Arterial disease in chronic leg ulceration: an underestimated hazard? Lothian and Forth Valley leg ulcer study

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Abstract
Six hundred patients with chronic leg ulcers were interviewed and examined for evidence of arterial impairment. There were 827 ulcerated legs. Pedal pulses could not be felt in 94 (11%). A Doppler resting pressure index of 0.9 or less was found in 176 legs (21%). Risk factors for arterial impairment included age, ulceration affecting the foot, and a history of claudication, ischaemic heart disease, or cerebrovascular disease. Roughly half the patients with arterial impairment also showed the clinical features of chronic venous insufficiency.

Careful assessment for arterial disease is mandatory before patients with chronic leg ulcers are treated with elastic compression when applied to limbs with arterial insufficiency but who may not recognise its presence.

The aim of this study was to ascertain how frequently arterial impairment could be detected by simple non-invasive means in a large population of patients with leg ulcers identified in the Lothian and Forth Valley leg ulcer study. Various factors were assessed to see if they were of value in predicting the patients likely to have arterial disease in association with leg ulceration. This paper is not concerned with microvascular disease as represented, for example, by the arteritis of rheumatoid disease and diabetes. These and other diseases associated with leg ulcers will be the subject of a separate report.

Introduction
Chronic ulceration of the leg affects roughly 1% of European populations. Most ulcers are the result of several aetiological factors operating together, though it is generally accepted that the most important and commonest of these is venous insufficiency, due either to previous deep vein thrombosis or to longstanding primary varicose veins.

Most ulcers are treated with some form of compression. Nevertheless, when a degree of arterial impairment is present not only may this type of treatment be of little benefit but it may be harmful. Most patients with chronic leg ulcers are managed by community nurses, who may know that compression bandages are dangerous

Patients and methods
This survey was conducted in the south east of Scotland in the adjoining health board areas of Lothian and Forth Valley (population 1 million) and has been fully described. All patients receiving treatment for chronic leg ulceration in any branch of the health service at the time of the survey were identified and asked if they would agree to an interview and examination.

The 600 patients in the sample were compared with the overall series of 1477 in terms of age, sex, source of referral, and geographical distribution in order to ensure that they were representative. The assessment consisted of a full medical history and examination plus relevant investigations. The data were recorded on an eight page document and subsequently entered into a computer database for analysis. History taking included specific questioning for angina, myocardial infarction, transient ischaemic attacks, and cerebrovascular accidents as well as intermittent claudication. Clinical assessment of the severity of arterial disease included palpation of distal pulses and measurement of brachial and ankle systolic blood pressures by Doppler ultrasonography.

The Doppler measurements were made with the patient lying with the upper part of the body at roughly 40° to horizontal for 15 minutes. Measurements of brachial and ankle systolic pressures were carried out at the same time and with the patient in the same position. Whichever of the three pedal pulses (posterior tibial, dorsalis pedis, or peroneal) gave the strongest signal was used to calculate the resting pressure index—that is, ankle systolic pressure/brachial systolic pressure. The resting pressure index allows for individual variation in blood pressure, and in normal people
without arterial disease it should always be greater than 1-0, ankle pressure normally being higher than brachial pressure. In a normal population the mean resting pressure index was 1.1 (SD 0.08). We have therefore taken the mean value minus two standard deviations as the lower limit of normal (0.94), corrected to 0.9.

Results

A total of 1477 patients were identified in the original postal survey. We set out to examine as many as possible in the time available and found it necessary to set a limit when we reached 600, a further eight having been rejected because of incomplete data. The 600 patients had 827 ulcerated legs. The total series of 1477 patients and the sample of 600 were similar in terms of age, sex, geographical distribution, and source of referral. Puls—Ninety four (11%) of the 827 ulcerated legs had no palpable dorsalis pedis, posterior tibial, or perforating peroneal pulses at the ankle. In the other 733 legs the pulses were palpable but, as expected, the pulses were weak in a considerable number or palpable at only one or two of the three sites. Oedema, inflammation, or open ulceration often made it impossible to assess the posterior tibial pulse.

Doppler pressures—One hundred and seventy six legs (21%) had an ankle resting pressure index of 0-9 or less and 78 (10%) had an index of 0-7 or less (table I).

Correlation between pulses and Doppler pressures—One hundred and fifteen of the 176 legs with low Doppler pressures had palpable pulses. Thirty three (5%) of the 651 legs with normal Doppler pressures had impalpable pulses (table II).

<table>
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<table>
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<td>618 (84)</td>
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<tr>
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<td>61 (65)</td>
<td>33 (35)</td>
</tr>
<tr>
<td>Total</td>
<td>176 (21)</td>
<td>651 (79)</td>
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</table>

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<th>Cerebrovascular or coronary vascular disease</th>
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<td>91 (67)</td>
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<tr>
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<td>131 (19)</td>
<td>560 (81)</td>
</tr>
<tr>
<td>Total</td>
<td>176 (21)</td>
<td>651 (79)</td>
</tr>
</tbody>
</table>

| PREDICTIVE FACTORS FOR REduced ARTERIAL PRESSURE INDEX IN ULCERATED LEG |

| History of arterial disease—Table III shows the correlation between a low resting pressure index and a history of stroke, transient ischaemic attack, angina, or myocardial infarction. Such a history increased the probability of arterial impairment in the ulcerated leg from 19% (131/691) to 33% (45/136) (p<0.01). A history of intermittent claudication was found in 31 patients and was associated with a low resting pressure index in every case. Sex—Fifty nine of the 232 ulcerated legs in men (25%) and 117 of the 595 ulcerated legs in women (20%) had low resting pressure indices. This difference was not significant (χ²=3.68, p=0.05). Age—The figure shows the relation between age and the percentage probability that the ulcer was associated with arterial disease. Under the age of 40 none of the patients had arterial disease in association with their ulcers but by the time the ninth decade was reached the probability had increased to 50%.

Side of ulceration—Eighty nine of the legs with ulcers and low pressures were left sided and 87 right sided. The distribution of arterial disease was therefore virtually equal between the two sides, in contrast with the distribution of venous disease, which is more common on the left.

Site of ulceration—Twenty seven of the 51 ulcers which affected the foot (53%) were associated with low resting pressure indices, whereas only 149 of the 776 more proximal ulcers (19%) were associated with low resting pressure indices. This difference was significant (p<0.01).

Relation between venous disease and arterial disease—Patients with venous disease were examined for the stigmata of chronic venous insufficiency—that is, varicose veins, pigmentation, eczema, or lipodermatosclerosis. Of the 176 legs with arterial impairment, 92 (52%) had varicose veins or the typical changes of chronic venous insufficiency in skin or subcutaneous tissues, or both.

Discussion

In this study 600 patients with chronic leg ulcers from the general population were interviewed, examined, and tested individually. This represents by a considerable margin the largest series yet published. The Czechoslovakian study by Bobek et al of a district population of some 22000 detected 153 patients with ulcers.1 This was the Basle study in which 4376 industrial workers were examined and yielded only 57 with ulcer disease.2

We could not screen the entire 1477 patients in detail but a comparison of demographic features between the 1477 patients in the total series and the 600 in the sample group suggested that the two groups were comparable.

PROPORTION OF PATIENTS WITH VASCULAR DISEASE

Slightly less than a quarter of the patients with leg ulceration had evidence of arterial insufficiency in the ulcerated limb as measured by Doppler pressures. This suggests that it is essential to look for arterial disease in any patient with a leg ulcer before compression treatment is applied. It is immaterial whether the arterial disease plays an important part in the pathogenesis of the ulcer. If compression is applied it is likely to contribute to failure to heal and may cause further ulceration over pressure points.

ARE PULSES ENOUGH?

Table II shows that, though there is considerable correlation between the absence of pulses and the presence of arterial impairment, both false negative and to a less extent false positive results abound. Pulses may frequently be impalpable when there is oedema, which is common in this group of patients. Conversely, pulsae may be felt by a skilled examiner even when there is a considerable degree of peripheral arterial disease. This study suggests that unless strong pulses can be felt in the affected leg the brachial to ankle Doppler pressure ratio should be measured before compression is applied.

WHAT IS SIGNIFICANT ARTERIAL DISEASE?

Opinion is divided among vascular specialists about what level of depressed Doppler pedal pressures represents "significant" arterial disease. Patients presenting with claudication usually have resting pressure indices in the range 0.5-0.9, whereas those with rest pain and impending gangrene generally have indices below 0.5. Patients with calcified vessels, however, may have deceptively high pressure readings. Our findings may therefore understate the reality of arterial disease.
frequency and severity of arterial disease. Only 31 patients gave a history of intermittent claudication, but many of the more elderly patients had such limited mobility that claudication would not be apparent.

Elastic bandages and compression hosiery exert pressures of up to 60 mm Hg (manufacturers' specifications). Above that level they are unlikely to be tolerated for long by patients with normal sensation. More usual pressures of a newly applied bandage are around 30 mm Hg at ankle level. Pressures are not evenly distributed around the circumference of the limb but, according to the Laplace formula, are much greater over bony or tendinous prominences, where convexity is greater; hence the greater vulnerability of the skin over the malleoli, the tendo Achillis, and the anterior tendons at the ankle to compression bandages. Indeed, it was our experience of seeing limbs lost as a result of necrosis induced by compression bandaging that stimulated this aspect of the survey. The two consultant surgeons in this study have encountered eight cases in recent years where the application of compression has precipitated amputation, and less degrees of damage are much more common. We are currently carrying out a survey among consultant surgeons in Scotland in an attempt to quantify the incidence of this complication. Analysis of the interim results shows over 100 cases of compression induced damage. The recent availability of higher quality bandages, especially those which maintain their pressure by self-adhesion, increases the hazard.

Studies of blood flow with external compression in normal subjects have shown severe diminution of flow under the area of compression. As the level of compression approaches that of the diastolic blood pressure flow halts in the compressed area. Palbst et al have shown that the skin perfusion pressure in ischaemic limbs, measured by laser Doppler flowmetry during external compression, is noticeably less than in normally perfused limbs. In ischaemic feet mean skin perfusion pressures as low as 11 mm Hg were recorded. Holstein found that in ischaemic legs the washout of a subcutaneous radioactive isotope can be prevented by external compression with pressures as low as 20 mm Hg.12 The experimental application of sustained pressure to patients known to have arterial insufficiency presents ethical difficulties. Nevertheless, the reviewed evidence leaves little doubt of the danger to these patients.

**PREDICTIVE FACTORS**

The first predictive factor—that is, age—was the most significant (see figure). Though the overall frequency of arterial disease in association with leg ulceration was 21%, there were no instances below the age of 40, and the frequency rose steadily to 50% in the very elderly. Therefore, in this group in particular the possibility of arterial disease should always be considered. The slightly higher proportion of men with arterial disease was expected, but the difference was not large enough to be of any practical value; in any case, the gap is likely to narrow further in the future with the relative increase in women smokers.

As atherosclerosis is a generalised condition we thought that the presence of ischaemic symptoms at other sites would increase the risk of arterial disease in the legs. This was found to be true, as the risk rose from 19% to 33% for this factor alone. Intermittent claudication was associated with Doppler evidence of arterial disease in every case. The results confirmed that manifestations of arterial disease are more likely to be found in the distal part of the leg. Patients with ulceration anywhere on the foot should be regarded as having arterial disease until proved otherwise. Nevertheless, it is important to emphasise that the presence of an ulcer in the classical venous site—that is, the gaiter area—should not lead to the assumption that arterial disease is not present.

The well known features of chronic venous insufficiency are varicose veins in association with changes of lipodermatosclerosis in the subcutaneous tissues consisting of pigmentation, eczema, inflammation, and fibrosis. When these are absent the doctor or nurse may be alerted to the possibility of other aetiologies, including arterial disease, and will probably appreciate the need to avoid compression bandaging. Less easy is the setting in which patients with arterial impairment also have evidence of venous insufficiency (52% of legs in this study with low resting pressure indices had evidence of chronic venous insufficiency). This is likely to lead to the arterial disease being overlooked. The use of elastic compression in such patients becomes a matter for individual judgment. It is our policy to use light compression—for example, a bandage pressure of not more than 20 mm Hg—in the gaiter area, provided that the brachial to ankle pressure index is not less than 0.7. This, however, is a somewhat empirical approach. The management of patients who have combined arterial and venous disease deserves more detailed study.

We conclude that patients with chronic leg ulcers should carefully be examined for arterial disease. Age, the presence of arterial disease at other sites, and distal ulceration on or partly on the foot should alert the clinician to the possibility of arterial disease. If the pulses are not easily felt Doppler pressures should be measured.

We thank the many medical, nursing, and physiotherapy colleagues in the community and in hospitals who made this study possible.

**References**