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Ankle brachial indices – do they have a role in the assessment of blood flow to the feet in people with diabetes?

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INTRODUCTION

The Ankle Brachial Index (ABI) is a non-invasive, quantitative measurement used to evaluate arterial blood flow to the feet, at the level of the ankle or dorsum of the foot where the measure is taken. The ABI has several indications for use in podiatry including: where there is a need to establish the likelihood of healing, such as in chronic wound care or pre-surgical evaluation; or where the current circulatory patency to the feet requires assessment. This test is often utilised in combination with other vascular assessments, such as pulse palpation and the evaluation for ischaemic symptoms such as intermittent claudication. It is simple, fast and requires minimal equipment. As with any clinical test the ABI must be interpreted in light of relevant limitations and in context of the individual patient presentation. Controversy exists over whether the ABI is a meaningful measurement to utilise in the pedal vascular assessment of people with diabetes. It has been suggested that it should be disbandoned in this population due to the confounding factor of medial calcific sclerosis, which can interfere with test accuracy. The following discussion aims to highlight the advantages and disadvantages of the ABI, with particular reference to this clinical group. Two case scenarios, involving chronic wounds in patients with diabetes mellitus, are drawn upon to illustrate the critical issues under debate. The final remarks in the section on *Clinical Implications* offers suggestions regarding the role of ABIs in the assessment of blood flow to the feet in people with diabetes, in context of the known limitations of the test.

Keywords: vascular assessment, ankle brachial index, diabetes mellitus, ankle systolic blood pressure

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OVERVIEW OF THE ANKLE BRACHIAL INDEX

The ABI involves the measurement and comparison of the systolic blood pressure in the ankle arteries undergoing investigation, to the systolic blood pressure in the arm arteries, which serve as reference values. It is based upon the logic that in a normal circulatory system (ie, where flow through the blood vessels is not impeded by pathology such as atherosclerosis) the systolic blood pressure in the leg arteries should be the same or slightly higher than the systolic blood pressure in the brachial arteries. Where there is significant arterial narrowing in the leg vessels a corresponding alteration in blood flow haemodynamics occurs, resulting in measurably reduced blood pressure in the affected vessels. This decrease in blood pressure is presumed to be proportional to the magnitude of pathology and the degree to which the vascular tree can compensate via collateral circulation. Overall therefore, the test is used to quantify the degree to which blood flow to the ankle and thus the foot is reduced, due to the impact of narrowing vessel pathology (aka Peripheral Arterial Disease – PAD/Peripheral Vascular Disease – PVD) in the proximal leg arteries. This information then contributes to the overall results of a vascular assessment, thereby assisting with clinical judgements regarding the patency of arterial flow to the feet.

HOW TO TAKE THE ABI

An overview of the procedure for taking an ABI is outlined below, however readers are directed to prior publications should further detail be required.[1] Figure One outlines equipment requirements for this procedure. Once the procedure has been fully explained to the patient and consent to continue obtained, the patient is required to lie resting in a supine position for approximately 15 minutes. After this time the respective blood pressure measurements are taken. The procedure for obtaining systolic blood pressure readings is the same for each artery undergoing measurement and involves the following steps:

- Place the sphygmomanometer around the limb just above the site of the vessel undergoing measurement.
- Palpate the pulse if possible and then place the Doppler probe at an angle of approximately 45 degrees over the site of the vessel.
- Slowly move the Doppler probe around the site of the artery in order to locate the position where the loudest, clearest sound is present.
- Inflate the blood pressure cuff until the sound of the pulse disappears and then approximately 10 to 20 mmHg higher.

Figure 1: Equipment required to take an ABI measurement.

- A sphygmomanometer – the standard cuff size is of 12cm width, if there is provision for more than one cuff to be purchased for the clinic a variety of sizes can assist in obtaining more accurate measures in cases where limb size varies significantly.[1]
- A Doppler ultrasound with a 5 or 8 MHz probe – there are several Doppler ultrasound machines commercially available, generally the small handheld units are easiest to use and most economical (ensure batteries are checked regularly).
- Water soluble, ultrasonic transmission gel.

- Slowly deflate the cuff and record the pressure reading from the sygmomanometer at the point which the pulse sound returns. This reading represents the systolic blood pressure in that vessel at that point. (Note: take care not to let the cuff down too quickly as this may lead to an inaccurate reading).

Using this method, systolic blood pressure readings should then be taken for the brachial artery in the left and right arms and the dorsalis pedis (DP) and posterior tibial (PT) arteries in the left and right feet/ankles. The ABI is calculated by dividing the ankle blood systolic pressure measurement (using the DP or PT reading) by the arm blood systolic pressure measurement (ie, the brachial artery) on the respective side. The ABI should be calculated in turn for both the dorsalis pedis artery and the posterior tibial artery as vascular pathology within the lower leg vessels can vary, resulting in different readings being obtained for either vessel. This has clinical significance when interpreting the results of the ABIs. Please refer to Figure Two for an example of calculation of the ABI and refer to other references for a variation on the above measurement approach.[2] Note that in 12% of the population the dorsalis pedis artery is reported as being congenitally absent and should be considered as a possible explanation if this vessel is inaudible on assessment.[3]

INTERPRETATION OF ABI VALUES

The ABI values are reported in the literature as indicating the following:

- An ABI of 1.30 or greater indicates incompressible vessels usually due to calcification, which may be caused from diabetes and renders the test results unusable, alternative testing such as toe pressures is recommended.
- An ABI of 0.91 to 1.30 indicates normal blood flow.
- An ABI of 0.41 to 0.90 indicates mild to moderate peripheral arterial disease.
- An ABI of 0.00 to 0.40 indicates severe peripheral arterial disease.[4]

While the above values can be used as a guide to the degree of PAD and the subsequent level of blood flow to the feet, it is crucial that test results are still interpreted in light of other factors. Firstly this involves careful observation of the clinical picture, where if ABI test results and the presenting clinical situation do not match up, further investigation and consideration to the case should be given. For example, a patient has an ABI of 0.8 but presents with a non-healing ischaemic digital ulcer. While the ABI is not substantially lowered (and is indicating only mild PAD) an alternative explanation might be that there is significant PAD in the pedal arteries which are below the ankle level where the ABI is measured. ABI test results did not fully explain the presenting complaint but further investigation (with tests such as toe pressure measurements or transcutaneous oxygen perfusion) would reveal that further information was needed for an

Figure 2: An example of ABI calculation.

Systolic blood pressure readings (mmHg)

Left		**Right**	
Brachial Artery	120	Brachial Artery	120
Dorsalis Pedis Artery	125	Dorsalis Pedis Artery	120
Posterior Tibial Artery	100	Posterior Tibial Artery	75

Ankle Brachial Index Calculations

For the Dorsalis Pedis Artery:

Left	**Right**
125 divided by 120 = 1.1 (ABI)	120 divided by 120 = 1.0 (ABI)

For the Posterior Tibial Artery:

Left	**Right**
100 divided by 120 = 0.9 (ABI)	75 divided by 120 = 0.6 (ABI)

adequate diagnosis. In addition, in interpreting ABI results in general there are issues surrounding the viability of collateral blood flow, measurement error and test sensitivity and specificity, that require consideration.

HOW RELIABLE IS THE MEASUREMENT OF THE ABI?

The reliability of the measurement of the ABI has been investigated and is generally reported as good.[3,5] In a study by Fowkes in 1988, it was found that the variability of the ABI due to factors such as different clinicians, repeated measures and timing of measurement, was substantially less than that which occurs due to true changes in the ABI measurement from disease.[6] For this reason it has been reported that "standard clinical measures of the ABI may be sufficiently accurate to guide clinical decision making."[7] In addition, when reviewing the literature, Bird et al 1999 report the 95% Confidence Interval (CI) of reproducibility to be in the +/- 0.10 to +/- 0.15 range.[8] That is, it can be reasonably expected that the effect of measurement error on an ABI measurement, will be within this range for the large majority of measures taken. Several authors do however, highlight the importance of experience in enhancing the accuracy of ABI measurement, both in screening for PAD and in the detection of critical limb ischaemia.[1,9] There is evidence to suggest that the reliability of ABI measurements is poorer in people with diabetes. In a study by Matzke et al 2003, diabetes was found to be a significant factor increasing inter-observer difference in the measurement of the ABI in people with critical leg ischaemia.[9] This study however is likely to be a worse case scenario as the sample was based on the more clinically severe cases, in addition to the limitation of a small sample size. It might be argued that those with diabetes in the study are also likely to present with co-morbidities which might make the ABI measurement more difficult to perform accurately. For this reason this study may not be a good representation of the general population with diabetes. Overall therefore, while the reliability generally appears to be relatively good, there is error associated with the measurement of the ABI (this may be higher in patients with complications from diabetes and inexperienced clinicians) which does introduce some margin for inaccuracy when assessing for PAD in this clinical group.

HOW WELL DOES THE ABI INDICATE PERIPHERAL ARTERIAL DISEASE, THE RISK OF MORBIDITY, MORTALITY AND OTHER RELATED PATIENT OUTCOMES?

The degree to which the ABI measurement truly indicates the presence and magnitude of PAD, that is its validity as a measurement, has also undergone investigation. The ABI measurement has been compared to the 'gold standard' assessment of PAD, angiography and a high correlation was found. The authors reported that an "ABI < 0.90 is 95% sensitive and 99% specific for angiographically significant PAD".[5,6] In one of the classic studies by Carter in 1969 however, it was found that the while the ABI was abnormal in 80% of cases which were shown to have severe stenosis, in cases where mild stenosis was detected the ABI was abnormal only 50% of the time.[10] This and further research would suggest that while the ABI measurement correlates well with angiographically identified disease, it is more accurate at detecting more severe PAD and is variable in its ability to indicate the presence and degree of milder PAD. In addition, the ABI has been demonstrated to be useful in monitoring disease progression.[8] It has been reported that a change that exceeds the measurement error margin of +/- 0.15 suggests a true change in vascular status has occurred, a highly useful indicator in clinical practice when monitoring patient progress. Conflicting information regarding ABI validity however, does exist in the literature and therefore further research evaluating the sensitivity and specificity of the ABI in its ability to detect PAD is needed, specifically in populations such as diabetes mellitus.

In regard to diabetes mellitus however, how do ABIs rate in their ability to detect PAD and related outcomes? It is consistently reported throughout the literature that there is an increased prevalence of medial calcific sclerosis in the tibio-peroneal arteries of patients with diabetes.[11,12,13] This has also been reported in diabetes to possibly extend more distally into the digital arteries of the feet.[14] Therefore due to this phenomena of arterial calcification which leads to stiffness of the arterial wall, measurements of systolic blood pressure and thus ABIs are falsely elevated in those people with diabetes affected by this arterial change. Given this, it would be reasonable to suggest that overall the validity of ABIs is reduced in this population. While it is generally well accepted that this is the case, questions remain around the following issues: to what extent will people with diabetes exhibit inaccurate test results due to medial calcification; and is it a predictable event, that is, can we tell which patients have inaccurate readings and which do not? The answers to these questions may better inform the diagnostic utility of this test in this population, however to date there is a lack of evidence on which to base a consensus. In a study by Emanuele in 1981, 24 patients with diabetes and a past or present history of ischaemic gangrene had ABI measurements of the affected limb taken. In this select group, only 33% had ABIs that measured 0.9 or lower.[13] The authors concluded that "a Doppler pressure index above 0.90 is not a reliable indicator of adequate blood flow in severe diabetic occlusive arterial disease".

The degree to which incompressibility of blood vessels due to medial calcification occurs however, is very difficult to measure. Therefore its prevalence is not truly known, but has been reported to occur in 18% to 30% of patients with diabetes.[12,13,15] It is generally accepted that an ABI of around 1.3 or above indicates incompressible vessels but it is less clear whether a mild amount of calcification can occur raising a lowered ABI into an apparently normal zone. It is feasible to consider that this may be the case and indeed creates a more difficult situation, as this is less able to be detected clinically. A study by Brookes et al in 2001, investigating related issues compared differences in ABIs and Toe Brachial Indices (TBIs) in 174 participants with diabetes and 53 controls.[14] This is significant as TBIs are thought not to be affected by medial calcification sclerosis and therefore serve as a reference point on which to evaluate possible changes which may occur to ABIs from this pathology. It was reported that only where overt calcification was evident, as demonstrated by an ABI of greater

then 1.3, was the ABI significantly different to the TBI. This finding supports the recommendation that an ABI of 1.3 or higher is inaccurate and therefore not useful. While this study also suggests that ABIs below 1.3 may not be hampered by the possible presence of medial calcification, there is acknowledgement that this study result may be due to toe vessels at lower pressures being equally affected by medial wall calcification, leading to no differences existing between the two measures. In some other studies investigating the use of ankle blood pressure measurements (the major component of the ABI) as a predictor of foot ulcer healing in diabetes mellitus, ankle pressures were found to be a useful tool in contributing to the prediction of primary healing.[11,16] This would suggest that the ABI may have some clinical use in this population when predicting patient outcomes such as healing. In addition, claudication and very low ABI has been associated with increased risk of disease progression, gangrene, ulcers and amputation in studies including participants with diabetes.[5,17] There is however, some literature which reports "a lack of an association between ABI and the predicted potential for healing".[18] The clinical implications of the research discussed above may justifiably appear somewhat contradictory and confusing. It is perhaps fair to suggest therefore, that while there are uses for the ABI in the vascular assessment of people with diabetes, there are also downfalls, however there is insufficient information at this stage on which to base a definitive and supportable consensus statement on this issue.

Until that research is available, how is the current information placed into a meaningful clinical context? While it is evident that the ABI is not a perfect measurement, it is likely that an abnormal ABI of 0.9 or lower is indicative of the presence of occlusive arterial pathology. It is also reasonable to suggest that the lower the ABI measurement, the worse the underlying disease and related patient outcomes are like to be. The clinician should be cognizant however of the possibility of false negative measurements, that is gaining a normal ABI result where underlying PAD is actually is present, particularly where milder arterial pathology exists. In addition, always consider the possible impact of medial calcification in patients with diabetes.

On a more overall note, ABIs have been described as a "surrogate measure of atherosclerosis" due to a high correlation between the two.[7] It is not surprising therefore that research investigating health issues of a more general nature, have found that "the ABI is well established as an independent predictor of cardiovascular morbidity and mortality."[18] Vogt et al 1993 and Newman et al 1993 "provide compelling evidence that a reduction in the ABI of 0.9 or less is associated with increased risk for both coronary heart disease and total cardiovascular disease morbidity and mortality as well as all-cause mortality".[5,17] Subsequent research investigating the incidence of stroke also reports a strong association between low ABI and ischaemic stroke, although this was somewhat reduced when adjusted for other risk factors.[19] Given the above findings and that further research strongly reinforce these relationships, it would be reasonable to view a low ABI as a risk factor or at least a significant risk marker for these adverse health events.[5,7,17,20,21,22,23,24] An additional consideration is the relationship between a low ABI and reduced lower extremity functional capacity, as indicated by findings such as reduced leg strength and slower walking velocity.[5]

In light of the above discussion it would appear that results of an ABI potentially have very important implications which may span a broad range of significant health issues. Podiatrists are often in the position of being the first practitioner to take an ABI. Where patients present with lowered ABIs, communication to the GP of test findings with a view to a cardio-vascular and general medical check-up, should be strongly considered if not already addressed.

CASE EXAMPLES

The following case studies aim to offer a more practical perspective on the use of ABIs in diabetes and are followed by a suggested framework by which this assessment might be used in this group until further knowledge comes to light.

Case One - Mr Z

Mr Z, a 62 year old gentleman of Italian background, initially presented to podiatry for the management of an ulcer on the lateral aspect of his right heel. On first presentation the wound measured 7mm horizontally at the widest point and 5 mm vertically at the widest point, with surrounding erythema extending from the edges of the wound for approximately 3 cm. The base of the wound was fully covered with thick black and yellow adherent slough and was dry and hard. Mr Z reported that the wound began about one month previously when he got a stone in his shoe, which he felt rubbed the skin in this area. The patient also relayed that the wound was slowly getting bigger and was very painful. Current self-treatment consisted of daily application of dry gauze after washing with warm water.

Mr Z's medical history consisted of type 2 diabetes mellitus diagnosed approximately 15 years ago, which was being managed with oral hypoglycaemic agents and diet. His last HbA1c was 9.6 m/mol, which was consistent with past readings. Mr Z also had a history of hypertension, hypercholesterolemia and ischaemic heart disease, all of which were under the management of his general practitioner. Social history included a long history of cigarette smoking that has increased since retirement two years previously due to boredom. Mr Z reported spending most of his days around home watching television and drove the car when he needed to leave home for anything.

Initial physical assessment findings revealed that the pulses on the right foot were diminished on palpation. ABI values obtained were 1.2 for the dorsalis pedis pulses on the right and left feet and 1.1 for the posterior tibial pulse on the left foot with an inability to take a reading on the right foot due to the presence of pitting oedema. Clinical sensory neurological assessment revealed normal monofilament and vibration sensations on both feet and other biomechanical/pressure assessments were non-significant. The patient was wearing slippers due to the pain at the site of the wound. At this stage it was unclear as to whether the base of the ulcer extended to bone due to the thick coverage of slough.

Treatment consisted of mechanical debridement of the sloughy tissue where possible (which was somewhat restricted by pain), the initiation of a moist wound dressing regime utilising daily changes of a hydrogel with a secondary dressing

and referral to the GP for antibiotic therapy and pain relief. Appropriate maintenance of good blood glucose control was reinforced and modification of risk factors, particular cessation of smoking, was discussed. No evidence of a stone or foreign body was found in the wound.

Management of Mr Z continued along the same lines for several months with slow improvements of the wound occurring. This was indicated by moistening and removal of the slough and the appearance of granulation tissue covering approximately 25% of the wound base. Pain was still present but decreased with ongoing use of pain relief medication. Antibiotic therapy was prescribed by the doctor as required during this time due to intermittent bouts of cellulitis. Plain film radiographs were ordered to evaluate for the presence of osteomyelitis in the underlying calcaneus, however these were clear. The patient continued to smoke heavily.

After several months of treatment it became evident that the clinical progress of the wound had not continued and signs of deterioration, such as increasing pain and increasing wound size, were now presenting. A review of the assessment of causative and contributing factors and the current management regime was conducted in an attempt to identify why the wound was not healing. Given the presence of symptoms that were consistent with an ischaemic aetiology, the vascular assessment was reviewed. While the initial ABIs fell within the normal range, the complicating factor of medial calcification due to diabetes was re-considered as possibly resulting in a false value being obtained. Given the subjectivity of the other clinical indicators, ie colour, temperature etc, and the lack of availability of other accurate clinical tests, it was difficult to make a clear, quantitative assessment of the vascular status at this time. Mr Z was therefore referred back to his doctor who sent the patient to a vascular laboratory for duplex scanning. The results of the vascular studies showed that an 80% stenosis existed at the level of the right popliteal artery. The result of the initial ABI had falsely indicated that the blood flow to the foot was adequate to heal the wound. Ongoing soft tissue infections delayed the recognition of the fact that significant PVD was the critical factor in this case that was impairing healing. Pulses were palpable but diminished which served to arouse some suspicion of an ischaemic aetiology, but other indicators were inconclusive. For example, intermittent claudication was not revealed due to the sedentary lifestyle lead by the patient and reports of rest pain were mixed and influenced by the patient's focus on the sore wound. Clearly, the ABI was not a useful clinical test in this patient. Mr Z subsequently had a balloon angioplasty and the wound healed over the several months to follow. He continues to smoke heavily.

Case Two – Mr M

Mr M, a 58 year old gentleman of Irish background, was referred to the podiatry clinic for the management of two small wounds on his left foot. One small but deep wound, measuring approximately 2 mm in diameter, was located medial to the left fifth toe nail and was partially covered in hyperkeratosis. The other wound was located on the apex of the third digit and measured 3 mm by 4 mm with a central necrotic, sloughy base. Both wounds were surrounded by localised erythema and the patient reported that the ulcer on the fifth toe often became infected but resolved when the doctor prescribed antibiotics. While the wound on the fifth toe had been present for over ten months, it was the other wound on the third toe which was only one month old that was troubling the patient, as it was very painful. The patient was not sure how the wounds started, current self treatment consisted of application of antiseptic liquid and plastic strips.

Mr M's medical history consisted of type 2 diabetes mellitus, diagnosed 20 years ago and was currently being managed with oral hypoglycaemic agents and diet. HbA1c readings were reported as usually being around 7.5 m/mol and apart from a minor cerebro-vascular accident in 1998, there was no other significant medical history. Mr M reported that he lead an active life and had a large extended family with whom he enjoyed spending much of his time.

Assessment findings revealed that the pulses on the left foot were palpable but diminished and Mr M reported symptoms consistent with intermittent claudication when walking, particularly if on an incline. Clinical sensory neurological, biomechanical and pressure assessments were all non significant. ABIs were subsequently conducted and were found to be 0.4 for both the dorsalis pedis and posterior tibial pulses on the left foot and 0.8 and 0.7 respectively for the right foot.

Management was initially aimed at confirming and addressing the aetiological factors for the foot wounds. The ABI was clearly significantly lowered on the side which was affected by ulceration and therefore Mr M was subsequently referred for further vascular lab testing, to establish the level of blood flow to the feet. He underwent Duplex scanning which revealed significant occlusive arterial pathology. In addition, plain film x-rays were ordered to evaluate for osteomyelitis in the fifth toe which were inconclusive. Bone scans however confirmed this diagnosis. Treatment therefore consisted of a balloon angioplasty in the left leg and a regime of long term antibiotic therapy was instigated. A moist wound dressing regime utilising third daily changes of a polyurethane foam was prescribed for the wound at the third digit, suitable footwear was purchased and appropriate maintenance of good blood glucose control was reinforced. After three months the lesions had fully resolved and the patient was able to walk for longer distances without intermittent claudication.

CLINICAL IMPLICATIONS

The current body of literature regarding the use of the ABI in the clinical vascular assessment of patients with diabetes is contradictory and there is insufficient information on which to base a definitive consensus as to its use. Given this may be one of only a few tests available in the clinical setting to assess the blood flow to the feet, ascertaining its usefulness has relevance to current clinical practice. The case studies outlined above demonstrate that in one scenario, the ABI was misleading and inaccurate due to false elevation, which in that situation lead to delayed diagnosis and management. The other case however illustrated that the ABIs, which were significantly lowered on the affected side, provided a meaningful result which was used to inform subsequent testing and development of the patient care plan. From a clinical perspective therefore, it is appropriate to suggest that the ABI can have a useful place in the vascular assessment of patients with diabetes, in combination with other assessments such as: questioning regarding ischaemic pain

(intermittent claudication, rest pain); signs of critical ischaemia (ulceration, gangrene); palpation of pulses; and the use of other tests such as toe pressures if the required equipment is available. The salient point is that clinicians must be aware of the limitations of the ABI and interpret individual assessment findings in light of this. Therefore instead of outright accepting or rejecting the use of this test in diabetes, it is proposed that the following considerations in Figure 3 offer a more suitable framework on which to base the interpretation of the ABI result in this clinical group.

Figure 3: Proposed framework for the interpretation of the ABI in people with diabetes.

- A low ABI (≤ 0.9) is likely to be abnormal and potentially problematic, therefore requires further attention.
- A normal ABI reading (0.91 to 1.3) indicates that PAD is unlikely, however in some situations this reading may be falsely elevated and therefore further testing should be considered if the clinical picture suggests ischaemia is present, eg: non healing wounds.[25]
- An elevated ABI (≥ 1.3) is abnormally high and renders the test results unusable, alternative testing is recommended if clinically indicated.
- In cases of acute/severe ischaemic presentations or where this is suspected, such as gangrene or invasive infection with possible PAD, immediate testing at a vascular laboratory and medical attention is strongly recommended.

Until further research is conducted into the use of the ABI in diabetes or new clinical tests are developed, it is worthwhile considering the potential usefulness of the ABI in light of its limitations. This does require an index of suspicion, where the ABI values are not just taken on face value but are evaluated in context of the other presenting findings. While it may be that the test will only serve of benefit to a proportion of this population, in the absence of another option, its use may be justified on the grounds that, in some instances, it can provide valuable information regarding the patient's vascular status, which may in turn impact positively on patient outcomes.

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