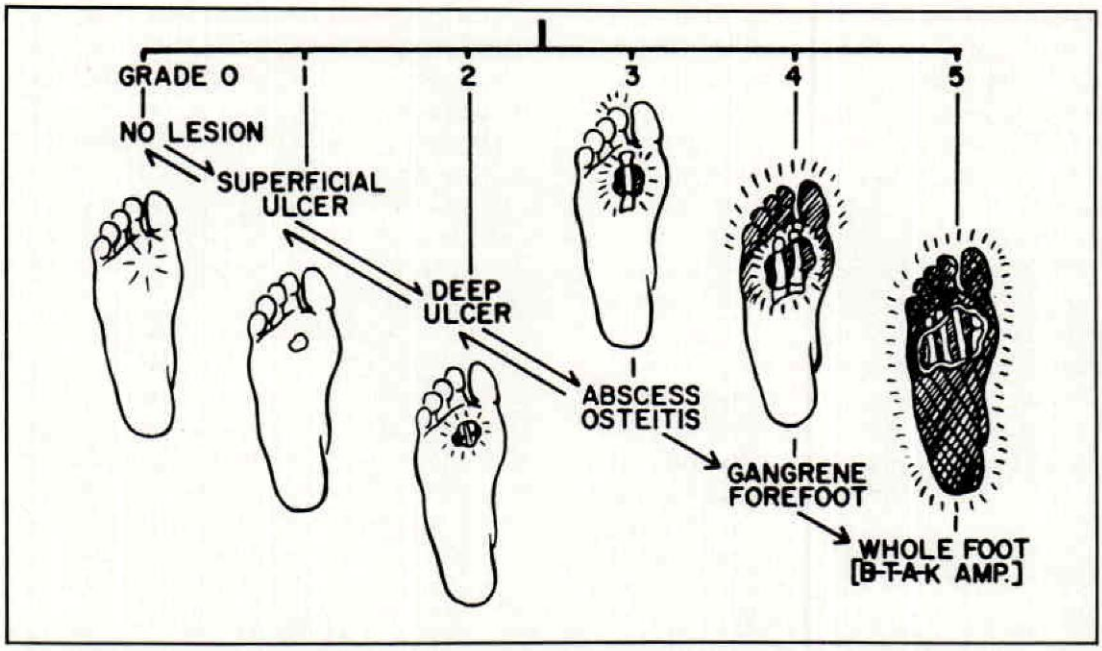

Fig. 1. The natural history of the breakdown of the diabetic foot (1).

cause tissue breakdown. Inflammation and edema prevent the body's own vascular healing process from taking place.

When arteriosclerosis or other contraindications such as undrained abscesses can be ruled out, or can be drained by surgery or treated with antibiotics, cast treatment can be started.

The function of the cast is to allow walking or other physical activities without added trauma. The encapsulation by the plaster cast results in a reduction of edema. The constant stress over a broad area not only controls the edema, but also helps to mature the affected tissue, and encourages faster wound healing.

Diabetic Foot Wear

When healing takes place, the Plastazote "forgiving" shoe is prescribed.

The Plastazote shoe (Fig. 3) constructed of the "forgiving" Plastazote insole and moulded Plastazote upper is combined with various heel and sole modifications to allow walking contact with the ground, without excessive trauma to the newly healed lesion.

The Plastazote insole, moulded to the foot or to a plaster cast, protects the insensitive, thin-skinned bony prominences of the plantar surface, while the molded Plastazote upper reduces pressure to the uneven dorsal surface of the toes and foot. Modifications consistences of the "SACH" heel, and metatarsal or rocker bars reduce trauma and excessive pressures when the patient walks.

A ready-made type of Plastazote shoe is available from Apex,[2] and the custom fabricated type as described by Guilford[3] can be used.

The depth inlay[4] or extra depth shoe[5], one which can accommodate a contoured Plastazote insole without compromising the dorsal surface of the toes and foot, is also used after healing of the lesion.

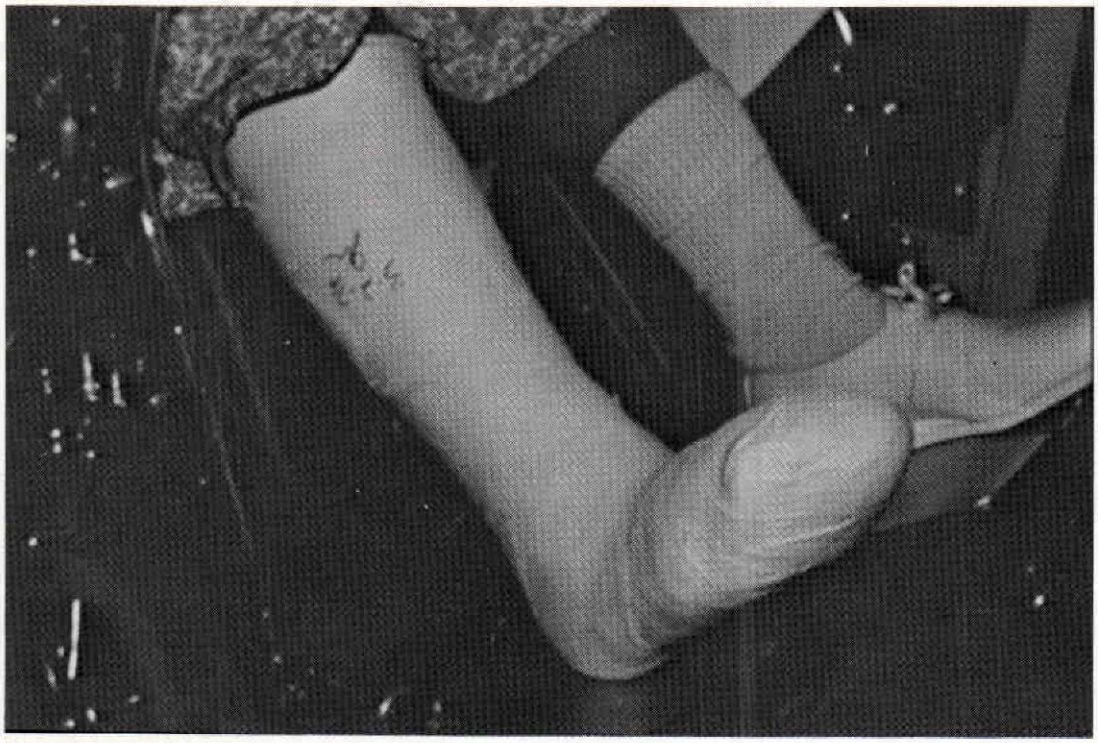

Fig. 2. Plaster casts to protect the foot is one conservative method of treating the diabetic foot.

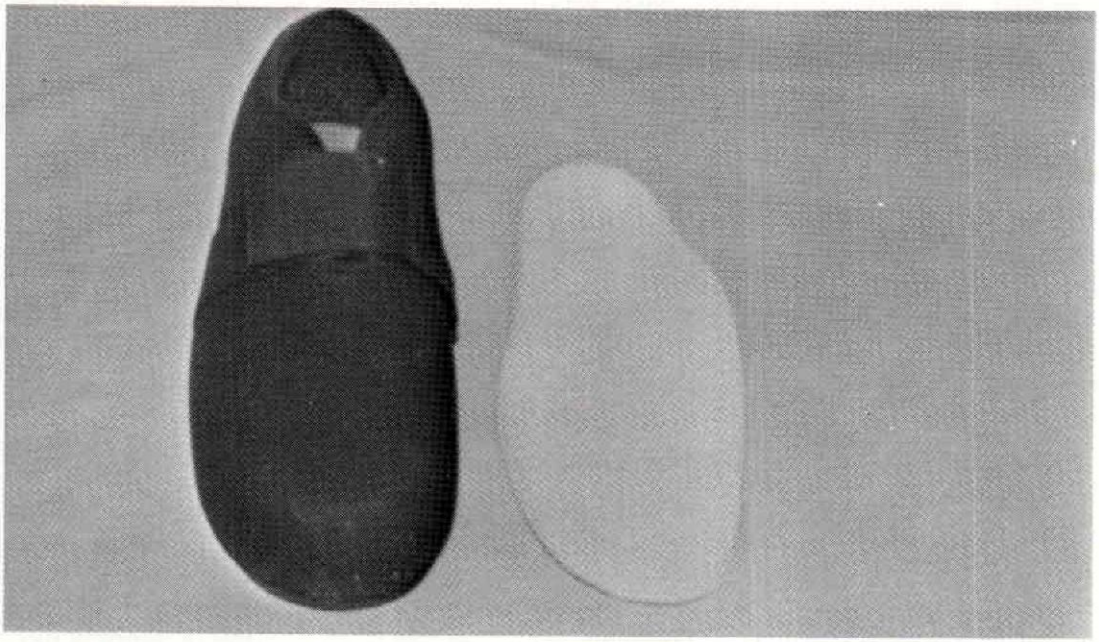

Fig. 3. The Plastazote shoe.

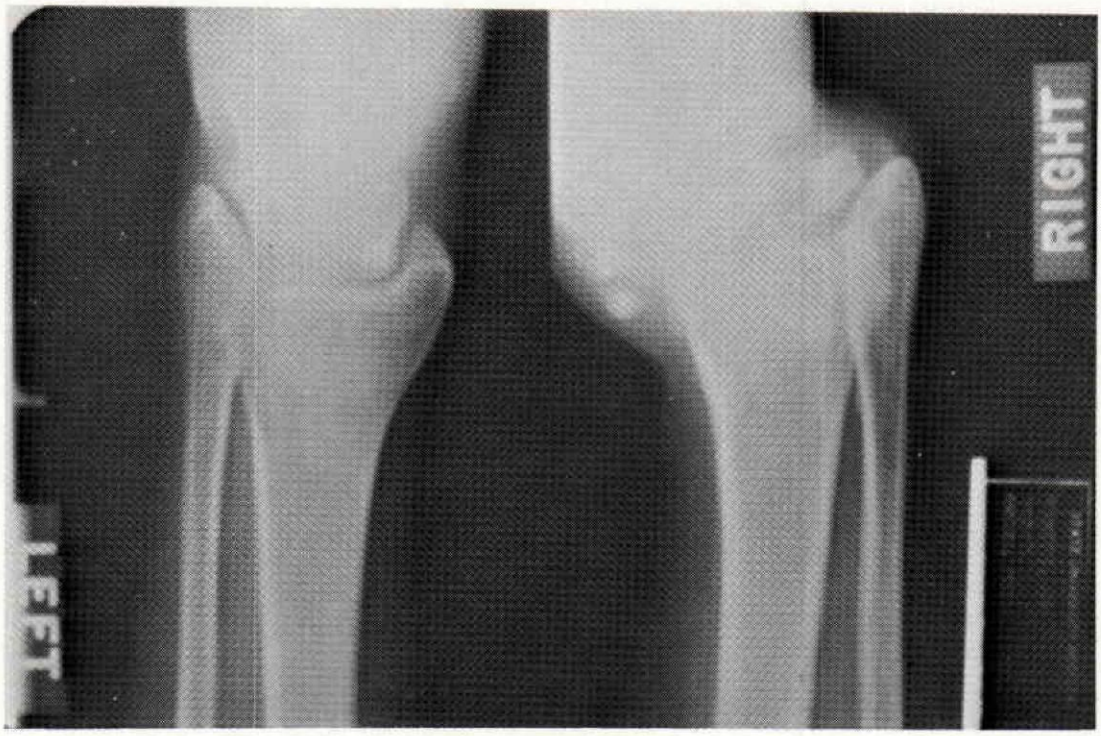

Fig. 4. X-Ray views of Charcot joints.

Extreme care is taken in the fitting and modification of footwear for diabetics with potential for, or healed, foot lesions. It is important that the patient be included in this effort, and made aware of his specific foot and footwear problems.

Diabetic Foot Care

Daily foot hygiene is mandatory and must be encouraged. Mild soap and warm water are used. Complete drying of the skin, especially between the toes, and square toenail trimming are mandatory, as well as close inspection of skin for blisters and pressure areas.

Periodic re-evaluation of the foot, shoe, and shoe modification will insure proper continued orthotic management.

Charcot Joints

A second complication responding to orthotic management is Charcot joint disease, usually in the ankle and foot, and is the result of recurrent trauma unappreciated because of diminished proprioception. The actual change in the joint is an exaggerated form of degenerative arthritis, instability, and swelling (Fig. 4).

Treatment is by support, unweighting, and protection from stress and further in-

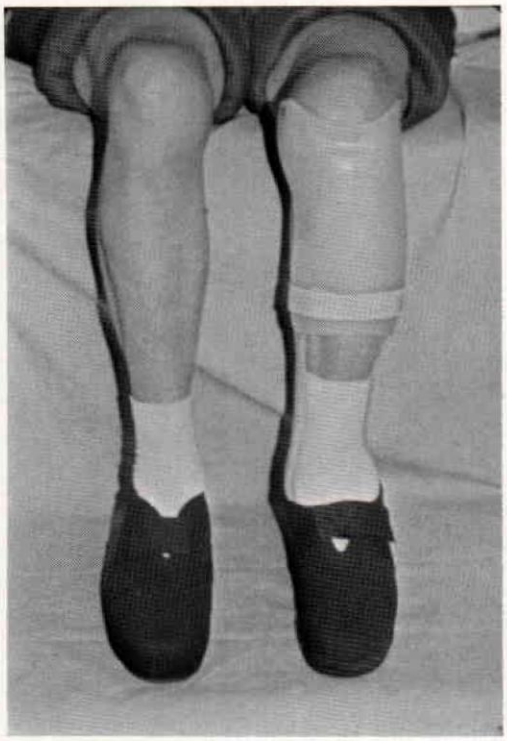

Fig. 5. An AFO with the patellar-tendon-bearing feature for treatment of Charcot joint.

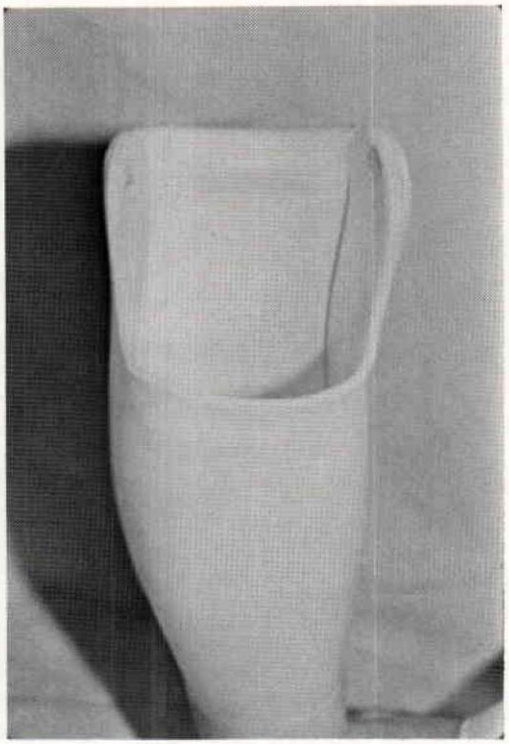

Fig. 6. Plastic foam is molded carefully over the patellar-tendon bridge and the proximal contours of the AFO to provide protection over the tibial crest.

jury. Orthotic protection of the distal joints of the foot requires the shoe modification, rigid sole and/or rocker bar. A Plastazote insole may also be used.

Orthotic management for protection of the ankle joint and proximal joints of the foot requires an AFO with a patella-tendon bearing area (Fig. 5).

Special care in design and fabrication is necessary because of the neuropathic complications presented by the diabetic patient.

1. Foam rubber is molded carefully over the patella tendon bridge and proximal contours to provide protection over the tibial crest (Fig. 6).

2. The trimlines at either malleolus must be sufficient to maintain rigidity in the flexion-extension plane, and to cover totally each side to maintain medial-lateral stability. Plastazote padding is used to prevent pressure to insensitive skin, and added or removed to accommodate changing contours and edema (Fig. 7).

3. A Plastazote insole is also incorporated in the moulding of the plastic to protect the insensitive and bony prominences of the foot.

4. A depth shoe or Plastazote shoe is used to accommodate the added inner soles without causing pressure to the dorsum of the foot and toes.

Muscle Weakness

A third complication in diabetic neuropathy is muscle weakness of the lower limb. Involved are the iliopsoas muscle and quadriceps group proximally, and the anterior peroneals below the knee. The involvement is usually isolated, prox-

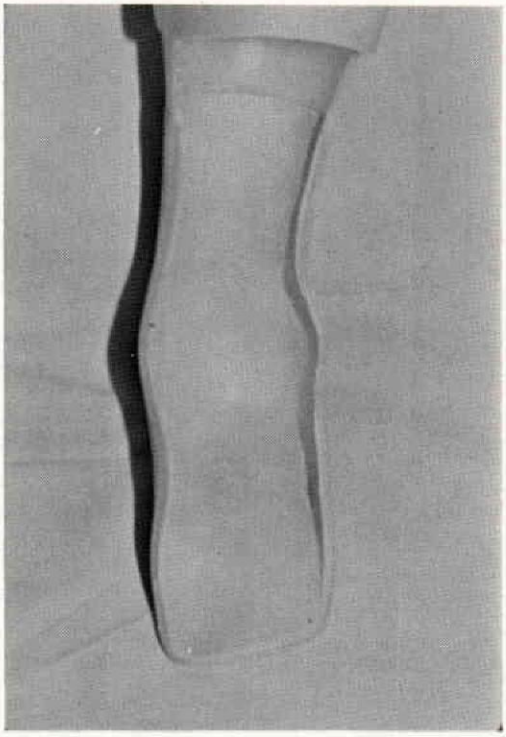

Fig. 7. Plastazote is used to prevent excessive pressure over insensitive skin in the AFO.

imal or distal, and is unilateral.

With a proximal muscle weakness, the orthosis of choice is dependent on many variables, but sufficient involvement of the quadriceps to cause knee instability requires a knee-ankle-foot orthosis (KAFO).

When the involvement is isolated to the peroneals resulting in a "drop foot", an AFO is sufficient.

The sensory involvement in the diabetic must be kept in mind during fabrication. Insensitive areas around the ankle and especially the plantar surfaces of the foot must be recognized and protected with adequate contours and/or Plastazote padding and insoles.

Conclusion

An overview of diabetes and the orthotic considerations of the diabetic have been presented.

We would re-emphasize that diabetes is an enormously complicated disease and carelessness or mistreatment can have catastrophic end results. The possible devastating problems of the diabetic patient demand knowledgeable orthotic management.

Footnotes

[1]Clinical Instructor, Dept. of Orthopaedic Surgery and Director Orthotics Research & Education, College of Medicine, State Univ. of New York, Upstate Medical Center, Syracuse, N.Y.

[2]Apex Foot Products, Corp., 200 Forest Avenue, Englewood, N.J. 07631

[3]Guilford, Arthur, C.O., Suite E, 19437 Mount Langley St., Fountain Valley, California 92708

[4]Alden Shoe Company, Taunton St., Middleborough, Mass.

[5]Miller Shoe Company, 4015 Cherry St., Cincinatti, Ohio

References

(1) Joslin, Elliott P., *"Joslin's diabetes mellitus"*, Eleventh Edition, 1971

(2) Wagner, F.W., Jr.: *"Classification and treatment outline for diabetic foot lesions"*; Instructional Course, American Academy of Orthopedic Surgeons, New Orleans, Louisiana, Feb. 1976.