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An Overview on Ankle-Brachial Index

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Abstract:
The Ankle-Brachial Index (ABI) is a simple, non-invasive diagnostic test used primarily to assess the presence and severity of peripheral artery disease (PAD) a condition where arteries in the legs or arms are narrowed or blocked, typically due to atherosclerosis.
Keywords: Ankle-brachial index, Peripheral Vascular Disease, Atherosclerosis.

Introduction:

The ABI has also been called the ankle-arm index, the ankle-brachial blood pressure index, the ankle-arm ratio, or the Winsor Index. The term ABI was recommended by the recent American Heart Association Proceeding on Atherosclerotic Peripheral Vascular Disease (1).

The ABI is a ratio of Doppler-recorded systolic pressures in the lower and upper extremities **(Figure)**. The ABI is a quick, easy, and cost-free diagnostic test. In healthy people without peripheral artery disease (PAD), arterial pressures increase with greater distance from the heart. This occurs because of retrograde wave reflection generated by resistance from peripheral arterioles that adds to retrograde flow (2).

Additionally, increasing impedance with increasing arterial taper contributes to increasing systolic pressures with increasing distance from the heart. This phenomenon results in higher systolic pressures at the ankle compared to the brachial arteries in people without lower extremity arterial obstruction. For these reasons, people without lower extremity atherosclerosis typically have an ABI value ≥1.10 and 1.30 is indicative of medial calcinosis of lower extremity peripheral arteries and may be commonly observed in people with and without PAD (2).

The ABI as a measure of the presence and severity of PAD has been validated against angiographically documented PAD. Using an angiogram-demonstrated stenosis of 50% or greater to diagnose PAD, Lijmer et al. reported that an ABI. An ABI of 1.19 had sensitivity of 94% and specificity of 29% for PAD. It has been reported that an ABI< 0.97 was 94% sensitive and 99% specific for PAD. ABI is both sensitive and specific for PAD, with lower ABI values indicative of more severe lower extremity atherosclerosis (3).

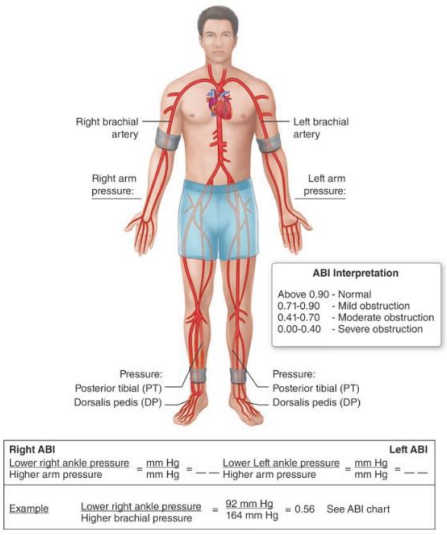

Figure 1: The ankle brachial index measurement (2)

Anatomical Consideration

ABI obtained by dividing the highest pressure at the ankle level (obtained in the posterior tibial artery, dorsal pedial, and when necessary, the peroneal arteries) by the systolic pressure in the upper arm (brachial artery) (4).

- **Dorsalis pedis artery:** The dorsalis pedis artery arises primarily from the anterior tibial artery and starts higher in the anterior compartment of the leg between the tibialis anterior and extensor hallucis longus (EHL). In most cases, the vessel above the ankle passes under the EHL to reside between EHL and the extensor digitorum longus (EDL).

  Other variations include later crossing sites either at the ankle or distal. Ranging the great toe can help identify the EHL for guidance about finding the dorsalis pedis pulse, especially in a patient with faint pulses.

  Additionally, the artery can be traced proximally from the first dorsal metatarsal artery extending into the great toe webspace or distally from the supplying artery (5).

- **Posterior tibial artery:** The posterior tibial artery passes posterior to the medial malleolus at the ankle between the tibialis posterior and flexor digitorum longus tendons (6).

- **Brachial artery:** The brachial artery is palpable medial to the biceps tendon in the antecubital fossa (7).

Brachial pressure is used as a surrogate for central aortic pressure, which is not readily available and is generally accurate unless there is an occlusive disease of the vessels supplying the upper extremity. For this reason, pressure is measured in both upper extremities, and the higher of the two is used (1).

This relationship allows a better appreciation of the degree of arterial occlusive disease. Without taking into account the brachial pressure, it would not be possible to know if low pressure in the ankle was caused by systemic hypotension or PAD; conversely, ankle pressure could be normal despite significant disease if the patient was hypertensive. The objective is to detect occlusive disease by identifying the pressure drops between the proximal aorta and the ankle arteries (8).

Ankle Brachial Index Measurement

The ABI should be measured with the patient in a supine position, after at least a 5 min rest **(Table 1)**. Appropriately sized blood pressure cuffs are placed over each brachial artery and at each ankle. At the ankle, the blood pressure cuff bladder should be positioned so that the artery marker is directly over the posterior tibial artery.

Patients should be instructed not to talk during the examination, since talking can alter the systolic pressures during the test (9).

Table 1: Measuring the Ankle Brachial Index (2).

Measuring the Ankle Brachial Index
• The ankle brachial index (ABI) should be measured in the supine position.
• The patient should rest supine for at least 5 min before the measurement is performed.
• A 5–10 mHz Doppler and appropriately sized blood pressure cuffs for each extremity are required.
• Pressures are measured beginning with the right brachial artery followed by the right dorsalis pedis, right posterior tibial, left dorsalis pedis, left posterior tibial, and left brachial arteries.
• The Doppler probe should be used to locate the strongest signal from each artery.
• The sphygmomanometer is inflated to at least 20 mm above the systolic pressure.

• The sphygmomanometer should be deflated no faster than 2 mm/s.

• The ABI may be calculated for each artery but is typically calculated for each leg by dividing the highest lower extremity pressure in each leg by the highest brachial artery pressure.

Blood pressures are typically measured sequentially starting with the right upper extremity to the right lower extremity, left lower extremity, and left upper extremity. In the lower extremities, the dorsalis pedis and the posterior tibial pressure are each measured. However, if time is insufficient for measuring both the dorsalis pedis and posterior tibial arteries in each extremity, accurate ABI values can also be obtained by measuring the posterior tibial artery alone (10).

A handheld Doppler is used to locate each artery before each arterial pressure measurement. The probe should be moved so that it detects the strongest signal from the artery prior to cuff inflation.

Accurate ABI measurement consists of inflating the cuff sphygmomanometer to at least 20 mm above the systolic pressure and deflating the pressure no faster than 2 mm/s. The systolic pressure at which the pulse reappears is measured and recorded for each artery and used to calculate the ABI as described below (11).

Calculating the ABI

The ABI is the ratio of Doppler-recorded systolic pressures in the lower and upper extremities. An ABI may be calculated for each lower extremity artery, by dividing the lower extremity artery pressure by the highest of the brachial artery pressures. The ABI is typically calculated for each leg, by dividing the highest of the two pressures in each leg by the highest of the left vs. the right brachial artery pressures. The highest pressure in each leg is traditionally selected when calculating the ABI, because the highest pressure represents the greatest arterial pressure reaching the foot (12).

However, it has been demonstrated that the ABI calculation using the average of the dorsalis pedis, and posterior tibial artery pressures correlates most closely with functional impairment in people with PAD. Using the lowest of the dorsalis pedis and posterior tibial pressures to calculate the ABI in each leg maximizes sensitivity of the ABI for the diagnosis of PAD but may be associated with lower specificity (1).

Interpreting ABI Values

A normal ABI value is defined as an ABI between 1.10 and 1.30 **(Table)**. An ABI value of < 0.90 is 99% specific and approximately 79% sensitive for the presence of PAD. ABI values < 1.00 are more sensitive for PAD than ABI values of > 0.90. For example, as noted above, an ABI<0.97 was reported to be 94% sensitive for PAD. Among people with ABI < 0.90, lower ABI values indicate more severe PAD.

ABI values < 0.50 are associated with increased risk of amputation compared to higher ABI values in patients with leg ulcers and in patients with leg ulcers and in patients with history of diabetes values (13).

Table 2: Ankle brachial index values and their clinical significance (2).

ABI value	Clinical relevance	Associations with clinically important outcomes
1.10–1.30 (reference)	Absence of lower extremity atherosclerosis	Associated with lower rates of cardiovascular events and better lower extremity functioning than ABI values <1.1.0
0.90–1.10	Small amounts of lower extremity atherosclerosis	People with ABI 0.90–1.10 have slightly higher rates of all-cause mortality, cardiovascular events, and mobility loss compared to the reference group
0.50–0.90	Indicates the presence of mild to moderate PAD	Risk of all-cause mortality, cardiovascular events, and mobility loss is significantly higher than the reference group
<0.50	Indicates severe PAD	Risk of all-cause mortality, cardiovascular events, and mobility loss is significantly higher than the reference. Increased risk of lower extremity limb loss or critical limb ischemia

> 1.30	Indicates lower extremity medial calcinosis and inability to assess the presence and severity of lower extremity atherosclerosis	Increased risk of mortality and cardiovascular events compared to the reference

Physiology of the ABI

- **Systolic Blood Pressure (SBP) Higher in the Ankles Than in the Arms**

The blood pressure waveform amplifies as it travels distally from the heart, resulting in a progressive increase in SBP and a decrease in diastolic blood pressure. The most widely accepted model used to explain the SBP amplification relies on retrograde wave reflection from resistant distal arterioles, which is additive to the antegrade wave. Several lines of evidence indicate that reflected waves occur at various sites in the vascular bed, with some attenuation along the arterial system. However, the reflected wave is not the sole explanation for the changes in pressure wave morphology (14).

In the legs, remodeling of vessel structure occurs, resulting from increased intraluminal pressure, characterized by increased wall thickening and unchanged inner radius. The changes in wall thickness resulting from increased hydrostatic pressure in the lower extremities with walking (vertical position) occur during the second year of life and plausibly explain why the ABI is <1.00 in the newborn and increases to adult values at 2 to 3 years of age. Therefore, both reflected waves and changes in vessel wall thickness and consequently stiffness contribute to SBP amplification (15).

- **Physiological variations of ankle-brachial index**

The force generated by the contraction of the heart is transmitted to the arteries as a pulse wave (ventriculoarterial coupling). It undergoes amplification while crossing the vascular bed, from proximal to distal. The explanation is the retrograde pulse wave reflection arising from arterioles which has an additive effect, stronger in arterial territories characterized by increased peripheral vascular resistance such as limbs. These reflected waves suffer an attenuation process, too (16).

Increased hydrostatic pressure (due to the vertical position of the body) which acts on the lower limb vessels, may cause a physiological thickening of the arterial wall, without changing the inner diameter of the vessel. This is supported by the ABI change, which in the neonate is 1, subsequently increasing to the normal value found in adults around the age of 2-3 years. These two mechanisms explain why blood pressure is physiologically in the legs than the upper limbs (17).

Many physiological factors can affect ABI, such as age, height, race or order of the measurements. Two observational studies showed a difference of 0.03 between ABI to the right leg compared to the left lower limb ABI. This minor difference can be explained through the order of measurements (usually first measured is the right leg), knowing that blood pressure diminishes slightly after the patient is accustomed with the doctor (18).

Although theoretically ABI should increase with age as a result of increased arterial stiffness, longitudinal population studies have shown the opposite, paradox explained by increased prevalence of PAD in the elderly. The contribution of the height at ABI value is negligible, about 0.01 per 20 cm, although in individuals without cardiovascular impairment, ABI should increase proportionally with height because of the higher systolic blood pressure in the legs due to increased hydrostatic pressure. Women have on average a lower ABI with 0.02 to 0.07, according to the study report, independent of other factors such as the height. This applies to populations without PAD / atherosclerosis (19).

Race also influences the ABI value, and this may be due to genetic factors. The blacks without PAD (MESA study participants) had on average an 0.02 lower ABI compared to the non-Hispanic whites of similar age. Also, the

European lineage is associated with a lower relative risk of developing PAD (RR=0.9) compared to Hispanic / Black. Increased heart rate is correlated inversely proportional to ABI values in patients without cardiovascular disease (20).

This can be explained by the increasing difference between central and peripheral systolic blood pressure with heart rate (brachial systolic blood pressure/central systolic blood pressure ratio increases with 0.012 units per 10 beats/min in addition) and the decreasing gain of the pulse wave (systolic shortening due to increased heart rate leads to displacement of the reflected wave from systole to diastole, which causes a decrease in blood pressure in the legs). This phenomenon was not observed in the MESA study participants (20).

Factors influencing blood pressure do not alter the diagnostic value of ABI, because ABI is a ratio of tensions, which in turn cancels any physiological factors of confusion (21).

ABI in Clinical Practice

- **ABI: A Diagnostic Method for Lower-Extremity PAD**

The natural history of PAD includes a decrease in the ABI over time. The level of ABI (and the corresponding ankle pressure) is useful to predict limb outcomes. PAD may not progress in a parallel manner in both limbs, so it is necessary to assess the ABI in both limbs during follow-up (22).

- **Post-exercise ABI**

Some patients with signs and/or symptoms of PAD have low normal or borderline ABI values (i.e., ABI values of 0.90–1.09), leaving uncertainty about the presence of PAD. In these patients, the ABI can be performed before and after treadmill exercise activity. A decline in ABI of 20% or greater after a treadmill exercise test indicates the presence of PAD (23).

The ABI drops after exercise in patients with PAD because during lower extremity exercise, such as treadmill walking activity, systolic blood pressure values increase centrally, while arterial vessels that supply the lower extremities dilate. Together, these phenomena result in an increase in the brachial artery pressure simultaneously with a drop in the ankle pressure.

These physiologic phenomena in response to exercise are observed even in healthy individuals. However, the magnitude of decline in the post-exercise ABI is approximately 5% in healthy people without lower extremity atherosclerosis vs. 20% in people with PAD (24)

A drop in a borderline ABI value (a borderline ABI value ranges from 0.90 to 1.09) of at least 20% after walking exercise is consistent with a diagnosis of PAD. Alternatively, a lower extremity pressure declines after exercise of >30 mm Hg can be used to diagnose PAD. In one study, the criterion of an ABI 30 mm Hg drop in lower extremity pressure after exercise was 33% sensitive and 85% specific for PAD. In a separate study, a recovery of the ABI to at least 90% of the baseline value within 3 min after completion of exercise was 94% specific for PAD (24).

The post-exercise ABI is typically measured before and after treadmill exercise in a vascular laboratory and requires treadmill equipment, and this testing may not be convenient in other settings. An alternative exercise ABI test is the heel-rise ABI, which does not require treadmill equipment and can be performed to elicit a diagnosis of PAD in people with borderline ABI values and suspected PAD (25).

In the heel rise ABI measurement, after the resting ABI, the patient lightly rests fingertips against a wall for balance, while rising up and down on the toes at a rate of one per second for 50 heel rises. The ABI is immediately repeated after the heel-rise exercise and has comparable sensitivity and specificity as a post-treadmill ABI for diagnosing PAD. Measuring the ABI as soon as possible after completion of the heel-rise exercise can improve the sensitivity of the post-heel-rise ABI for the diagnosis of PAD **(2).**

- **Abnormally High ABI**

In some cases, the ankle artery is incompressible and the systolic pressure at that location cannot be measured despite cuff inflation >250 mm Hg. In other cases, the ankle artery systolic pressure is measurable but

is much higher than the brachial artery systolic pressure, leading to an ABI that exceeds the normal range. These situations are related to calcification of the arterial wall and may occur in patients with medial calcinosis, diabetes mellitus, or end-stage renal disease. Vascular calcification does not imply that occlusive lesions are present, although these 2 conditions frequently coexist. When vascular calcification is present, however, stenotic disease cannot be detected by the ABI (22)

Other noninvasive tests such as measurement of the toe-brachial index or analysis of the Doppler waveform enable detection of occlusive disease despite a falsely high ABI. Measurement of the toe-brachial index is useful in such circumstances because the digital vessels rarely develop calcification and can provide an accurate determination of vascular disease in this setting. With these alternative tests, the rates of coexistent peripheral artery occlusive disease in patients with high ABIs range from 60% to 80% (22).

- **ABI and Cardiovascular diseases**

1. A Marker of Cardiovascular Risk and Atherosclerosis

A. Association of Low ABI With Cardiovascular Risk Factors and Prevalent Disease.

The ABI serves as a measure of systemic atherosclerosis and thus is associated with both atherosclerotic risk factors and prevalent CVD in other vascular beds. Atherosclerosis is a disease that affects medium, large sized arteries which is characterized by lipids, inflammatory cells accumulation within the arterial wall and scar tissue development that is covered by a fibrous cap (26).

There is an association between peripheral artery disease (PAD) and coronary artery disease (CAD) as they are mainly caused by atherosclerosis, so they share similar risk factor profiles. They are mainly caused by atherosclerosis, so they share the same risk factors and pathogenesis. PAD has been identified in many studies to correlate significantly with worse cardiovascular outcomes. The ABI has been introduced to diagnose PAD with high sensitivity and specificity compared against invasive angiography (27, 28).

A low ABI is associated with many cardiovascular risk factors, including hypertension, diabetes mellitus, dyslipidemia, smoking history, and several novel cardiovascular risk factors (eg, C-reactive protein, interleukin-6, homocysteine, and chronic kidney disease). ABI less than 0.9 is associated with 2-3 folds of increased risk of C.V and total mortality (29).

B. Association of High ABI With Cardiovascular Risk Factors and Prevalent Disease.

It has been shown that there is an association of an abnormally high ABI, indicative of vascular calcification, with cardiovascular risk factors or with prevalent CVD. High ABI is associated directly with male sex, diabetes mellitus, and hypertension but is inversely associated with smoking and hyperlipidemia. ABI more than 1.4 is also associated with higher C.V events and mortality (30).

2. ABI and coronary artery disease

The ABI has the advantage of being a noninvasive, inexpensive measurement and could be used as an indirect measure of CAD severity. It has been found out that there is a relation between ABI and the severity of CAD as reflected by SYNTAX score, Gensini score and number of diseased vessels. It has been found that there is a significant association between a history of myocardial infarction and a higher grade of SYNTAX score (31).

This result could be attributed to a higher rate of coronary artery occlusion, hence the higher SYNTAX score. It has been illustrated that there is a highly significant association between abnormal ABI and a high SYNTAX score. The ABI had a sensitivity of 54% and a specificity of 83% to predict high SYNTAX score (32, 33).

It has been reported to have a sensitivity of 34%, specificity of 87% of ABI< 0.9 in predicting coronary artery involvement. This means that ABI could serve as a prognostic marker for cardiovascular events (31).

Limitations of the ABI:

There are several limitations of ABI testing. First, the ABI is not sensitive to improvements in walking performance that occur in response to supervised exercise interventions. Although supervised treadmill exercise dramatically improves the 6-minute walk distance, the ABI value does not change concomitantly (34).

Second, as indicated above, the ABI may not be sensitive to progression of lower extremity atherosclerosis. Third, the ABI is not a useful measure of the presence or severity of PAD in people with medial calcinosis of the lower extremity arteries. Despite these limitations, the noninvasive and inexpensive nature of the ABI, along with its generally high sensitivity and specificity for PAD, makes it a highly useful clinical diagnostic and prognostic tool (34).

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