Venous distension in the diabetic neuropathic foot (physical sign of arteriovenous shunting)[1]

J D Ward MD FRCP
J M Simms MB FRCS
G Knight MB MRCP
A J M Boulton MB MRCP
D A Sandler MB MRCP
Departments of Medicine and Surgery, Royal Hallamshire Hospital, Sheffield

Summary: A new physical sign is described in the feet of a group of diabetic patients with ulcerating neuropathic problems, in which major venous distension of the veins on the dorsum of the foot and lower calf is seen. Elevation of the leg is required to an average height of 32.3 cm to cause collapse of these distended veins. It is suggested that this clinical sign indicates the presence of arteriovenous shunting in such neuropathic legs, and as such is a simple and useful measure of this abnormality.

Introduction

When considering blood flow in the diabetic leg it is usual to think in terms of arteriosclerotic vascular disease, which of course is present in many diabetic subjects with an increased incidence when compared to normal. This degenerative vascular disease is distributed more proximally than in the non-diabetic and is commonly associated with calcification of the vessel wall. The concept has thus grown up that the blood supply to the diabetic leg is impaired mainly because of obliterative degenerative arterial disease.

In the course of studying many aspects of diabetic neuropathy, a common observation has been of a diabetic leg and foot which was warm and often had an easily palpable and visible pulse at the dorsalis pedis, and yet within centimetres of such an apparently adequate blood supply there would be gangrene of a digit or a penetrating neuropathic ulcer. The conventional explanation for this is that between the palpable pulse and the area of gangrene or ulceration there exists specific diabetic angiopathy occluding many distal small vessels. However, examination of the diabetic neuropathic foot, with so-called ischaemia and ulceration, revealed that it was almost always warm and that the veins seemed prominent over the dorsum of the foot. This led to a series of studies with Doppler ultrasound techniques, which indicated extremely fast rapid forward flow of blood in such neuropathic feet (Scarpello *et al.* 1980, 1978), an observation confirmed by Edmonds *et al.* (1980), who described the pulsatility index as a quantitative measure indicative of this fast forward flow. At this stage it seemed that such fast forward flow was highly suggestive of arteriovenous shunting of blood. Subsequently, blood was sampled from these distended veins and analysed for oxygen concentration, this concentration being shown to approach that of arterial blood (Boulton *et al.* 1981). Thus was developed the concept of arteriovenous shunting in the diabetic neuropathic leg and foot, a totally different concept from that of obliterative vascular disease.

In the course of studying many patients with diabetic neuropathy, 15 patients were identified with a typical history of neuropathic symptoms along with past or present foot ulceration in whom venous distension was a very prominent clinical physical sign. Slight variations in the degree of venous distension were observed from day to day, but this abnormality was always easily observed in such patients. This paper reports on this clinical observation, describes the height to which the leg has to be raised to cause collapse of the veins, and discusses the background and reasons for arteriovenous shunting in this situation.

[1]Based on paper read to Section of Neurology, 7 April 1983. Accepted 14 September 1983

0141–0768/83/121011–04/$01.00

Methods

Fifteen patients are described, all of whom had previously taken part in clinical and therapeutic studies of diabetic neuropathy. Their ages ranged from 31 to 68 years (mean 50 years) with a duration of diabetes between one and 38 years (mean 11.7 years). Eight of these patients were taking oral hypoglycaemic agents or diet alone, and 7 were being treated with insulin. All subjects described some history of sensory neuropathy with the typical symptoms of tingling, paraesthesiae and numb cold sensations in the feet, and 25% of patients experienced hyperaesthesiae to touch. However, none of these patients had experienced the more severe and extremely painful type of sensory neuropathy. Physical signs in all patients consisted of patchy sensory loss in the legs with absent knee and ankle jerks, and there was a varying degree of muscle wasting particularly prominent in the quadriceps muscles and small muscles of the feet. A prominent physical sign was almost total lack of pain sensation. This gross clinical neuropathy was confirmed in all patients by the demonstration of a motor conduction velocity in the lateral popliteal nerve of below 42 m/s and vibration perception threshold, measured by the biothesiometer, of over 15 units. Major obliterative arterial disease was excluded by the absence of typical symptoms of ischaemia and the presence of easily palpable bounding foot pulses at the dorsalis pedis.

At the time of study 4 patients had healing clean neuropathic ulcers, the other 11 giving a history of ulcer in the past. The foot of one patient was excluded from the study due to the presence of active infection.

In the supine position all patients had easily observed marked distension of the veins on the dorsum of the foot and the lower part of the calf, varying from obvious to so gross a degree of distension that the vein could be easily palpated (Figures 1 and 2). From the diabetic clinic 200 consecutive diabetic subjects were observed in the supine position and no such venous distension was seen, apart from a few cases with obvious varicose veins. This physical sign would seem, therefore, to be confined to patients with the ulcerating type of peripheral neuropathy, although from personal observation two patients with acute painful neuropathy have been observed in which this physical sign seemed to resolve as the clinical symptoms improved.

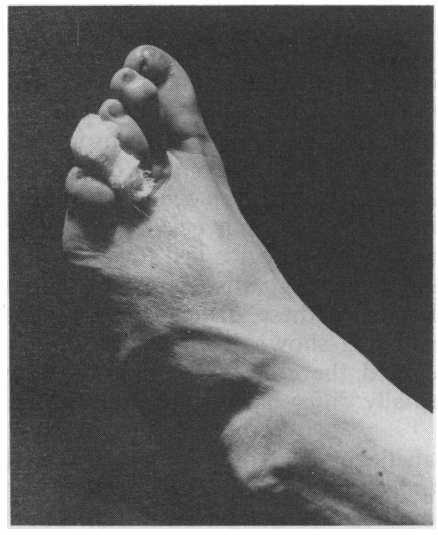

Figure 1. A 57-year-old diabetic with longstanding neuropathic problems and a healed ulcer on the sole of the left foot. Gross venous distension in the supine position. Collapse of veins when foot elevated to 36 cm

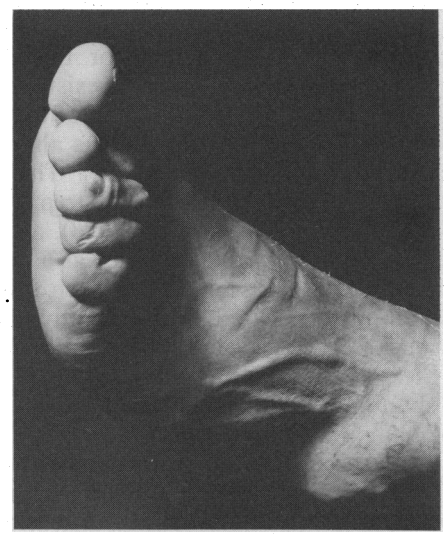

Figure 2. A 54-year-old male diabetic with past history of painful neuropathy. One-month history of extremely unpleasant aching and tingling sensations in both thighs and calves and clinical picture of acute painful neuropathy. Gross venous distension seen in both feet extending up into calves

Subjects were rested in a room in which the temperature did not vary outside the range of 22–27°C (mean 24.5°C) and were allowed to lie flat at rest for 15 minutes before any studies or observations were made. Obvious venous distension was observed and recorded and the leg was then slowly elevated until collapse of the veins occurred, the height of the leg from the horizontal being recorded. The leg was then replaced flat and the time recorded for return of the original venous distension. An intravenous butterfly type catheter was then introduced with ease into the distended vein and connected to a pressure transducer, allowing a measurement of venous pressure to be made. At the same time a sample was taken for analysis of oxygen content, the temperature of the leg itself was also recorded and a sample of venous blood was taken from the dorsum of one hand for calculation of hand vein venous oxygen. The venous oxygen content was calculated from two aliquots of each sample and results are given as a mean of these two estimations. As there is no significant difference between the results in the right and left leg, all results will be presented as a mean of both legs.

Results

The mean temperature of the neuropathic legs was 29.9°C (range 26.2–34.0°C), which was significantly higher than the room temperature. There was no correlation between room temperature and any of the other measurements – temperature of the leg, venous pressure, venous oxygenation, or height to collapse of veins. The mean height to which the leg had to be elevated before collapse of the veins occurred was 32.3 cm, with a range from 12 cm to, in one instance, 80 cm. On returning the leg to the horizontal, the mean time for refilling to distend the vein was 30.4 seconds (range 2–39 seconds). Estimation of venous oxygen confirmed previous studies in showing venous oxygen concentrations of nearly arterial levels: mean 7.80 kPa, and range 4.70–10.47 kPa (artery range 9.8–14.2 kPa). As in our previous studies, venous oxygen saturation from the hand was not elevated: mean 4.49 kPa (range 2.65–8.50). The mean venous pressure was 18.41 mmH_2O (range 13–31).

Discussion

There seems little doubt that arteriovenous shunting is occurring in the diabetic neuropathic foot and leg, particularly in those with a history of foot ulceration, thus explaining the observation of an easily palpable bounding pulse at the dorsalis pedis only centimetres away from an ulcer or ischaemic toe. Rapidly-flowing fluid does not adequately fill small branches and side channels, suggesting that this abnormality is partly responsible for further reducing the supply of blood through these distal small channels. Evidence from Doppler ultrasound studies and the near arterial oxygenation of venous blood from distended veins in these feet strongly supports the suggestion of arteriovenous shunting. There is ample evidence for the anatomical presence of such channels (Sherman 1963) which can open up in response to increased blood flow (Richards 1970). It is known that there is increased blood flow in diabetic subjects, particularly at times of poor metabolic control (Gundersen 1974). Partsch (1977), in a study of ulceromutilating neuropathies with a number of aetiologies, demonstrated the rapid accumulation of radioactive microspheres in the lungs of such subjects following injection into the femoral artery – a strong indication of shunting.

Autonomic dysfunction leading to a widely dilated vascular bed seems to be the single most likely explanation for this phenomenon, and there is ample evidence for this. Edmonds *et al.* (1981) clearly show a correlation of autonomic dysfunction with the presence of foot ulceration, correlating this with abnormal sonograms obtained with the Doppler ultrasound system. In animal work, sympathectomy and the hyperaemia of infection have been shown to result in arteriovenous shunting (Ronenwett & Lindenaur 1977). There is little evidence in the literature for the reversibility of significant autonomic dysfunction, but recently Edmonds *et al.* (1983) have demonstrated changes back towards normality of the Doppler sonogram and the pulsatility index in 4 patients with 'neuropathic oedema' treated with ephedrine. Presumably these patients with advanced diabetic peripheral neuropathy responded due to the sympathomimetic stimulation causing vasoconstriction and a

reduction in the degree of vasodilatation to allow a more normal pattern of flow. Two other patients have been observed with typical acute sensory neuropathy, in whom obvious clinical venous distension in the feet and legs has been seen to resolve over a six-week period, during which blood glucose control was improved with resolution of uncomfortable symptoms. The physical properties of the vessels themselves may well be playing an important part. In diabetes it has long been known that vessels in many parts of the body – especially the legs – are heavily calcified and hence rigid. Such vessels will therefore resist pressure to collapse and for this reason it is very likely that the Doppler ultrasound ratio is less reliable in the diabetic (Lazarus *et al.* 1978, Emanuele *et al.* 1981). The patients described here did not all have evidence of heavy vascular calcification, but certainly rigid calcified vessels would potentiate the tendency to fast-flowing blood.

The metabolic state may also be contributing to increased flow, for it is known that there is such increased flow at times of poor metabolic control. Another possible factor is related to a degree of blockage of small vessels distally, perhaps forcing blood to find more proximal channels, again potentiating the tendency to shunting. In the legs of patients with peripheral nerve damage in diabetes there is known to be considerable blockage of small vessels in peripheral nerve (Timperley *et al.* 1976).

It is therefore possible to build up a postulate of how these interrelated factors are playing their part in causing arteriovenous shunting and hence potentiating distal ischaemia. Autonomic dysfunction leads to a widely-dilated vascular tree and this is aggravated by rigid arteries, thus allowing very rapid flow of blood which will tend not to fill small vessels and side channels and therefore makes its way through established shunting channels to the venous system – hence distending the veins in the way described above. Any contribution from the metabolic state in causing further dilatation of vessels or increased blood flow will only serve to aggravate the condition. Now a very important effect develops: as a result of the fast flow in the larger vessels and the much less adequate flow in small distal vessels, the known abnormalities of platelet function and fibrinolysis become critical in such slowly-moving blood, leading to more and more occlusion of small vessels. This causes further ischaemia distally, but also potentiates the need for fast-flowing blood to find more proximal channels.

This state of arteriovenous shunting in diabetic neuropathy can be seen to be a potent factor allowing ischaemia and ulceration to develop. A more detailed understanding of the exact mechanisms will allow a more logical approach to therapy. In further studies use will be made of measurements of autonomic function, ultrasound measurements of blood flow characteristics including intravenous Doppler studies, combined with measurements of venous oxygenation. The physical sign of venous distension in the feet and legs of such patients will add a further very simple measure and it is hoped that the presence of this sign will alert the physician to the potential danger of ischaemia and ulceration in such legs.

Acknowledgment: We are most grateful for the help given by Mr B Parkinson, Photographic Department, Royal Hallamshire Hospital, who produced the photographs.

References

Boulton A J M, Scarpello J H B & Ward J D (1981) *Diabetologia* **22,** 6–8
Edmonds M E, Archer A G & Watkins P J (1983) *Lancet* i, 548–551
Edmonds M E, Nicolaides K & Watkins P J 1981) *Diabetologia* **21,** 506 (Abstract)
Edmonds M E, Wilton G N, Roberts V C & Watkins P J (1980) *Diabetologia* **19,** 272 (Abstract)
Emanuele M A, Buchanan B J & Abraira C (1981) *Diabetes Care* **4,** 289–292
Gundersen H J G (1974) *Diabetologia* **10,** 225–231
Lazarus H M, Albo D jr, Welling D & Hutto W (1978) In: Non-invasive cardiovascular diagnosis: current concepts. Ed. E B Diethrich. University Park Press, Baltimore; pp 127–135
Partsch H (1977) *Vasa,* Suppl 6; pp 1–48
Richards R L (1970) In: Peripheral Arterial Disease. Ed. R L Richards. Livingstone, Edinburgh & London; pp 6–25
Ronenwett J L & Lindenaur S M (1977) *Surgery* **82,** 82–89
Scarpello J H B, Martin T R P & Ward J D (1978) *Clinical Science* **55,** 3P (Abstract)
Scarpello J H B, Martin T R P & Ward J D (1980) *Clinical Science* **58,** 53–57
Sherman J L (1963) *Medicine (Baltimore)* **42,** 247–267
Timperley W R, Preston F E, Duckworth T & O'Malley B C (1976) *Diabetologia* **12,** 237–243