The Validity, Reliability, Reproducibility and Extended Utility of Ankle to Brachial Pressure Index in Current Vascular Surgical Practice

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Background. Despite the increasing sophistication of vascular surgical practice, more than three decades after its introduction to clinical practice, the ankle to brachial pressure index (ABPI) remains the cornerstone of non-invasive assessment of the patient with symptomatic peripheral arterial disease (PAD).

Aim. To summarise what is known about ABPI and critically appraise its validity, reliability, reproducibility and extended utility.

Methods. A MEDLINE (1966–2004) and Cochrane library search for articles relating to measurement of ABPI was undertaken; see text for further details.

Results. There is considerable disagreement as to how ABPI should be measured. Furthermore, various factors, including the type of equipment used, and the experience of the operator, can result in significant inter- and intra-observer error. As such, care must be taken when interpreting data in the literature. ABPI is valuable in the assessment of patients with atypical symptoms, venous leg ulcers and after vascular and endovascular interventions. However, absolute pressures are probably more valuable in patients with critical limb ischaemia. ABPI is also useful in subjects with asymptomatic PAD where it correlates well with, and may be used in screening studies to quantify, cardiovascular risk.

Conclusions. While its apparent simplicity can beguile the unwary, ABPI will continue to have a key role in the assessment of symptomatic PAD. ABPI is also likely to have extended utility in health screening and institution of best medical therapy (BMT) in asymptomatic subjects.

Keywords: Ankle brachial pressure index; Peripheral arterial disease.

Introduction

Despite the increasing sophistication of vascular surgical practice, more than three decades after its introduction into clinical practice, the ankle to brachial pressure index (ABI or ABPI) remains the cornerstone of non-invasive assessment of the patient with symptomatic peripheral arterial disease (PAD). While Travis Winsor was the first to report in 1950 that the ankle pressure is usually decreased if the arteries in the lower limb are obstructed, Yao from St Mary’s Hospital in London was the first to report in 1970 that the severity of disease correlated to the drop in the ABPI. More recently, it has been developed as an epidemiological tool for defining the natural history of PAD. Furthermore, ABPI is likely to play a key role in screening for individuals with asymptomatic PAD who are at high cardiovascular risk and would benefit from lifestyle advice and ‘best medical therapy’ (BMT). The purpose of this review is to critically appraise the validity, reliability, reproducibility and extended utility of the ABPI in clinical practice, and as a research tool, in primary and secondary care.

Methods

A MEDLINE (1966–2004) and Cochrane library search looking for articles relating to ABPI was performed. The terms ankle pressure, ABI, ABPI, ankle arm index...
(AAI), toe pressures, cuff width, claudication and angioplasty were amongst those included. These were linked with terms such as cardiovascular disease, diabetes and PAD. Further articles were identified by following MEDLINE links, by cross-referencing from the reference lists of major articles and by following citations for these studies. The studies were then graded with highest priority given according to the level of evidence.

Results

ABPI methodology

Several methods for measuring ABPI have been described. As well as producing different results in individual patients, these different methodologies are open to different forms of bias and will, therefore, be associated with different levels of reproducibility depending on the clinical circumstances. The higher of the two pressures in the dorsalis pedis (DP) or the posterior tibial (PT) arteries (or the peroneal artery if no audible signal is forthcoming from these two vessels) is conventionally taken as the ABPI numerator and the higher of the two brachial pressures as the denominator. In the absence of significant stenosis or occlusion in these vessels the two values are usually within 10 mmHg of each other even in the presence of more proximal disease. Other workers have suggested that averaging the DP and PT pressures correlates more closely with limb function. Sometimes brachial blood pressures are averaged and/or the brachial pressure is only measured in one arm; usually the right. It is worth noting that a pressure difference between the right and left brachial arteries of at least 20 mmHg is present in 3.5% of the normal healthy population and over 20% of patients with PAD. As such, the pressure should be measured in both arms. Although the higher of the two pressures will most closely reflect central aortic pressure, it is possible for patients with PAD to have bilateral subclavian-axillary artery occlusive disease although the precise incidence of this situation is difficult to determine. In these circumstances, both brachial pressures will be artificially low and the ABPI artificially elevated.

Another potential source of variability, particularly in longitudinal studies, is the non-linear relationship between the central and ankle systolic pressures and between central pressure and ABPI. In other words, for the same degree of lower limb disease, ABPI will be relatively lower in the presence of hypertension. Furthermore, as hypertension is treated the ABPI will appear to ‘improve’. Finally, given the phenomenon of ‘white coat hypertension’, it is likely that the ABPI measured by a vascular surgeon in hospital will appear lower that it would when measured by a nurse in the patient’s own home. It is wise, therefore, to record the absolute brachial and ankle pressures as well as ABPI.

The method used to measure the brachial arterial pressure also significantly affects the ABPI. As mercury sphygmomanometers are gradually phased out of clinical practice due to safety concerns, automated devices using the oscillometric principle (e.g. Dinamap) are becoming increasingly popular. Some studies suggest that the brachial and ankle pressures can be accurately obtained using these devices, and values closely resemble those obtained using a pneumatic cuff and Doppler probe.

The technique used to measure the brachial systolic pressure can also influence the value of the ABPI. Use of oscillometric devices tends to overestimate the systolic blood pressure when compared to using a standard sphygmomanometer and this discrepancy is more pronounced in subjects with stiff arteries. This methodological variability is not limited to ABPI. Toe pressures and cutaneous oxygen tension measurements are also significantly affected, particularly when taken from the second toe rather than the hallux.

The importance of blood pressure cuff position

It is worth re-emphasising that the ‘ankle pressure’ is actually the pressure under the cuff where it is positioned on the calf and not where the Doppler probe is positioned at the ankle. In patients with crural disease the recorded ABPI may, therefore, vary significantly depending on whether the cuff is placed proximally over the bulk of the calf muscles, as may be the case in a patient with a circumferential ulcer, or distally, just proximal to the malleoli. This may lead to inappropriate clinical decision making; for example, regarding the use of compression in patients with mixed arterial and venous ulcers. It is also worth pointing out that as the ABPI is based upon the highest of the three ankle pressures it ‘puts the best light’ on the perfusion of the foot. For example, a patient with occlusion of PT and peroneal arteries but a relatively normal anterior tibial artery may appear to have an adequate ABPI and yet have little or no perfusion of
the posterior part of the foot and heel. Specifically, patients with diabetes often have segmental crural and pedal arterial disease resulting in very different levels of perfusion in different parts of the foot. Furthermore, ABPI may be falsely elevated because the arterial disease is distal to the cuff and even the Doppler probe.

The importance of blood pressure cuff size

Cuff size has an important effect on the indirect measurement of blood pressure. Specifically, larger cuffs should be used in the obese so as to avoid over-estimating systolic brachial blood pressure and thus under-estimating ABPI. Provided that the cuff is placed as distally as possible on the calf, this may be less of an issue in the measurement of ankle pressures as even the morbidly obese tend not to have fat ankles. However, it is highly relevant in patients with oedema (lymphoedema and venous oedema) and in children. Most clinicians tend to opt for a cuff that is as wide as two-thirds to three-quarters of the upper arm length and this has been shown to significantly under-estimate both the systolic and diastolic blood pressure when compared to measurements obtained directly via a radial artery transducer. Early work in this field by Geddes et al. has shown that using a cuff with a bladder width of 40% of the upper arm circumference gives a more accurate estimate of the systolic blood pressure. In neonates and infants the ABPI is physiologically lower than in older children and adult values are reached during the second year of life. The above facts must be kept in mind both when assessing young infants for instance following iatrogenic catheterisation injuries and when such patients are followed up into later childhood.

Reproducibility of ABPI

The significance of ABPI reproducibility, or lack of it, depends upon the context in which it is being used. For example, as inter-observer variability is considerably less than the biological variability between normal subjects and those affected by different stages of disease, lack of reproducibility is generally not an issue in epidemiological studies. This is not necessarily the case when ABPI is being used to define the natural history of PAD or treatment outcomes in individual patients being studied longitudinally. In these circumstances, multiple measurements both at baseline and during follow-up are recommended.

For example, one hospital-based study found that 30% of the ABPIs performed by junior doctors were incorrect when compared to the values obtained by vascular technicians; this figure improved to 15% after formal training. This may partially relate to the fact that within the vascular laboratory, the brachial systolic pressure is more likely to be estimated using a Doppler probe, whereas house officers tend to rely on the Korotkoff method which tends to yield lower values for the systolic pressure. Significant variability between measurements depending upon degree of experience has also been shown in other more recent studies. The impact of both inter-observer and intra-observer error on reproducibility has been quantified in a Dutch general practice based study. When the ABPI was used to follow up patients with PAD, the difference between two sequential ratios had to be at least 19% in order to exclude an intra-observer error. This reaffirms the generally accepted notion that the ABPI must change by at least 0.15 before this can be considered to be clinically significant.

Implications of variability and methodology for practice

Taken together the various factors influencing the measurement and reproducibility of ABPI can have a profound effect upon the data being collected and recorded for clinical practice, as well as scientific reporting. While there may not be ‘one best way’ of measuring ABPI, it is important that individual clinical units and research teams:

- Formally train their observers (whether they be doctors, nurses, or technologists) using a standardised methodology
- Record their results in a standardized manner for day to day clinical practice
- State their preferred method explicitly when reporting in the literature
- Audit their results

to ensure that the apparent simplicity of the ABPI does not beguile them in to making inappropriate clinical decisions, drawing erroneous conclusions with regard to the outcome from vascular interventions, or publishing spurious data.

Association between ABPI and clinical stage of PAD

In the aggregate, ABPI correlates well with the angiographic severity of lower limb arterial occlusive disease and the resulting functional impairment both objectively and as perceived by the patient. In healthy individuals the ABPI is usually 1.0–1.2 in the supine position. The peak systolic ankle pressure
tends to be higher at the ankle than at the arm because of pressure augmentation by the muscular peripheral arteries as well as the summation of reflected pressure waves. As stressed below, ankle pressures must be measured with the patient supine, not least because of the veno-arteriolar reflex. In healthy individuals, standing up activates this reflex which leads to arteriolar vasoconstriction and an overall restriction in arterial flow. This mechanism is significantly impaired in patients with SCLI and CLI, chronic venous disease and in diabetics with autonomic neuropathy. Patients with intermittent claudication (IC) usually have an ABPI of 0.5–0.8 while those with sub-critical (SCLI) and critical limb ischaemia (CLI) usually have ABPI of <0.5 and <0.3, respectively. The association between disease severity and the ABPI applies not only to hospital patients, but also to community-based studies. However, there can be considerable overlap in ABPI between these major clinical groups and, as with many clinical measurements, it is the trend over time, rather than the absolute index, that is most clinically important on an individual patient basis.

A significant number of patients presenting to vascular clinics can be difficult to assess either because they have:

- PAD with atypical symptoms or
- other pathology (e.g. spinal stenosis) to explain their leg symptoms in the presence of asymptomatic PAD

ABPI can be particularly helpful in such circumstances by allowing the clinician to correlate the patient’s subjective symptoms with an objective measure of arterial disease severity.

Value of ABPI in critical limb ischaemia

In patients with potentially limb-threatening ischaemia there appears to be a better relationship between symptoms, limb viability, treatment opportunities and outcome and absolute pressures than there is with ABPI. A review of the results of 20 publications reporting 6118 patients, demonstrated that patients with SCLI (defined as rest pain and/or an absolute ankle pressure of more than 40 mmHg) were at significantly lower risk of limb loss than patients with CLI (tissue loss or an absolute ankle pressure of less than 40 mmHg). However, while easy to recognise clinically, CLI has proved surprisingly difficult to define for purposes of scientific reporting. The European Consensus Document definition of CLI, which remains controversial and is by no means strictly observed in the literature, includes an absolute ankle pressure <50 mmHg or toe pressure less than 30 mmHg. The latter is also included in the Trans-Atlantic Inter-Society Consensus recommendations and is recommended for patients with calcified crural arteries. However, toe pressures have a low positive and a high negative predictive value when used to detect CLI and are, therefore, more useful in the exclusion of CLI than its confirmation. See section on the ‘pole test’ below.

ABPI and chronic venous ulceration

In patients with chronic venous ulceration (CVU), it is currently recommended that the ABPI should be >0.8 if compression bandaging is to be applied safely in the community. This is a major issue with district and practice nurses increasingly referring patients with CVU to vascular clinics for measurement of ankle pressures and ABPI; or for confirmation of ankle pressures and ABPI measured in primary care. ABPI in patients with CVU must be interpreted with caution for a number of reasons:

- In patients with low central systolic pressure, or in whom this is underestimated because of PAD in the upper extremity, the ABPI under-represents the degree of arterial disease in the lower extremity. Absolute systolic ankle pressures may be more meaningful in this situation.
- The ABPI is representative of the highest systolic pressure measured at the ankle. In patients where individual calf vessels are heavily diseased or occluded while a single tibial vessel is relatively preserved, the ABPI would fail to indicate the fact that part of the calf may be significantly under-perfused and, therefore, more susceptible to pressure damage. A difference of >10 mmHg between systolic pressure readings taken from different pedal vessels should alert the clinician to this possibility.
- The ABPI may be inaccurate if the cuff is placed proximally over the bulk of the calf muscles, as may be the case in a patient with a circumferential ulcer. This may lead to inappropriate use of compression therapy in patients with mixed arterio-venous ulcers.

Non-compressibility of vessels

Many patients with PAD, particularly those with diabetes and end-stage renal failure, have calcified
and incompressible crural vessels leading to spuriously elevated ABPI. The degree to which the ABPI can be raised is variable and difficult to quantify, once again emphasising the need to interpret the index in the light of clinical findings and other investigations. If there is doubt about the validity of the ABPI then two alternatives are to use toe pressures to calculate the TBI or to utilize the ‘pole test’. In the latter, with the patient supine, the foot is elevated against a calibrated pole. The height above the heart at which the ankle or toe Doppler signal disappears approximates the perfusion pressure at the site of the probe in centimetres of water from which a value in mmHg can be derived (1.36 cm of water equals 1 mmHg). In practice, if the Doppler signals are still present at the ankle with the hip elevated to 90° then this effectively excludes CLI (i.e. the perfusion pressure exceeds 50 mmHg) and is a useful semi-quantitative screening test.

**Effect of exercise on the ABPI**

Exercise results in local vasodilation and this rapidly overcomes the vasoconstrictive response to postural changes. In normal individuals this results in a significant increase in the total blood flow and the blood pressure is maintained. This increased flow would amplify the resistance across both a significant stenosis in an arterial trunk feeding the intramuscular arterioles and across a collateral system bypassing an occluded artery. Consequently, the pressure in a vascular bed distal to such a lesion would fall and remains low while exercise continues and until the local vasodilator mediators are washed out of the limb. This explains how the presence of mild to moderate PAD causes the ABPI to drop after exercise and why the length of the recovery period is proportional to the severity of the disease. To obtain a true resting ABPI, it is necessary for the patient to rest supine for 10–20 min after walking to the clinic.

**ABPI and the outcome of vascular surgery**

It is widely accepted that successful aorto-iliac and infra-inguinal arterial reconstruction is accompanied by a significant increase in ABPI. The magnitude as well as time scale over which this increase occurs depends very much upon the extent of the underlying disease as well as the type and extent of intervention. For instance, following an aorto-bifemoral bypass for isolated iliac vessel disease, one would expect a near instantaneous rise in the ABPI to normal. Following a femoro-popliteal bypass the rise in ABPI may take up to 4 h to complete, and after profundaplasty this may take over 24 h. Indeed, there is some evidence that ABPI may continue to rise for several months following successful surgery.

Serial ABPI measurement alone has been shown to have a very low positive predictive value for vein graft occlusion following infra-inguinal bypass. It is also insensitive in predicting impending graft failure. In one study only 38% of limbs demonstrated an ABPI drop of 0.15 or more on graft occlusion. Long-term outcome for aorto-iliac and infra-inguinal bypass depends primarily upon progression of PAD, and the ABPI appears to be relatively insensitive in detecting disease progression when compared to angiography or duplex scanning. Despite these points, a significant drop in the ABPI (>0.15) following surgery should, if clinically indicated, prompt further graft assessment. This is supported by data from the recent vein graft surveillance trial (VGST). This European Multicentre randomised control trial has shown that routine duplex graft surveillance is unjustified in terms of cost and clinical outcome when compared to standard clinical follow-up and ABPI measurement followed by selective duplex scanning. The importance of ensuring standardisation and applying local quality control to ABPI measurement cannot be overemphasized in this respect.

**ABPI and the outcome of endovascular intervention**

ABPI appears to increase more slowly after successful angioplasty and may continue to increase for at least a month after the procedure. This has been shown to correlate with changes in flow rate and is independent of any calf swelling related to reperfusion. This suggests that relying on a single measurement of ABPI taken soon after endovascular intervention (or surgical bypass for that matter) may underestimate the success of the procedure as well as the deterioration associated with post-procedural failure or disease progression. The ABPI also appears to be relatively insensitive as a tool for longitudinal studies of patients following angioplasty. In one prospective study of patients following successful superficial femoral artery angioplasty, deterioration in the ABPI by 0.15 was highly specific for reocclusion or stenosis but pressure measurements showed poor sensitivity in detecting re-stenosis or occlusion.

**ABPI and the outcome of medical treatment**

While intermittent claudication can often be attributed to specific abnormalities in the arterial tree, the
pathophysiology is more complex than simply altered large vessel haemodynamics. Various local metabolic effects distal to a stenosis or occlusion may all play a role in the progression of the disease independently from changes in the ABPI: changes in the production of local vasodilating agents such as adenosine, and systemic haematologic effects such as impaired fibrinolysis and increased blood viscosity may contribute. Conversely, medical treatment such as cilostazol and pentoxifylline may improve symptoms without necessarily affecting the ABPI. Cilostazol is a selective phosphodiesterase III inhibitor which has beneficial effects on platelet function and cholesterol metabolism, causes vasodilation and inhibits smooth muscle proliferation. Cilostazol has been shown to increase resting ABPI and improve the post-exercise recovery time but this may not fully represent its therapeutic effect. For this reason the large trials of cilostazol used initial claudication distance and absolute claudication distance as their primary endpoints.

Current evidence would suggest that supervised exercise programmes for intermittent claudication are both safe and effective. Exercise is postulated to increase walking distance by enhancing the oxidative capacity of skeletal muscle cells, increasing the reliance on non-ischaemic muscles and promoting the development of collateral vessels. These effects would be under-represented by changes in the ABPI and, therefore, endpoints such as changes in quality of life scores and treadmill walking distances are of more relevance for instance when comparing different approaches to exercise therapy.

It is clear, therefore, that the ABPI, being a measure of changes in the pressure gradient within the axial arteries, cannot be solely and directly used as a marker for proof of concept or mechanism for new treatment modalities that act at the molecular level. This point may become even more relevant in future as newer treatment modalities emerge aimed at promoting angiogenesis.

**ABPI and asymptomatic PAD**

The relationship between lower limb symptoms, ABPI and cardiovascular risk is an important one. As described above, a normal ABPI at rest does not exclude the presence of haemodynamically significant lower limb atherosclerosis and if there is strong suspicion that the patient’s symptoms are vascular in aetiology, then ABPI should be repeated after exercise testing. Similarly, the absence of symptoms does not exclude the presence of significant lower limb PAD. A lack of lower limb symptoms in the presence of a low resting or post-exercise ABPI (typically claudication) may simply be due to the patient’s choice not to walk, or due to other exercise limiting pathology (e.g. osteoarthritis, angina, breathlessness) or diabetic neuropathy.

It is now quite clear from several large longitudinal studies (Table 1) that a low ABPI, usually taken as < 0.8 or < 0.9, is associated with a marked increase in cardiovascular events, recurrent events and mortality, whether lower limb symptoms are present or not.

Furthermore, the Edinburgh Artery Study has shown that even a near-normal ABPI (0.91–1.0) is associated with reduced 5 year survival.

The data shown in Table 1 suggests that ABPI can be used in primary care to screen for early asymptomatic and symptomatic PAD. The institution of BMT in such individuals would lead to a significant reduction in disease progression, cardiovascular events and mortality, and health care spending on more advanced vascular disease in secondary care. The results of further ongoing studies (e.g. the POPADAD study) in this respect where the ABPI is a primary endpoint are eagerly awaited. ABPI could also be used selectively to identify those individuals engaged in certain critical occupations (e.g. heavy goods vehicle drivers, airline pilots) who are at particularly high risk of sudden and incapacitating cardiovascular events such as myocardial infarction and stroke.

**Discussion**

Although it is used day-in day-out across the world in primary and secondary care, it is clear that there is more to the ABPI than first meets the eye and that its apparent simplicity may beguile the unwary. In particular, care needs to be taken with methodology and training, reproducibility, interpretation, clinical recording and scientific reporting. However, when used properly, the ABPI remains an invaluable tool in assessment of vascular patients, especially those with atypical presentation, and in determining the success or otherwise of surgical, endovascular and medical interventions. But perhaps the most exciting area is the extended utility of the ABPI as a basis for community and occupational health screening and the opportunities that would herald for the early detection and evidence-based, clinically and cost-effective, treatment of vascular disease.
Table 1. Studies examining the relationship between ABPI and cardiovascular outcome

<table>
<thead>
<tr>
<th>Study</th>
<th>N (=)</th>
<th>Cohort</th>
<th>Study method</th>
<th>Follow-up</th>
<th>Major findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>McKenna M et al.</td>
<td>744</td>
<td>Patients investigated non-invasively for PAD</td>
<td>Retrospective</td>
<td>–</td>
<td>RR for death 2.36 if ABPI &lt;0.85. RR for MI 4.49 if ABPI &lt;0.4</td>
</tr>
<tr>
<td>Vogt MT et al.</td>
<td>1930</td>
<td>Patients aged &gt;50 years referred for arterial assessment</td>
<td>Prospective cohort</td>
<td>13 years</td>
<td>ABPI &lt;0.9, strong predictor for all-cause mortality (RR for men =1.8, RR for women =1.5) and CHD mortality (RR for men =2.0, RR for women =2.1)</td>
</tr>
<tr>
<td>Fishbane S et al.</td>
<td>132</td>
<td>Haemodialysis patients</td>
<td>Prospective observational</td>
<td>1 year</td>
<td>ABPI &lt;0.9, RR for cardiovascular mortality =7.5</td>
</tr>
<tr>
<td>Leng GC et al.</td>
<td>1592</td>
<td>Age stratified population sample aged 55–74 years</td>
<td>Prospective cohort</td>
<td>5 years</td>
<td>ABPI &lt;0.9, RR for non-fatal MI =1.38, RR for CVA =1.98, RR for cardiovascular mortality =1.85 and RR for all-cause mortality =1.58</td>
</tr>
<tr>
<td>Newman AB et al.</td>
<td>5888</td>
<td>Population sample aged &gt;65 years</td>
<td>Population observational</td>
<td>10 years</td>
<td>ABPI&lt;0.9, RR for all-cause mortality =1.62; incidence of cardiovascular events increase with each ABPI decrement of 0.1</td>
</tr>
<tr>
<td>Tsi AW et al.</td>
<td>14839</td>
<td>Population sample aged 45–64 years</td>
<td>Prospective observational</td>
<td>7 years</td>
<td>Overall inverse trend between ABPI and incidence of CVA, RR for CVA =1.93 if ABPI &lt;0.8</td>
</tr>
<tr>
<td>Powell J et al.</td>
<td>2305</td>
<td>Patients with abdominal aortic aneurysm</td>
<td>Prospective observational</td>
<td>5.7 years</td>
<td>RR for mortality (adjusted) increases by 1.17 per 0.2 decrement in ABPI</td>
</tr>
<tr>
<td>Jonsson B et al.</td>
<td>353</td>
<td>Population sample aged 50–89 years</td>
<td>Prospective cohort</td>
<td>10 years</td>
<td>RR for all-cause mortality =2.1 if ABPI 0.51–0.8, and 3.4 if ABPI &lt;0.5</td>
</tr>
<tr>
<td>Murabito JM et al.</td>
<td>674</td>
<td>Elderly healthy population: mean age 80 years</td>
<td>Prospective observational study</td>
<td>4 years</td>
<td>Significant increase in risk of CVA or TIA if ABPI &lt;0.9</td>
</tr>
</tbody>
</table>

RR, relative risk; CHD, coronary heart disease; MI, myocardial infarction; CVA, stroke; TIA, transient ischaemic attack.

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