

Ankle Brachial Pressure Index (ABPI): An update for practitioners

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Abstract: Peripheral vascular disease affects some 12%–14% of the general population, and the majority of people with the disease are asymptomatic. The Ankle Brachial Pressure Index (ABPI) test is widely used by a diverse range of practitioners (in the community and hospital setting) in order to screen asymptomatic patients, diagnose patients with clinical symptoms, and to monitor patients who have had radiological or surgical intervention. This paper explains the theoretical basis of the ABPI test, as well as the relevance of the common modifications of the test. It explores the background to the quoted normal ranges for the ABPI test. It reviews the large body of literature that has developed on the association between ABPI and cardiovascular risk, as well as ABPI as a predictor for cardiovascular morbidity and mortality, highlighting the evidence that can inform practice. The review looks critically at the limitations of the ABPI test, providing practitioners with an evidence-based update on the importance and challenges of standardizing ABPI methodology. This paper highlights the influence of the key technical aspects of the ABPI test that all practitioners need to consider in order to be able to make more reliable and informed management decisions based on ABPI findings.

Keywords: ankle, brachial, pressure, index, ABPI, update

Introduction

The Ankle Brachial Pressure Index (ABPI) test is widely used in the setting of peripheral vascular disease by a diverse range of practitioners. The advantages and limitations of the ABPI test are not widely understood, and this review paper aims to inform and update practice by presenting the evidence about the performance of the ABPI test, allowing practitioners to make an evidence-based interpretation of ABPI findings.

Diagnosing peripheral vascular disease: the ABPI test

Peripheral arterial disease affects some 12% to 14% of the general population. The prevalence of peripheral arterial disease is age-dependent, reaching 10% in people aged over 60 years,^{1,2} and some 20% aged over 75 years.^{3–5} Peripheral vascular disease is estimated to affect some 27 million people in Europe and North America.⁶

Unfortunately, the majority of patients are asymptomatic and undiagnosed; although intermittent claudication is the primary, and most often only, symptom, over 16 million of the patients with peripheral arterial disease are asymptomatic. Not only this, but one third of patients with symptoms do not report them to their doctor. As a result of this,

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a high-risk group of patients are underdiagnosed and receive no (or suboptimal) treatment(s) or intervention(s).^{7,8}

The Fontaine classification provides a framework for clinical staging (from I to IV) of peripheral vascular disease.⁹ However, given the large number of asymptomatic peripheral arteriopathies, relying on clinical history has a very low sensitivity for determining the presence of peripheral arterial disease. Fewer than 10% of patients with peripheral arterial disease will provide symptoms consistent with the Rose criteria for intermittent claudication.^{6,10} Criqui and colleagues¹¹ claim that a history of intermittent claudication underestimates the presence of peripheral arterial disease by a factor of two to five. Similarly, Marinelli and colleagues¹² claim that relying on an absent pedal pulse also underestimates the prevalence of disease. Hirsch and colleagues¹³ claim that clinicians who screen patients for peripheral arterial disease on the basis of finding a complaint of intermittent claudication will miss up to 90% of high-risk patients with the disease.

Therefore, given the limitations of relying on the clinical history or physical examination, an additional noninvasive test, the ABPI, has been widely adopted for confirmation of a clinical diagnosis of peripheral arterial disease and its quantification. The ABPI is a measure of the blood pressure in the arteries supplying legs relative to central, aortic pressure (approximated by measuring the blood pressure in the arm).

The Society for Vascular Technology¹⁴ has produced comprehensive laboratory guidelines for the measurement of ABPI, aiming to control the methodology under clinical conditions. Similarly, Vowden and colleagues,¹⁵ Stubbing and colleagues,¹⁶ Hiatt and colleagues,³ and McDermott and colleagues¹⁷ try to provide comprehensive methodology guidance for the measurement of ABPI.

In essence, ABPI is calculated by dividing the systolic blood pressure measured in the arterial conduits at the level of the ankle by the systolic blood pressure measured in the brachial artery as seen in the following equation:

$$\text{ABPI} = \frac{\text{Ankle systolic blood pressure}}{\text{Brachial systolic pressure}}$$

The ABPI is used to assess patients for peripheral arterial disease as a fall in blood pressure in an artery at the ankle relative to the central blood pressure would suggest a stenosis in the arterial conduits somewhere in between the aorta and the ankle.

Theoretical basis of the ABPI test

The idea of comparing peripheral systolic pressure with central systolic pressure was first introduced by Winsor¹⁸ using

a mercury and rubber strain gauge method. The theoretical basis of ABPI is supported by obtaining ABPI ratios against findings in peripheral angiography,^{19–24} as well as from Doppler ultrasound as gold standard.²⁵

ABPI is claimed to be a simple, noninvasive, and reproducible test.^{26–28} When used in university settings, ABPI measurements have been found to have good interobserver reliability between experienced physicians, with a reported κ -statistic of 0.77 to 1.0.²⁹ Estimates of intraobserver variability range from 7.3%–12% for ABPI in the hospital^{26,27,30,32} as well as in the community setting.^{31,32} Holland-Letz and colleagues³³ point out that the previous studies of reliability had only small sample sizes of patients with disease; therefore they performed a large study in “normal” (asymptomatic) elderly patients (only two with low ABPI) and, by contrast, they found no differences in mean ABPI recordings between medical and nonmedical primary care practitioners; their estimate of intra- and interobserver error was 8% and 9%, respectively.

Similarly, Endres and colleagues³⁴ found little interobserver variability (nearly zero) between different practitioner groups in a large study of unselected (asymptomatic) elderly patients. Well-controlled, repeat measurements of ABPI by expert practitioners are accurate enough to be used as a clinical gatekeeper in decision-making.^{27,30}

The American Diabetes Association consensus paper³⁵ quotes the normal range of ABPI as being 0.91–1.3. This same consensus paper claims that mild disease falls into the range of 0.7–0.9, whilst moderate ischemic disease is quoted for ratios of 0.41–0.69. Patients with mild or moderate disease will be claudicants. Ratios of less than or equal to 0.4 are quoted in severe disease, presenting clinically as critical ischemia.

Table 1 summarizes the Society for Vascular Technology’s¹⁴ interpretation of resting ABPI measurements.

Understanding normal ranges for the ABPI test

Neither the Society for Vascular Technology¹⁴ nor the consensus paper³⁵ explain how the limits of this normal ABPI range are derived. Male and colleagues³⁶ claim that the majority of authors quoting ranges for ABPI refer back to original data from Yao and colleagues,²² Cornwall and colleagues,³⁷ and Sumner.³⁸ They also assert that there is a lack of evidence-based literature supporting the traditional interpretations of ABPI.

Male and colleagues³⁶ point out that ABPI is age- and blood pressure-dependent (and these two factors are themselves linked). The accepted methodology of ABPI does

Table I Interpretation of ABPI

Resting ABPI	Severity of disease
>1.4	Calcification may be present
>1.0	Probably no arterial disease
0.81–1.00	No significant arterial disease, or mild/insignificant disease
0.5–0.80	Moderate disease
<0.5	Severe disease
<0.3	Critical ischemia

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Abbreviation: ABPI, Ankle Brachial Pressure Index.

not make any corrections for physiological variation over time in baseline brachial systolic blood pressure, or other confounders for blood pressure measurement like respiratory phase necessitating a range to be used, rather than a point figure. It is easy to see that ABPI values over the upper limit of 1.3 may be associated with arterial incompressibility at the ankle (secondary to arterial wall medial calcification), a condition very commonly found in diabetics, renal patients, as well as a physiological function of age.^{14,35,39,40}

It is less easy to explain why the lower limit of the normal ABPI ratio should ever be less than 1.0. In the normal individual, resting supine for at least ten minutes, the systolic blood pressure at the ankle will be slightly higher than the systolic blood pressure at the brachial artery. This is mostly due to the differences in the magnitude of pressure pulse reflections from the vascular beds immediately downstream of the ankle and elbow (ie, the vascular bed of the foot and that of the forearm and hand, respectively), but also partially because of the differences in distance between the point of measurement at the ankle and the elbow from the peripheral bed. The ankle is immediately proximal to a pressure antinode (the peripheral bed represented by the foot), whereas the elbow is one segment upstream from the analogous peripheral bed of the hand. Therefore one would assume from this that a normal ABPI ratio should be at least 1.0 or slightly more. And, in fact, Male and colleagues³⁶ found that the mean normal ABPI in a small sample of young, healthy patients aged twenty to forty is 1.14, with a range of 1.05 to 1.25.

Ouriel and Zarins²³ found that ABPI in a group of 34 “normal” controls (asymptomatic, nondiabetic volunteers under 30 who had never smoked, and with triphasic Doppler waveforms) came to be 1.09 (± 0.02 for 95% confidence windows). Carser⁴¹ claims that the normal corrected ABPI range is 1.02 to 1.78 (admittedly, Carser was studying

ABPI ranges in the context of background hypertension). Of 100 studies of ABPI methodology included in their review, Klein and Hage⁴² found that the most frequently quoted normal value for ABPI was 1.0. Hiatt and colleagues³ found the mean ABPI was 1.13 ± 0.08 (right leg) and 1.11 ± 0.09 (left leg); later, Hiatt and colleagues⁴³ derived a set of normal ranges for ABPI sub-classified according to sex, leg, and ankle vessel; all their data being drawn from a nondiabetic population screened to have low risk for peripheral vascular disease (men, right: dorsalis pedis 1.15 ± 0.11 ; posterior tibial 1.19 ± 0.12 ; men, left: dorsalis pedis 1.14 ± 0.10 ; posterior tibial 1.18 ± 0.12 ; women, right: dorsalis pedis 1.09 ± 0.09 ; posterior tibial 1.13 ± 0.08 ; women, left: dorsalis pedis 1.06 ± 0.10 ; posterior tibial 1.12 ± 0.08).

So why do the American Diabetes Association³⁵ quote the lower limit of ABPI as 0.91? A number of groups, in fact, quote a range of abnormal lower cut-off points of less than 1.0, from 0.80–0.98.^{2,3,19,44} An answer is partially deduced by looking again at ABPI methodology.¹⁴ Although all the most important aspects have been standardized, the respiratory phase is not controlled during measurement of ABPI. Therefore it is perfectly possible to measure the systolic pressure at the ankle at peak expiration and coincidentally measure the systolic pressure at the brachial at peak inspiration. Respiration is well known to modulate blood pressure in mammals, with limited data from normal humans in one study suggesting a 3% fall in systolic pressure during inspiration.⁴⁵

As ABPI ranges are most likely the result of analyses of data from observational studies, the lower limit of 0.91 is therefore possibly a reflection of the inherent limitation of a noninvasive method of measuring blood pressure (using an occlusive cuff) without controlling physiological variation from, for example, respiration. Beat-to-beat physiological variation in systolic blood pressure can only be controlled by synchronous measurements at the arm and ankle, which is cumbersome and not prescribed by any of the authoritative guides on ABPI measurement. Additionally, technical factors such as over-rapid deflation of the ankle cuff can also give artifactually low systolic pressure readings at the ankle.

Association between the ABPI ratio and cardiovascular morbidity and mortality

Having a low ABPI ratio is an independent risk factor for cardiovascular disease including fatal and nonfatal complications.^{46,47} In addition, the lower the ABPI value, the higher the risk of all-cause and cardiac mortality in patients with peripheral vascular disease.⁴⁸

The predictive value of ABPI in cardiovascular morbidity and mortality is similar to that of traditional Framingham risk factors.⁴⁹ McKenna and colleagues⁵⁰ evaluated a low ABPI as an independent risk factor for mortality. Doobay and Anand⁵¹ found that low ABPI was highly specific for predicting future cardiovascular events. A recent meta-analysis found that ABPI is an independent risk factor for cardiovascular mortality alongside the traditional Framingham risk factors, and having an ABPI of <0.9 resulted in a doubling of cardiovascular mortality and morbidity across all of the Framingham risk categories; furthermore, combining ABPI with traditional Framingham risk assessment would re-classify 19% of men and 36% of women into more appropriate treatment categories.⁵²

An ABPI ratio of less than 0.9 has been associated with up to a three-fold relative increase in all-cause and cardiac mortality, in both men and women.^{50,53–55}

Similarly, having an elevated ABPI > 1.40 (even if the observation was nondiagnostic because of arterial incompressibility secondary to calcification) is a predictor for an increase in all-cause mortality as well as cardiovascular mortality.⁵⁶

The association between peripheral arterial disease and increased mortality is a result of the fact that the underlying pathological process, atherosclerosis, is a systemic one. Atherosclerosis, if present in the periphery, is also likely in other parts of the arterial tree. Ness and Aronow⁵⁷ found that 68% of patients with peripheral arterial disease also had coronary artery disease in an elderly population. Patients with peripheral arterial disease have more than a sixfold increase in the risk of death from coronary artery disease when compared to patients without the disease.⁵³ Even if patients with peripheral arterial disease are asymptomatic, they have an increased risk of future cardiac and cerebrovascular events, as well as being six times more likely to die within ten years when compared to healthy individuals.⁵⁸ A number of groups support the use of ABPI not only as a diagnostic tool, but also as a risk assessment tool in the setting of peripheral vascular disease.^{59–61}

Modifications of the ABPI technique

The literature is replete with validations of modifications or refinements of traditional ABPI methodology or its calculation.^{62,63}

Clinically, the most common addition to the resting ABPI is that of postexercise measurements. Carter⁶⁴ claimed that exercise testing would unmask patients with mild peripheral vascular disease (ie, patients with degrees

of diameter reduction in the arteries of less than 50%). Some researchers claim that resting ABPI correlates well with functional walking capacity in patients with peripheral arterial disease.^{65–67} Szuba and colleagues⁶⁸ outright reject this position, claiming that only postexercise ABPI, and not ABPI itself (or other resting hemodynamic measures) has any predictive ability for or correlation with walking distance in peripheral arterial disease. The value of exercise testing (over and above resting ABPIs) was questioned by Allen and colleagues²⁵ and Ouriel and colleagues.⁴⁴

Allen and colleagues²⁵ compared resting and postexercise ABPI to color Duplex ultrasound (as gold standard). When postexercise ABPI measurements were used in addition to resting ABPI, the correlation with color Duplex ultrasound for detecting significant arterial disease increased by only 2% (from 83% for resting ABPI alone to 85% in the combined resting and postexercise ABPI method). However, the clinical utility of any exercise test is limited in bilateral disease by the most symptomatic limb in the patient; and Stein and colleagues⁶⁹ point out that exercise testing is not even an option in patients with poor mobility due to comorbidity (eg, cardiac disease, respiratory disease, or disability). Hiatt and colleagues³ estimated that at least 15% of patients were unsuitable for exercise testing or hyperemia induction (simulating exercising with an occlusive cuff).

Limitations of ABPI

In their original paper on ankle systolic pressures, Yao and colleagues²² claimed 98% overall accuracy (sensitivity 97%, specificity 100%) against angiography using an ABPI threshold of 1.0 as being normal. This pioneering work, preceded by that of Carter,¹⁹ assumed an ABPI threshold of 1.0 using normal (clinically asymptomatic) volunteers as controls, rather than angiographically-proven normals.

Other papers from the same authors^{20,21} as well as that from Ouriel and Zarins²³ make similar claims for accuracy, but these studies also rely on the same the type of control group (normal volunteers rather than angiographically-proven normals).

Later, Allen and colleagues,²⁵ using color Duplex ultrasound as the gold standard in nondiabetic patients with peripheral vascular disease, came up with an overall figure of 83% accuracy using an ABPI threshold of 0.8. This study used patients who could tolerate both a resting ABPI and an exercise ABPI. The data did not include any

normal volunteers. Agreement with Duplex ultrasound was shown to drop in patients with higher ABPI values, down to 76% in patients with ABPIs of ≥ 0.9 . At the same time, Allen and colleagues²⁵ found 100% agreement with Duplex ultrasound in patients with an ABPI < 0.6 . Allen and colleagues²⁵ concluded that “ABPI is biased toward the detection of more severe disease and is more consistent with colour Duplex ultrasound when the most symptomatic limbs are compared”. Therefore in the clinical setting, where it is possible to do an ABPI on patients with peripheral vascular disease, up to 17% of these ABPI tests might be unreliable.

Belch and colleagues⁷ point out that it is also possible to have a high-grade aorto-iliac stenosis or occlusion clinically masked by a rich collateral network; in these situations, using ABPI would also fail to unmask the underlying lesion. In fact, Belch and colleagues⁷ highlight the wider problem that nearly 60% of patients with peripheral arterial disease are asymptomatic, and if collateral vessels are the reason, they are likely to be unreliable candidates for identification by the ABPI test.

Stein and colleagues⁶⁹ question the utility of the ABPI test in patients with low-grade stenosis after reporting that nearly half of all patients with symptoms of peripheral arterial disease had a normal resting ABPI. Although Stein and colleagues⁶⁹ concede that some of these patients will have intermittent claudication or ulceration from another cause, they also point out that the original studies on ABPI were limited to patients with advanced disease. Ouriel and Zarins²³ did not find a significant difference between ABPI for asymptomatic patients and patients with angiographically-proven nonocclusive disease.

At the other end of the clinical spectrum, in certain groups of patients at high risk of heavy arterial calcification, ABPI becomes impractical and nondiagnostic. For example, in elderly, diabetic and renal patients (and also other groups with rarer systemic diseases, like systemic sclerosis, or rheumatic diseases) calcification of the peripheral arteries can make the arteries incompressible, and therefore the ABPI test (relying on measurement of systolic pressure with an occlusive cuff) becomes nondiagnostic or inaccurate due to artefactually-raised occlusion pressures secondary to medial calcification in the arterial wall.⁷⁰

This is all the more problematic since, in some of these subgroups of patients, peripheral arterial disease is common. Estimates of the prevalence of peripheral arterial disease in diabetic patients vary from 33% to 50%.^{3,71}

In diabetic patients, it is not uncommon to use a pressure cuff at 300 mmHg and still fail to occlude an artery at the

ankle due to calcification and hardening of the arterial wall. Raines and colleagues⁷² estimated that occlusive ankle pressures could not be measured in 5%–10% of diabetic patients. Therefore, for a group of patients with long-standing diabetes, or renal failure, presenting with peripheral vascular symptoms, and in whom ABPI is nondiagnostic, there is a need for a noninvasive, clinical alternative to ABPI for assessing the presence of significant arterial disease.

Standardizing ABPI methodology

The exact methodology of ABPI is far from standardized on a large number of technical aspects, starting with the choice of artery at the ankle. Because there are three arteries at the ankle, ABPI can be calculated in a number of ways and combinations eg, based on using the highest pressure,^{44,73} the lowest pressure,⁷⁴ the best Doppler single,²⁴ or a combination of ankle vessel pressures.⁴³ The Society for Vascular Technology allows practitioner to choose between two prescribed methods for the ankle systolic.¹⁴ Similarly, it is possible to measure pressure in either arm, and there is no consensus position on how central pressure should be approximated from the arm pressure. Both Carter²¹ and Sumner³⁸ pointed out that atherosclerosis is systemic and can therefore affect the pressure in the upper limbs directly or indirectly via disease in the aortic arch. Grim⁷⁵ pointed out that, the length of the rest period, “patient-white coat” phenomena, cuff placement, cuff size, and the speed of inflation/deflation can affect blood pressure recordings – all of which are relevant to the context of ABPI. In patients who cannot lie supine, a further inaccuracy is added by the hydrostatic pressure difference between the arm and leg.⁷⁶ The definition of “resting” (as part of the resting ABPI) is also important; patients with severe multilevel arterial disease need a longer period of “rest” prior to taking systolic pressure measurements and this is difficult to know beforehand.⁷⁷ Doppler probe selection (with respect to frequency, and level of quality assurance maintenance) can also be a source of inconsistency between practitioners.⁷⁶

Apart from the inconvenience and difficulty of controlling a large number of environmental factors that can affect vasomotor tone (eg, temperature) and therefore blood pressure readings, there is also an intrinsic, physiological variability found in blood pressure measurements (and their ratios, like ABPI) so that some authors have recommended multiple measurements to allow consistent longitudinal follow-up, and to dismiss changes of less than 0.15 in serial readings of the ABPI ratio.^{78,79}

Interestingly, Holland-Letz and colleagues³³ pointed out that the validity of noninvasive blood pressure measurement (by whatever method) in the context of ABPI has never been assessed (eg, compared in real time against the invasive blood pressure reading in a controlled setting).

Furthermore, when measuring blood pressure using an occlusive cuff, there is a reduction in blood flow through any proximal lesion at the moment of systolic pressure measurement, thereby giving an underestimate of true resting pressure drop across the lesion. Hence the occlusive cuff method on which ABPI relies is liable to underestimate the presence of peripheral arterial disease. This underestimation of true pressure drop is most marked when the lesion is immediately proximal to the ankle cuff.^{80–82}

As of yet, there is no consensus agreement on the methodology for ABPI, and no vessel-specific criteria for the diagnosis of peripheral vascular disease. Carser⁴¹ found that the ABPI ratio was influenced by whether patients had baseline hypotension or hypertension (as represented by the brachial systolic pressure), and that baseline systemic pressure was therefore a confounder of ABPI measurement. Klein and Hage⁴² try to make sense of the diverse methodologies in practice for measuring ABPI and try to synthesize a standard approach. They identify a long list of technical factors (see Table 2 below) that can confound serial ABPI values and make empirical recommendations for their standardization. They point out, rightly, that without standardization, studies using ABPI testing cannot be compared to each other adequately. And patients moving from centre to centre might find their ABPI drifts as well.⁸³

Klein and Hage⁴² point out that there is no agreement on the precise normal value (or normal range) for ABPI, not least due to methodological inconsistencies between groups; some authorities apply a lower limit of ABPI for

the absence of disease, some apply an upper limit for the presence of disease, and others still consider both these limits to be one and the same (so defining a point value for ABPI below which there is disease, and above which disease is absent). When trying to relate ABPI to symptomatic patients who complained of intermittent claudication, Yao and colleagues⁷⁷ found that the ABPI varied from between 0.2 to 1.0 in these patients.

Notably, Hiatt and colleagues⁴ add that, in any case, all “normal” ranges for ABPI need to be corrected for sex, order of measurement of the limbs, and which ankle vessel(s) is/are being used for the calculation.

As a result of the lack of consensus on standardization, the accuracy and repeatability of ABPI readings has certainly been questioned in the literature, especially between diseased (symptomatic) and “normal” (asymptomatic) patient groups, and for nonexpert and expert practitioners.^{27,84–88} Vowden and Vowden⁷⁶ surveyed nurse practitioners and found poor understanding of the methodological requirements for accurate and reproducible ABPI measurement, as well as a lack of awareness of its limitations. It is interesting to note that ABPI has not been included in the UK Department of Health’s five-yearly screening program for vascular risk for those aged 40–74 years.⁸⁹

Furthermore, ABPI has been found to be insensitive to detecting progression of atherosclerosis. In a study by McLafferty and colleagues,⁹⁰ ABPI had a sensitivity of 41%, a specificity of 84%, and an overall accuracy of 68% for detecting changes in peripheral arterial disease status compared to catheter angiography and duplex ultrasound imaging (as gold standards), leading the authors to recommend that imaging studies are superior to ABPI to monitor the progression of peripheral vascular disease.

Conclusion

Although regarded as a routine test, a great deal of further work is required before a consensus can be reached on a standardized technique for ABPI. A practitioner should be familiar with the evidence around the theoretical basis for the ABPI test in order to make evidence-based interpretations of the results. In particular, practitioners need to be aware of the technical limitations of using ABPI and how variations in ABPI methodology can affect the findings. In certain common clinical situations, for example diabetic patients with calcified ankle arteries, ABPI can give artifactual results, or be nondiagnostic altogether. In these cases, practitioners need to consider alternative tests to confirm or exclude peripheral vascular disease.

Table 2 Nonstandard aspects of ABPI measurement⁴²

Position of patient during measurement
Width and level of sphygmometer cuffs
Use of Korotkoff method vs Dinamap vs Doppler probe to define systolic pressures
Whether to use both brachial pressures or not in calculation of ABPI
Whether to use all three crural vessels, two, or just one in calculation of ABPI
Whether to use highest or mean values of pressure in calculation of ABPI
Normal ranges for ABPI ratios

Abbreviation: ABPI, Ankle Brachial Pressure Index.

Disclosures

The authors report no conflicts of interest in this work.

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