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Falsely Elevated Ankle Pressures in Severe Leg Ischaemia: The Pole Test—An Alternative Approach*

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Ankle–brachial pressure indices (ABPIs), measured by sphygmomanometer and Doppler probe, are an accepted index of chronic leg ischaemia. However, tibial artery sclerosis or calcification decreases compliance, producing falsely elevated cuff occlusion pressures. Arterial cannulation is invasive and impractical, but by elevating the foot and measuring the height at which the Doppler signal disappears, ankle systolic pressure in mmHg can be derived. Using an 8MHz Doppler apparatus and calibrated pole, ankle systolic pressures measured by sphygmomanometer and elevation were compared in 49 severely ischaemic legs (40 patients). ABPIs were derived by dividing ankle systolic pressure by brachial pressure. Median (interquartile range) ABPI assessed by sphygmomanometry was 0.46 (0.35–0.56). Median ABPI measured by leg elevation was significantly lower at 0.21 (0.14–0.30), $p < 0.0001$, Wilcoxon. In 20 patients undergoing in situ *vein bypass grafting, direct transducer-derived pressure measurements were obtained. Median ABPI for this method was 0.15 (0.11–0.27). No significant difference was found when compared with ABPIs derived by elevation, median 0.2 (0.13–0.31), $p = 0.324$, however median ABPI measured by sphygmomanometry was significantly higher at 0.37 (0.27–0.6), $p = 0.0008$. Correlation of elevation with transducer-assessed pressure measurements ($r = 0.88$) was closer than with cuff-derived measurements ($r = 0.69$). Pressures derived by leg elevation provide a more accurate index of severe leg ischaemia than sphygmomanometry, although the technique is limited to assessing pressures of less than approximately 60 mmHg. Falsely elevated ABPIs may underestimate the extent of disease in patients assessed for vascular reconstruction.*

Key Words: Critical leg ischaemia; Ankle systolic pressure; Pole Test; Elevation; Doppler ultrasound.

Introduction

Quantification of the severity of lower limb ischaemia remains an elusive and topical goal.[1–4] Introduction of ankle systolic pressure measurements and derivation of an ankle–brachial pressure index (ABPI) originally provided a simple means of quantifying leg ischaemia which appeared to correlate with the severity of symptoms.[5] However, patients often present with disproportionately high ankle pressures compared to their clinical symptoms. Causes of reduced arterial wall or tissue compliance, resulting in abnormally elevated ankle occlusion pressures, include vascular calcification in diabetes,[6] arterial sclerosis, or chronic induration of overlying soft tissues.[3,7,8] High occlusion pressures may also occur in non-diabetic patients with lesser degrees of arterial disease.[9]

Buergers' sign of foot pallor on elevation and erythema in dependency is a reliable clinical sign denoting severe chronic lower limb ischaemia.[10] We observed that the level at which the foot becomes pale on elevation is the level at which the Doppler signal over the dominant ankle artery becomes inaudible. The height of the ankle above the heart at which this occurs is determined by systolic arterial pressure, and the elasticity of the arterial wall. If the arterial wall is non-compliant then the height of elevation of the ankle at which the Doppler signal disappears should represent systolic blood pressure. A pole calibrated to measure systolic pressure in mmHg was designed. This was used to measure the height at which the arterial Doppler signal at the ankle became inaudible. ABPIs derived by this method were compared with those obtained by sphygmomanometry in patients with severely ischaemic legs.

Patients and Methods

Patients presenting to our Vascular Surgical Units with severe leg ischaemia were assessed. All measurements were performed after the patients had rested for a minimum of 30 minutes.

The three ankle arteries were insonated with an 8MHz Doppler probe (Huntleigh, U.K. Ltd.), the posterior tibial artery behind the medial malleolus, the dorsalis pedis artery over the tarsal bones, and

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the anterior communicating branch of the peronea artery, antero-superior to the lateral malleolus Sphygmomanometer pressures were derived for eacl artery by placing a 15 cm wide cuff around the ankl immediately proximal to the malleoli. The cuff wa inflated with the patient supine until the Doppler signal disappeared, and then deflated slowly. The pressure at which a pulsatile signal first reappeared was taken as the systolic pressure for that artery. Measurements were repeated for the three ankle arteries if present and ABPIs were derived by dividing highest systolic ankle pressure by brachial systolic pressure (obtained by the same method).

Pressure measurements were then repeated using the calibrated pole. Zero pressure with the patient supine was regarded as the level of the left ventricle and taken to be the anterior axillary line. In 20 patients mean (s.d.) vertical distance between the bed and the anterior axillary line was 18 (± 1.25) cm, and this was permanently marked as zero pressure on the pole. Calibration marks on the pole were sited at 13 cm intervals, corresponding to pressure increments of 10 mmHg, corrected for the relative density of blood and gravity. Pressure calibrations continued to 80 mmHg. This was sufficient to assess systolic pressure in all leg lengths encountered in this study. The pole was mounted in a block to allow it to stand freely in a vertical position.

With the patient lying supine, and the Doppler probe held in place over the artery to give an optimum signal, the foot was slowly elevated until the signal disappeared (Fig. 1). The height of the ankle at which this occurred was marked on the pole, recorded as arterial systolic pressure in mmHg, and divided by the brachial pressure measured by sphygmomanometry to obtain the ABPI(Pole). The elevation manoeuvre was repeated until loss of the signal at a given height was observed consistently, to avoid probe displacement errors.

Direct blood pressure measurements were obtained in patients who underwent femorodistal vascular reconstruction using *in situ* long saphenous vein as the graft conduit. Peroperatively arterial blood pressure was recorded via an indwelling radial arterial cannula connected through a heparinised saline-filled fine bore tube to an electronic pressure transducer and monitor. With the patient supine, radial pressure was found to be equal to brachial pressure. After establishing graft flow and ensuring all patent venous tributaries were ligated,[11] a second arterial cannula was secured into the graft via a distal sidebranch and connected to the transducer and monitor. With the graft fully open, an arterial pressure wave analogous to the radial artery waveform was obtained. Temporary occlusion of graft inflow produced a fall in intragraft pressure measured at the distal anastomosis, to the level of that maintained by the remaining collateral circulation. This pressure was then divided by the synchronous radial systolic pressure to derive a peroperative intraarterial ankle-brachial (radial) pressure index. ABPIs measured by pressure transducer were compared with ABPIs derived by elevation and sphygmomanometry after induction of anaesthesia but prior to surgery.

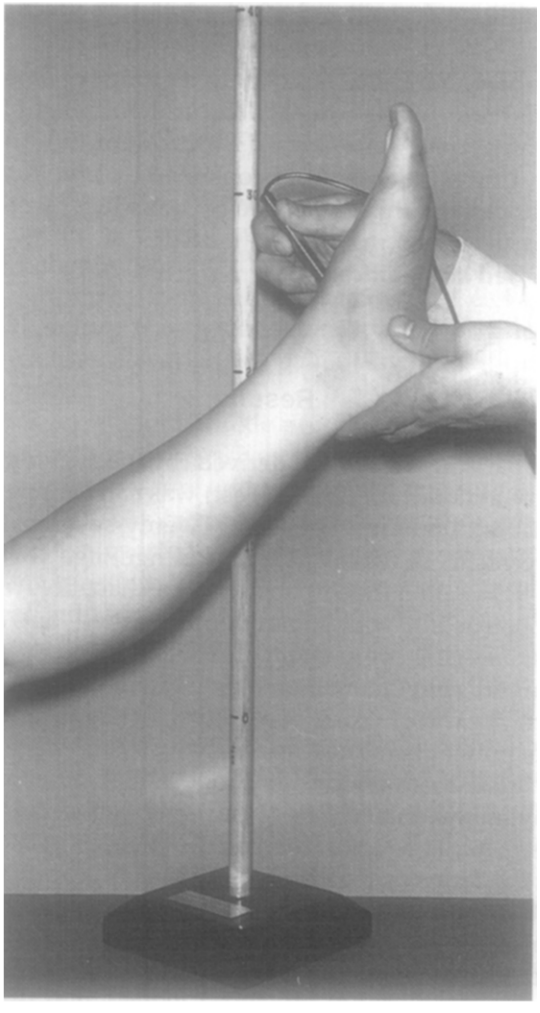

Fig. 1. Use of an 8MHz Doppler probe and calibrated pole to assess ankle systolic pressure. Systolic pressure in mmHg is determined from the pole as a function of height above the left ventricle at which the Doppler signal is lost on leg elevation.

By these means it was possible to compare three measurement techniques for determining both absolute ankle systolic pressures and ABPIs; two indirect, (sphygmomanometer and pole), and one direct, (graft pressure transducer).

Statistical Analysis

Non-parametric analysis of paired groups of data was carried out using the Wilcoxon Rank Sign Test. Unpaired data was analysed by the Mann-Whitney U Test. Correlations of ABPIs obtained by non-invasive methods with those obtained by transducer were derived by linear regression. p values of <0.05 were regarded as significant.

Results

Comparison of ABPIs derived by sphygmomanometry and elevation (Pole Test) was carried out in 49 ischaemic limbs in 40 patients. Twenty-seven patients were male; 13 female. Median (range) age was 71.4 (52–91) years. Fifteen patients were diabetic. Arterial reconstruction was subsequently carried out in 20 legs, allowing comparison of ABPIs derived by elevation and sphygmomanometry immediately preoperatively, with direct operative arterial pressure measurements in these cases.

Median (interquartile range) ABPI assessed by sphygmomanometry in 49 severely ischaemic limbs was 0.46 (0.35–0.56). Median ABPI measured in the same arteries by leg elevation was significantly lower at 0.21 (0.14–0.30), $p < 0.0001$, Wilcoxon. Fig. 2 shows the range of differences in individual limbs. There was wide disparity between pressures measured by cuff and elevation and in five cases cuff occlusion pressures were more than four times greater than those recorded using the elevation tech-

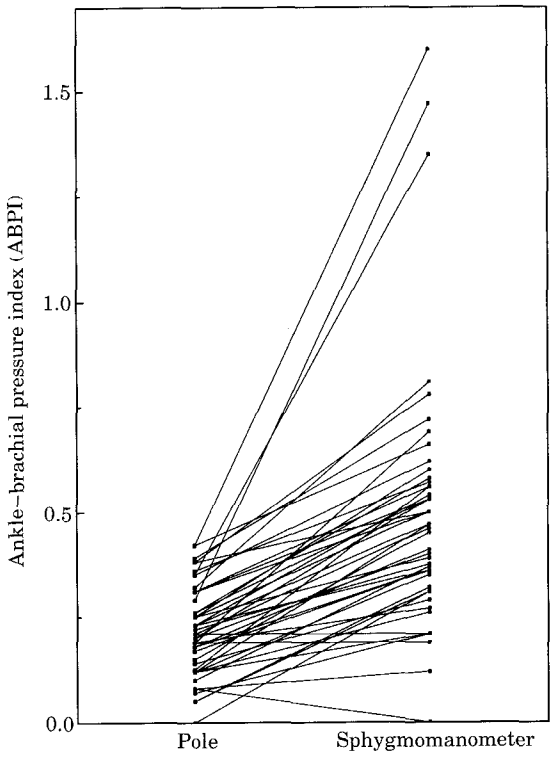

Fig. 2. Variation of ABPIs assessed by sphygmomanometry and elevation in 49 severely ischaemic limbs. Median (interquartile range) ABPI—pole 0.21 (0.14–0.30); sphygmomanometry 0.46 (0.35–0.56), ($p < 0.0001$, Wilcoxon).

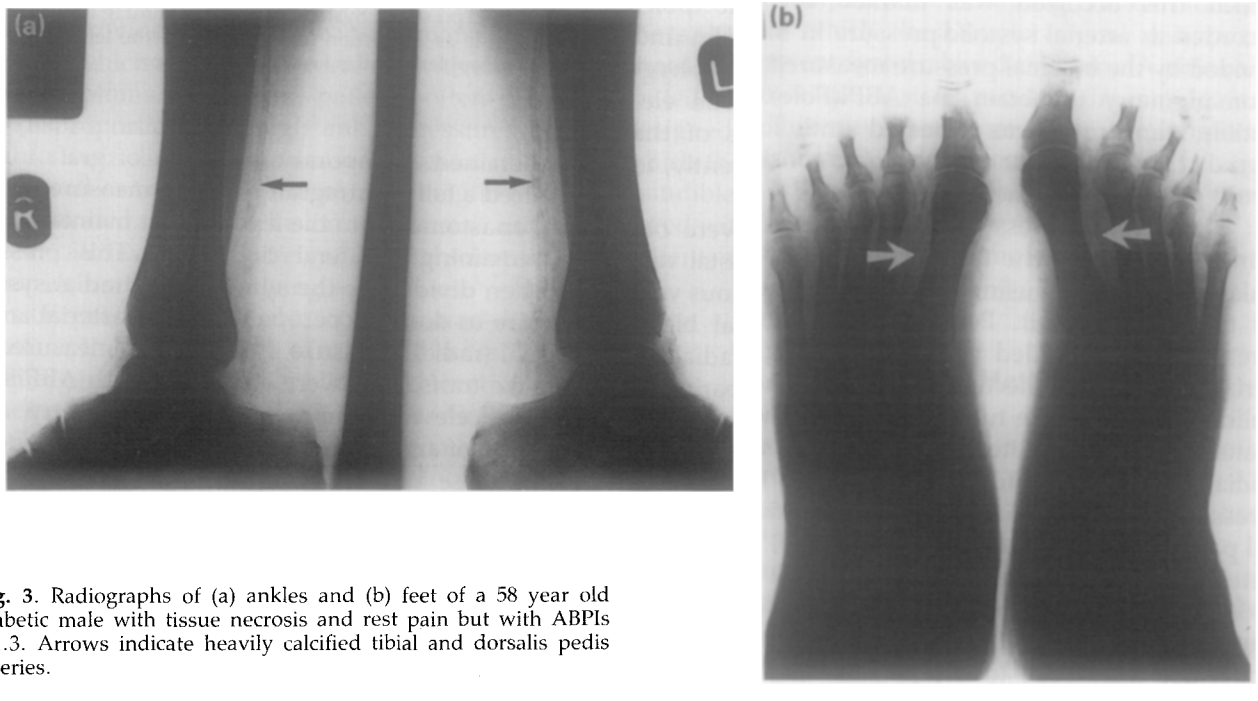

Fig. 3. Radiographs of (a) ankles and (b) feet of a 58 year old diabetic male with tissue necrosis and rest pain but with ABPIs >1.3. Arrows indicate heavily calcified tibial and dorsalis pedis arteries.

nique. Two patients with rest pain and sphygmomanometry-derived ABPIs of >1.3 were found to have severe tibial artery calcification on lower limb radiography (Fig. 3).

In 20 patients undergoing *in situ* vein bypass grafting, direct transducer-derived measurements were obtained. There was closer correlation between transducer and pole-derived measurements ($r = 0.88$) than with those obtained by sphygmomanometry ($r = 0.69$) (Fig. 4). Median ABPI derived by graft pressure transducer was 0.15 (0.11–0.27). No significant difference was found when this method was compared with ABPIs derived by elevation, median 0.2 (0.13–0.31), $p = 0.324$, Wilcoxon, however median ABPI measured by sphygmomanometry was significantly higher, 0.37 (0.27–0.6), $p = 0.0008$, Wilcoxon.

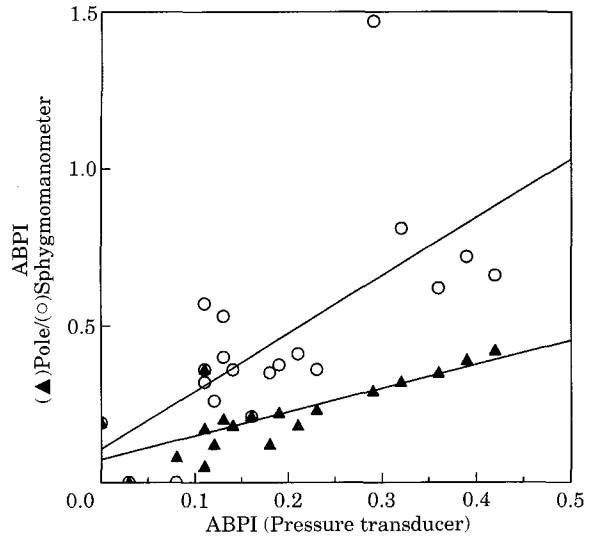

Fig. 4. Correlation of operative pressure transducer-derived ABPI measurements with ABPIs obtained by elevation (▲) ($r = 0.88$), and sphygmomanometry (○) ($r = 0.69$) in 20 patients undergoing femorodistal bypass.

Although median ABPIs measured by both elevation and sphygmomanometry were found to be higher in diabetics (0.23 and 0.54 respectively), than in non-diabetics, (0.2; 0.45), the differences did not achieve statistical significance in this relatively small group of patients ($p > 0.05$, Mann-Whitney). The difference between the two methods was greater in diabetics than in non-diabetics but this also was insignificant.

Discussion

ABPIs measured by sphygmomanometry were higher in all cases than when determined by elevation using the Pole Test. Intraoperative pressure measurements correlated closely with those derived by elevation, but significant differences occurred between ABPIs derived by cuff occlusion and both the other methods. No specific clinical markers enabled us to predict which cuff pressures were highly inaccurate rather than slightly inaccurate, other than a high index of clinical suspicion. The leg length of most patients prevented arterial pressures of greater than 60 mmHg from being measured by elevation, and therefore this technique is particularly useful only in the elevation of patients with critical leg ischaemia.[4]

Measurement of ankle pressure by the elevation method assumes a passive relationship between systolic arterial pressure and hydrostatic pressure at this level.[12] This has been questioned by other authors who have suggested than an increase in calf vascular resistance results in a smaller decrease in popliteal arterial pressure on leg elevation than might be expected.[13] However this was not found in this study of patients with critical ischaemia, where there was close correlation between pressures measured directly and by elevation. A generalised loss of arterial compliance in patients with severe vascular disease may be a major factor contributing to this almost linear relationship. The results of Goss *et al.* using a similar elevation technique also suggest a directly proportional relationship between arterial and hydrostatic pressure in the leg.[12]

Although median ABPI was higher in diabetics than in non-diabetics, this did not achieve statistical significance in this small group of patients, when measured by either sphygmomanometry or elevation, and there was also no significance in the difference between the two methods in diabetics and non-diabetics.

Recent attempts to redefine chronic critical leg ischaemia have drawn attention to the potential inaccuracy of sphygmomanometry-derived ankle pressure measurements.[4, 14] This is especially important in advanced peripheral vascular disease, and in the presence of vascular calcification, when arterial wall compliance and compressibility may be reduced. Ankle pressure measurements often play an important part in determining the initial management of such patients and falsely elevated pressures could result in inappropriate treatment or complacent management.

In routine usage in our vascular clinics, the "Pole Test" has proved a valuable adjunctive investigation for assessing the true severity of peripheral vascular disease in patients with signs or symptoms of critical leg ischaemia, but with paradoxically high ankle

pressures measured by sphygmomanometry. It is simple to carry out, requires minimal apparatus and appears to be more accurate than standard cuff occlusion techniques.

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