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AHA Scientific Statement

Measurement and Interpretation of the Ankle-Brachial Index

A Scientific Statement From the American Heart Association

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The ankle-brachial index (ABI) is the ratio of the systolic blood pressure (SBP) measured at the ankle to that measured at the brachial artery. Originally described by Winsor[1] in 1950, this index was initially proposed for the noninvasive diagnosis of lower-extremity peripheral artery disease (PAD).[2,3] Later, it was shown that the ABI is an indicator of atherosclerosis at other vascular sites and can serve as a prognostic marker for cardiovascular events and functional impairment, even in the absence of symptoms of PAD.[4–6]

Rationale for Standardization of the ABI

The current lack of standards for measurement and calculation of the ABI leads to discrepant results with significant impact from clinical, public health, and economic standpoints. Indeed, the estimated prevalence of PAD may vary substantially according to the mode of ABI calculation.[7–9] In a review of 100 randomly selected reports using the ABI, multiple variations in technique were identified, including the position of the patient during measurement, the sizes of the arm and leg cuffs, the location of the cuff on the extremity, the method of pulse detection over the brachial artery and at the ankles, whether the arm and ankle pressures were measured bilaterally, which ankle pulses were used, and whether a single or replicate measures were obtained.[10]

There is controversy about what ABI threshold should be used to diagnose PAD. The ABI threshold most commonly used is $\leq$0.90 based on studies reporting $>$90% sensitivity and specificity to detect PAD compared with angiography.[2,3] These studies were limited in that they included mostly older white men with PAD or who were at high risk for PAD and compared them with a younger healthy group. A recent meta-analysis of 8 studies of diverse populations, including diabetic patients, confirmed a high specificity but lower sensitivity (at best $<$80%) than that reported in earlier studies.[11]

Similar to other vascular markers such as carotid intima-media thickness[12] or coronary artery calcium score,[13] standardization of the techniques used to measure the ABI and the calculation and interpretation of its values is necessary.

Aims and Scope

The goals for this document are to provide a comprehensive review of the relevant literature on the measurement of the

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ABI, to provide recommendations for a standardized method to determine the ABI, to provide guidance on the interpretation of the ABI in the clinical setting, to propose standards for reporting ABI data in the scientific literature, and to delineate methodological issues requiring further research.

ABI Terminology

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[14] on the basis of its current widespread use in contemporary literature and accordingly is used throughout this document.

Physiology of the ABI

Why Is 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.[15] Several lines of evidence indicate that reflected waves occur at various sites in the vascular bed,[16,17] with some attenuation along the arterial system.[18,19] However, the reflected wave is not the sole explanation for the changes in pressure wave morphology.[18] In the legs, remodeling of vessel structure occurs, resulting from increased intraluminal pressure, characterized by increased wall thickening and unchanged inner radius.[20,21] 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.[22] Therefore, both reflected waves and changes in vessel wall thickness and consequently stiffness contribute to SBP amplification.

Physiological Conditions Affecting the ABI at Rest

Age, height, ethnicity, and even the order of measurement can affect the ABI. In 2 population studies, the ABI of the right leg was on average 0.03 higher than that of the left leg.[23,24] This observation may be due to the order of measurements (usually the right leg first) and the resulting temporal reduction in systemic pressure over time (white coat attenuation effect). An increased ABI may be expected with aging as a result of arterial stiffening. Cross-sectional and longitudinal population studies indicate that the ABI decreases with age, probably because of the increased prevalence and progression of PAD.[23,25]

It might be expected that taller people would have higher ABIs than shorter people as a consequence of the progressive SBP increase with greater distance from the heart. Indeed, in populations without clinical cardiovascular disease (CVD), there is a direct correlation between height and ABI.[24,26] In the Multi-Ethnic Study of Atherosclerosis (MESA), however, the adjusted contribution of height to ABI was negligible, <0.01 higher for every 20-cm height increase, after accounting for sex, ethnicity, and risk factors.[27]

Sex differences in ABI have been reported in many population studies.[23,26–29] Among participants without traditional CVD risk factors in the San Luis Valley Diabetes Study,[24] the average ABI was 0.07 less in women than in men. Adjustment for height reduces but does not eliminate observed differences.[24,27,30] After multivariate adjustments, ABI was 0.02 lower in women than men in a subset of MESA participants free of PAD and traditional risk factors for atherosclerosis.[27]

Black PAD-free participants in MESA had an ABI 0.02 unit lower than non-Hispanic white counterparts after multivariate adjustment,[27] consistent with a previous observation from the Atherosclerosis Risk in Communities Study (ARIC).[30] Ethnic differences are likely to result from genetic influences. Carmelli et al[31] measured the ABI of monozygotic and dizygotic pairs of elderly, white, male twins and estimated that 48% of the variability in ABI values could be attributed to genetic factors. European ancestry was associated with lower odds for PAD (ABI ≤ 0.90) than among Hispanic and black participants in MESA.[32]

An inverse relationship between the ABI and heart rate has been reported in subjects without heart disease[33,34] and in subjects referred to a vascular laboratory.[35] In 1 study,[34] an increased difference between peripheral and central SBP was observed during cardiac pacing as heart rate increased from 60 to 110 bpm. With increasing heart rate, the ratio of brachial to central pressure rose by 0.012 unit for every 10 bpm, whereas the amplification index (the difference between the first and second peaks of the central arterial waveform) decreased. This was attributed to the ejection duration reduction, which causes a shift of the reflected wave into diastole associated with an increasing heart rate. In MESA, a population-based study, heart rate did not correlate with the ABI.[27]

Because the ABI is a ratio, it is in theory not affected by factors that raise or lower blood pressure. For example, changes in blood volume after hemodialysis do not alter the ABI, despite significant removal of fluid and reduction in blood pressure.[36]

Overall, all these factors that affect the ABI at an individual level are minor but may be relevant in large population studies, especially when the epidemiology of PAD is being studied.

ABI in Clinical Practice

Background

ABI: A Diagnostic Method for Lower-Extremity PAD

ABI Versus Angiography and Other Imaging Methods

Compared with a variety of imaging methods to determine the presence of PAD, the diagnostic performance of the ABI varies according to the population studied, the cutoff threshold, and the technique used to detect flow in the ankle arteries. Table I in the online-only Data Supplement summarizes these disparities and provides diagnostic performances.[2,3,28,37–55] The sensitivity and specificity of the ABI with the Doppler technique range from 0.17 to 1.0 and from 0.80 to 1.0, respectively. Lower sensitivities (0.53–0.70) are reported

Table 1. The Diagnostic Performances of the Ankle-Brachial Index Versus Other Methods: Receiver-Operating Characteristic Curve Analysis

Authors, Year	Population Study	Gold Standard	Method for ABI Measurement	Area Under the Curve
Lijmer et al,[38] 1996	441 Patients (PAD suspicion)	Angiography limited to 53 patients	Doppler	Entire limb ≥50% stenosis: 0.95 (0.02)
		Criteria: ≥50% or occlusion	(Higher ankle artery pressure/ higher brachial pressure)	Occlusion: 0.80 (0.05) Aortoiliac ≥50% stenosis: 0.69 (0.05)
				Occlusion: 0.83 (0.05)
				Femoral-popliteal ≥50% stenosis and occlusion: 0.77 (0.04)
				Infrapopliteal ≥50% stenosis: 0.59 (0.06)
				Occlusion: 0.57 (0.07)
Parameswaran et al,[42] 2005	57 Type 2 diabetics with no clinical evidence of PAD	Doppler waveform analysis	Doppler (PT or DP if PT absent/high)	0.88 (0.80–0.96)
Guo et al,[50] 2008	298 Patients (cardiology), PAD in 7%	Angiography: 50% stenosis	Oscillometry	0.93 (0.87–0.96)
Clairotte et al,[48] 2009	146 Patients (296 limbs), vascular laboratory (diabetes group, 83)	Color duplex	Doppler and oscillometry	Doppler: 0.87 Oscillometric: 0.81 (P=0.006)

ABI indicates ankle-brachial index; PAD, peripheral artery disease; PT, posterior tibial; and DP, dorsalis pedis.

in diabetic patients.[43,47,48] The sensitivities and specificities of the ABI measured with oscillometric methods vary from 0.29 to 0.93 and from 0.96 to 0.98, respectively. The overall diagnostic ability may be provided by the receiver-operating characteristic (ROC) curves. The reported areas under the ROC curve are higher for ABI measured by Doppler (0.87–0.95) than that measured with the oscillometric method (0.80–0.93; Table 1).[38,42,48,50] Studies used to determine the accuracy of the ABI generally included severe cases of PAD in which arterial imaging was performed after initial ABI measurements were found to be abnormal. To avoid verification bias, Lijmer et al[38] estimated the corrected area under the curve of the Doppler ABI to diagnose >50% angiographic stenosis as very satisfactory (0.95±0.02). Diagnostic performance was higher for detecting proximal compared with distal lesions. Using the plethysmographic method to detect flow, 1 study[49] reported a specificity of 0.99 but a sensitivity of 0.39, and only about half the participants in that study had isolated occlusive disease of the posterior tibial (PT) artery.

Data on the optimal ABI threshold for the diagnosis of PAD are scarce, with different criteria having been used to determine the optimal ABI cutoff value (Table 2).[28,38,40,45,48,50,56,57] In older studies, the lower limit of the 95% confidence interval (CI)

Table 2. Studies Assessing Optimal Ankle-Brachial Index Cutoff for the Diagnosis of Peripheral Artery Disease

Authors, Year	Study Population	Method for Determination of Optimal ABI	Optimal ABI Cutoff Proposed
Carter,[56] 1969	Inpatients: 202 diseased limbs, 86 control subjects	95% Confidence limit for limbs without PAD	0.97
Sumner and Strandness,[45] 1979	48 Control subjects	Normal minus 2 SD (1.08±0.08)	0.92
Bernstein et al,[57] 1982	Patients with angiographically significant PAD	95% Confidence limit for limbs without PAD	0.85
Ouriel et al,[40] 1982	218 PAD patients (56 limbs not tested, 247 limbs with claudication, 58 with rest pain, ulcers, or gangrene), 25 control subjects (<30 y old, no RF, triphasic Doppler waveforms)	ROC curve analysis	0.97
Stoffers et al,[28] 1996	Community and vascular laboratory	ROC curve analysis	0.97 (If pretest probability 33%) 0.92 (If pretest probability 50%)
Lijmer et al,[38] 1996	441 Inpatients (PAD suspicion)	ROC curve analysis	0.98 (Corrected)
Guo et al,[50] 2008	298 Inpatients, cardiology PAD prevalence (angiography): 7%	ROC curve analysis	0.95
Clairotte et al,[48] 2009	146 Patients (296 limbs) undergoing color duplex (diabetes group, 83), PAD prevalence: 33% non–diabetes mellitus, 27% diabetes mellitus	ROC curve analysis	1.00 (1.04 in the absence of diabetes mellitus)

ABI indicates ankle-brachial index; PAD, peripheral artery disease; RF, radiofrequency; and ROC, receiver-operating characteristic.

ranged from 0.85 to 0.97. Subsequent studies using the ROC curve recommended a threshold value of either 0.97 or 0.92.[41,45,56] Clairotte et al[48] reported a cutoff value between 1.00 and 1.04 for people with and without diabetes mellitus, with slightly higher values recommended for the oscillometric method than the Doppler technique. Serial ABI measurements can influence the optimal threshold value for detecting PAD. In a study based on ROC curve analysis, Stoffers et al[28] proposed a cutoff value of 0.97 for a single measurement and of 0.92 for 3 measurements. They argued that the optimal cutoff might be influenced by population characteristics and disease prevalence.[28] From a bayesian perspective, the optimal cutoff for identifying PAD patients depends on the pretest probability of PAD. The pretest probability is based on multiple clinical parameters, including the presence, characteristics, and intensity of symptoms; the presence of CVD risk factors; and other information derived from the medical history and physical examination. Although an ABI ≤0.90 remains the most common and consensual threshold, this value should not be considered a binary marker for the diagnosis of PAD. Eight studies assessed the diagnostic performances of an ABI ≤0.90 (Doppler method) to detect >50% stenosis identified by imaging methods, including color duplex ultrasound,[37,43,44,46] magnetic resonance angiography,[34] or angiography (Table I in the online-only Data Supplement).[38,39,50] All these studies found reasonably high specificity (83%–99%) but lower sensitivity (69%–79%, except 1 outlier[51] reporting 20% sensitivity). With an ABI ≤1.0 used as a threshold for detecting PAD, sensitivities as high as 100% have been reported.[2,52] Yet, ABI should be interpreted according to the a priori probability of PAD, and values between 0.91 and 1.00 should be considered borderline. For example, for a 47-year-old woman with atypical calf pain, no history of CVD or risk factors, and an ABI of 0.91, the probability of PAD is low; however, the probability of PAD is high for a man with classic intermittent claudication who smokes and whose ABI is 0.96. Thus, clinical judgment is important when interpreting the ABI results. The sensitivity of the ABI can be significantly increased when it is measured immediately after treadmill exercise.

Postexercise ABI

With leg exercise, systolic pressure increases in the central circulation, as measured in the arms, concordant with an increase in left ventricular systolic pressure. Peripheral vasoconstriction occurs in nonexercising limbs and other organs, whereas it decreases at the ankle owing to vasodilation in exercising muscle. This leads to a mild decrease in the ABI in healthy patients when measured immediately after exercise cessation.[41,58] The ankle pressure then increases rapidly and reaches the pre-exercise values within 1 to 2 minutes.[58,59] In the case of even moderate occlusive PAD (typically in the proximal vessels), the ankle pressure decreases more during treadmill exercise compared with healthy patients, and the recovery time to the pre-exercise value after exercise cessation is prolonged, proportional to the severity of PAD.[40,58–60] The ABI recovery time also is affected by the duration of exercise.[61] Ouriel et al[40] reported an average ABI decrease of 5% from resting to postexercise values after treadmill exercise in healthy people compared with 20% in patients with PAD. A recovery of at least 90% of the ABI to baseline value within the first 3 minutes after exercise was found to have a specificity of 94% to rule out PAD. Compared with angiography, the ROC curves of ABI at rest and after exercise were comparable for the detection of PAD.[40] Augmentation of the ankle-brachial pressure gradient after exercise improves the sensitivity of the ABI to detect PAD, especially for borderline ABI values (0.91–1.00). Laing and Greenhalgh[60] proposed an absolute decline of 30-mm Hg ankle pressure for the diagnosis of PAD according to the 95% CI of the change in ankle pressure change after 1 minute of treadmill exercise in a study of healthy subjects. Others[62] reported 33% sensitivity and 85% specificity for a postexercise ABI <0.90- and/or >30-mm Hg drop in ankle pressure after exercise. Diagnostic criteria for postexercise ABI should also take into account the reproducibility of this measurement (see below). A challenge for establishing diagnostic criteria for the postexercise ABI is the heterogeneity of exercise protocols. Although treadmill testing requires specific equipment, an alternative method, the active pedal plantar flexion technique, has been proposed for an office-based assessment of postexercise ABI.[63,64] This technique consists of repetitive active plantar flexion (heel raising) while standing, with an excellent correlation between ABI obtained after this method compared with treadmill exercise in claudicants.[63,64]

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.[65,66] 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%.[65,66]

ABI and Monitoring Patients With PAD

ABI as a Marker of PAD Progression. The natural history of PAD includes a decrease in the ABI over time. In a series of patients assessed in a vascular laboratory,[67] the ABI decreased by a mean of 0.06 over 4.6 years. A smaller ABI change (0.025 decrease over 5 years) was reported in the general population.[23] Nicoloff et al[68] defined PAD progression as a decrease in ABI of >0.15, a condition observed at 3 and 5 years in 19% and 37% of their vascular laboratory

patients, respectively. Among patients with intermittent claudication followed up for a mean period of 2.5 years, Cronenwett et al[69] found no correlation between baseline ABI and clinical outcome of the limb, whereas an ABI decrease of at least 0.15 was associated with an increased risk for bypass interventions (2.5-fold) and symptom progression (1.8-fold). In the absence of revascularization, an ABI decrease is correlated with clinical deterioration. Clinical improvement in terms of an increased walking distance, however, is not correlated with an ABI increase.[70]

The level of ABI (and the corresponding ankle pressure) is useful to predict limb outcomes. An ankle pressure <50 mm Hg is associated with higher risk for amputation.[71] An increased risk of amputation has been reported when the ABI is <0.50 in nonrevascularized patients with leg ulcers.[72] An ABI ≤0.90 is strongly associated (odds ratio: 8.2) with a 7-year risk of amputation in people with diabetes mellitus.[73] Several studies reported greater accuracy of the ankle pressure per se, rather than the ABI, to predict the clinical prognosis of the limb.[41,74–76]

From a clinical perspective, 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.

ABI and Monitoring Patients After Revascularization. The ABI change correlates poorly with improvement in symptoms or functional performance. After angioplasty, an ABI increase of 0.10 and 0.15 in the revascularized limb predicted no residual stenosis >50% with sensitivities of 79% and 67% and specificities of 92% and 100%, respectively.[77] The ABI may continue to improve from that measured in the immediate postoperative period for several weeks or months after revascularization.[3,78–80] The accuracy of the ABI in predicting revascularization failure is poor, as shown in Table II in the online-only Data Supplement,[77,81–87] because the ABI is a global estimator of whole-limb perfusion and cannot distinguish between graft failure and progression of PAD in native arteries. The ABI is not site specific and may reflect changes elsewhere in the arterial tree. Considering its low sensitivity for predicting graft failure, the measurement of the ABI alone is not a reliable method of surveillance after revascularization.

The ABI and Functional Impairment and Decline

Compared with individuals without PAD, those with PAD have poorer walking endurance, slower walking velocity, and lower physical activity levels.[88–91] A thorough medical history is an important means for assessing the degree of functional impairment in men and women with PAD. However, some PAD patients restrict their physical activity to avoid exertional leg symptoms[88]; therefore, patient report of symptoms cannot be construed as a reliable measure of the degree of functional limitation.[92] Several studies have demonstrated that in cohorts including men and women with and without PAD, lower ABI values are associated with greater functional impairment or faster functional decline compared with higher ABI values.[5,89,90,92] The Walking and Leg Circulation Study (WALCS) cohort further demonstrated that even individuals with borderline baseline ABI values (0.91–0.99) and those with low-normal ABI values (1.00–1.09) had significantly higher rates of mobility loss than participants with a baseline ABI of 1.10 to 1.30.[5]

The association of lower ABI values with greater functional impairment in cohorts restricted to men and women with PAD is less consistent. Several studies that included only PAD participants reported that lower ABI values are not associated with greater functional limitations.[93–95] These prior studies were limited by small sample sizes, by exclusion of functional measures other than treadmill walking performance, and by exclusion of participants without classic symptoms of intermittent claudication.[93–95] In other studies of patients with PAD, both with and without intermittent claudication symptoms, strong and independent associations of lower ABI values were observed with poorer 6-minute walk performance, slower walking velocity at usual and fastest pace, greater limitation in maximum treadmill walking performance, and lower Walking Impairment Questionnaire distance score.[90,96,97] No prospective studies in cohorts restricted to patients with PAD have demonstrated that lower ABI values are associated with a faster decline in functioning. However, it is important to point out that characteristics contributing to functional impairment and decline in people with PAD are multifactorial and include muscle size and composition, inflammation, lower-extremity strength, mitochondrial function, and behavioral factors.[98–102] Therefore, the ABI is just one of many characteristics associated with functional impairment and decline in patients with PAD.

ABI: A Marker for CVD Risk and Events

ABI: A Marker of Cardiovascular Risk and Atherosclerosis

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. 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).[30,103–105] The majority of studies use an ABI of 0.90 as a threshold to define PAD and use Doppler for ABI measurement. Therefore, it is not known whether the strength of the associations between low ABI and cardiovascular risk factors differs with alternative measurement methods and thresholds of ABI. Some studies have shown a graded inverse association of CVD risk factors across ABI thresholds.[103,106]

A strong and consistent relationship between low ABI and prevalent coronary artery disease and cerebrovascular disease has been demonstrated in several population-based cohort studies that included individuals with existing CVD.[29,103,104,107,108] The strength of the relationship between low ABI and coronary artery disease varies, depending on the underlying risk of the population studied. In most studies, odds ratios range from 1.4 to 3.0, with 1 study reporting the association to be as high as 9.3 in individuals with type 1 diabetes mellitus.[103,109–111] The prevalence of coronary artery disease among PAD patients ranges from 10.5% to 71% compared with 5.3% to 45.4% among subjects without PAD. Low ABI is also associated with prevalent cerebrovascular disease, with odds ratios in the range of 1.3 to 4.2 among 9 studies.[29,104,111–113] The majority of these studies use Doppler to measure ABI and 0.90 as a threshold for defining PAD. Whether the association of low ABI with prevalent CVD would differ with alternative measurement methods or definitions is unknown.

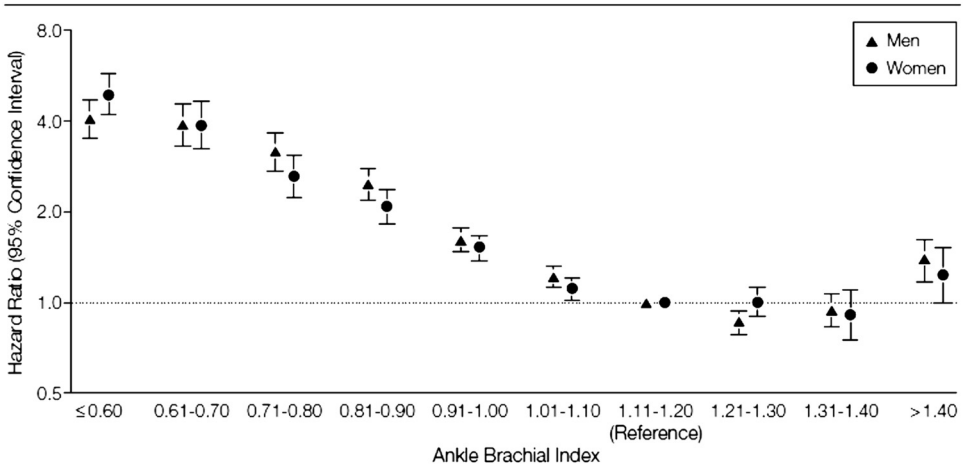

Figure 1. Hazard ratios for total mortality in men and women by ankle-brachial index at baseline for all studies combined in the ABI Collaboration. Reproduced from Fowkes et al[6] with permission from the publisher. Copyright © 2008, American Medical Association.

There is little information to determine whether the associations of abnormal ABI and CVD differ by sex. In the ARIC study,[29] the association of low ABI and coronary artery disease was strong in both men and women, but there was no association of low ABI with stroke in women despite a strong association reported in men. In a Spanish study, low ABI was associated with coronary artery disease in both men (odds ratio, 2.1) and women (odds ratio, 3.3).[114]

Association of High ABI With Cardiovascular Risk Factors and Prevalent Disease. Few studies have evaluated the 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.[66,115] Allison et al[115] demonstrated an ABI >1.40 to be associated with stroke and congestive heart failure but not with myocardial infarction or angina. In MESA, high ABI was associated with incident CVD.[116] Other studies have reported inconsistent results.[117–119]

ABI and Risk of Future Cardiovascular Events

The ABI is a measure of the severity of atherosclerosis in the legs but is also an independent indicator of the risk of subsequent atherothrombotic events elsewhere in the vascular system. The ABI may be used as a risk marker both in the general population free of clinical CVD and in patients with established CVD.

In the general population, cardiovascular risk equations incorporating traditional risk factors such as age, sex, cigarette smoking, hypercholesterolemia, hypertension, and diabetes mellitus have been used to predict future risk of events.[120] These predictive scores, however, have limited accuracy,[121] leading to the evaluation of other risk predictors such as C-reactive protein[122] or measures of subclinical atherosclerosis such as coronary artery calcium,[123] used alone or in combination with traditional risk factors. More precise identification of high-risk individuals may permit appropriate targeting of aggressive risk reduction therapies, although this strategy has not been properly evaluated.

The ABI has been investigated as a risk predictor in several population-based cohort studies, mostly in Europe[124–127] and North America.[106,107,128–130] These studies have consistently found that a low ABI is associated with an increased risk of myocardial infarction, stroke, and both total and cardiovascular-related mortality. Furthermore, the increased risks are independent of established CVD and risk factors at baseline, suggesting that the ABI, as an indicator of atherosclerosis, might enhance the accuracy of risk prediction with established scoring systems.[6]

The ABI Collaboration performed an individual-based meta-analysis of 16 population cohorts to investigate in a large data set whether the ABI provided information on the risk of cardiovascular events and mortality independent of the Framingham Risk Score (FRS) and might improve risk prediction when combined with the FRS.[6] An ABI ≤0.90 was associated with approximately twice the age-adjusted 10-year total mortality, cardiovascular mortality, and major coronary event rate compared with the overall rate in each FRS category. Use of the ABI resulted in reclassification of the risk category in both men and women.[6] In men, the greatest incremental benefit of ABI for predicting risk was in those with an FRS >20%; a normal ABI, found in 43% of cases, reclassified them to the intermediate-risk category. Conversely, 9% of women at low (<10%) or intermediate (10%–19%) risk estimated by the FRS presented abnormal ABI (<0.90 or >1.40) and were reclassified as high risk. Since this meta-analysis, a recent report from MESA presented consistent data in different ethnic groups in the United States.[116] Thus, a low or high ABI is associated with increased cardiovascular risk, and the risk prediction extends beyond that of the FRS alone.[6,116] Further work is warranted to refine these results and to establish whether the ABI is of more value in certain subgroups in the population. Additional analyses are encouraged to use several recent metrics assessing the improvement of CVD risk prediction with the ABI. Specifically, criteria such as discrimination, calibration, and net reclassification improvement are awaited.

Although an ABI cut point of 0.90 is used in many studies to identify high-risk individuals, the ABI Collaboration confirmed that the risk increases as the ABI decreases below a threshold of 1.10 (Figure 1).[6] Clinical risk prediction could

conceivably benefit from using ABI categories rather than 1 cut point for high risk. Individuals with a high ABI >1.40 are also at increased risk. Thus, the graph of mortality or other cardiovascular outcome by ABI level is a reverse J-shaped curve in which the lowest level of risk (normal) is from 1.11 to 1.40 (Figure 1).[6] One explanation for an increased risk associated with a high ABI is that a high ABI caused by calcified arteries is associated frequently with occlusive PAD.[131]

Patients with established CVD who also have a low ABI are at higher risk compared with patients with CVD who have a normal ABI.[132–134] This is consistent with the observation that in patients with evidence of disease in >1 vascular bed, the 3-year vascular event rate is >60% higher than in those with disease in only 1 vascular territory.[135] The magnitude of the increased risk associated with a low ABI would appear to be slightly less for those with known CVD than the 2- to 3-fold increased relative risk in healthy individuals. In the Heart Outcomes Prevention Evaluation (HOPE) study of patients with coronary heart disease, stroke, or diabetes mellitus, ABIs in the range of 0.60 to 0.90 were associated with a risk ratio for future nonfatal myocardial infarction of 1.4, nonfatal stroke of 1.2, and cardiovascular mortality of 1.6 compared with higher ABIs.[135] In patients with prior CVD, the Cardiovascular Health Study found that those with a low ABI of ≤0.90 had an increased risk of congestive heart failure (risk ratio, 1.3) and cardiovascular mortality (risk ratio, 1.5).[107] These increased risks were independent of established cardiovascular risk factors. Furthermore, in patients with PAD, not only is a low ABI associated independently with an increased risk of cardiovascular morbidity and mortality, but a decrease in ABI of >0.15 over time is associated with a 2-fold increase in mortality independently of the absolute ABI level.[136] Thus, risk of vascular events in cardiovascular patients with a low or declining ABI is higher than in those with a normal ABI.

The postexercise ABI is also predictive of risk. In the case of a normal ABI at rest, the presence of an abnormal ABI after exercise is associated with increased mortality.[137]

The Use of ABI in Primary Care

As one of the least expensive and most available markers of atherosclerosis, the ABI is a highly appropriate measurement for CVD risk assessment in primary care. In the PAD Awareness, Risk, and Treatment: New Resources for Survival (PARTNERS) study, several barriers to the use of the ABI in the primary care, including time constraints, reimbursement, staff availability, and staff training, were identified.[138] Yet, in this study, the time needed for ABI measurement was <15 minutes.[138] In a Dutch study, which included 955 general practices, the time needed for an ABI measurement varied between 12 and 20 minutes (average, 17 minutes).[139] The lack of reimbursement for ABI measurement is a hurdle for its broader use in general practice. The standardized ABI measurement proposed in this document has very good test characteristics for the diagnosis of PAD and should be considered for appropriate reimbursement.

Conditions for the Measurement of the ABI

The Patient

Body position and knee or hip flexion influence the ABI.[140] Gornik et al[141] showed that arm pressure is not different in the sitting and supine positions when the arm is kept at heart level. These positions affect ankle pressure because the ankle is lower than the heart in the seated but not in the supine position, and consequently, the pressure is higher. The ABI averages 0.35 higher in the seated than in the supine position. Therefore, patients should be lying flat for an accurate ABI measurement, with the head and heels fully supported, ie, not hanging over the end of the examination table. Gornik et al[141] recommended a formula to correct the seated ABI (under standardized conditions) in patients who cannot lie down. However, no external validation of this formula is available.

The effect of the duration of the rest period on the reliability of ABI measurement is unknown. The length of the rest period before performing the ABI measurement has varied among studies,[10] with most studies using a 5- to 10-minute period. Longer delays are impractical in the clinical setting. Even after a resting period, the first limb measurement tends to provide higher systolic pressures during a sequential (limb by limb) measurement. Smoking cigarettes also may affect the ABI. Smoking 10 minutes before the measurement significantly decreases the ABI (−0.09) compared with the ABI measured after 12 hours of smoking abstinence.[142] The effect on the ABI was specifically related to a decrease in ankle pressures without a corresponding change in brachial artery pressure.[142]

The Cuff

Studies of brachial blood pressure measurement highlight the importance of an appropriate cuff size to avoid inaccurate measurements.[143,144] Comparable information is not available on the size of the ankle cuff. If the same concept of cuff size used for the arm is applied to that of the ankle, the width of the cuff should be at least 40% of the limb circumference.[144] The cuff should always be clean and dry. The cuff wrapping method (spiral or parallel) affects the ankle SBP, with lower values occurring with the spiral cuff wrapping method.[145] In a comparative study, similar intraobserver reproducibility was observed between both wrapping methods when an automated cuff was used, but a slightly better intraobserver reproducibility was observed for the spiral wrap when a manual cuff inflation was used with the Doppler technique.[145] Takahashi et al[146] found good correlation of parallel and spiral wrapping with intra-arterial pressure, similar intraobserver variability with both wrapping methods, but better interobserver variability with parallel wrapping. Given these data and the fact that the straight method is used to assess arm blood pressure, parallel wrapping is also preferred for the ankles.

Although the measurement of the ABI by a pressure cuff is noninvasive, safe, and well tolerated in most circumstances, cuff inflation should be interrupted if it is painful. Caution is advised in 2 clinical situations. Direct apposition of the ankle cuff over open wounds and ulcers should be avoided or prevented by an impermeable dressing. In addition, cuff inflation should be avoided over a recently placed bypass graft because of the potential risk of causing graft thrombosis.

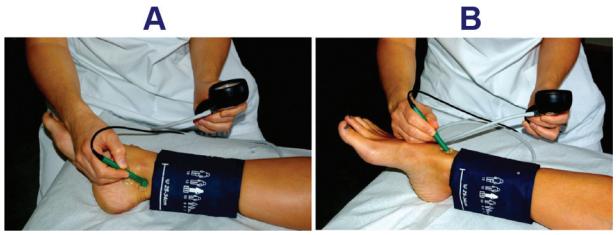

Figure 2. Ankle pressure measurement with a Doppler probe: posterior tibial (**A**) and dorsalis pedis (**B**) arteries.

The Measurement of the ABI

Methods of Pressure Measurement

Several noninvasive techniques are used to detect limb flow or pulse volume for measuring the ABI, primarily Doppler ultrasound and oscillometric methods. The former uses a continuous-wave Doppler probe for detection of arterial flow (Figure 2). The SBP is determined with a pneumatic cuff, which is first inflated until flow ceases and then deflated slowly until there is reappearance of the flow signal. The corresponding cuff pressure is the SBP. The oscillometric technique is based on the assumptions that the maximum oscillations appearing during cuff deflation correspond to the mean arterial pressure and that SBP and diastolic blood pressure can be calculated from this pressure with mathematical algorithms. These algorithms, based on empirical data from healthy subjects, were originally developed to measure arm blood pressure. The validation studies for oscillometric methods[48,145,147–174] are summarized in Table III in the online-only Data Supplement. Some studies, but not others, have questioned the validity of the oscillometric method for the detection of PAD.[145,155,175–177] The correlation between Doppler-derived and oscillometry-determined ankle pressures and ABIs in healthy subjects or subjects with mild PAD has been acceptable in most studies[151,152,155,156,162,178] with 1 exception.[164] However, when the ABI determined by the Doppler method is in the low range, the oscillometric method results in an overestimation of the actual pressure value,[148,155,156,160,161,165,179] as illustrated in Figure 3.[156] In addition, most oscillometric blood pressure devices are unable to detect low pressures, eg, <50 mm Hg[148] or even 80 mm Hg,[178] and as a consequence, recording failures are frequent (from 11%[161] to 44%[178]) in patients with advanced PAD.[153,158,160,161,178,179] The sensitivity (67%–97%) and specificity (62%–96%) of the ABI measured with oscillometry compared with the Doppler method have been reported in multiple studies (Table III in the online-only Data Supplement).[48,145,147–174] Bland-Altman plots were used in several studies to assess the agreement between the Doppler and oscillometric techniques.[48,147,149,152–155,162,164,176,178] The limits of agreement (±2 SD) for the ABI were 0.25[149] and 0.23[158] in 2 studies in which it was calculated. In a third study, the limit of agreement of the ankle pressure in non-PAD subjects was ±20 mm Hg but more than ±70 mm Hg in patients with PAD.[155] The 95% CI of the difference between the 2 methods in 2 additional studies varied from −0.19 to 0.14[164] and −0.18 to 0.35,[176] respectively.

Other methods used to measure ABI include plethysmography,[180] photoplethysmography,[169,173,174] auscultation,[146] and pulse palpation.[147,171] Strain-gauge plethysmography is not suitable for use in most settings other than a vascular laboratory. The photoplethysmography method, in which a sensor is placed on the great toe to detect flow after cuff deflation, correlated well with Doppler in several series of patients with PAD.[169,173,174] However, the reproducibility of this method has not been reported. In 1 series, the limits of agreement (±2 SD) for the differences compared with the Doppler method ranged from −0.23 to 0.24.[169] In addition, photoplethysmography of the toe is affected by temperature. A cool environment causes digital vasoconstriction. A laser Doppler probe placed on the dorsum of the foot to detect flow was used for ABI measurements in 1 study.[170] The mean difference compared with Doppler was negligible, but agreement and reproducibility were not reported.

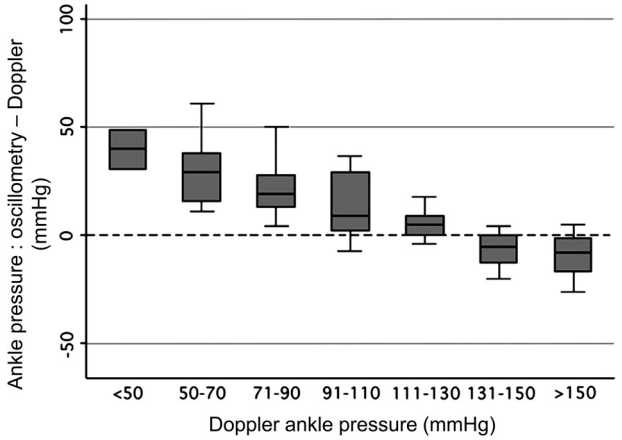

Figure 3. Difference between ankle pressures measured with an oscillometric device (CASMED 740) and Doppler (*y* axis) according to the ankle pressure bands obtained with Doppler (*x* axis). In the box plot, the line indicates median percentiles and outer markers indicate 5% and 95% percentiles. Reprinted from Korno et al[156] with permission from the publisher. © Copyright 2009, Elsevier.

Measurement of ABI using auscultation with a stethoscope was assessed in a Japanese study.[146] Korotkoff sounds, however, are not always audible in the ankles (inaudible in ≈40% of cases), and there is an unacceptable difference in ankle pressures determined by this method compared with Doppler (−15.2 mm Hg). Compared with Doppler, pulse palpation to measure the ABI has a sensitivity of 88% and a specificity ranging from 75% to 82%.[147,171] The palpation method underestimates (−0.14) the ABI compared with the Doppler method.[147]

Are the Different Methods of ABI Measurement Similarly Reproducible?

Several studies assessed the intraobserver and interobserver reproducibilities of the ABI, with mixed findings (Tables IV and V in the online-only Data Supplement).[40,147,156,162,165,181–196] Direct comparisons of studies are difficult because different statistical approaches were used or because of methodological limitations (eg, small samples of observers or patients, selective inclusion of symptomatic PAD patients).

The intraobserver coefficient of variation (CoV) of the ABI with the Doppler method varies widely in the literature, from 4.7%[189] to 13.0%[28] (on average, ≈10%). Overall, these

results are superior to those obtained with an automated oscillometric method, which has a CoV ranging from 5.1%[156] to 20.2%.[185] This general observation is confirmed by 2 comparative studies[147,184] but has been challenged recently by Richart et al,[165] who used a 4-cuff oscillometric device.

The palpation method has poor reproducibility (CoV, 23%).[147] Similarly, the intraobserver and interobserver reproducibilities are poorer for the auscultation than for the Doppler method.[197] No reproducibility data are available for the plethysmographic method.

The interobserver variability has been studied extensively for the Doppler method, but there are few data for other methods.[147,156,177,181,182,184,188,190–196,198] The interobserver variability of the oscillometric method has been assessed only in the ARIC study, showing a CoV of 11%.[184] All other studies (Table V in the online-only Data Supplement)[147,156,177,181,182,184,188,190–196] used the Doppler method, with CoVs varying from 5.4% to 24% (mean, 13%). The ABI measured by Doppler in all limbs showed significantly better reproducibility than the 2 alternative methods of using a stethoscope or an oscillometry for the arms.[194,195] Considering the evidence, Doppler appears to be the most reliable method to determine the ABI.

The Examiner's Experience

Several studies reported higher ABI reproducibility when measured by skilled examiners.[183,199] Endres et al[200] found no systematic bias between examiners from 3 distinct occupational groups with diverse training backgrounds, but all the examiners were well trained to measure the ABI. In patients with critical limb ischemia, comparison of ABIs obtained by inexperienced physicians and skilled vascular technicians revealed a higher interobserver difference for the former, especially when the dorsalis pedis (DP) artery was used.[75] The ABI is more reproducible in "nonexpert" hands for healthy people compared with patients with PAD.[188]

Overall Reliability and Reproducibility of ABI Measurement

The confidence of any particular point estimate of the "true" ABI depends on the number of measurements. Theoretically, the 95% CI is reduced by the square root of the number of measurements. As an illustration, in the ARIC ABI reliability study, the actual ABI value after 1 measurement could be the point estimate ±0.21.[190] Considering this, the CI for an ABI based on the average of 2 visits would be ±0.15; it would be ±0.12 if based on 3 measures.

For a given method of ABI measurement and calculation, Fowkes et al[175] reported several factors that contribute to the within-subject ABI variability, including the interactions among the subject, the subject's leg (right versus left), the observer, and the delay between measurements. However, the variability resulting from these interactions is considered trivial compared with the greater ABI variability between different subjects. The variability of ankle pressures was found to be similar to that of arm pressures in 3 reports,[2,181,189] whereas in 5 other studies,[157,166,167,196,201] a better reproducibility of the arm pressures was reported. Overall, data demonstrate that the ABI is a valid biological parameter.[181] Nevertheless, establishing the ABI method with the best reproducibility is warranted to keep the single measurement error to a minimum and to improve the ability of repeated ABI measurements over time to detect an actual change in PAD severity.

In addition to methodological aspects and variability of the measurements in different laboratories,[202] the CoV depends on the average ABI of the population studied (Figure I in the online-only Data Supplement), with a better ABI reproducibility in healthy people than in those with PAD. At an individual level, the size and direction of change between 2 ABI measurements do not vary with the average ABI.[190,193] However, Osmundson et al[194] and Fowkes et al[175] reported lower variability in healthy subjects compared with PAD patients. Additionally, in patients with critical limb ischemia, significantly higher interobserver variability occurs in those with an ABI <0.50 than in those with an ABI >0.50.[75]

Data on postexercise ABI variability are scarce. In 20 patients with intermittent claudication, the interobserver variability for the ABI at rest and after exercise was 10% and 21%, respectively.[196] Similarly, the intraobserver variability was higher for the ABI measured after exercise than for that measured at rest.[40]

The specific steps for an adequate measurement of the ABI are summarized in Table 3.

Recommendations for the Measurement of the ABI

1. **The Doppler method should be used to measure the SBP in each arm and each ankle for the determination of the ABI (*Class I; Level of Evidence A*).[38,42,48,50,147,156,165,181–189]**
2. **The cuff size should be appropriate with a width at least 40% of the limb circumference (*Class I; Level of Evidence B*).[143,144]**
3. **The ankle cuff should be placed just above the malleoli with the straight wrapping method (*Class I; Level of Evidence B*).[146]**
4. **Any open lesion with the potential for contamination should be covered with an impermeable dressing (*Class I; Level of Evidence C*).**
5. **The use of the cuff over a distal bypass should be avoided (risk of bypass thrombosis) (*Class III harm; Level of Evidence C*).**

Standard Calculation of the ABI

The Denominator (Arm)

The highest SBP of that measured in each arm is used most often as the denominator, although some studies report the average SBP of both arms, except in cases of interarm blood pressure differences. Differences in SBP between arms may occur in the case of subclavian artery stenosis. Osborn et al[201] reported 100% sensitivity and specificity to detect >50% subclavian stenosis when the interarm blood pressure difference exceeded 15 mm Hg. Thus, subclavian artery stenosis should be suspected when the SBP difference between both arms is ≥15 mm Hg. In an analysis of 3 cohorts derived from the general population or from patients visiting a vascular laboratory, the presence of subclavian artery stenosis was associated with an increased risk of mortality,[203] and several studies found a significant association between high interarm

Table 3. Limb Pressure Measurement Protocol for the Determination of the Ankle-Brachial Index With the Doppler Method

The patient should be at rest 5 to 10 min in the supine position, relaxed, head and heels supported, in a room with comfortable temperature (19°C–22°C/66°F–72°F).
The patient should not smoke at least 2 hours before the ABI measurement.
The cuff should be chosen adequately according to the limb size. The width should contour at least 40% of the limb circumference.
The cuff should not be applied over a distal bypass (risk of thrombosis) or over ulcers. Any open lesion posing potential contamination should be covered with an impermeable dressing.
The patient should stay still during the pressure measurement. If the patient is unable to not move his/her limbs (eg, tremor), other methods should be considered.
Similar to the brachial blood pressure measurement, the cuff should be placed around the ankle using the straight wrapping method. The lower edge of the cuff should be 2 cm above the superior aspect of the medial malleolus (Figure 2).
An 8- to 10-MHz Doppler probe should be used. Doppler gel should be applied over the sensor.
After the Doppler device is turned on, the probe should be placed in the area of the pulse at a 45° to 60° angle to the surface of the skin. The probe should be moved around until the clearest signal is heard.
The cuff should be inflated progressively up to 20 mm Hg above the level of flow signal disappearance and then deflated slowly to detect the pressure level of flow signal reappearance. The maximum inflation is 300 mm Hg; if the flow is still detected, the cuff should be deflated rapidly to avoid pain.
The detection of the brachial blood flow during the arm pressure measurement should also be done by Doppler.
The same sequence of limb pressure measurements should be used. The sequence should be the same for clinicians within a same center.
During the sequence of measurement, the first measurement should be repeated at the end of the sequence and both results averaged to temper the white coat effect of the first measurement, except if the difference between the 2 measurements of the first arm exceeds 10 mm Hg. In that case, the first measurement should be disregarded and only the second measurement should be considered. For example, when the counterclockwise sequence—right arm, right PT, right DP, left PT, left DP, left arm—is used, the measurement of the right arm should be repeated at the end of the sequence and both results obtained at the right arm should be averaged unless the difference between the 2 measurements of the right arm exceeds 10 mm Hg. In this case, only the second measurement of right arm pressure should be considered.
In case of repeat measurement of the 4 limb pressures (see indications in the text), the measurements should be repeated in the reverse order of the first series (eg, in the case of the initial counterclockwise sequence [right arm, right PT, right DP, left PT, left DP, left arm, right arm], the clockwise sequence should be used, starting and ending with the left arm).

ABI indicates ankle-brachial index; PT, posterior tibial; and DP, dorsalis pedis.

blood pressure difference and other cardiovascular conditions, including PAD.[179,204–207] Apparent differences also may be observed in an anxious patient (white coat effect) when the first measurement (usually the right arm) is higher than the last one (left arm). This issue justifies a second measurement of the SBP in the first arm measured. To minimize the risk of ABI overestimation by a falsely lower denominator, the higher SBP between both arms should be used systematically for the ABI denominator.

The Numerator (Ankle)

The numerator for the calculation of the ABI incorporates the SBP of the PT and/or the DP artery separately or the average of both. The intraobserver variability of the ABI is the lowest when the average pressures of PT and DP artery are used for the numerator, although the differences with other methods that take either the highest or the lowest pressure are trivial in direct comparisons.[178,183] No significant difference in interobserver variability was reported between the ABI obtained by the PT versus the DP artery.[75,195] The ABI reproducibility is affected more by the technique used to record pressure at the ankle than by which artery is used.[183,181,190,202]

The Effect of the Mode of Determination of the Ankle Pressure on the Ability of the ABI to Diagnose PAD. Two studies[39,44] assessed the performance of the ABI with 2 methods for determining the numerator, comparing the higher with the lower pressure between the PT and DP arteries at each ankle. In both studies, the higher brachial pressure was selected as the denominator, and the ABI cutoff value was 0.90. One study compared Doppler ABI <0.90 with the presence of $\geq 70\%$ stenosis detected by color duplex ultrasound.[44] The other study compared Doppler ABI ≤ 0.90 with angiographic stenosis $\geq 50\%$ of any lower-limb artery. Choosing the lower compared with the higher ankle pressure as the ABI numerator was associated with better sensitivity (0.89 versus 0.66 in the former and 0.83 versus 0.79 in the latter study).[39,44] Using the higher ankle pressure, however, resulted in higher specificity (0.99 versus 0.93 in the former and 0.93 versus 0.83 in the latter study, respectively).[39,44] Neither of these studies assessed the average of both pressures as the numerator; however, the average of the PT and DP would likely not change overall accuracy and would result in intermediate values for sensitivity and specificity. Of note, if arterial flow in the ankle is not detected, the reason is seldom arterial agenesis but is most likely related to arterial occlusion or technical difficulties in localizing the artery. When an ankle artery signal is absent and the ABI based on the other ankle artery is within the normal range, it is reasonable to perform other vascular tests (eg, duplex ultrasound) to determine whether PAD is present.

In calculations of the ABI to confirm a suspected diagnosis of PAD, use of the higher pressure at the ankle (high specificity) is preferred to minimize overdiagnosis in healthy subjects and thus to avoid further unnecessary tests and treatment. Although more false-negative tests will occur compared with using the lower ankle pressure, the clinical suspicion of PAD should lead to further investigation in such patients so that the diagnosis is unlikely to be missed.

The Effect of the Mode of Determination of the Ankle Pressure on the Association of PAD With Cardiovascular Risk Factors and Localization of Atherosclerosis. In MESA,[9] the association of PAD (ABI ≤ 0.90) with CVD risk factors was assessed with 3 alternative numerators: the higher, the average, and the lower of the PT and DP arteries. The use of the lower of the PT and DP arteries for the calculation led to the weakest association between PAD and cardiovascular risk factors and subclinical atherosclerosis in the coronary or carotid arteries. This is plausibly related to the inclusion of

participants with less burden of disease (perhaps affecting only 1 ankle artery) in the PAD group.

The Effect of the Mode of Determination of the Ankle Pressure on the Ability of the ABI to Predict Cardiovascular Events. In the population cohort studies that participated in the ABI Collaboration, the associations of ABI with total mortality, cardiovascular mortality, and major coronary events were consistent between studies despite some differences in ABI protocols.[6] For an ABI ≤0.90 compared with a reference ABI range of 1.11 to 1.40, the pooled hazard ratio for cardiovascular mortality in men was 4.2 (95% CI, 3.3–5.4) and in women was 3.5 (95% CI, 2.4–5.1). In approximately half of the studies, the ABI was determined with only 1 arm, only the PT, and the lower ABI of the 2 legs.

Direct comparisons of methods that measure the ABI for prediction of events are limited.[208,209] In 1 study, the ABI was measured in >800 patients undergoing coronary angiography who were then followed up for 6 years to detect myocardial infarction, stroke, and CVD death.[208] The prevalence of patients with an ABI <0.90 in either leg was 25% with the use of the higher of the PT and DP pressure compared with 36% with the use of the lower pressure. The cardiovascular event rate in subjects with an ABI <0.90 was almost identical with each mode of ABI calculation (28.1% and 27.4%, respectively). Thus, the lower of the PT and DP identified more patients at risk. A secondary analysis in the Cardiovascular Health Study assessed the prognostic value of the ABI to predict cardiovascular events.[209] Using the lower ABI of the 2 legs identified more individuals with an ABI below the traditional high-risk cut point of 0.90. There were, however, no significant differences in the relative risks of a cardiovascular event based on calculations using the lower or higher ABI. Thus, taking the lower ABI of both legs will identify more individuals at risk of cardiovascular events. This conclusion is not surprising given that PAD may be unilateral or more severe in 1 leg than another. When the higher ABI of the 2 legs is used, individuals with significant disease who are at high risk of cardiovascular events may be missed.

Recommendations for the Measurement of the Systolic Pressures of the 4 Limbs

1. **Each clinician should adopt the following sequence of limb pressure measurement for the ABI at rest: first arm, first PT artery, first DP artery, other PT artery, other DP artery, and other arm (*Class I; Level of Evidence C*).**
2. **After the measurement of systolic pressures of the 4 limbs, if the SBP of the first arm exceeds the SBP of the other arm by ≥10 mm Hg, the blood pressure of the first arm should be repeated, and the first measurement of the first arm should be disregarded (*Class I; Level of Evidence C*).**

In clinical practice, one should consider that reproducibility is crucial only when the ABI obtained after the first set of measurements is close to the threshold values. Taking into consideration the threshold ABI value of 0.90 for the diagnosis of PAD, with 95% CI of differences between 2 measurements reported as ±0.10, an ABI <0.80 is sufficient to detect PAD and an ABI >1.00 is high enough to rule it out, whereas repeat measurements are needed within the interval of 0.80 to 1.00 for a definitive diagnosis. Thus, repeated measurements are indicated if the initial ABI is between 0.80 and 1.00; a single ABI result <0.80 has a 95% positive predictive value for the diagnosis of PAD; and a single ABI >1.00 has a 99% negative predictive value for PAD.[28]

The Public Health Consequences of the Mode of Calculation of the ABI

ABI Mode of Calculation and the Epidemiology of PAD. Several studies have demonstrated that the mode of calculation of the ABI affects the estimation of PAD prevalence within a population.[7–9] In MESA, in which the lower pressure between PT and DP was used instead of the higher one for the ABI numerator, the prevalence of PAD was 3.95 times higher in women (14.6% instead of 3.7%) and 2.74 times higher in men (9.3% instead 3.4%).[9]

The ABI Mode of Calculation and the Prevention of CVDs. The ABI can be used to stratify the risk of individuals initially classified as intermediate risk on the basis of cardiovascular risk scores (eg, FRS). Subjects with an ABI ≤0.90 are considered at high risk of CVD events, primarily on the basis of using the higher of PT and DP pressures as the numerator or exclusively using the PT artery (Table 4).[4,24,89,104,107,109,124–130,190,210,212–215] Less is known about the prognostic value of the ABI in the general population if calculated using the lower of the PT and DP pressures. Although the use of this mode of calculation may slightly increase the sensitivity for identification of high-risk patients, the overall level of risk of those with an ABI ≤0.90 would be lower because of less specificity and the inclusion of numerous cases with early disease. The use of the lower of the PT and DP pressures may lead to the overdiagnosis of PAD, with important consequences in terms of resource use and cost.

The appropriate management of patients with an asymptomatic low ABI is still unclear. The Aspirin for Asymptomatic Atherosclerosis trial failed to show any benefit of the use of aspirin in patients with an ABI <0.95, with no trend to any benefit when the ABI was <0.90, although the ABI was calculated from the lowest of the 4 ankle arteries.[210] Using a technique that reduces specificity for PAD in a clinical trial may limit the ability to show efficacy of therapeutic interventions.

Recommendations for the Calculation of the ABI

1. **The ABI of each leg should be calculated by dividing the higher of the PT or DP pressure by the higher of the right or left arm SBP (*Class I; Level of Evidence A*).[39,44,189]**
2. **When ABI is used as a diagnostic tool to assess patients with symptoms of PAD, the ABI should be reported separately for each leg (*Class I; Level of Evidence C*).**
3. **When the ABI is used as a prognostic marker of cardiovascular events and mortality, the lower of the ABIs of the left and right leg should be used as the prognostic marker of cardiovascular events and mortality. The exception to this recommendation is the case of noncompressible arteries (*Class I; Level of Evidence C*).**

Table 4. Ankle-Brachial Index Modes of Calculation in the 16 Population Studies Included in the ABI Collaboration Study[210]

		Arm			Ankle Artery					Repeat Measures	
Study	Measurement Method	1 Measured	Higher L+R	Average L+R	1 Measured	Higher PT+DP	Average PT+DP	Lower PT+DP	Other	Higher	Average
Atherosclerosis Risk in Communities Study[184]	Oscillometry	√			√						√
Belgian Men study[128]	Doppler	√			√						
Cardiovascular Health Study[104,107]	Doppler	√			√						√
Edinburgh artery study[124]	Doppler	√			√						
Framingham Offspring Study[109]	Doppler		√		√						√
Health in Men study[212]	Doppler	√				√					
Honolulu study[129]	Doppler	√			√						√
Hoorn study[213]	Doppler		Not available								
InCHIANTI[214]	Doppler	√			√					√	
Limburg study[125]	Doppler		√		√						
Men Born in 1914[126]	Plethysmography		√		√						
Rotterdam Study[127]	Doppler	√			√						√*
San Diego study[4]	Plethysmography			√†							
San Luis Valley study[24]	Doppler			√†					√		
Strong Heart Study[130]	Doppler	√			√						√
Women's Health and Ageing[89]	Doppler	√			√					√	

*Average done only for arms.
†Except for large interarm difference (highest pressure taken in this case).

4. **For any situation, when the ABI is initially determined to be between 0.80 and 1.00, it is reasonable to repeat the measurement (*Class IIa; Level of Evidence B*).[28]**

Recommendations for the Use and Interpretation of the ABI in Case of Clinical Presentation of Lower-Extremity PAD

1. **In the case of clinical suspicion based on symptoms and clinical findings, the ABI should be used as the first-line noninvasive test for the diagnosis of PAD (*Class I; Level of Evidence A*).[11,38,41,50,56]**
2. **An ABI ≤0.90 should be considered the threshold for confirming the diagnosis of lower-extremity PAD (*Class I; Level of Evidence A*).[11,37–39,42–44,46,50,51]**
3. **When the ABI is >0.90 but there is clinical suspicion of PAD, postexercise ABI or other noninvasive tests, which may include imaging, should be used (*Class I; Level of Evidence A*).[40,58,60,212]**
4. **It is reasonable to consider a postexercise ankle pressure decrease of >30 mm Hg or a postexercise ABI decrease of >20% as a diagnostic criterion for PAD (*Class IIa; Level of Evidence A*).[40,60,62]**
5. **When the ABI is >1.40 but there is clinical suspicion of PAD, a toe-brachial index or other noninvasive tests, which may include imaging, should be used (*Class I; Level of Evidence A*).[65,66]**

Recommendations for the Interpretation of the ABI During Follow-Up

1. **An ABI decrease of >0.15 over time can be effective to detect significant PAD progression (*Class IIa; Level of Evidence B*).[68,69]**
2. **The ABI should not be used alone to follow revascularized patients (*Class III no benefit; Level of Evidence C*).**

Recommendations for the Interpretation of the ABI as a Marker of Subclinical CVD and Risk in Asymptomatic Individuals

1. **The ABI can be used to provide incremental information beyond standard risk scores in predicting future cardiovascular events (*Class IIA; Level of Evidence A*).[6,116]**
2. **Individuals with an ABI ≤0.90 or ≥1.40 should be considered at increased risk of cardiovascular events and mortality independently of the presence of symptoms of PAD and other cardiovascular risk factors (*Class I; Level of Evidence A*).[6,116]**
3. **Subjects with an ABI between 0.91 and 1.00 are considered "borderline" in terms of cardiovascular risk. Further evaluation is appropriate (*Class IIa; Level of Evidence A*).[6]**

Training for the Use of the ABI

The ABI should be performed by qualified individuals, including physicians, nurses, vascular technicians, and other allied health professionals. The amount of education and training required depends on prior knowledge and experience. Training should consist of both didactic and experiential learning. The individual performing the ABI should have basic knowledge of vascular anatomy, physiology, and the clinical presentation of PAD, as well as a basic understanding of how a Doppler device functions. Training should include demonstration of performance of an ABI with clear delineation of each step

and emphasis on correct technique. To become proficient in performance of the ABI, it is necessary to practice the ABI measurement over time to ensure comfort and competence with the equipment and the procedures. The trainee should be asked to correctly demonstrate the independent performance of each step of the ABI in both healthy individuals and those with PAD. Trainees should also be able to demonstrate reproducible results. Trainees should be able to demonstrate correct calculation of the ABI and interpretation of results with a clear understanding of normal and abnormal values.

Recommendations for ABI Measurement Training

1. **The measurement and interpretation of the ABI should be part of the standard curriculum for medical and nursing students (*Class I; Level of Evidence C*).**
2. **All health professionals who perform the ABI should have didactic and experiential learning under the supervision of a qualified and experienced health professional (*Class I; Level of Evidence C*).**
3. **Professionals using the ABI should be proficient in performing the technique as determined by quality control measures (*Class I; Level of Evidence C*).**

Standards to Report ABI in Scientific Papers

One of the aims of this scientific statement is to recommend uniform methods of ABI measurement in research. Controversial results reported in the literature are related in part to discrepancies in the ABI method (see "ABI Mode of Calculation and the Epidemiology of PAD").

The results of studies using the ABI need to be translated into clinical practice. Consequently, most of the recommendations on the clinical use of the ABI apply also to research protocols. However, time constraints for performing a comprehensive ABI should not apply to research protocols. A comprehensive ABI calculation for research protocols includes measurement of SBP in all 4 limbs, including both the PT and DP arteries at each ankle. Given that the reproducibility and accuracy of ABI values are augmented with repeat measurements, it is reasonable to require systematically at least 2 sets of ABI measurements with averaging of the measurements in research studies. This is especially true when the ABI is used as the sole method to determine PAD (as in most epidemiological studies) or when repeated measurements are planned over time. In these situations, duplicate ABI measurements provide increased accuracy and limit measurement bias. In addition, the reduced CI enables the detection of individual ABI changes of a smaller magnitude. It is suggested that ABI results in research reports include intraobserver and interobserver variation measured in a subset of the study population or in a population similar to the one assessed in the study. The prevalence of incompressible arteries or absent flow signals also should be reported. Finally, to compare more appropriately the population between different reports, it is suitable to report also the population's absolute pressure values in arms and legs.

Recommendations for the Use of the ABI in Scientific Reports

1. **The ABI intraobserver and interobserver variability of the research team should be reported (*Class I; Level of Evidence C*).**
2. **To improve the precision of the test, it is reasonable to measure each limb pressure twice and to average the results of each artery to calculate the ABI (*Class IIa; Level of Evidence C*).**

Unmet Needs: Fields of Research for the Future

The following issues have been identified as gaps for evidence on the use and interpretation of the ABI:

- Although several studies report differences in the normal values of ABI according to sex and ethnicity, it is still unclear whether specific thresholds should be used in different sex and ethnic groups in both population studies and clinical practice and research.
- Further research should explore potentially easier and faster alternative methods for ABI measurement that would likely be implemented more broadly in primary care.
- Standards of accreditation are necessary for the ABI measurement devices using methods other than Doppler devices (eg, oscillometric methods).
- Further research to identify the optimal method of ABI calculation for predicting cardiovascular events and mobility loss is encouraged.

A major aim of this document is to provide evidence-based recommendations for ABI measurement. However, separate but related ABI issues need to be addressed in future research. Two examples are in whom the ABI should be measured and how often the ABI should be measured.

The current recommendations for the target population for ABI screening in American Heart Association/American College of Cardiology guidelines[215] reflect the criteria used by investigators in the PARTNERS[108] and the German Epidemiological Trial on Ankle-Brachial Index (getABI)[216] studies, and the American Diabetes Association has suggested minor modifications of these criteria for diabetic patients.[217] However, these recommendations are based on observational epidemiology. Ideally, the criteria would be established by a randomized, clinical trial, but such a trial seems unlikely in the near future. An attractive alternative is a cost-effectiveness analysis in different population subgroups; several such analyses are currently under way.

How often the ABI should be repeated is also unknown. On average, the ABI decreases with age as PAD incidence increases. Some evidence exists on the rates of ABI progression in clinical populations[25,67,68] and in the general population.[23,218] However, there is little evidence on the cost-effectiveness of repeat measurement of the ABI in different patient groups, and with increasing use of the ABI, this will become an important question.

Disclosures

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This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (a) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (b) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

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*Modest.

†Significant.

References

1. Winsor T. Influence of arterial disease on the systolic blood pressure gradients of the extremity. *Am J Med Sci*. 1950;220:117–126.
2. Carter SA. Indirect systolic pressures and pulse waves in arterial occlusive diseases of the lower extremities. *Circulation*. 1968;37:624–637.
3. Yao ST, Hobbs JT, Irvine WT. Ankle systolic pressure measurements in arterial disease affecting the lower extremities. *Br J Surg*. 1969;56:676–679.
4. Criqui MH, Langer RD, Fronek A, Feigelson HS, Klauber MR, McCann TJ, Browner D. Mortality over a period of 10 years in patients with peripheral arterial disease. *N Engl J Med*. 1992;326:381–386.
5. McDermott MM, Guralnik JM, Tian L, Liu K, Ferrucci L, Liao Y, Sharma L, Criqui MH. Associations of borderline and low normal ankle-brachial index values with functional decline at 5-year follow-up: the WALCS (Walking and Leg Circulation Study). *J Am Coll Cardiol*. 2009;53:1056–1062.
6. Ankle Brachial Index Collaboration, Fowkes FG, Murray GD, Butcher I, Heald CL, Lee RJ, Chambless LE, Folsom AR, Hirsch AT, Dramaix M, deBacker G, Wautrecht JC, Kornitzer M, Newman AB, Cushman M, Sutton-Tyrrell K, Lee AJ, Price JF, d'Agostino RB, Murabito JM, Norman PE, Jamrozik K, Curb JD, Masaki KH, Rodriguez BL, Dekker JM, Bouter LM, Heine RJ, Nijpels G, Stehouwer CD, Ferrucci L, McDermott MM, Stoffers HE, Hooi JD, Knottnerus JA, Ogren M, Hedblad B, Witteman JC, Breteler MM, Hunink MG, Hofman A, Criqui MH, Langer RD, Fronek A, Hiatt WR, Hamman R, Resnick HE, Guralnik J. Ankle brachial index combined with Framingham risk score to predict cardiovascular events and mortality: a meta-analysis. *JAMA*. 2008;300:197–208.
7. Lange SF, Trampisch HJ, Pittrow D, Darius H, Mahn M, Allenberg JR, Tepohl G, Haberl RL, Diehm C; getABI Study Group. Profound influence of different methods for determination of the ankle brachial index on the prevalence estimate of peripheral arterial disease. *BMC Public Health*. 2007:147.
8. Aboyans V, Lacroix P, Preux PM, Vergnenegre A, Ferrieres J, Laskar M. Variability of ankle-arm index in general population according to its mode of calculation. *Int Angiol*. 2002;21:237–243.
9. Allison MA, Aboyans V, Granston T, McDermott MM, Kamineni A, Ni H, Criqui MH. The relevance of different methods of calculating the ankle-brachial index: the Multi-Ethnic Study of Atherosclerosis. *Am J Epidemiol*. 2010;171:368–376.
10. Klein S, Hage JJ. Measurement, calculation, and normal range of the ankle-arm index: a bibliometric analysis and recommendation for standardization. *Ann Vasc Surg*. 2006;20:282–292.
11. Dachun X, Jue L, Liling Z, Yawei X, Dayi H, Pagoto SL, Yunsheng M. Sensitivity and specificity of the ankle-brachial index to diagnose peripheral artery disease: a structured review. *Vasc Med*. 2010;15:361–369.
12. Stein JH, Korcarz CE, Hurst RT, Lonn E, Kendall CB, Mohler ER, Najjar SS, Rembold CM, Post WS; American Society of Echocardiography Carotid Intima-Media Thickness Task Force. Use of carotid ultrasound to identify subclinical vascular disease and evaluate cardiovascular disease risk: a consensus statement from the American Society of Echocardiography Carotid Intima-Media Thickness Task Force: endorsed by the Society for Vascular Medicine. *J Am Soc Echocardiogr*. 2008;21:93–111; quiz 189–190.
13. Greenland P, Bonow RO, Brundage BH, Budoff MJ, Eisenberg MJ, Grundy SM, Lauer MS, Post WS, Raggi P, Redberg RF, Rodgers GP, Shaw LJ, Taylor AJ, Weintraub WS. ACCF/AHA 2007 clinical expert consensus document on coronary artery calcium scoring by computed tomography in global cardiovascular risk assessment and in evaluation of patients with chest pain: a report of the American College of Cardiology Foundation Clinical Expert Consensus Task Force (ACCF/AHA Writing Committee to Update the 2000 Expert Consensus Document on Electron Beam Computed Tomography) developed in collaboration with the Society of Atherosclerosis Imaging and Prevention and the Society of Cardiovascular Computed Tomography. *Circulation*. 2007;115:402–426.
14. Hiatt WR, Goldstone J, Smith SC Jr, McDermott M, Moneta G, Oka R, Newman AB, Pearce WH; American Heart Association Writing Group 1. Atherosclerotic peripheral vascular disease symposium II: nomenclature for vascular diseases. *Circulation*. 2008;118:2826–2829.
15. Safar ME, Protogerou AD, Blacher J. Statins, central blood pressure, and blood pressure amplification. *Circulation*. 2009;119:9–12.
16. Murgo JP, Westerhof N, Giolma JP, Altobelli SA. Aortic input impedance in normal man: relationship to pressure wave forms. *Circulation*. 1980;62:105–116.
17. Latham RD, Westerhof N, Sipkema P, Rubal BJ, Reuderink P, Murgo JP. Regional wave travel and reflections along the human aorta: a study with six simultaneous micromanometric pressures. *Circulation*. 1985;72:1257–1269.
18. Hope SA, Tay DB, Meredith IT, Cameron JD. Waveform dispersion, not reflection, may be the major determinant of aortic pressure wave morphology. *Am J Physiol Heart Circ Physiol*. 2005;289:H2497–H2502.
19. Wang JJ, Parker KH. Wave propagation in a model of the arterial circulation. *J Biomech*. 2004;37:457–470.
20. Tsamis A, Stergiopulos N. Arterial remodeling in response to hypertension using a constituent-based model. *Am J Physiol Heart Circ Physiol*. 2007;293:H3130–H3139.
21. Humphrey JD. Mechanisms of arterial remodeling in hypertension: coupled roles of wall shear and intramural stress. *Hypertension*. 2008;52:195–200.
22. Katz S, Globerman A, Avitzour M, Dolfin T. The ankle-brachial index in normal neonates and infants is significantly lower than in older children and adults. *J Pediatr Surg*. 1997;32:269–271.
23. Smith FB, Lee AJ, Price JF, van Wijk MC, Fowkes FG. Changes in ankle brachial index in symptomatic and asymptomatic subjects in the general population. *J Vasc Surg*. 2003;38:1323–1330.
24. Hiatt WR, Hoag S, Hamman RF. Effect of diagnostic criteria on the prevalence of peripheral arterial disease: the San Luis Valley Diabetes Study. *Circulation*. 1995;91:1472–1479.
25. Bird CE, Criqui MH, Fronek A, Denenberg JO, Klauber MR, Langer RD. Quantitative and qualitative progression of peripheral arterial disease by non-invasive testing. *Vasc Med*. 1999;4:15–21.
26. London GM, Guerin AP, Pannier B, Marchais SJ, Stimpel M. Influence of sex on arterial hemodynamics and blood pressure: role of body height. *Hypertension*. 1995;26:514–519.
27. Aboyans V, Criqui MH, McClelland RL, Allison MA, McDermott MM, Goff DC Jr, Manolio TA. Intrinsic contribution of gender and ethnicity to normal ankle-brachial index values: the Multi-Ethnic Study of Atherosclerosis (MESA). *J Vasc Surg*. 2007;45:319–327.
28. Stoffers HE, Kester AD, Kaiser V, Rinkens PE, Kitslaar PJ, Knottnerus JA. The diagnostic value of the measurement of the ankle-brachial

systolic pressure index in primary health care. *J Clin Epidemiol*. 1996; 49:1401–1405.
29. Zheng ZJ, Sharrett AR, Chambless LE, Rosamond WD, Nieto FJ, Sheps DS, Dobs A, Evans GW, Heiss G. Associations of ankle-brachial index with clinical coronary heart disease, stroke and preclinical carotid and popliteal atherosclerosis: the Atherosclerosis Risk in Communities (ARIC) Study. *Atherosclerosis*. 1997;131:115–125.
30. Zheng ZJ, Rosamond WD, Chambless LE, Nieto FJ, Barnes RW, Hutchinson RG, Tyroler HA, Heiss G; ARIC Investigators. Lower extremity arterial disease assessed by ankle-brachial index in a middle-aged population of African Americans and whites: the Atherosclerosis Risk in Communities (ARIC) Study. *Am J Prev Med*. 2005; 29(suppl 1):42–49.
31. Carmelli D, Fabsitz RR, Swan GE, Reed T, Miller B, Wolf PA. Contribution of genetic and environmental influences to ankle-brachial blood pressure index in the NHLBI Twin Study: National Heart, Lung, and Blood Institute. *Am J Epidemiol*. 2000;151:452–458.
32. Allison MA, Peralta CA, Wassel CL, Aboyans V, Arnett DK, Cushman M, Eng J, Ix J, Rich SS, Criqui MH. Genetic ancestry and lower extremity peripheral artery disease in the Multi-Ethnic Study of Atherosclerosis. *Vasc Med*. 2010;15:351–359.
33. Su HM, Lee KT, Chu CS, Lee MY, Lin TH, Voon WC, Sheu SH, Lai WT. Effects of heart rate on brachial-ankle pulse wave velocity and ankle-brachial pressure index in patients without significant organic heart disease. *Angiology*. 2007;58:67–74.
34. Wilkinson IB, MacCallum H, Flint L, Cockcroft JR, Newby DE, Webb DJ. The influence of heart rate on augmentation index and central arterial pressure in humans. *J Physiol*. 2000;525(pt 1):263–270.
35. Abraham P, Desvaux B, Colin D, Leftheriotis G, Saumet JL. Heart rate-corrected ankle-to-arm index in the diagnosis of moderate lower extremity arterial disease. *Angiology*. 1995;46:673–677.
36. Su HM, Chang JM, Lin FH, Chen SC, Voon WC, Cheng KH, Wang CS, Lin TH, Lai WT, Sheu SH. Influence of different measurement time points on brachial-ankle pulse wave velocity and ankle-brachial index in hemodialysis patients. *Hypertens Res*. 2007;30:965–970.
37. Allen J, Oates CP, Henderson J, Jago J, Whittingham TA, Chamberlain J, Jones NA, Murray A. Comparison of lower limb arterial assessments using color-duplex ultrasound and ankle/brachial pressure index measurements. *Angiology*. 1996;47:225–232.
38. Lijmer JG, Hunink MG, van den Dungen JJ, Loonstra J, Smit AJ. ROC analysis of noninvasive tests for peripheral arterial disease. *Ultrasound Med Biol*. 1996;22:391–398.
39. Niazi K, Khan TH, Easley KA. Diagnostic utility of the two methods of ankle brachial index in the detection of peripheral arterial disease of lower extremities. *Catheter Cardiovasc Interv*. 2006;68:788–792.
40. Ouriel K, McDonnell AE, Metz CE, Zarins CK. Critical evaluation of stress testing in the diagnosis of peripheral vascular disease. *Surgery*. 1982;91:686–693.
41. Ouriel K, Zarins CK. Doppler ankle pressure: an evaluation of three methods of expression. *Arch Surg*. 1982;117:1297–1300.
42. Parameswaran GI, Brand K, Dolan J. Pulse oximetry as a potential screening tool for lower extremity arterial disease in asymptomatic patients with diabetes mellitus. *Arch Intern Med*. 2005;165:442–446.
43. Premalatha G, Ravikumar R, Sanjay R, Deepa R, Mohan V. Comparison of colour duplex ultrasound and ankle-brachial pressure index measurements in peripheral vascular disease in type 2 diabetic patients with foot infections. *J Assoc Physicians India*. 2002;50:1240–1244.
44. Schroder F, Diehm N, Kareem S, Ames M, Pira A, Zwettler U, Lawall H, Diehm C. A modified calculation of ankle-brachial pressure index is far more sensitive in the detection of peripheral arterial disease. *J Vasc Surg*. 2006;44:531–536.
45. Sumner DS, Strandness DE Jr. The relationship between calf blood flow and ankle blood pressure in patients with intermittent claudication. *Surgery*. 1969;65:763–771.
46. Williams DT, Harding KG, Price P. An evaluation of the efficacy of methods used in screening for lower-limb arterial disease in diabetes. *Diabetes Care*. 2005;28:2206–2210.
47. Alnaeb ME, Crabtree VP, Boutin A, Mikhailidis DP, Seifalian AM, Hamilton G. Prospective assessment of lower-extremity peripheral arterial disease in diabetic patients using a novel automated optical device. *Angiology*. 2007;58:579–585.
48. Clairotte C, Retout S, Potier L, Roussel R, Escoubet B. Automated ankle-brachial pressure index measurement by clinical staff for peripheral arterial disease diagnosis in nondiabetic and diabetic patients. *Diabetes Care*. 2009;32:1231–1236.
49. Feigelson HS, Criqui MH, Fronek A, Langer RD, Molgaard CA. Screening for peripheral arterial disease: the sensitivity, specificity, and predictive value of noninvasive tests in a defined population. *Am J Epidemiol*. 1994;140:526–534.
50. Guo X, Li J, Pang W, Zhao M, Luo Y, Sun Y, Hu D. Sensitivity and specificity of ankle-brachial index for detecting angiographic stenosis of peripheral arteries. *Circ J*. 2008;72:605–610.
51. Wikstrom J, Hansen T, Johansson L, Lind L, Ahlstrom H. Ankle brachial index <0.9 underestimates the prevalence of peripheral artery occlusive disease assessed with whole-body magnetic resonance angiography in the elderly. *Acta Radiol*. 2008;49:143–149.
52. Baxter GM, Polak JF. Lower limb colour flow imaging: a comparison with ankle:brachial measurements and angiography. *Clin Radiol*. 1993; 47:91–95.
53. de Groote P, Millaire A, Deklunder G, Marache P, Decoulx E, Ducloux G. Comparative diagnostic value of ankle-to-brachial index and transcutaneous oxygen tension at rest and after exercise in patients with intermittent claudication. *Angiology*. 1995;46:115–122.
54. Flanigan DP, Ballard JL, Robinson D, Galliano M, Blecker G, Harward TR. Duplex ultrasound of the superficial femoral artery is a better screening tool than ankle-brachial index to identify at risk patients with lower extremity atherosclerosis. *J Vasc Surg*. 2008;47:789–792.
55. Alnaeb ME, Boutin A, Crabtree VP, Mikhailidis DP, Seifalian AM, Hamilton G. Assessment of lower extremity peripheral arterial disease using a novel automated optical device. *Vasc Endovascular Surg*. 2007; 41:522–527.
56. Carter SA. Clinical measurement of systolic pressures in limbs with arterial occlusive disease. *JAMA*. 1969;207:1869–1874.
57. Bernstein EF, Fronek A. Current status of noninvasive tests in the diagnosis of peripheral arterial disease. *Surg Clin North Am*. 1982;62: 473–487.
58. Carter SA. Response of ankle systolic pressure to leg exercise in mild or questionable arterial disease. *N Engl J Med*. 1972;287:578–582.
59. Winsor T. Conditioned vasoconstrictive responses of digital vessels. *AMA Arch Surg*. 1958;76:193–199.
60. Laing S, Greenhalgh RM. The detection and progression of asymptomatic peripheral arterial disease. *Br J Surg*. 1983;70:628–630.
61. Sakurai T, Matsushita M, Nishikimi N, Nimura Y. Effect of walking distance on the change in ankle-brachial pressure index in patients with intermittent claudication. *Eur J Vasc Endovasc Surg*. 1997;13:486–490.
62. Hoogeveen EK, Mackaay AJ, Beks PJ, Kostense PJ, Dekker JM, Heine RJ, Nijpels G, Rauwerda JA, Stehouwer CD. Evaluation of the one-minute exercise test to detect peripheral arterial disease. *Eur J Clin Invest*. 2008;38:290–295.
63. McPhail IR, Spittel PC, Weston SA, Bailey KR. Intermittent claudication: an objective office-based assessment. *J Am Coll Cardiol*. 2001; 37:1381–1385.
64. Amirhamzeh MM, Chant HJ, Rees JL, Powel RJ, Campbell WB. A comparative study of treadmill tests and heel raising exercise for peripheral arterial disease. *Eur J Vasc Endovasc Surg*. 1997;13:301–305.
65. Suominen V, Rantanen T, Venermo M, Saarinen J, Salenius J. Prevalence and risk factors of PAD among patients with elevated ABI. *Eur J Vasc Endovasc Surg*. 2008;35:709–714.
66. Aboyans V, Ho E, Denenberg JO, Ho LA, Natarajan L, Criqui MH. The association between elevated ankle systolic pressures and peripheral occlusive arterial disease in diabetic and nondiabetic subjects. *J Vasc Surg*. 2008;48:1197–1203.
67. Aboyans V, Criqui MH, Denenberg JO, Knoke JD, Ridker PM, Fronek A. Risk factors for progression of peripheral arterial disease in large and small vessels. *Circulation*. 2006;113:2623–2629.
68. Nicoloff AD, Taylor LM Jr, Sexton GJ, Schuff RA, Edwards JM, Yeager RA, Landry GJ, Moneta GL, Porter JM; Homocysteine and Progression of Atherosclerosis Study Investigators. Relationship between site of initial symptoms and subsequent progression of disease in a prospective study of atherosclerosis progression in patients receiving long-term treatment for symptomatic peripheral arterial disease. *J Vasc Surg*. 2002;35:38–46.
69. Cronenwett JL, Warner KG, Zelenock GB, Whitehouse WM Jr, Graham LM, Lindenauer M, Stanley JC. Intermittent claudication: current results of nonoperative management. *Arch Surg*. 1984;119:430–436.
70. Amighi J, Sabeti S, Schlager O, Francesconi M, Ahmadi R, Minar E, Schillinger M. Outcome of conservative therapy of patients with severe intermittent claudication. *Eur J Vasc Endovasc Surg*. 2004;27:254–258.
71. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG; TASC II Working Group. Inter-society consensus for the man-

agement of peripheral arterial disease (TASC II). *J Vasc Surg*. 2007; 45(suppl S):S5–S67.
72. Marston WA, Davies SW, Armstrong B, Farber MA, Mendes RC, Fulton JJ, Keagy BA. Natural history of limbs with arterial insufficiency and chronic ulceration treated without revascularization. *J Vasc Surg*. 2006;44:108–114.
73. Hamalainen H, Ronnemaa T, Halonen JP, Toikka T. Factors predicting lower extremity amputations in patients with type 1 or type 2 diabetes mellitus: a population-based 7-year follow-up study. *J Intern Med*. 1999;246:97–103.
74. Brothers TE, Esteban R, Robison JG, Elliott BM. Symptoms of chronic arterial insufficiency correlate with absolute ankle pressure better than with ankle:brachial index. *Minerva Cardioangiol*. 2000;48:103–109.
75. Matzke S, Ollgren J, Lepantalo M. Predictive value of distal pressure measurements in critical leg ischaemia. *Ann Chir Gynaecol*. 1996;85: 316–321.
76. Fowl RJ, Gewirtz RJ, Love MC, Kempczinski RF. Natural history of claudicants with critical hemodynamic indices. *Ann Vasc Surg*. 1992;6: 31–33.
77. Decrinis M, Doder S, Stark G, Pilger E. A prospective evaluation of sensitivity and specificity of the ankle/brachial index in the follow-up of superficial femoral artery occlusions treated by angioplasty. *Clin Investig*. 1994;72:592–597.
78. Motukuru V, Suresh KR, Vivekanand V, Raj S, Girija KR. Therapeutic angiogenesis in Buerger's disease (thromboangiitis obliterans) patients with critical limb ischemia by autologous transplantation of bone marrow mononuclear cells. *J Vasc Surg*. 2008;48(suppl):53S–60S.
79. Allouche-Cometto L, Leger P, Rousseau H, Lefebvre D, Bendayan P, Elefterion P, Boccalon H. Comparative of blood flow to the ankle-brachial index after iliac angioplasty. *Int Angiol*. 1999;18:154–157.
80. Matoba S, Tatsumi T, Murohara T, Imaizumi T, Katsuda Y, Ito M, Saito Y, Uemura S, Suzuki H, Fukumoto S, Yamamoto Y, Onodera R, Teramukai S, Fukushima M, Matsubara H; TACT Follow-Up Study Investigators. Long-term clinical outcome after intramuscular implantation of bone marrow mononuclear cells (Therapeutic Angiogenesis by Cell Transplantation [TACT] trial) in patients with chronic limb ischemia. *Am Heart J*. 2008;156:1010–1018.
81. Barnes RW, Thompson BW, MacDonald CM, Nix ML, Lambeth A, Nix AD, Johnson DW, Wallace BH. Serial noninvasive studies do not herald postoperative failure of femoropopliteal or femorotibial bypass grafts. *Ann Surg*. 1989;210:486–493.
82. Stierli P, Aeberhard P, Livers M. The role of colour flow duplex screening in infra-inguinal vein grafts. *Eur J Vasc Surg*. 1992;6: 293–298.
83. Laborde AL, Synn AY, Worsey MJ, Bower TR, Hoballah JJ, Sharp WJ, Kresowik TF, Corson JD. A prospective comparison of ankle/brachial indices and color duplex imaging in surveillance of the in situ saphenous vein bypass. *J Cardiovasc Surg (Torino)*. 1992;33:420–425.
84. Idu MM, Blankenstein JD, de Gier P, Truyen E, Buth J. Impact of a color-flow duplex surveillance program on infrainguinal vein graft patency: a five-year experience. *J Vasc Surg*. 1993;17:42–52.
85. Dalsing MC, Cikrit DF, Lalka SG, Sawchuk AP, Schulz C. Femorodistal vein grafts: the utility of graft surveillance criteria. *J Vasc Surg*. 1995; 21:127–134.
86. Lundell A, Lindblad B, Bergqvist D, Hansen F. Femoropopliteal-crural graft patency is improved by an intensive surveillance program: a prospective randomized study. *J Vasc Surg*. 1995;21:26–33.
87. Radak D, Labs KH, Jager KA, Bojic M, Popovic AD. Doppler-based diagnosis of restenosis after femoropopliteal percutaneous transluminal angioplasty: sensitivity and specificity of the ankle/brachial pressure index versus changes in absolute pressure values. *Angiology*. 1999;50: 111–122.
88. McDermott MM, Greenland P, Liu K, Guralnik JM, Criqui MH, Dolan NC, Chan C, Celic L, Pearce WH, Schneider JR, Sharma L, Clark E, Gibson D, Martin GJ. Leg symptoms in peripheral arterial disease: associated clinical characteristics and functional impairment. *JAMA*. 2001;286:1599–1606.
89. McDermott MM, Fried L, Simonsick E, Ling S, Guralnik JM. Asymptomatic peripheral arterial disease is independently associated with impaired lower extremity functioning: the Women's Health and Aging Study. *Circulation*. 2000;101:1007–1012.
90. McDermott MM, Greenland P, Liu K, Guralnik JM, Celic L, Criqui MH, Chan C, Martin GJ, Schneider J, Pearce WH, Taylor LM, Clark E. The ankle brachial index is associated with leg function and physical activity: the Walking and Leg Circulation Study. *Ann Intern Med*. 2002;136: 873–883.
91. McDermott MM, Ohlmiller SM, Liu K, Guralnik JM, Martin GJ, Pearce WH, Greenland P. Gait alterations associated with walking impairment in people with peripheral arterial disease with and without intermittent claudication. *J Am Geriatr Soc*. 2001;49:747–754.
92. McDermott MM, Liu K, Greenland P, Guralnik JM, Criqui MH, Chan C, Pearce WH, Schneider JR, Ferrucci L, Celic L, Taylor LM, Vonesh E, Martin GJ, Clark E. Functional decline in peripheral arterial disease: associations with the ankle brachial index and leg symptoms. *JAMA*. 2004;292:453–461.
93. Szuba A, Oka RK, Harada R, Cooke JP. Limb hemodynamics are not predictive of functional capacity in patients with PAD. *Vasc Med*. 2006;11:155–163.
94. Gardner AW, Skinner JS, Cantwell BW, Smith LK. Prediction of claudication pain from clinical measurements obtained at rest. *Med Sci Sports Exerc*. 1992;24:163–170.
95. Parr B, Noakes TD, Derman EW. Factors predicting walking intolerance in patients with peripheral arterial disease and intermittent claudication. *S Afr Med J*. 2008;98:958–962.
96. McDermott MM, Criqui MH, Liu K, Guralnik JM, Greenland P, Martin GJ, Pearce W. Lower ankle/brachial index, as calculated by averaging the dorsalis pedis and posterior tibial arterial pressures, and association with leg functioning in peripheral arterial disease. *J Vasc Surg*. 2000; 32:1164–1171.
97. McDermott MM, Ferrucci L, Guralnik JM, Dyer AR, Liu K, Pearce WH, Clark E, Liao Y, Criqui MH. The ankle-brachial index is associated with the magnitude of impaired walking endurance among men and women with peripheral arterial disease. *Vasc Med*. 2010;15:251–257.
98. McDermott MM, Liu K, Ferrucci L, Tian L, Guralnik JM, Green D, Tan J, Liao Y, Pearce WH, Schneider JR, McCue K, Ridker P, Rifai N, Criqui MH. Circulating blood markers and functional impairment in peripheral arterial disease. *J Am Geriatr Soc*. 2008;56:1504–1510.
99. Herman SD, Liu K, Tian L, Guralnik JM, Ferrucci L, Criqui MH, Liao Y, McDermott MM. Baseline lower extremity strength and subsequent decline in functional performance at 6-year follow-up in persons with lower extremity peripheral arterial disease. *J Am Geriatr Soc*. 2009;57: 2246–2252.
100. Anderson JD, Epstein FH, Meyer CH, Hagspiel KD, Wang H, Berr SS, Harthun NL, Weltman A, Dimaria JM, West AM, Kramer CM. Multifactorial determinants of functional capacity in peripheral arterial disease: uncoupling of calf muscle perfusion and metabolism. *J Am Coll Cardiol*. 2009;54:628–635.
101. McDermott MM, Liu K, Ferrucci L, Tian L, Guralnik JM, Liao Y, Criqui MH. Greater sedentary hours and slower walking speed outside the home predict faster declines in functioning and adverse calf muscle changes in peripheral arterial disease. *J Am Coll Cardiol*. 2011;57: 2356–2364.
102. McDermott MM, Liu K, Ferrucci L, Criqui MH, Greenland P, Guralnik JM, Tian L, Schneider JR, Pearce WH, Tan J, Martin GJ. Physical performance in peripheral arterial disease: a slower rate of decline in patients who walk more. *Ann Intern Med*. 2006;144:10–20.
103. Selvin E, Erlinger TP. Prevalence of and risk factors for peripheral arterial disease in the United States: results from the National Health and Nutrition Examination Survey, 1999–2000. *Circulation*. 2004;110: 738–743.
104. Newman AB, Siscovick DS, Manolio TA, Polak J, Fried LP, Borhani NO, Wolfson SK. Ankle-arm index as a marker of atherosclerosis in the Cardiovascular Health Study: Cardiovascular Heart Study (CHS) Collaborative Research Group. *Circulation*. 1993;88:837–845.
105. Allison MA, Criqui MH, McClelland RL, Scott JM, McDermott MM, Liu K, Folsom AR, Bertoni AG, Sharrett AR, Homma S, Kori S. The effect of novel cardiovascular risk factors on the ethnic-specific odds for peripheral arterial disease in the Multi-Ethnic Study of Atherosclerosis (MESA). *J Am Coll Cardiol*. 2006;48:1190–1197.
106. Weatherley BD, Nelson JJ, Heiss G, Chambless LE, Sharrett AR, Nieto FJ, Folsom AR, Rosamond WD. The association of the ankle-brachial index with incident coronary heart disease: the Atherosclerosis Risk in Communities (ARIC) study, 1987–2001. *BMC Cardiovasc Disord*. 2007:3.
107. Newman AB, Shemanski L, Manolio TA, Cushman M, Mittelmark M, Polak JF, Powe NR, Siscovick D. Ankle-arm index as a predictor of cardiovascular disease and mortality in the Cardiovascular Health Study: the Cardiovascular Health Study Group. *Arterioscler Thromb Vasc Biol*. 1999;19:538–545.

108. Hirsch AT, Criqui MH, Treat-Jacobson D, Regensteiner JG, Creager MA, Olin JW, Krook SH, Hunninghake DB, Comerota AJ, Walsh ME, McDermott MM, Hiatt WR. Peripheral arterial disease detection, awareness, and treatment in primary care. *JAMA*. 2001;286:1317–1324.
109. Murabito JM, Evans JC, Nieto K, Larson MG, Levy D, Wilson PW. Prevalence and clinical correlates of peripheral arterial disease in the Framingham Offspring Study. *Am Heart J*. 2002;143:961–965.
110. Zander E, Heinke P, Reindel J, Kohnert KD, Kairies U, Braun J, Eckel L, Kerner W. Peripheral arterial disease in diabetes mellitus type 1 and type 2: are there different risk factors? *Vasa*. 2002;31:249–254.
111. Hayashi C, Ogawa O, Kubo S, Mitsuhashi N, Onuma T, Kawamori R. Ankle brachial pressure index and carotid intima-media thickness as atherosclerosis markers in Japanese diabetics. *Diabetes Res Clin Pract*. 2004;66:269–275.
112. Yang X, Sun K, Zhang W, Wu H, Zhang H, Hui R. Prevalence of and risk factors for peripheral arterial disease in the patients with hypertension among Han Chinese. *J Vasc Surg*. 2007;46:296–302.
113. Ovbiagele B. Association of ankle-brachial index level with stroke. *J Neurol Sci*. 2009;276:14–17.
114. Ramos R, Quesada M, Solanas P, Subirana I, Sala J, Vila J, Masia R, Cerezo C, Elosua R, Grau M, Cordon F, Juvinya D, Fito M, Isabel Covas M, Clara A, Angel Munoz M, Marrugat J; REGICOR Investigators. Prevalence of symptomatic and asymptomatic peripheral arterial disease and the value of the ankle-brachial index to stratify cardiovascular risk. *Eur J Vasc Endovasc Surg*. 2009;38:305–311.
115. Allison MA, Hiatt WR, Hirsch AT, Coll JR, Criqui MH. A high ankle-brachial index is associated with increased cardiovascular disease morbidity and lower quality of life. *J Am Coll Cardiol*. 2008;51:1292–1298.
116. Criqui MH, McClelland RL, McDermott MM, Allison MA, Blumenthal RS, Aboyans V, Ix JH, Burke GL, Liu K, Shea S. The ankle-brachial index and incident cardiovascular events in the MESA (Multi-Ethnic Study of Atherosclerosis). *J Am Coll Cardiol*. 2010;56:1506–1512.
117. Sutton-Tyrrell K, Venkitachalam L, Kanaya AM, Boudreau R, Harris T, Thompson T, Mackey RH, Visser M, Vaidean GD, Newman AB. Relationship of ankle blood pressures to cardiovascular events in older adults. *Stroke*. 2008;39:863–869.
118. Wattanakit K, Folsom AR, Duprez DA, Weatherley BD, Hirsch AT. Clinical significance of a high ankle-brachial index: insights from the Atherosclerosis Risk in Communities (ARIC) Study. *Atherosclerosis*. 2007;190:459–464.
119. Resnick HE, Foster GL. Prevalence of elevated ankle-brachial index in the United States 1999 to 2002. *Am J Med*. 2005;118:676–679.
120. Greenland P, Smith SC Jr, Grundy SM. Improving coronary heart disease risk assessment in asymptomatic people: role of traditional risk factors and noninvasive cardiovascular tests. *Circulation*. 2001;104:1863–1867.
121. Brindle P, Beswick A, Fahey T, Ebrahim S. Accuracy and impact of risk assessment in the primary prevention of cardiovascular disease: a systematic review. *Heart*. 2006;92:1752–1759.
122. Tsimikas S, Willerson JT, Ridker PM. C-reactive protein and other emerging blood biomarkers to optimize risk stratification of vulnerable patients. *J Am Coll Cardiol*. 2006;47(suppl):C19–C31.
123. Greenland P, LaBree L, Azen SP, Doherty TM, Detrano RC. Coronary artery calcium score combined with Framingham score for risk prediction in asymptomatic individuals. *JAMA*. 2004;291:210–215.
124. Leng GC, Fowkes FG, Lee AJ, Dunbar J, Housley E, Ruckley CV. Use of ankle brachial pressure index to predict cardiovascular events and death: a cohort study. *BMJ*. 1996;313:1440–1444.
125. Hooi JD, Kester AD, Stoffers HE, Rinkens PE, Knottnerus JA, van Ree JW. Asymptomatic peripheral arterial occlusive disease predicted cardiovascular morbidity and mortality in a 7-year follow-up study. *J Clin Epidemiol*. 2004;57:294–300.
126. Ogren M, Hedblad B, Isacsson SO, Janzon L, Jungquist G, Lindell SE. Non-invasively detected carotid stenosis and ischaemic heart disease in men with leg arteriosclerosis. *Lancet*. 1993;342:1138–1141.
127. van der Meer IM, Bots ML, Hofman A, del Sol AI, van der Kuip DA, Witteman JC. Predictive value of noninvasive measures of atherosclerosis for incident myocardial infarction: the Rotterdam Study. *Circulation*. 2004;109:1089–1094.
128. Kornitzer M, Dramaix M, Sobolski J, Degre S, De Backer G. Ankle/arm pressure index in asymptomatic middle-aged males: an independent predictor of ten-year coronary heart disease mortality. *Angiology*. 1995;46:211–219.
129. Abbott RD, Petrovitch H, Rodriguez BL, Yano K, Schatz IJ, Popper JS, Masaki KH, Ross GW, Curb JD. Ankle/brachial blood pressure in men >70 years of age and the risk of coronary heart disease. *Am J Cardiol*. 2000;86:280–284.
130. Resnick HE, Lindsay RS, McDermott MM, Devereux RB, Jones KL, Fabsitz RR, Howard BV. Relationship of high and low ankle brachial index to all-cause and cardiovascular disease mortality: the Strong Heart Study. *Circulation*. 2004;109:733–739.
131. Aboyans V, Lacroix P, Tran MH, Salamagne C, Galinat S, Archambeaud F, Criqui MH, Laskar M. The prognosis of diabetic patients with high ankle-brachial index depends on the coexistence of occlusive peripheral artery disease. *J Vasc Surg*. 2011;53:984–991.
132. Aboyans V, Lacroix P, Postil A, Guilloux J, Rolle F, Cornu E, Laskar M. Subclinical peripheral arterial disease and incompressible ankle arteries are both long-term prognostic factors in patients undergoing coronary artery bypass grafting. *J Am Coll Cardiol*. 2005;46:815–820.
133. Agnelli G, Cimminiello C, Meneghetti G, Urbinati S; Polyvascular Atherothrombosis Observational Survey (PATHOS) Investigators. Low ankle-brachial index predicts an adverse 1-year outcome after acute coronary and cerebrovascular events. *J Thromb Haemost*. 2006;4:2599–2606.
134. Purroy F, Coll B, Oro M, Seto E, Pinol-Ripoll G, Plana A, Quilez A, Sanahuja J, Brieva L, Vega L, Fernandez E. Predictive value of ankle brachial index in patients with acute ischaemic stroke. *Eur J Neurol*. 2010;17:602–606.
135. Alberts MJ, Bhatt DL, Mas JL, Ohman EM, Hirsch AT, Rother J, Salette G, Goto S, Smith SC Jr, Liau CS, Wilson PW, Steg PG; Reduction of Atherothrombosis for Continued Health Registry Investigators. Three-year follow-up and event rates in the international Reduction of Atherothrombosis for Continued Health Registry. *Eur Heart J*. 2009;30:2318–2326.
136. Criqui MH, Ninomiya JK, Wingard DL, Ji M, Fronek A. Progression of peripheral arterial disease predicts cardiovascular disease morbidity and mortality. *J Am Coll Cardiol*. 2008;52:1736–1742.
137. Sheikh MA, Bhatt DL, Li J, Lin S, Bartholomew JR. Usefulness of postexercise ankle-brachial index to predict all-cause mortality. *Am J Cardiol*. 2011;107:778–782.
138. Mohler ER 3rd, Treat-Jacobson D, Reilly MP, Cunningham KE, Miani M, Criqui MH, Hiatt WR, Hirsch AT. Utility and barriers to performance of the ankle-brachial index in primary care practice. *Vasc Med*. 2004;9:253–260.
139. Bendermacher BLW, Teijink JAW, Willigendael EM. Applicability of the ankle brachial index measurement as screening device in general practice for high cardiovascular risk. In: Bendermacher B. *Peripheral Arterial Disease. Screening, Diagnosis and Conservative Treatment* [dissertation]. Maastricht, Netherlands: Maastricht University; 2007.
140. Pollak EW, Chavis P, Wolfman EF. The effect of postural changes upon the ankle arterial perfusion pressure. *Vasc Surg*. 1976;10:219–222.
141. Gornik HL, Garcia B, Wolski K, Jones DC, Macdonald KA, Fronek A. Validation of a method for determination of the ankle-brachial index in the seated position. *J Vasc Surg*. 2008;48:1204–1210.
142. Yataco AR, Gardner AW. Acute reduction in ankle/brachial index following smoking in chronic smokers with peripheral arterial occlusive disease. *Angiology*. 1999;50:355–360.
143. Manning DM, Kuchirka C, Kaminski J. Miscuffing: inappropriate blood pressure cuff application. *Circulation*. 1983;68:763–766.
144. Pickering TG, Hall JE, Appel LJ, Falkner BE, Graves J, Hill MN, Jones DW, Kurtz T, Sheps SG, Roccella EJ. Recommendations for blood pressure measurement in humans and experimental animals, part 1: blood pressure measurement in humans: a statement for professionals from the Subcommittee of Professional and Public Education of the American Heart Association Council on High Blood Pressure Research. *Circulation*. 2005;111:697–716.
145. Mundt KA, Chambless LE, Burnham CB, Heiss G. Measuring ankle systolic blood pressure: validation of the Dinamap 1846 SX. *Angiology*. 1992;43:555–566.
146. Takahashi O, Shimbo T, Rahman M, Musa R, Kurokawa W, Yoshinaka T, Fukui T. Validation of the auscultatory method for diagnosing peripheral arterial disease. *Fam Pract*. 2006;23:10–14.
147. Aboyans V, Lacroix P, Doucet S, Preux PM, Criqui MH, Laskar M. Diagnosis of peripheral arterial disease in general practice: can the ankle-brachial index be measured either by pulse palpation or an automatic blood pressure device? *Int J Clin Pract*. 2008;62:1001–1007.
148. Adiseshiah M, Cross FW, Belsham PA. Ankle blood pressure measured by automatic oscillotonometry: a comparison with Doppler pressure measurements. *Ann R Coll Surg Engl*. 1987;69:271–273.

149. Beckman JA, Higgins CO, Gerhard-Herman M. Automated oscillometric determination of the ankle-brachial index provides accuracy necessary for office practice. *Hypertension*. 2006;47:35–38.
150. Benchimol A, Bernard V, Pillois X, Hong NT, Benchimol D, Bonnet J. Validation of a new method of detecting peripheral artery disease by determination of ankle-brachial index using an automatic blood pressure device. *Angiology*. 2004;55:127–134.
151. Blebea J, Ali MK, Love M, Bodenham R, Bacik B. Automatic postoperative monitoring of infrainguinal bypass procedures. *Arch Surg*. 1997; 132:286–291.
152. Cortez-Cooper MY, Supak JA, Tanaka H. A new device for automatic measurements of arterial stiffness and ankle-brachial index. *Am J Cardiol*. 2003;91:1519–1522, A9.
153. Diehm N, Dick F, Czuprin C, Lawall H, Baumgartner I, Diehm C. Oscillometric measurement of ankle-brachial index in patients with suspected peripheral disease: comparison with Doppler method. *Swiss Med Wkly*. 2009;139:357–363.
154. Ena J, Lozano T, Verdú G, Argente CR, González VL. Accuracy of ankle-brachial index obtained by automated blood pressure measuring devices in patients with diabetes mellitus. *Diabetes Res Clin Pract*. 2011;92:329–336.
155. Jonsson B, Lindberg LG, Skau T, Thulesius O. Is oscillometric ankle pressure reliable in leg vascular disease? *Clin Physiol*. 2001;21: 155–163.
156. Korno M, Eldrup N, Sillesen H. Comparison of ankle-brachial index measured by an automated oscillometric apparatus with that by standard Doppler technique in vascular patients. *Eur J Vasc Endovasc Surg*. 2009;38:610–615.
157. Lee BY, Campbell JS, Berkowitz P. The correlation of ankle oscillometric blood pressures and segmental pulse volumes to Doppler systolic pressures in arterial occlusive disease. *J Vasc Surg*. 1996;23:116–122.
158. MacDonald E, Froggatt P, Lawrence G, Blair S. Are automated blood pressure monitors accurate enough to calculate the ankle brachial pressure index? *J Clin Monit Comput*. 2008;22:381–384.
159. MacDougall AM, Tandon V, Wilson MP, Wilson TW. Oscillometric measurement of ankle-brachial index. *Can J Cardiol*. 2008;24:49–51.
160. Mehlsen J, Wiinberg N, Bruce C. Oscillometric blood pressure measurement: a simple method in screening for peripheral arterial disease. *Clin Physiol Funct Imaging*. 2008;28:426–429.
161. Nukumizu Y, Matsushita M, Sakurai T, Kobayashi M, Nishikimi N, Komori K. Comparison of Doppler and oscillometric ankle blood pressure measurement in patients with angiographically documented lower extremity arterial occlusive disease. *Angiology*. 2007;58:303–308.
162. Pan CR, Staessen JA, Li Y, Wang JG. Comparison of three measures of the ankle-brachial blood pressure index in a general population. *Hypertens Res*. 2007;30:555–561.
163. Raines JK, Farrar J, Noicely K, Pena J, Davis WW, Willens HJ, Wallace DD. Ankle/brachial index in the primary care setting. *Vasc Endovascular Surg*. 2004;38:131–136.
164. Ramanathan A, Conaghan PJ, Jenkinson AD, Bishop CR. Comparison of ankle-brachial pressure index measurements using an automated oscillometric device with the standard Doppler ultrasound technique. *ANZ J Surg*. 2003;73:105–108.
165. Richart T, Kuznetsova T, Wizner B, Struijker-Boudier HA, Staessen JA. Validation of automated oscillometric versus manual measurement of the ankle-brachial index. *Hypertens Res*. 2009;32:884–888.
166. Salles-Cunha SX, Vincent DG, Towne JB, Bernhard VM. Noninvasive ankle pressure measurements by oscillometry. *Tex Heart Inst J*. 1982; 9:349–357.
167. Bonham PA, Cappuccio M, Hulsey T, Michel Y, Kelechi T, Jenkins C, Robison J. Are ankle and toe brachial indices (ABI-TBI) obtained by a pocket Doppler interchangeable with those obtained by standard laboratory equipment? *J Wound Ostomy Continence Nurs*. 2007;34:35–44.
168. Carmo GA, Mandil A, Nascimento BR, Arantes BD, Bittencourt JC, Falqueto EB, Ribeiro AL. Can we measure the ankle-brachial index using only a stethoscope? A pilot study. *Fam Pract*. 2009;26:22–26.
169. Khandanpour N, Armon MP, Jennings B, Clark A, Meyer FJ. Photoplethysmography, an easy and accurate method for measuring ankle brachial pressure index: can photoplethysmography replace Doppler? *Vasc Endovascular Surg*. 2009;43:578–582.
170. Ludyga T, Kuczmik WB, Kazibudzki M, Nowakowski P, Orawczyk T, Glanowski M, Kucharzewski M, Ziaja D, Szaniewski K, Ziaja K. Ankle-brachial pressure index estimated by laser Doppler in patients suffering from peripheral arterial obstructive disease. *Ann Vasc Surg*. 2007;21: 452–457.
171. Migliacci R, Nasorri R, Ricciarini P, Gresele P. Ankle-brachial index measured by palpation for the diagnosis of peripheral arterial disease. *Fam Pract*. 2008;25:228–232.
172. Nicolai SP, Kruidenier LM, Rouwet EV, Wetzels-Gulpers L, Rozeman CA, Prins MH, Teijink JA. Pocket Doppler and vascular laboratory equipment yield comparable results for ankle brachial index measurement. *BMC Cardiovasc Disord*. 2008:26.
173. Sadiq S, Chithriki M. Arterial pressure measurements using infrared photosensors: comparison with CW Doppler. *Clin Physiol*. 2001;21: 129–132.
174. Whiteley MS, Fox AD, Horrocks M. Photoplethysmography can replace hand-held Doppler in the measurement of ankle/brachial indices. *Ann R Coll Surg Engl*. 1998;80:96–98.
175. Fowkes FG, Housley E, Macintyre CC, Prescott RJ, Ruckley CV. Variability of ankle and brachial systolic pressures in the measurement of atherosclerotic peripheral arterial disease. *J Epidemiol Community Health*. 1988;42:128–133.
176. Stoffers J, Kaiser V, Kester A, Schouten H, Knottnerus A. Peripheral arterial occlusive disease in general practice: the reproducibility of the ankle-arm systolic pressure ratio. *Scand J Prim Health Care*. 1991;9: 109–114.
177. Kaiser V, Kester AD, Stoffers HE, Kitslaar PJ, Knottnerus JA. The influence of experience on the reproducibility of the ankle-brachial systolic pressure ratio in peripheral arterial occlusive disease. *Eur J Vasc Endovasc Surg*. 1999;18:25–29.
178. Aboyans V, Lacroix P, Lebourdon A, Preux PM, Ferrieres J, Laskar M. The intra- and interobserver variability of ankle-arm blood pressure index according to its mode of calculation. *J Clin Epidemiol*. 2003;56: 215–220.
179. Baker JD, Dix DE. Variability of Doppler ankle pressures with arterial occlusive disease: an evaluation of ankle index and brachial-ankle pressure gradient. *Surgery*. 1981;89:134–137.
180. Johnston KW, Hosang MY, Andrews DF. Reproducibility of noninvasive vascular laboratory measurements of the peripheral circulation. *J Vasc Surg*. 1987;6:147–151.
181. de Graaff JC, Ubbink DT, Legemate DA, de Haan RJ, Jacobs MJ. Interobserver and intraobserver reproducibility of peripheral blood and oxygen pressure measurements in the assessment of lower extremity arterial disease. *J Vasc Surg*. 2001;33:1033–1040.
182. Holland-Letz T, Endres HG, Biedermann S, Mahn M, Kunert J, Groh S, Pittrow D, von Bilderling P, Sternitzky R, Diehm C. Reproducibility and reliability of the ankle-brachial index as assessed by vascular experts, family physicians and nurses. *Vasc Med*. 2007;12:105–112.
183. Espeland MA, Regensteiner JG, Jaramillo SA, Gregg E, Knowler WC, Wagenknecht LE, Bahnson J, Haffner S, Hill J, Hiatt WR; Look AHEAD Study Group. Measurement characteristics of the ankle-brachial index: results from the Action for Health in Diabetes study. *Vasc Med*. 2008;13:225–233.
184. Weatherley BD, Chambless LE, Heiss G, Catellier DJ, Ellison CR. The reliability of the ankle-brachial index in the Atherosclerosis Risk in Communities (ARIC) study and the NHLBI Family Heart Study (FHS). *BMC Cardiovasc Disord*. 2006:7.
185. Forster FK, Turney D. Oscillometric determination of diastolic, mean and systolic blood pressure: a numerical model. *J Biomech Eng*. 1986; 108:359–364.
186. Hamel JF, Foucaud D, Fanello S. Comparison of the automated oscillometric method with the gold standard Doppler ultrasound method to access the ankle-brachial pressure index. *Angiology*. 2010;61:487–491.
187. Ursino M, Cristalli C. A mathematical study of some biomechanical factors affecting the oscillometric blood pressure measurement. *IEEE Trans Biomed Eng*. 1996;43:761–778.
188. van Montfrans GA. Oscillometric blood pressure measurement: progress and problems. *Blood Press Monit*. 2001;6:287–290.
189. Benchimol D, Pillois X, Benchimol A, Houitte A, Sagardiluz P, Tortelier L, Bonnet J. Accuracy of ankle-brachial index using an automatic blood pressure device to detect peripheral artery disease in preventive medicine. *Arch Cardiovasc Dis*. 2009;102:519–524.
190. Kawamura T. Assessing ankle-brachial index (ABI) by using automated oscillometric devices. *Arq Bras Cardiol*. 2008;90:294–298.
191. Arveschoug AK, Revsbech P, Brochner-Mortensen J. Sources of variation in the determination of distal blood pressure measured using the strain gauge technique. *Clin Physiol*. 1998;18:361–368.
192. Brown J, Asongwed E, Chesbro S, John E. Inter-rater and intra-rater reliability of ankle brachial index (ABI) measurements using a stethoscope and Doppler. Paper presented at: American Physical

Therapy Association Meeting; 2008. http://apps.Apta.Org/custom/abstracts/pt2008/abstractspt.Cfm?M_id_17314. Accessed February 10, 2011.
193. Clyne CA, Jones T, Moss S, Ensell J. The use of radioactive oxygen to study muscle function in peripheral vascular disease. *Surg Gynecol Obstet*. 1979;149:225–228.
194. Osmundson PJ, O'Fallon WM, Clements IP, Kazmier FJ, Zimmerman BR, Palumbo PJ. Reproducibility of noninvasive tests of peripheral occlusive arterial disease. *J Vasc Surg*. 1985;2:678–683.
195. Fisher CM, Burnett A, Makeham V, Kidd J, Glasson M, Harris JP. Variation in measurement of ankle-brachial pressure index in routine clinical practice. *J Vasc Surg*. 1996;24:871–875.
196. Jeelani NU, Braithwaite BD, Tomlin C, MacSweeney ST. Variation of method for measurement of brachial artery pressure significantly affects ankle-brachial pressure index values. *Eur J Vasc Endovasc Surg*. 2000; 20:25–28.
197. Atsma F, Bartelink ML, Grobbee DE, van der Schouw YT. Best reproducibility of the ankle-arm index was calculated using Doppler and dividing highest ankle pressure by highest arm pressure. *J Clin Epidemiol*. 2005;58:1282–1288.
198. van Langen H, van Gurp J, Rubbens L. Interobserver variability of ankle-brachial index measurements at rest and post exercise in patients with intermittent claudication. *Vasc Med*. 2009;14:221–226.
199. Ray SA, Srodon PD, Taylor RS, Dormandy JA. Reliability of ankle: brachial pressure index measurement by junior doctors. *Br J Surg*. 1994;81:188–190.
200. Endres HG, Hucke C, Holland-Letz T, Trampisch HJ. A new efficient trial design for assessing reliability of ankle-brachial index measures by three different observer groups. *BMC Cardiovasc Disord*. 2006:33.
201. Osborn LA, Vernon SM, Reynolds B, Timm TC, Allen K. Screening for subclavian artery stenosis in patients who are candidates for coronary bypass surgery. *Catheter Cardiovasc Interv*. 2002;56:162–165.
202. Vierron E, Halimi JM, Tichet J, Balkau B, Cogneau J, Giraudeau B; DESIR Study Group. Center effect on ankle-brachial index measurement when using the reference method (Doppler and manometer): results from a large cohort study. *Am J Hypertens*. 2009;22:718–722.
203. Aboyans V, Criqui MH, McDermott MM, Allison MA, Denenberg JO, Shadman R, Fronek A. The vital prognosis of subclavian stenosis. *J Am Coll Cardiol*. 2007;49:1540–1545.
204. Shadman R, Criqui MH, Bundens WP, Fronek A, Denenberg JO, Gamst AC, McDermott MM. Subclavian artery stenosis: prevalence, risk factors, and association with cardiovascular diseases. *J Am Coll Cardiol*. 2004;44:618–623.
205. Clark CE, Campbell JL, Powell RJ. The interarm blood pressure difference as predictor of cardiovascular events in patients with hypertension in primary care: cohort study. *J Hum Hypertens*. 2007;21: 633–638.
206. Orme S, Ralph SG, Birchall A, Lawson-Matthew P, McLean K, Channer KS. The normal range for inter-arm differences in blood pressure. *Age Ageing*. 1999;28:537–542.
207. Aboyans V, Kamineni A, Allison MA, McDermott MM, Crouse JR, Ni H, Szklo M, Criqui MH. The epidemiology of subclavian stenosis and its association with markers of subclinical atherosclerosis: the Multi-Ethnic Study of Atherosclerosis (MESA). *Atherosclerosis*. 2010;211:266–270.
208. Espinola-Klein C, Rupprecht HJ, Bickel C, Lackner K, Savvidis S, Messow CM, Munzel T, Blankenberg S; AtheroGene Investigators. Different calculations of ankle-brachial index and their impact on cardiovascular risk prediction. *Circulation*. 2008;118:961–967.
209. O'Hare AM, Katz R, Shlipak MG, Cushman M, Newman AB. Mortality and cardiovascular risk across the ankle-arm index spectrum: results from the Cardiovascular Health Study. *Circulation*. 2006;113:388–393.
210. Fowkes FG, Price JF, Stewart MC, Butcher I, Leng GC, Pell AC, Sandercock PA, Fox KA, Lowe GD, Murray GD; Aspirin for Asymptomatic Atherosclerosis Trialists. Aspirin for prevention of cardiovascular events in a general population screened for a low ankle brachial index: a randomized controlled trial. *JAMA*. 2010;303:841–848.
211. Hoogeveen EK, Kostense PJ, Beks PJ, Mackaay AJ, Jakobs C, Bouter LM, Heine RJ, Stehouwer CD. Hyperhomocysteinemia is associated with an increased risk of cardiovascular disease, especially in non-insulin-dependent diabetes mellitus: a population-based study. *Arterioscler Thromb Vasc Biol*. 1998;18:133–138.
212. Fowler B, Jamrozik K, Norman P, Allen Y. Prevalence of peripheral arterial disease: persistence of excess risk in former smokers. *Aust N Z J Public Health*. 2002;26:219–224.
213. Jager A, Kostense PJ, Ruhe HG, Heine RJ, Nijpels G, Dekker JM, Bouter LM, Stehouwer CD. Microalbuminuria and peripheral arterial disease are independent predictors of cardiovascular and all-cause mortality, especially among hypertensive subjects: five-year follow-up of the Hoorn Study. *Arterioscler Thromb Vasc Biol*. 1999;19:617–624.
214. McDermott MM, Guralnik JM, Albay M, Bandinelli S, Miniati B, Ferrucci L. Impairments of muscles and nerves associated with peripheral arterial disease and their relationship with lower extremity functioning: the InCHIANTI Study. *J Am Geriatr Soc*. 2004;52:405–410.
215. Rooke TW, Hirsch AT, Misra S, Sidawy AN, Beckman JA, Findeiss LK, Golzarian J, Gornik HL, Halperin JL, Jaff MR, Moneta GL, Olin JW, Stanley JC, White CJ, White JV, Zierler RE. ACCF/AHA focused update of the guideline for the management of patients with peripheral artery disease (updating the 2005 guideline): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2011;124:2020–2045.
216. Diehm C, Allenberg JR, Pittrow D, Mahn M, Tepohl G, Haberl RL, Darius H, Burghaus I, Trampisch HJ; German Epidemiological Trial on Ankle Brachial Index Study Group. Mortality and vascular morbidity in older adults with asymptomatic versus symptomatic peripheral artery disease. *Circulation*. 2009;120:2053–61.
217. American Diabetes Association. Peripheral arterial disease in people with diabetes. *Diabetes Care*. 2003;26:3333–3341.
218. Kennedy M, Solomon C, Manolio TA, Criqui MH, Newman AB, Polak JF, Burke GL, Enright P, Cushman M. Risk factors for declining ankle-brachial index in men and women 65 years or older: the Cardiovascular Health Study. *Arch Intern Med*. 2005;165:1896–1902.

KEY WORDS: AHA Scientific Statements ■ ankle brachial index

Measurement and Interpretation of the Ankle-Brachial Index

A Scientific Statement From the American Heart Association

Table I: Diagnostic characteristics of ABI to detect PAD, versus imaging methods.

First Author, Year	Study population	*Gold standard*	Threshold - (ankle/arm)	SE	SP	PPV	NPV
Doppler							
Yao, 1969[3]	110 pts -183 limbs - 25 control	Angiography	1.0	0.97	1.00*	1.00*	0.83*
Carter, 1968[2]	202 limbs all diseased - 86 control (normal pulse)	Cases: angio stenosis >40% below CFA, >50% above.	1.0 (High/High)	1.0*	1.00*		
Sumner, 1979[45]	100 pts with PAD & 48 controls	angiography – criteria : ≥50%, occlusion	0.92 (Pt or DP/arm)	0.98	0.94		
Ouriel, 1982[40]	218 pts with PAD 25 young controls (no RF, triphasic Doppler waveform)	Cases: Angio (criteria?) Doppler (controls)	0.97 (Highest?/arm)	0.97	1.00		
Ouriel, 1982[41]	260 limbs of 133 PAD pts & 68 limbs of 34 controls	Angiography (cases) Doppler (controls)	0.97 (High/High)	0.94	0.99		
Baxter, 1992[52]	20 PAD patients	Angiography (≥ 50% stenosis)	1.0 (High/High)	1.00	0.40		
De Groote, 1995[53]	111 claudicants (138 limbs)	Angiography (≥ 50% stenosis)	0.80	0.82			
Allen, 1996[37]	200 vascular lab pts (290 limbs)	Duplex criteria ≥ 50% stenosis	0.90 (High/High)	0.82*	0.84*	1.00*	0.83*
Lijmer, 1996[38]	Suspected PAD: 441 pts Angiography in only 53 pts	Angiography (≥ 50% stenosis)	0.97 (High/High)	0.84*	0.88*		
Stoffers, 1996[28]	Community, 117 subjects	technician diagnosis	0.97 (1 measure)	0.79	0.82		
	Vascular laboratory: 54 pts	technician diagnosis	0.92 (3 measures)	0.87	0.91		
Premalatha, 2002[43]	Vasc. unit: 100 diabetic pts	Color Doppler (criteria ?)	0.90	0.71	0.89		
Parameswaran, 2005[42]	57 diabetic pts w/o clinical PAD	Monophasic Doppler Wave	0.90 (PT, or DP if no PT /High)	0.63	0.97	1.00*	0.83*
Williams, 2005[46]	Non-diabetic pts with or w/o PAD (41 limbs) - Diabetics with (57 limbs) or w/o (32 limbs) neuropathy.	Color Doppler limited to below iliac artery	0.90	Non-diabetic : 0.83 Diabetic w/o	1.00	1.00	0.95

				neuropathy:	1.00	0.88	0.70	0.91
				Diabetic with neuropathy:	0.53	0.95	0.80	0.84
Niazi, 2006[39]	107 pts (208 limbs)	Angiography (≥ 50% stenosis)	0.90 (High/High) (Low/High)	0.69 0.83		0.83 0.64	1.00*	0.83*
Schröder, 2006[44]	216 outpatients, (81 PAD and 74 diabetes)	duplex - criteria: ≥ 70% (peak velocity ratio >2) – angio in 42 pts	0.90 (High/High) (Low/High)	0.68 0.89		0.99 0.93	0.99 0.93	0.74 0.88
Alnaeb, 2007[47]	24 type II diabetics (47 limbs) 15 control (30 limbs)	duplex – criteria: Rutherford scoring scheme - cut-off ?	? (High/Right)	0.80		0.93		
Alnaeb, 2008[58]	50 PAD patients 18 control	duplex –criteria: Rutherford scoring scheme - score >7	0.84 (High / ?)	0.94		0.79	0.96	0.93
Flanigan, 2008[54]	vascular laboratory - 585 Patients - PAD screening	Duplex: SFA atheroma	< 0.90 and >1.2	0.17		1.00		
Wikström, 2008[51]	306 pts >70 yrs Population based cohort	MRA (≥50% stenosis)	0.90 (PT/Right arm)	0.20		0.99	0.83	0.84
Clairotte, 2009[48]	Vasc lab 146 pts (296 limbs) – (diabetes: 83)	Duplex: ≥ 50% stenosis (peak velocity ratio >2)	0.90 (PT&DP/High)	Overall: Non-diabetic: Diabetic:	0.63 0.73 0.54	0.98 0.95 0.96	0.95 0.98 0.93	0.76 0.78 0.75
Oscillometry								
Carter, 1968[2]	146 limbs with PAD 85 controls	Cases: stenosis >40% below CFA, >50% above Controls: normal pulse	1.0	0.93		0.98		
Guo, 2008[50]	298 patients hospitalized in cardiology	Angiography: >50% stenosis	0.90	0.76		0.90		
Clairotte, 2009[48]	Vascular lab 146 pts (296 limbs) – (diabetes: 83)	Duplex: ≥ 50% stenosis (peak velocity ratio >2)	0.90	Overall: Non-diabetic: Diabetic:	0.40 0.57 0.29	0.96 0.95 0.96	0.88 0.92 0.83	0.66 0.66 0.66
Plethysmography								
Feigelson, 1994[49]	63 cases & 421 controls	Doppler and segmental pressure	0.80	0.40		0.99	0.91	0.53

CFA indicates common femoral artery; DP, dorsal pedis artery; High, highest pressure (arm or ankle artery); Low, lowest pressure (arm or ankle artery); LR, likelihood ratio; MRA, magnetic resonance angiography; NPV, negative predictive value; PPV, positive predictive value; PT, posterior tibial artery; Pts, patients; RF, risk factor; SE, sensitivity; SFA, superficial femoral artery; SP, specificity.
* Calculated results.

Table II: ABI During the Follow-Up of Revascularized PAD Patients

Author, Year	Study population	Endpoint	Reference method	ABI decrease Cut-off value	Se	Sp	PPV	NPV	Comments
Barnes, 1989[81]	232 infra-inguinal grafts FU 17 months	Primary graft failure	Clinical (angiography and/or redo operation)	≥0.20	0.14	0.84	0.22	0.27	Immediate post-op ABI as reference. Bias: subject w/o post-op ABI change >0.20 included and considered as stable –Accuracy : 0.63
Stierli, 1992[82]	42 infra-inguinal vein bypasses	Bypass stenosis	color Duplex (unknown criteria) – all suspect cases had confirmatory angiography	0.10	1.0	0.73			
Laborde 1992[83]	124 infra-inguinal vein bypasses	Bypass stenosis	angiography (criteria?)	0.15 All stenoses: Stenoses >70%	0.43 0.47				ABI calculation: highest ankle, and brachial – Doppler mode. Incompressible arteries included
Idu 1993[84]	201 infra-inguinal bypass FU: 21 months	Bypass stenosis	Angiography (44): only when suspect Duplex or ABI decrease > 0.15%	15% decrease	0.38				ABI method and calculation ? Different surveillance protocols during FU (bias). Duplex criteria: (PSVratio >3 or PSV <45 cm/s):
Decrinis, 1994[77]	116 PTA occluded SFA – FU 1 year - prospective	Reoclusion or stenosis (from <50% to >70%) or >10% predilatation stenosis)	Angiography at 1 year: Reocclusion or stenosis (limited to PTA vessel) Stenosis or occlusion (all vessels)	0.10 0.15 0.10 0.15	0.64 0.60 0.72 0.66	0.88 0.97 0.83 1.00			Doppler criteria: higher ankle and brachial. Reference ABI at day 1.
Dalsing, 1995[85]	80 pts – 102 femoro-distal grafts – 329 FU visits	Graft occlusion (duplex) or reintervention		10% decrease 15% decrease 20% decrease	0.62 0.56 0.51	0.71 0.81 0.87	0.19 0.24 0.33	0.94 0.94 0.94	ABI: highest ankle / highest brachial No data beyond the graft patency – Protocol modified during the FU (ABI alone, then ABI and Duplex: bias) Accuracy: 0.70.
Lundell 1995[86]	156 pts	failing graft FU: 3 yrs	Angiography if clinical signs, ABI decrease >0.15 or duplex +50% stenosis	ABI decrease > 0.15	0.72				Femoro-popliteal grafts only. ABI methods and criteria ?
Radak, 1999[87]	171 PAD: femoro-popliteal PTA	Restenosis	Duplex	50% loss of ABI increase after PTA	0.67	0.80			Duplex criteria: restenosis: > 50% residual diameter reduction of the dilated artery or inflow or outflow tract.

FU indicates follow-up, Se, sensitivity, Sp, specificity, NPV, negative predictive value, PAD, peripheral artery disease, PPV, positive predictive value, PSV, peak systolic velocity, PTA, percutaneous angioplasty, PTS, patients, SFA, superficial femoral artery.

Table III. Comparisons Between Different Techniques for Ankle Pressure Measurement

First author, Year	Study population	Reference method	Cut-point	Mean (95%CI) difference: Reference – index	Sensitivity (%)	Specificity (%)	Accuracy (%)	Correlation coefficient
Index method : Oscillometry								
Aboyans, 2008[147]	54 participants: 19 claudicants; 19 high risk for PAD; 10 healthy subjects	Doppler	0.90	ABI 1.03±0.26 vs. 1.09±0.31 (one of two observers)	76 / 58 (2 observers)	96 / 89	87 (calculated mean)	-
Adiseshiah, 1987[148]	AP in 43 pts; ABI in 19 of them	Doppler	-	AP 120 ± 35 vs. 132 ± 25 mmHg	-	-	-	0.88
Beckman, 2006[149]	201 subjects referred to vascular lab, 14% excluded ("calcification artifact")	Doppler	0.90	ABI -0.06 (0.2) right leg; 0.04 (0.2) left leg	- 73 / 88 (right/left)	95 / 85	-	-
Benchimol, 2004[150]	219 patients referred for cardiological consultation	Doppler	0.90	ABI 1.00±0.20 vs. 1.03±0.18	76	95	89	-
Blebea, 1997[151]	10 healthy subjects; 10 PAD pts; 20 post-op. fem-pop bypass	Doppler	-	ABI 0.83 vs. 0.87	-	-	-	0.89
Clairotte, 2009[48]	146 patients (83 diabetics) referred for PAD evaluation.	Doppler	0.90	ABI -0.021±0.27	Doppler 63 Oscillometry 40	Doppler 98 Oscillometry 96	-	No diabetes 0.60; Diabetes 0.49
Cortez-Cooper 2003[152]	52 healthy subjects	Doppler	-	AP -2.2 ± 6.8 mmHg	-	-	-	0.95
Diehm, 2009[153]	50 PAD patients	Doppler	-	ABI -0.07	-	-	-	0.77
Ena, 2011[154]	104 diabetics > 54 years old	Doppler	0.90	ABI -0.05 (-0.50 – 0.39)	67	87	-	-
Jönsson, 2001[155]	47 PAD pts and 34 without PAD (14 diabetics, 20 healthy volunteers)	Doppler	-	AP: no PAD +1.7±10.5 PAD -28.8 ± 41.4 mmHg	-	-	-	No PAD 0.81 PAD 0.38
Korno, 2009[156]	61 patients in a vascular surgery unit	Doppler	0.90	+0.08 ± 0.15	71	92	82	0.61
Lee, 1996[157]	110 patients referred to vascular lab	Doppler	-	ABI 0.84 ± 0.27 vs. 0.94 ± 0.24	-	-	-	0.90 (excluding failures)
MacDonald, 2008[158]	36 patients referred to vascular clinic	Doppler	-	AP -2.69 mmHg	-	-	-	0.87
MacDougall, 2008[159]	26 PAD-free, 11 at risk, 57 with PAD suspicion	Doppler	0.90	AP -3 mmHg	71	89	-	0.71
Mehlsen, 2008[160]	80 patients with possible PAD; 1258 primary care patients	Plethysmography (strain gauge)	0.90	-	97 (PAD group)	62 (PAD group)	80 (PAD group)	0.88
Mundt, 1992[145]	71 healthy volunteers	Doppler	-	AP +1.5 ± 1.5 mmHg	-	-	-	-
Nukumizu, 2007[161]	168 vascular patients	Doppler	-	-	-	-	-	0.93
Pan, 2007[162]	Population study; 946 subjects 12 – 84 (mean 45) years old	Doppler	-	AP: men + 0.02. women + 0.04 mmHg.	-	-	-	-

Raines, 2004[163]	2 phases: 54 & 69 (healthy?) subjects	Doppler	-	AP –2 ± 6.7 & -3.1 ± 5.1 mmHg	-	-	-	-
Ramanathan, 2003[164]	50 healthy volunteers (mean age 23)	Doppler	-	ABI -0.024	-	-	-	0.42
Richart, 2009[165]	105 (population study), mean age 56	Doppler	0.90	ABI 1.12 ± 0.10 vs. 1.13 ± 0.07	-	-	-	-
Salles-Cunha, 1982[166]	18 PAD-free & 26 PAD pts	Doppler	-	AP (mmHg): Healthy 134 ±11 vs. 133 ±10; PAD 99 ± 14 vs. 111 ± 15	67	87	-	-
Index method: Pocket Doppler								
Bonham , 2007[167]	30 subjects with PAD suspicion	(automatic) Doppler	-	ABI +0.02 ± 0.08	-	-	-	-
Nikolai, 2008[172]	99 subjects with PAD suspicion	(automatic) Doppler	-	ABI +0.05	-	-	-	-
Index method: Palpation								
Aboyans , 2008[147]	54, mixed (healthy + PAD)	Doppler	0.90	ABI +0.22	88	75	79	-
Migliacci, 2008[171]	196 subjects with PAD suspicion	Doppler	0.90	-	88	82	83	-
Index method: Auscultation								
Carmo, 2008[168]	88 subjects with PAD or suspicion	Doppler	0.90	ABI -0.03 (-0.07- 0.00)	71	91	87	-
Index method: Digital photoplesmygraphy								
Khandanpour, 2009[169]	131 claudicants (no diabetics)	Doppler	-	ABI +0.004 (-0.23 - 0.24)	-	-	-	0.79
Sadiq, 2001[173]	91 patients referred to a vascular lab.	Doppler	-	-	-	-	-	AP: 0.96; ABI: 0.95
Whiteley, 1998[174]	32 PAD patients	Doppler	-	-	-	-	-	0.88
Index method: Laser Doppler								
Ludyga, 2007[170]	30 claudicants	Doppler	-	ABI +0.001	-	-	-	-

AP indicates ankle pressure mm Hg; ABI, ankle-brachial index. Others: see Table I.

Table IV. Intra-Observer Reproducibility of the ABI, According to Different Measurement and Calculation Methods

First Author, Year	Study Population	Observers	Measurements /subject (N)	*ABI calculation* (ankle arteries/arm)[a]	Mean ABI	Coef. Variation	95%CI diff. between 2 measurements
Doppler							
Numerator = PT pressure							
Fowkes, 1988[181]	24 pts	Multiple	8	PT/right arm	0.88	7.6%	± 0.13
Stoffers, 1991[182]	9 subjects (3 normal)	59	4 to 9	PT (or DP if PT=0) / Left arm	0.81	13.0%	± 0.21
Kaiser, 1999[183]	6 patients	2 experienced	2	PT (or DP if PT=0)/Highest	NA	NA	± 0.15
		24 less experienced	2	PT (or DP if PT=0)/Highest	NA	NA	± 0.22
Aboyans, 2003[184]	194 subjects w/o known PAD	Multiple	2	PT / Highest	0.97	8.1%	± 0.16
				PT pressure / Mean[b]	0.99	8.5%	± 0.17
				PT pressure / Lowest	1.02	9.3%	± 0.19
Numerator = Highest of PT & DP							
Baker, 1981[185]	35 pts, stable claudication	Single	6.8	Highest / Highest	0.62	12.1%	± 0.15
Ouriel, 1982[40]	10 pts, stable claudication	NA	10	Highest / Highest	NA	9.7%	NA
Johnston, 1987[186]	15 pts	Multiple	Multiple	Highest / Highest	0.64	7.8%	± 0.10
de Graaf, 2001[187]	54 PAD patients	Single	2	Highest / Highest	NA	NA	± 0.09[c]
Holland-Leitz, 2007[188]	108 unselected subjects	Multiple	2	Highest / Mean[d]	1.10	8.0%	± 0.18
Espeland, 2008[189]	870 diabetic patients	Multiple	2	Highest / Highest	1.11	4.7%	± 0.10
Aboyans, 2008[147]	55 healthy/PAD suspicion	Multiple	2	Highest / Mean[e]	1.03	10.7%	± 0.22
Korno, 2009[156]	10 vascular lab pts	Single	2	Highest / Highest	NA	NA	± 0.11
Numerator = Mean PT & DP							
Aboyans, 2003[185]	194 subjects w/o known PAD	Multiple	2	Mean / Highest	0.95	5.8%	± 0.11
				Mean / Mean[b]	0.97	6.1%	± 0.12
				Mean / Lowest	0.99	6.8%	± 0.13
Espeland, 2008[189]	870 diabetic patients	Multiple	2	Mean / Highest	1.07	4.6%	± 0.10
Richart, 2009[165]	105 healthy participants	Single	2	Mean / Right arm	1.12	4.5%	± 0.10
Numerator = Lowest of PT & DP							
Aboyans, 2003[185]	194 subjects w/o known PAD	Multiple	2	Lowest / Mean[b]	0.97	7.6%	± 0.14
				Lowest / Lowest	0.89	8.2%	± 0.14
Espeland, 2008[189]	870 diabetic patients	Multiple	2	Lowest / Highest	1.02	5.3%	± 0.11

Oscillometric							
Weatherley, 2006[190]	119 participants	11	2	Ankle / Right arm	1.18	8.9%	± 0.21
Pan, 2007[162]	41 healthy volonteers	Single	2	Right ankle / Right arm	NA	5.1%	NA
Aboyans, 2008[147]	57 healthy or PAD suspicion	Multiple	2	Ankle / Highest	0.84	20.2%	± 0.34
Korno, 2009[156]	10 vascular lab pts	Single	2	Ankle / Highest	NA	NA	± 0.14
Richart, 2009[165]	105 healthy participants	Single	2	Ankle / NA	1.13	4.4%	± 0.10
Palpation method							
Aboyans, 2008[147]	56 healthy or PAD suspicion	Multiple	2	Highest / Mean[e]	0.84	23.0%	± 0.39

DP indicates dorsalis pedis artery; NA, information not available; PT, posterior tibial artery; PTS: patients.
[a]The ankle and arm arteries chosen in each study are abstracted as follows: Ankle: “Highest”, “Mean” and “Lowest” = respectively the highest, the average and the lowest systolic blood pressure between PT and DP arteries of the same ankle, Arms: “Highest”, “Mean” and “Lowest” = respectively the highest, the average and the lowest systolic blood pressure between both arms.
[b]except if inter-arm BP difference >20 mm Hg.
[c]2 measurements within the same day, other wise ± 0.22 if 1-week interval.
[d]Except if inter-arm BP difference >10 mmHg. [e]except if inter-arm BP difference >15 mmHg.

Table V. The Inter-Observer Reproducibility of the ABI : Literature Review

First Author (Year)	Study Population	Method (ankles/arms)	ABI calculation (ankle artery/arm artery)[a]	Mean ABI	Coef. Variation	95%CI of diff. between 2 measurements
Doppler						
Clyne, 1979[191]	117 PAD pts	Doppler/Doppler	NA	0.50	24.0%	± 0.24
Osmundson, 1985[192]	32 pts + 22 healthy	Doppler/Doppler	Mean /"arm"	0.80	10.0%	± 0.16
Johnston, 1987[186]	15 patients	Doppler/Doppler	PT / right arm	0.64	8.0%	± 0.16
Fowkes, 1988[181]	24 pts + 12 healthy volunteers	Doppler/Doppler	PT / right arm	0.88	6.3%	± 0.11
Stoffers, 1991[182]	9 subjects (3 healthy)	Doppler / Doppler	PT or DP / Left arm	0.81	13.6%	± 0.22
Fisher, 1996[193]	123 PAD patients	Doppler/Doppler	Highest / Highest	0.72	15.2%	± 0.21
Kaiser, 1999[177]	6 pts (experienced obs.)	Doppler / Doppler	PT (or DP if PT=0) / Highest	NA	NA	± 0.15
	6 pts (less experienced obs.)			NA	NA	± 0.20
Jeelani, 2000[194]	14 pts with PAD	Doppler / Doppler	NA	0.70	20.0%	± 0.28
		Doppler / Dinamap		0.79	20.0%	± 0.32
		Doppler / Auscultation		0.73	22.0%	± 0.32
de Graaf, 2001[187]	54 PAD pts	Doppler/DINAMAP	Highest / Highest	NA	NA	± 0.20[b]
Aboyans, 2003[184]	194 subjects w/o known PAD	Doppler/Doppler	Mean / Highest	0.95	13.2%	± 0.25
			Mean / Mean[c]	0.97	12.9%	± 0.25
			Mean / Lowest	0.99	12.6%	± 0.25
			PT / Highest	0.97	17.6%	± 0.34
			PT / Mean[c]	0.99	17.6%	± 0.35
			PT / Lowest	1.02	18.1%	± 0.37
			Lowest / Highest	0.85	17.6%	± 0.30
			Lowest / Mean[c]	0.87	18.4%	± 0.32
			Lowest / Lowest	0.89	18.0%	± 0.32
Atsma, 2005[195]	320 post-menopausal women	Doppler/Doppler	PT / either arm	1.11	5.4%	± 0.12
			DP / either arm	1.08	6.0%	± 0.13
			Lowest / either arm	NA	NA	± 0.12
			Highest / either arm	NA	NA	± 0.12
			Mean / either arm	1.10	4.5%	± 0.10
		Doppler/DINAMAP	PT / either arm	1.14	6.6%	± 0.15
			DP / either arm	1.12	6.7%	± 0.15
			Lowest / either arm	NA	NA	± 0.15
			Highest / either arm	NA	NA	± 0.14
			Mean / either arm	1.12	6.3%	± 0.14
Holland-Leitz, 2007[188]	192 volunteers	Doppler/Doppler	Highest / Mean[d]	1.08	9.3%	± 0.20

Aboyans, 2008[147]	44 for PAD suspicion + 10 healthy volonteers	Doppler/Doppler	Highest / Mean[e]	1.05	13.8%	± 0.29
Van Langen, 2009[196]	20 patients suspect of IC	Doppler/Doppler	Highest / Highest	0.84	9.5%	± 0.16
Korno, 2009[156]	61 vascular lab pts	Doppler/Doppler	Highest / Highest			
Oscillometric						
Weatherley, 2006[190]	119 participants	Oscillo/oscillo	1 ankle artery / right arm	1.18	11.3%	± 0.27
Palpation						
Aboyans, 2008[147]	44 suspect for PAD + 10 healthy volonteers	Palpation/Palpation	Highest / Mean[e]	1.09	22.0%	± 0.48

DP indicates dorsalis pedis artery; IC, intermittent claudication; NA, information not available; Obs, observers; PT, posterior tibial artery; Pts, patients.

[a]The ankle and arm arteries chosen in each study are abstracted as follows: Ankle: “Highest”, “Mean” and “Lowest” = respectively the highest, the average and the lowest systolic blood pressure between PT and DP arteries of the same ankle, Arms: “Highest”, “Mean” and “Lowest” = respectively the highest, the average and the lowest systolic blood pressure between both arms.

[b]both measurements within the same day, otherwise 0,27 if 1 week interval between the measurements

[c]except for inter-arm BP difference >20 mm Hg,

[d]except for inter-arm BP difference >10 mm Hg,

[e]except for inter-arm BP difference >15 mm Hg.

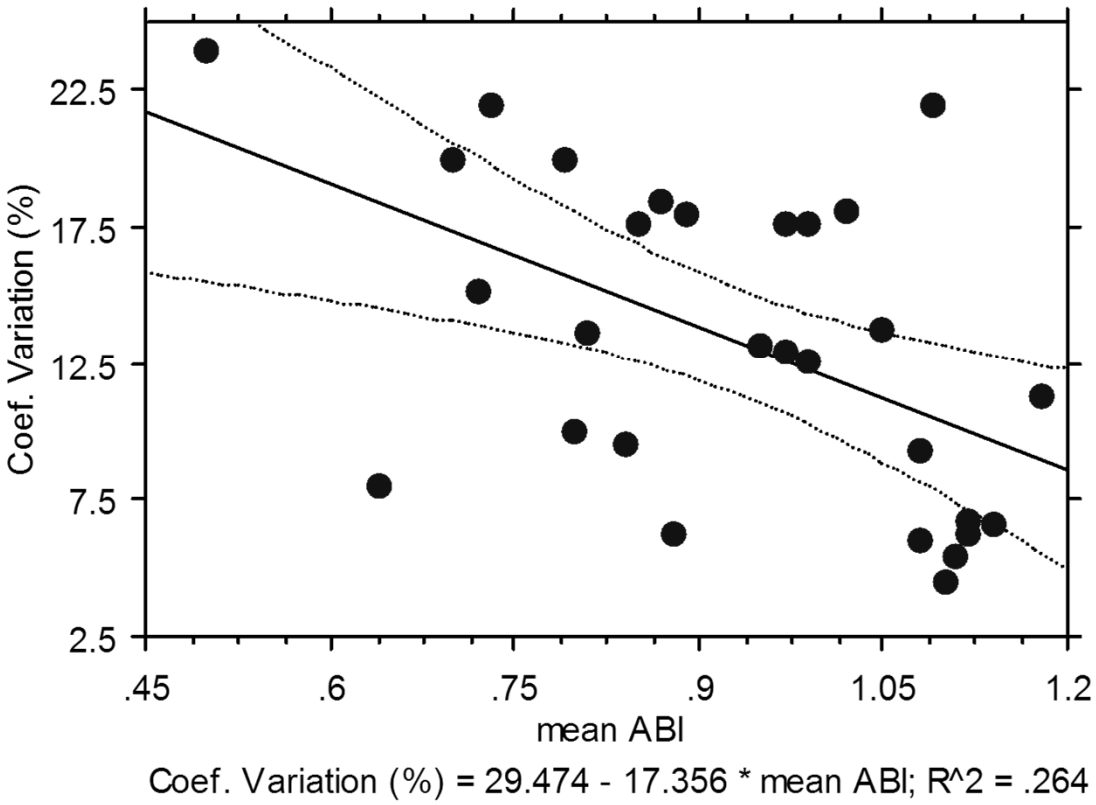

Figure I. - Meta-regression between the average ABI and the inter-observer coefficient of variation of the ABI reported in the same study. Each dot corresponds to one study (see Table V).

Correction Notice

In the article by Aboyans et al, "Measurement and Interpretation of the Ankle-Brachial Index: A Scientific Statement From the American Heart Association," which published ahead of print on November 16, 2012, and appeared in the December 11, 2012, issue of the journal (*Circulation*. 2012;126:2890-2909), a correction was needed.

In the author list on page 2890, the degree listing for Dr. Pande read, "Reena L. Pande MD, PhD, FAHA." It has been changed to read, Reena L. Pande MD, MSc."

This correction has been made to the print version and to the current online version of the article, which is available at http://circ.ahajournals.org/content/126/24/2890.

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