Sensitivity and Specificity of the Ankle–Brachial Index to Predict Future Cardiovascular Outcomes  
A Systematic Review  
Anand V. Doobay, Sonia S. Anand

Objective — The ankle–brachial index is the ratio of the ankle and the brachial systolic blood pressure and is used to assess individuals with peripheral arterial disease. An ankle–brachial index <0.90 suggests the presence of peripheral arterial disease and is a marker of cardiovascular risk. The objective of this review is to determine the sensitivity and specificity of an ankle–brachial index <0.90 to predict future cardiovascular events, including coronary heart disease, stroke, and death.

Methods and Results — We conducted a systematic review of the literature and included studies that used an ankle–brachial index cutoff between 0.80 and 0.90 to classify patients with or without peripheral arterial disease, followed patients prospectively, and recorded cardiovascular outcomes (ie, myocardial infarction, stroke, or mortality). Data were combined using a random-effects model meta-analysis to determine the sensitivity, specificity, relative risks, and likelihood ratios of a low ankle–brachial index to predict future cardiovascular disease. A total of 22 studies were identified, 13 were excluded, and 9 studies were included in the meta-analysis. The sensitivity and specificity of a low ankle–brachial index to predict incident coronary heart diseases were 16.5% and 92.7%, for incident stroke were 16.0% and 92.2%, and for cardiovascular mortality were 41.0% and 87.9%, respectively. The corresponding positive likelihood ratios were 2.53 (95% CI, 1.45 to 4.40) for coronary heart disease, 2.45 (95% CI, 1.76 to 3.41) for stroke, and 5.61 (95% CI, 3.45 to 9.13) for cardiovascular death.

Conclusion — The specificity of a low ankle–brachial index to predict future cardiovascular outcomes is high, but its sensitivity is low. The ankle–brachial index should become part of the vascular risk assessment among selected individuals. (Arterioscler Thromb Vasc Biol. 2005;25:1463-1469.)

Key Words: atherosclerosis ■ cardiovascular disease ■ ankle–brachial index ■ diagnostic test ■ systematic review

Cardiovascular disease is the leading cause of death in North America and all developed nations. As the population ages, the burden of cardiovascular disease is expected to increase. Although the primary determinants of cardiovascular disease have been clearly established and are used to predict future cardiovascular risk, simple, noninvasive, and inexpensive tests that aid in vascular risk prediction are useful.

Individuals with peripheral arterial disease of the lower extremities are among the highest-risk vascular patients. The presence of peripheral arterial disease is an indicator of widespread atherosclerosis in other vascular territories such as the coronary, carotid, and cerebrovascular arteries. There is substantial evidence that peripheral arterial disease is a predictor of future cardiovascular outcomes such as myocardial infarction, stroke, and death.

The ankle–brachial index (ABI) is the ratio of the ankle to brachial systolic blood pressure, and a value of <0.90 indicates the presence of flow-limiting arterial disease affecting the limb. The ABI is used in the diagnosis of peripheral arterial disease of the extremities in symptomatic patients and in the assessment of vascular risk in asymptomatic patients. The ABI is a simple and noninvasive test that can be performed in the office or clinic setting. The intraobserver variability of the test in trained observers is low at $\approx 7\%$. The validity of the ABI for detecting $\geq 50\%$ stenosis in the leg arteries is high (90% sensitivity and 98% specificity).

Although the ABI has been promoted as an adjunct to the office-based assessment of cardiovascular risk, to our knowledge, there have been no systematic reviews evaluating the sensitivity and specificity of the ABI used in this way. To determine the validity of a low ABI to predict future cardiovascular outcomes, we conducted a systematic review of all available studies in which the ABI was measured at baseline and the incidence of cardiovascular events was reported.

Methods

We attempted to identify all studies published before January 2004 by searching the MEDLINE database and reviewing reference lists.
Computer searches used combinations of words related to ABI (or ankle–arm index, ankle arm pressure, or ankle brachial pressure) and words related to cardiovascular outcomes (myocardial infarction, stroke, transient ischemic attack [TIA], coronary artery disease, coronary heart disease [CHD], unstable angina, mortality, or atherosclerosis). Studies were included if they (1) measured ABI in all subjects at baseline, (2) used an ABI cutoff between 0.80 and 0.90 to categorize individuals, (3) excluded participants with previous myocardial infarction and stroke, and (4) collected data on ≥1 of CHD, stroke, or mortality. Studies were subclassified as population-based cohorts, defined as populations with no evidence of pre-existing peripheral arterial disease, or “high-risk” cohorts, defined as populations referred to a vascular laboratory for evaluation of peripheral arterial disease.

**Outcome Definitions**

The reported events of CHD, stroke, all-cause mortality, and cardiovascular mortality were extracted from the selected publications. CHD was defined as a report of incident ischemic or CHD, myocardial infarction, coronary angioplasty, or coronary artery bypass graft surgery. Stroke was defined as a report of incident stroke or stroke and TIA together. For mortality, all-cause mortality and cardiovascular mortality were distinguished from each other, and cardiovascular mortality included atherosclerotic causes of death such as fatal myocardial infarction or stroke but did not include thromboembolism or aneurysmal causes.

**Statistical Methods**

A meta-analysis was performed using a Mantel–Haenszel random-effects model, which incorporates within-study and between-study variation, generally provides wider CIs, and is preferred for analyses with significant heterogeneity. Sensitivity, specificity, and relative risks were calculated for each study outcome using standard formulas. The totals for sensitivity, specificity, and positive likelihood ratios shown are weighted estimates from the random-effects meta-analysis. The positive and negative predictive values for each outcome were derived from the cumulative event rates for each outcome. All analyses were performed using Comprehensive Meta-Analysis version 1.0.23.

**Results**

We identified 22 reports  that evaluated the relationship between the ABI and cardiovascular outcomes and excluded 13% 28,33 studies. Seven studies were excluded because they measured only prevalent disease, 6.22,24–27.35 2 studies were excluded because they did not collect any data specifically on CHD, stroke, or mortality, 28,34 2 studies were excluded because they did not present data for CHD, stroke, or mortality in ABI categories. 29,31 1 study was excluded because the ABI cutoff was not between 0.80 and 0.90, 30 and 1 study was excluded because it included subjects with previous cardiovascular events. 9 Of the 9 studies that were included, 7,8,10–13,23,28,33 7 were population-based cohort studies of relatively healthy individuals, 7,10–13,28,33 2 were cohort studies that included people with suspected peripheral arterial disease who were referred to a vascular laboratory. 5,23

**Demographics and Risk Factors**

Combining data from 28 679 individuals from the 7 population-based cohort studies, 7,10–13,28,33 the mean age was 68.1, 48.0% were women, 29.4% reported tobacco exposure, 10.8% had diabetes, and 32.1% had hypertension. A total of 7.7% of subjects (2214 of 28 679) had a low ABI, and the average duration of follow-up of the cohort was 6.2 years.

**TABLE 1. Demographic and Risk Factor Characteristics of All Subjects**

<table>
<thead>
<tr>
<th></th>
<th>Population-Based Cohort Studies</th>
<th>High-Risk Cohort Studies</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>No. of studies</strong></td>
<td>7</td>
<td>2</td>
</tr>
<tr>
<td><strong>No. of subjects</strong></td>
<td>28 679 (366–14 306)</td>
<td>2674 (744–1930)</td>
</tr>
<tr>
<td><strong>Average follow-up (years)</strong></td>
<td>6.2 (4–10)</td>
<td>10 (10–10)</td>
</tr>
<tr>
<td><strong>Average age</strong></td>
<td>68.1 (61.4–77.8)</td>
<td>68.4 (66.2–69.2)</td>
</tr>
<tr>
<td><strong>ABI &lt;0.90 (%)</strong></td>
<td>7.7 (2.9–25.5)</td>
<td>72.3 (64.8–75.2)</td>
</tr>
<tr>
<td><strong>Women (%)</strong></td>
<td>48.0 (0–100)</td>
<td>48.8 (46.8–54.0)</td>
</tr>
<tr>
<td><strong>Tobacco exposure (%)</strong></td>
<td>29.4 (10.1–59.6)</td>
<td>66.3 (63.2–67.6)</td>
</tr>
<tr>
<td><strong>Diabetes mellitus (%)</strong></td>
<td>10.6 (7.2–27.0)</td>
<td>30.5 (30.0–31.9)</td>
</tr>
<tr>
<td><strong>Hypertension (%)</strong></td>
<td>32.1 (15.2–100)</td>
<td>48.1 (48.1–48.1)</td>
</tr>
</tbody>
</table>

Combining data from 2674 individuals from the 2 high-risk cohort studies, 8,23 the mean age was 68.4 years, 48.8% were women, 66.3% reported some exposure to tobacco, 30.5% had diabetes, and 48.1% had hypertension. A total of 72.3% individuals (1934 of 2674) had a low ABI at baseline, and the average duration of follow-up of the cohort was 10 years (Table 1).

**CHD and Stroke Incidence**

Two of the 9 studies reported data on CHD incidence, and both were population-based studies (Table 2; $n=7131$). 11,33 Three of the 9 studies reported stroke incidence, and all were population based ($n=21 341$). 11–13 For CHD incidence, the sensitivity of a low ABI was 16.5% (95% CI, 12.8 to 20.2), and the specificity was 92.7% (95% CI, 92.1 to 93.3). The positive likelihood ratio for CHD incidence was 2.53 (95% CI, 1.45 to 4.40). For stroke incidence, the sensitivity of a low ABI was 16.0% (95% CI, 12.9 to 19.1), the specificity was 92.2% (95% CI, 91.9 to 92.5), and the positive likelihood ratio was 2.45 (95% CI, 1.76 to 3.41).

**All-Cause Mortality**

Five studies reported information on all-cause mortality ($n=8908$), 7,8,10,11,28 and 4 of these studies were population based ($n=6978$). 7,10,11,28 (Table 3). Combining data from the population-based studies for all-cause mortality, the sensitivity of a low ABI was 31.2% (95% CI, 27.8 to 34.6), and the specificity was 88.9% (95% CI, 88.2 to 89.6). The likelihood ratio of a low ABI predicting all-cause mortality was 3.97 (95% CI, 3.17 to 4.96). Among 1 high-risk cohort, the sensitivity of a low ABI for all-cause mortality was 85.0% (95% CI, 82.1 to 87.5), the specificity was 30.1% (95% CI, 27.6 to 32.7), and the positive likelihood ratio was 1.22 (95% CI, 1.16 to 1.28).
TABLE 3. All-Cause Mortality by ABI Category

<table>
<thead>
<tr>
<th>Source, Year</th>
<th>No./Total (%) ABI &lt; 0.90</th>
<th>No./Total (%) ABI &gt; 0.90</th>
<th>Relative Risk (95% CI)</th>
<th>Adjusted Relative Risk (95% CI)</th>
<th>% Sensitivity of ABI (95% CI)</th>
<th>% Specificity of ABI (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population-based cohorts</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Criqui et al, 1992*20</td>
<td>22/50 (44.0)</td>
<td>46/366 (12.6)</td>
<td>3.50 (2.32–5.30)</td>
<td>3.1 abcdefghijkl (1.8–5.3)</td>
<td>32.4 (21.2–43.5)</td>
<td>92.0 (89.1–94.8)</td>
</tr>
<tr>
<td>Vogt et al, 1993*7</td>
<td>11/51 (21.6)</td>
<td>40/976 (4.1)</td>
<td>5.26 (2.87–9.64)</td>
<td>3.1 abcdefghijkl (1.5–6.7)</td>
<td>21.6 (10.3–32.9)</td>
<td>95.4 (97.7–92.3)</td>
</tr>
<tr>
<td>Newman et al, 1997*10</td>
<td>52/296 (17.4)</td>
<td>53/969 (5.5)</td>
<td>3.19 (2.34–4.57)</td>
<td>2.7 efghijkl (1.8–4.13)</td>
<td>49.5 (40.0–59.1)</td>
<td>78.8 (76.5–81.2)</td>
</tr>
<tr>
<td>Newman et al, 1999*11</td>
<td>104/409 (25.4)</td>
<td>337/3859 (8.7)</td>
<td>2.91 (2.40–3.54)</td>
<td>1.6 efghijkl (1.2–2.12)</td>
<td>23.6 (19.6–27.5)</td>
<td>92.0 (91.2–92.9)</td>
</tr>
<tr>
<td>Total (population-based)*7,10,11,28</td>
<td>189/808 (23.4)</td>
<td>476/6170 (7.7)</td>
<td>3.23 (2.68–3.88)</td>
<td>—</td>
<td>31.2 (27.8–34.6)</td>
<td>89.8 (88.2–89.6)</td>
</tr>
<tr>
<td>High-risk cohorts</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Vogt et al, 1993*8</td>
<td>577/1452 (39.7)</td>
<td>102/478 (21.3)</td>
<td>1.86 (1.55–2.24)</td>
<td>—</td>
<td>85.0 (82.1–87.5)</td>
<td>30.1 (27.6–32.7)</td>
</tr>
</tbody>
</table>

*This study used an ABI cutoff of 0.80; \# for population-based cohorts, positive predictive value was 23.4% (95% CI, 20.5–26.3), negative predictive value was 92.3% (95% CI, 91.6–93.0), the positive likelihood ratio was 3.97 (95% CI, 3.17–4.96), and disease incidence was 9.5%; ¥for the high-risk cohort, positive predictive value was 39.7%; negative predictive value was 78.7%; positive likelihood ratio was 1.22 (95% CI, 1.16–1.28), and disease incidence was 35.2%; €adjusted relative risk ratios are shown for studies that calculated the incremental effect of a low ABI on the risk of all-cause mortality after statistical adjustment for ≥1 cardiovascular risk factors including age, gender, race, smoking, diabetes, total cholesterol, hypertension, fasting glucose, body mass index, waist-hip ratio, fibrinogen, physical activity, history of coronary heart disease, amputation, CHD, or cardiovascular disease.
TABLE 4. Cardiovascular Mortality by ABI Category

<table>
<thead>
<tr>
<th>Source, Year</th>
<th>No./Total Subjects With ABI &lt;0.90</th>
<th>No./Total Subjects With ABI &gt;0.90</th>
<th>Relative Risk (95% CI)</th>
<th>Adjusted Relative Risk (95% CI)</th>
<th>% Sensitivity of ABI (95% CI)</th>
<th>% Specificity of ABI (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population-based cohorts</td>
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</tr>
<tr>
<td>Criqui et al, 1992*28</td>
<td>12/50 (24.0)</td>
<td>15/366 (4.1)</td>
<td>5.86 (2.91–11.8)</td>
<td>6.3 (2.6–15.0)</td>
<td>44.4 (25.7–63.2)</td>
<td>90.2 (87.3–93.2)</td>
</tr>
<tr>
<td>Vogt et al, 19937</td>
<td>6/51 (11.8)</td>
<td>9/976 (1.0)</td>
<td>12.76 (4.72–34.5)</td>
<td>4.5 (1.5–6.7)</td>
<td>40.0 (15.2–64.8)</td>
<td>95.6 (94.3–96.8)</td>
</tr>
<tr>
<td>Newman et al, 199710</td>
<td>19/298 (6.4)</td>
<td>16/969 (1.7)</td>
<td>3.86 (2.01–7.41)</td>
<td>3.2 (1.6–6.4)</td>
<td>54.3 (37.8–70.8)</td>
<td>77.4 (75.0–79.7)</td>
</tr>
<tr>
<td>Newman et al, 199911</td>
<td>28/409 (6.8)</td>
<td>67/3859 (1.7)</td>
<td>3.94 (2.57–6.06)</td>
<td>2.0 (1.2–3.3)</td>
<td>29.5 (20.3–38.6)</td>
<td>90.9 (90.0–91.7)</td>
</tr>
<tr>
<td>Total (population-based)7,10,11,28</td>
<td>65/808 (8.0)</td>
<td>107/6170 (1.7)</td>
<td>5.09 (3.30–7.88)</td>
<td>—</td>
<td>41.0 (33.8–48.2)</td>
<td>87.9 (82.7–88.6)</td>
</tr>
<tr>
<td>High-risk cohorts</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McKenna et al, 1991¥£23</td>
<td>86/482 (17.8)</td>
<td>15/262 (5.7)</td>
<td>3.12 (1.84–5.28)</td>
<td>—</td>
<td>5.61 (3.45–9.13)</td>
<td>—</td>
</tr>
</tbody>
</table>

*This study used an ABI cutoff of 0.80; £this study used an ABI cut-off of 0.85; ¥for population-based cohorts, positive predictive value was 8.0% (95% CI, 6.2–9.9), negative predictive value was 98.3% (95% CI, 98.0–98.6), positive likelihood ratio was 5.61 (95% CI, 3.45–9.13), and disease incidence was 2.5%; ¥for the high-risk cohort, positive predictive value was 17.8%, negative predictive value was 94.3%, positive likelihood ratio was 1.38 (1.25–1.53), and disease incidence was 13.6%; €adjusted relative risk ratios are shown for studies that calculated the incremental effect of a low ABI on the risk of all-cause mortality after statistical adjustment for ≥1 cardiovascular risk factors including age, gender, race, smoking, diabetes, total cholesterol, hypertension, fasting glucose, body mass index, waist-hip ratio, fibrinogen, physical activity history of coronary heart failure, amputation, CHD, or cardiovascular disease.

disease, and if the pretest probability is high, further testing is needed. Because of its low sensitivity (yet high specificity), the ABI cannot be used as a generic screening test. Rather, it must be used in a focused manner, choosing individuals for whom the yield of the test is expected to be higher.

When applying the results of a diagnostic test practically, using likelihood ratios is superior to using the specificity and sensitivity because likelihood ratios are less likely to change with the prevalence of the disorder. They can be calculated for several levels of the symptom/sign or test, they can be used to combine the results of multiple diagnostic tests, and they can be used to calculate post-test probability for a target disorder.36 This effect is reflected when comparing the diagnostic test properties of the population-based cohort to the high-risk cohort in our study (Tables 3 and 4). The high-risk cohort consists of individuals who are older, have more cardiovascular risk factors, and in whom the follow-up time is longer (ie, 10 years versus 6.8 years) compared with the population-based cohorts. As a result, the incidence of CHD, stroke, and mortality occur at a much higher rate in the high-risk cohort compared with the population-based cohorts (35.2% versus 9.5% for all-cause mortality). The sensitivity of a low ABI to predict all-cause mortality is much higher in the high-risk cohorts compared with the population-based cohorts (85.0% versus 31.2%), and the reverse is true for the specificity (30.1% versus 88.9%). The likelihood ratios for all-cause mortality are also different between the groups (1.22 versus 3.97) and indicate that among patients who have multiple risk factors or established vascular disease, the finding of a low ABI does not add significant additional information in determining their future risk of death (eg, pretest to post-test probability changes from 35.2% to 38.5%), whereas among lower-risk patients, the finding of a low ABI does provide important prognostic information because its associated with a significantly increased risk of mortality (eg, pretest to post-test probability changes from 9.5% to 28.0%).

It is important to determine whether a low ABI provides additional risk information over and above the assessment of conventional cardiovascular risk factors. The finding of a low ABI indicates the presence of flow-limiting atherosclerosis in a peripheral artery and likely reflects the presence of generalized atherosclerosis. Furthermore, the ABI should be used as an adjunct to a global risk assessment, which considers conventional cardiovascular risk factors as being additive in their predictive power. This is in contrast to the use of single risk factors to assess vascular prognosis (ie, low-density lipoprotein cholesterol) because on their own, they have poor predictive accuracy.37 A low ABI is more prevalent in patients with cardiovascular risk factors such as smoking, diabetes, and hypertension and is inversely correlated with other measures of vascular disease, including microalbuminuria38 and carotid intimal-medial thickness.22,39 Several studies have demonstrated that after adjusting for conventional cardiovascular risk factors, a low ABI is an independent predictor of cardiovascular risk.7,10–12,23,27–29,33,40 Murabito et al, using the Framingham data set, demonstrated that CHD prevalence remains associated with a low ABI after adjusting

TABLE 5. Summary Table of Population-Based Studies

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Sensitivity of Low ABI (95% CI)</th>
<th>Specificity of Low ABI (95% CI)</th>
<th>Positive Likelihood Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CHD11,33</td>
<td>16.5 (12.8–20.2)</td>
<td>92.7 (92.1–93.3)</td>
<td>2.53 (1.45–4.40)</td>
</tr>
<tr>
<td>Stroke11–13</td>
<td>16.0 (12.9–19.1)</td>
<td>92.2 (91.9–92.5)</td>
<td>2.45 (1.76–3.41)</td>
</tr>
<tr>
<td>All-cause mortality7,10,11,28</td>
<td>31.2 (27.8–34.6)</td>
<td>88.9 (88.2–89.6)</td>
<td>3.97 (3.17–4.96)</td>
</tr>
<tr>
<td>Cardiovascular mortality7,10,11,28</td>
<td>41.0 (33.8–48.2)</td>
<td>87.9 (87.2–88.6)</td>
<td>5.61 (3.45–9.13)</td>
</tr>
</tbody>
</table>
for conventional risk factors, indicating that there is some incremental value of a low ABI in this setting.\textsuperscript{27} Using the likelihood ratio of a low ABI for predicting incident CHD derived in our analysis (2.53), together with the pretest probability that a patient will experience future CHD, can assist a clinician in determining the post-test probability of future CHD. For example, a 55-year-old male smoker on an antihypertensive medication with a high-density lipoprotein cholesterol of 38.5 mg/dL is at a 20% risk of developing clinical CHD in the next 10 years by conventional risk assessment. If his ABI is 0.90, the likelihood ratio of 2.53 increases his post-test probability of developing CHD to 42%. This information will help inform the clinician that the patient is at much higher risk than that derived from standard risk assessments and may influence his/her choice of diagnostic and therapeutic strategies for this patient.

The American Heart Association (AHA) Prevention Conference V described the ABI as a strong and independent risk factor for cardiovascular mortality and recommended it be used to detect subclinical disease in the prevention of cardiovascular mortality and stroke.\textsuperscript{19,40} AHA recommended that the ABI might be a useful addition to the assessment of CHD risk in selected populations, especially among people \( \geq 50 \) years of age or among those who appear to be at intermediate or higher risk for cardiovascular disease on the basis of traditional risk factor assessment, such as cigarette smokers or individuals with diabetes mellitus. Their recommendation takes into account that the prevalence of a low ABI increases significantly with age and is 4 to 5× higher among individuals \( \geq 70 \) years of age compared with those \( <50 \) years of age.\textsuperscript{41} In our analysis, the mean age of individuals was 68 years, and the prevalence of a low ABI was \( \approx 13\% \); accepting that the prevalence of a low ABI is \( \approx 4\times \) lower among individuals aged 50, this is consistent with an expected prevalence of 3% in this age group. Although the prevalence of a low ABI may be lower in a younger population, the relative impact of this finding on their cardiovascular prognosis would be expected to be quantitatively similar. This suggests that although identifying younger individuals is important, advocating routine screening of all adults with the ABI is beyond the scope of primary care practice. Thus, the AHA recommendation to streamline the use of the ABI by age or the presence of cardiovascular risk factors is reasonable.

This model was tested in the Partners Program\textsuperscript{42} involving 350 primary care practices across the United States in which ABI screening was performed if subjects were \( \geq 70 \) years of age or if they were 50 to 69 years of age and had a history of smoking or diabetes. After exclusion of individuals with established diagnosis of peripheral arterial disease or previous cardiovascular disease, a low ABI was identified in 13.1% of individuals screened. In primary care settings, it seems reasonable to expand the Partners approach to screen people \( \geq 70 \) years of age or among those aged 50 to 69 years who have \( \geq 1 \) cardiovascular risk factor (ie, elevated serum cholesterol, hypertension, dysglycemia, tobacco exposure, or a family history of atherosclerotic vascular disease; Figure). Among patients with established vascular disease, there is clear evidence that supports aggressive risk factor control and treatment with antiplatelet agents,\textsuperscript{43–45} statins,\textsuperscript{46–48} and angiotensin-converting enzyme (ACE) inhibition,\textsuperscript{49,50} and routine screening with the ABI would not alter drug treatment. However, the ABI may be useful in identifying certain high-risk patients before their first event. Many of these patients would normally not receive treatment with antiplatelets, statins, and ACE inhibitors because most clinical trials have focused on patients with previous cardiovascular events. Future prospective studies are needed to determine the cost and effectiveness of a screening program using the ABI, followed by early aggressive medical treatment among individuals with a low ABI to determine whether such an approach significantly improves cardiovascular prognosis.

The major limitation of the present analysis is our inability to determine the exact incremental value of the ABI over and above conventional risk factors because individual data were not available to us. Therefore, our weighted estimates of
sensitivity, specificity, relative risk, and positive likelihood ratios are unadjusted for conventional risk factors such as age, gender, hyperlipidemia, hypertension, smoking, and diabetes. However, many of the included studies, but not all, showed an increased relative risk of CHD, stroke, and mortality in subjects with a low ABI even after adjustment of these risk factors (Tables 2 through 4). Therefore, an essential contribution to the current understanding of ABI and cardiovascular risk prediction would be a large-scale study adequately powered to assess the predictability of ABI after adjusting for all conventional risk factors such as diabetes, hypertension, smoking, and hyperlipidemia. Conducting such a study is the only reliable way to show conclusively that low ABI has an incremental predictive value over traditional methods of risk assessment. A second limitation is the variation in measurement technique of the ABI across studies. Some investigators used the average ABI of the 2 lower extremities, some used the lower of the 2 measured values, whereas others used the lowest pressure found among multiple points in the leg. However, despite variations in measurement technique between studies, no systematic bias in the classification of low versus normal ABI would have occurred.

Conclusion
A low ABI is highly specific but not sensitive for predicting future vascular risk. Given that it is simple to perform, noninvasive, and inexpensive, among selected individuals, the ABI is a useful vascular risk prediction tool.

Acknowledgments
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References


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