Venous Disease is Associated with an Impaired Range of Ankle Movement*

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Objectives: to investigate the relationship between clinical severity of venous disease, calf muscle pump dysfunction and range of ankle movement (ROAM).

Materials and Methods: ROAM was assessed by goniometry in the supine, non-weightbearing position. Calf muscle pump function was assessed by ambulatory venous pressure (AVP), calculating the pressure relief index (PRI). Venous disease was classified according to the CEAP classification (International Consensus Committee reporting standards on venous disease). Forty seven limbs in 38 adults were recruited and matched for age: 11 normal controls CEAP0, 12 varicose veins CEAP2, 12 chronic venous insufficiency CEAP 4,5 and 12 active ulceration CEAP 6.

Results: mean (s.e.m.) age was 60.7 (1.3) years. Mean (s.e.m.) PRI was 1959.6 (313.7) in CEAP0, 905.3 (139.3) in CEAP2, 596.5 (148.5) in CEAP4,5 and 170.6 (2.6) in CEAP6 (p < 0.001, ANOVA). Mean (s.e.m.) ROAM was 61.3 (2.0)° in CEAP0, but significantly reduced to 49.7 (2.0)° in CEAP2, 42.1 (2.6)° in CEAP4,5 and 40.9 (2.7)° in CEAP6 (p < 0.004, ANOVA post hoc Tukey). PRI correlated with ROAM (p < 0.001, Pearson correlation coefficient r = +0.52).

Conclusions: limbs with venous hypertension have a reduced range of ankle movement related to the clinical severity of venous disease.

Key Words: Venous insufficiency; Ambulatory venous pressure; Goniometry.

Introduction

Chronic venous disease may be caused by calf muscle pump dysfunction. Sustained elevation of venous pressure at the ankle is the main cause of venous ulceration, whether the raised pressures are due to an impairment of the calf muscle pump, or whether they are due to superficial or deep vein disease. The incidence of venous ulceration is also increased in patients who have a decreased calf muscle pump ejection fraction indicating that calf pump failure is an important factor in venous disease leading to ulceration.1 Ulceration and ambulatory venous pressure (AVP) is also closely related, in that an increased incidence of ulceration is associated with an increase in AVP.2 Measurable deficiencies of the calf muscle pump in limbs with active ulceration compared to limbs with healed ulceration further indicate that deficiency of the pump is related to the severity of venous disease.3

Although duplex imaging accurately identifies anatomical sites of venous incompetence and obstruction, it gives little information on venous function. Calf pump function and the degree of venous incompetence are more reliably assessed by AVP studies which are regarded as the gold standard. Several published studies have used air plethysmography (APG) to measure venous volume changes but the ability of APG to quantitatively separate different classes of venous insufficiency is inconsistent and unreliable.1,4,5 Effectiveness of the calf muscle pump may also be related to the range of movement of the ankle joint.6 Normal functioning of the pump relies on normal movement of the ankle joint, particularly dorsiflexion. The aim of this study was to investigate whether range of ankle movement (ROAM) measured by goniometry is related to the severity of venous insufficiency measured by AVP and the clinical severity of venous disease.
Materials and Methods

Patients and controls

Following ethical committee approval and informed consent in writing, 38 adult subjects (22 male, 16 female) with various clinical severities of venous disease and normal controls without venous disease, were recruited from the venous clinics of South Manchester University Hospitals NHS Trust and from general practitioner (GP) lists, respectively. Patients and controls were taken from the same urban, inner-city population. Patient selection was prospective by clinical presentation and aimed at obtaining an age-matched population using a birth date within two years of subjects matched to each group. Controls were recruited by contacting the GP of a matched patient, requesting a list of five subjects having a birth date within two years of that patient, one of whom was then recruited. Subjects with diabetes, ankle-brachial pressure index ≤0.9, arthritis, immobility, previous stroke, previous limb injury or fracture, ulcers over the area of the ankle joint, painful ulcers and subjects with a body mass index ≥30 were excluded from the study.

Limbs were classified according to the International Consensus Committee protocol on reporting standards in venous disease based on Clinical, aEtiological, Anatomical and Pathophysiological data (CEAP):7 C0 no visible or palpable signs of venous disease, C1 telangiectasis or reticular veins, C2 varicose veins, C3 oedema, C4 skin changes ascribed to venous disease (pigmentation, venous eczema, lipodermatosclerosis), C5 skin changes ascribed to venous disease as above with healed ulceration and C6 skin changes ascribed to venous disease as above with active ulceration. Where subjects had limbs of different clinical severities and therefore of different CEAP clinical class, only the most severe limb was included in the study to avoid bias from other factors such as immobility. Limbs were included in only one CEAP class in the study and not double counted in separate groups.

Four groups of subjects (47 limbs) were recruited with varying clinical grades of venous disease:

(i) 11 limbs (eight subjects) with no evidence of venous disease CEAP0
(ii) 12 limbs (10 subjects) with varicose veins and no evidence of chronic venous insufficiency CEAP2
(iii) 12 limbs (nine subjects) with chronic venous insufficiency CEAP4 (eight), CEAP5 (four),
(iv) 12 limbs (11 subjects) with active ulceration CEAP6.

Patients wearing four layer bandaging or compression hosiery had this removed prior to undergoing investigations. All patients underwent goniometry, duplex imaging of the deep and superficial veins, and AVP measurement of venous function.

Goniometry

ROAM was measured by a chartered physiotherapist. Measurements of maximal, voluntary, plantar flexion and dorsiflexion in the supine, nonweightbearing position (active unassisted motion) were taken from a neutral 90° position using a portable goniometer.8 ROAM was defined as the sum of plantar flexion and dorsiflexion.

Duplex imaging

All subjects underwent colour duplex imaging while standing (Advanced Technology Laboratories, Bothel, Washington, HDI 5000) performed by an experienced vascular technologist using B-mode real time ultrasound and a 4–7 MHz probe. The leg being examined was relaxed with the knee slightly bent and weight supported on the opposite leg. Venous reflux was identified by reverse blood flow of one second or greater following the release of firm calf compression. The deep veins, sapheno-femoral junction, long saphenous vein, sapheno-popliteal junction, short saphenous vein and all perforating veins were examined for patency and competence.

Ambulatory venous pressure studies

Following goniometry and duplex imaging, AVP studies were performed in the afternoon. A 21-gauge “butterfly” needle was inserted into a dorsal foot vein and connected to a pressure transducer at floor level, an amplifier and recorder (Lectromed II). Continuous pressure readings (mmHg) were recorded on standing. The patient was instructed to perform ten tip-toe exercises, one per second, whilst holding on to a frame for support. The resting pressure (AVPr), fall in pressure (AVPf), minimum pressure following exercise (AVPmin) and recovery time to 90% of original resting pressure (AVPrt90) were recorded. The pressure relief index (PRI) was calculated from the fall in pressure (AVPf = AVPr – AVPmin) multiplied by the recovery time, giving an overall measure of venous function (PRI = AVPf × AVPrt90). Measurements were repeated with an above-knee 2.5 cm tourniquet inflated to 180 mmHg to obstruct the long saphenous vein, a below-knee 2.5 cm tourniquet inflated to 140 mmHg.
to obstruct the short saphenous vein and distal long saphenous vein, then with both tourniquets inflated simultaneously to obstruct both saphenous veins. Measurements for each stage were repeated three times and the mean of these taken in analysis.

Statistical analysis

Investigators were blinded to each others results. Data was inserted into an SPSS database and analysed using SPSS 10.1 for Windows. The data presented is regarded as being normal in distribution and the results are presented as means with standard error of the mean (s.e.m.) and standard deviation (s.d.). Differences between groups were compared through analysis of variance (ANOVA) and differences among groups were compared through analysis of variance with post hoc testing by Tukey’s studentized test (reducing the likelihood of variables being interpreted as significant when in fact significance has arisen by chance). Where four groups are compared to each other (six comparisons), the Tukey test states that the p-value should be divided by six (0.05/6 ^ 0.0083). Values smaller than this are regarded as significant and values larger than this as nonsignificant. Correlation was by Pearson’s correlation coefficient.

Results

The mean (s.e.m.) age of all subjects was 60.7 (1.3) years. There was no significant difference in age between the groups (p = 0.85, ANOVA).

The results of clinical staging and duplex imaging (CEAP classifications) are shown in Table 1. There were no subjects in CEAP1 (telangiectasis or reticular veins only) and none in CEAP3 (oedema alone). One of the 12 limbs in CEAP2 had deep vein and perforator disease but the majority of limbs in this class had superficial venous disease alone as would be expected. Deep venous disease and “secondary” venous disease increased in frequency as the clinical severity of disease worsened.

Table 2 shows the results of the AVP measurements in the 47 limbs. Resting pressure AVPr, was not significantly different between the groups (p = 0.15, ANOVA). AVPmin was significantly different between groups (p < 0.001, ANOVA) and showed a step-wise increase as severity of venous disease

<table>
<thead>
<tr>
<th>Clinical severity of venous disease</th>
<th>CEAP class</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal controls</td>
<td>C0</td>
<td>11</td>
</tr>
<tr>
<td>Varicose veins</td>
<td>C2EP AsPr</td>
<td>9</td>
</tr>
<tr>
<td>Chronic venous insufficiency</td>
<td>C4EP AsDPr</td>
<td>8</td>
</tr>
<tr>
<td>Active ulceration</td>
<td>C6EP AsDPr</td>
<td>1</td>
</tr>
</tbody>
</table>

Table 2. Age, AVP measurements and PRI in the CEAP groups.

<table>
<thead>
<tr>
<th>CEAP class</th>
<th>n</th>
<th>Age (years)</th>
<th>AVPr (mmHg)</th>
<th>AVPmin (mmHg)</th>
<th>AVPf (mmHg)</th>
<th>AVPrt 90 (s)</th>
<th>PRI (AVPf / AVPrt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CEAP0</td>
<td>11</td>
<td>59.6 (2.3)</td>
<td>92.3 (3.9)</td>
<td>24.9 (3.3)</td>
<td>67.4 (5.0)</td>
<td>30.9 (5.5)</td>
<td>1959.6 (313.7)</td>
</tr>
<tr>
<td>CEAP2</td>
<td>12</td>
<td>60.9 (3.4)</td>
<td>104.3 (1.8)</td>
<td>41.1 (2.5)</td>
<td>63.5 (2.3)</td>
<td>14.4 (2.2)</td>
<td>905.3 (139.3)</td>
</tr>
<tr>
<td>CEAP4,5</td>
<td>12</td>
<td>62.5 (1.9)</td>
<td>105.6 (4.1)</td>
<td>46.8 (6.0)</td>
<td>58.8 (7.4)</td>
<td>9.2 (1.5)</td>
<td>596.5 (148.5)</td>
</tr>
<tr>
<td>CEAP6</td>
<td>12</td>
<td>59.8 (2.4)</td>
<td>109.8 (4.8)</td>
<td>72.1 (5.1)</td>
<td>37.8 (5.8)</td>
<td>3.8 (1.1)</td>
<td>170.6 (59.0)</td>
</tr>
</tbody>
</table>

p-value ANOVA

AVPr = resting pressure, AVPmin = minimum pressure, AVPf = fall in pressure (AVPr – AVPmin), AVPrt 90 = recovery time to 90% of original, PRI = pressure relief index (AVPf / AVPrt 90).

Data is shown as the mean (s.e.m.) and [s.d.].

Significantly different to controls (CEAP0) p < 0.008, ANOVA, post hoc Tukey.

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worsened from 24.9 (3.3) mmHg in CEAP0 to 72.1 (5.1) mmHg in CEAP6 ($p < 0.001$, ANOVA, post hoc Tukey). AVP$f$ was also significantly different between groups ($p = 0.04$, ANOVA) although there was no significant difference among groups. AVP$r$ was significantly different between groups ($p < 0.04$, ANOVA) although there was no significant difference among groups. AVP$r_{90}$ was significantly different between groups ($p < 0.001$, ANOVA) showing a significant shortening compared to controls in all CEAP classes as clinical severity of venous disease worsened ($p < 0.001$, ANOVA, Tukey).

PRI showed a step-wise decrease related to the severity of venous disease (Fig. 1, Table 2). PRI was significantly different between groups ($p < 0.001$, ANOVA). Mean (s.e.m.) PRI in CEAP0 was 1959.6 (313.7). Limbs in CEAP2 had a reduction in mean PRI to 905.3 (139.3) ($p < 0.003$, ANOVA, Tukey) with PRI even lower in CEAP4,5 at 596.5 (148.5). The lowest PRI at only 170.6 (69.0) was in CEAP6.

Results of plantar flexion, dorsiflexion and range of ankle movement are shown in Table 3. ROAM was significantly different between groups ($p < 0.001$, ANOVA) and reduced in all groups when compared to CEAP0 controls ($p < 0.004$, ANOVA, Tukey). In CEAP0, mean (s.e.m.) ROAM was 61.3 (6.6)°, reduced in CEAP2 to 49.7 (2.0), in CEAP4,5 to 42.1 (2.6) and in CEAP6 to 40.9 (2.7)° (Fig. 2).

The correlation coefficients between PRI and plantar flexion are $r^* = 0.36$ ($p = 0.014$), between PRI and dorsiflexion $r^* = 0.43$ ($p = 0.03$) and between PRI and ROAM $r^* = 0.52$ ($p < 0.001$) (Fig. 3).

![Fig. 1. Pressure relief index in the CEAP groups. Bars (–) show the mean values.](image)

![Fig. 2. Range of ankle movement (°) in the CEAP groups. Bars (–) show the mean values.](image)

![Fig. 3. Graph showing the correlation between pressure relief index and range of ankle movement. Pearson correlation coefficient $r = 0.52$ ($p < 0.001$).](image)

**Table 3. Plantar flexion, dorsiflexion and range of ankle movement (ROAM) in the CEAP groups.**

<table>
<thead>
<tr>
<th>CEAP</th>
<th>Plantar flexion (°)</th>
<th>Dorsiflexion (°)</th>
<th>ROAM (°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>47.0 (1.7)</td>
<td>14.4 (1.7)</td>
<td>61.3 (2.0)</td>
</tr>
<tr>
<td>2</td>
<td>39.5 (1.9)</td>
<td>10.2 (1.0)</td>
<td>49.7 (2.0) *</td>
</tr>
<tr>
<td>4,5</td>
<td>32.9 (2.1) *</td>
<td>9.2 (2.4)</td>
<td>42.1 (2.6) *</td>
</tr>
<tr>
<td>6</td>
<td>37.5 (2.6) *</td>
<td>3.4 (0.6) *</td>
<td>40.9 (2.7) *</td>
</tr>
<tr>
<td>p-value ANOVA</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

ROAM = range of ankle movement.

Data is shown as the mean (s.e.m.) and [s.d.].

*Significantly different to controls (CEAP0) $p < 0.008$, ANOVA, post hoc Tukey.
Discussion

Our results show that the clinical severity of venous disease is related to the severity of venous hypertension as measured by AVP. We have also shown that venous hypertension is associated with a reduced ROAM which becomes more severe with increased severity of venous hypertension and clinical severity of venous disease.

AVP measurement, first performed by Barber and Shatara in 1925, is regarded as the gold standard method of assessment of venous pressure. Pollack and Wood published a thorough review of AVP during exercise and changes in posture showing that the average venous pressure at the ankle is equal to the hydrostatic pressure exerted by a column of blood extending from the ankle to the heart and that contraction of the calf muscles by standing on the toes causes a fall in venous pressure at the ankle. Stretch of the plantar veins and calf muscle during weightbearing and dorsiflexion may be a key mechanism by which blood is pumped from leg veins reducing venous pressures at the ankle.

Normal movement of the ankle joint relies on a functioning system of bones, joints, muscles, nerves and blood supply. Factors such as pain, immobility, rheumatoid arthritis and osteoarthritis may all reduce ankle movement and we therefore excluded subjects with any of these conditions to avoid confounding the results. ROAM reduces with age such that it is 15% less in the eighth decade of life compared to the third. In this study patients were matched for age, the mean age of subjects being 61 years.

Normal nonweightbearing range of movement of the ankle joint has been reported as 40–54° for plantar flexion and 7–15° for dorsiflexion, giving a total ROAM of 47–69°. The same authors also present cases in which both dorsiflexion and plantar flexion are much less than or vastly in excess of the reference ranges which are based on means ± two s.d. In our study mean (s.e.m.) plantar flexion in controls was 47 (1.7)°, dorsiflexion was 14.4 (1.7)° and ROAM was 61.3 (2.5)°, all of which lie within the reference ranges for ankle movements. The reduction in ROAM in our study appears to be due to a combination of a reduction in both plantar flexion and dorsiflexion.

Tierney et al. showed a significant and prolonged impairment in venous pump function (assessed using APG) following ankle fracture in the absence of significant obstruction, persisting for up to 18 weeks. The association between ulcers and loss of mobility of the ankle joint was also noted by Dickson Wright in 1931 and later by Ruckley who showed that 32% of patients with ulcers had severe limitation of ankle movement but only 9% had rheumatoid arthritis. This suggests that ankle disease alone is not the only factor involved and there may be a mechanism whereby venous hypertension leads to an ankle arthropathy. Indeed, our results unexpectedly indicate that even in CEAP2 there is a reduction in ankle movement related to venous hypertension. Impairment of venous return due to an arthropathy and an arthropathy due to venous hypertension could perpetuate further calf muscle pump dysfunction.

Our study has indicated that patients with varicose veins and no evidence of chronic venous insufficiency (CEAP2) have a reduced ROAM. Lipodermatosclerosis is known to be associated with periostitis, subcutaneous calcification and subcutaneous fibrosis and there is some evidence that venous insufficiency may cause inflammatory change in the skin and periostium. These factors could explain a reduction in ROAM in CEAP classes four and above. Oedema may cause a reduction in ankle movement in patients in CEAP class three and above but cannot account for the reduction in patients in CEAP class two. The mechanism by which venous hypertension may lead to a reduction in ROAM even in patients with varicose veins requires further investigation.

Klyscz et al. investigated the effect in patients with chronic venous insufficiency of an intensified six-week-long physical training programme aimed at improving function of the calf muscle pump, strengthening the calf muscles and improving ankle joint mobility. Their results indicated an improvement in plantar flexion and dorsiflexion in patients compared to controls, although whether or not this effect is preserved long term was not assessed. Similarly, poor calf muscle pump function in patients with chronic venous ulceration can be improved by physical exercise.

Our study has demonstrated that limbs with all grades of venous disease from simple varicose veins to venous ulceration are associated with venous hypertension and a significantly reduced ROAM. Whether this is cause or effect remains uncertain but therapies to improve ROAM may improve venous function. The study was conducted with researchers blinded to each others results so as to reduce potential bias. We also excluded as many confounding factors as possible such as arthritis, ankle fractures, obesity and immobility all of which could have an effect on venous disease and ankle movement and we used post hoc statistical tests to reduce the potential for results to become significant when in fact they are not. The weaknesses of our study are that the measurement of ankle movement was passive in the supine, nonweightbearing position. Active ankle
movements in the upright, walking, weightbearing position may provide a more accurate assessment of ROAM with more relevance to normal functioning of the calf muscle pump. Further studies in our unit will focus upon the relationship of venous pressures to active, weightbearing movement in an attempt to answer some of these questions. Long term compression bandaging in patients with ulcers may produce some degree of ankle atrophy which could confound results in CEAP groups five and six. However, the reduction in ROAM in patients with varicose veins suggests that venous hypertension causes a reduction in range of movement of the ankle joint rather than vice versa.

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References


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