

Editorial

## Usefulness of the Ankle-Brachial Index

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The preferred method to calculate the Ankle-Brachial Index (ABI) for the diagnosis of Peripheral Arterial Disease (PAD) is to measure by the Doppler method the systolic pressures of the left and right brachial arteries in addition to the dorsalispedis and posterior tibial arteries in each leg [1,2]. For each leg, the higher of the dorsalispedis and posterior tibial pressures is divided by the higher brachial pressure, and the lower overall ABI of the 2 legs is used to diagnose the presence or absence of PAD. The cuff size should be appropriate with a width at least 40% of the limb circumference [2]. The ankle cuff should be placed just above the malleoli with the straight wrapping method [2]. The cuff should not be placed over a distal bypass to avoid the risk of bypass thrombosis [2]. The patient should be at rest for 5 to 10 minutes in the supine position, relaxed, head and heels supported, in a room with a temperature between 66° to 72°Farenheit. The patient should not smoke for at least 2 hours prior to measurement of the ABI [2].

The ABI should be used as the first-line non-invasive test for diagnosing PAD [2]. An ABI  $\leq$  0.90 should be considered the threshold for confirming the diagnosis of lower extremity PAD [2]. When the ABI is >0.90 but PAD is suspected, a post exercise ABI or other non-invasive tests should be used. A post exercise ankle pressure decrease of >30 mm Hg or a post exercise ABI reduction of >20% is a diagnostic criterion for PAD [2]. When the ABI is >1.40, a toe-brachial index or other non-invasive tests should be used to confirm the diagnosis of PAD [2]. An ABI reduction of >0.15 over time can be effective in diagnosing progression of PAD [2]. A high ABI may be caused by calcification of the arterial wall and may occur in patients with medial calcinosis, diabetes mellitus, or end-stage renal disease [2].

An ABI of less than 0.90 is 95% sensitive and 99% specific for the diagnosis of PAD [3]. The lower the ABI, the more severe the restriction of arterial blood flow, and the more serious the ischemia. ABIs of 0.6 to 0.9 usually correlate with mild to moderate intermittent claudication. ABIs of 0.4 to 0.6 usually correlate with severe intermittent claudication. With ABIs between 0.25 to 0.4, rest pain and tissue loss are often found. Patients with calcified arteries from diabetes mellitus or renal failure occasionally have relatively non-compressible arteries leading to falsely elevated ABI values in the normal range. Persons with an ABI of 1.4 or higher also have an increased incidence of cardiovascular events and lower quality of life [4,5].

In 740 men and women (460 with PAD), fewer than 40% of patients with an ABI <0.40 walked continuously for 6 minutes compared with more than 95% of patients with an ABI between 1.00 and 1.50 [3]. Associations between ABI and accelerometer-measured physical activity over 7 days, 6-minute walk, 4-meter walking velocity, and standing balance were stronger than associations between leg symptoms and function [3]. In the PARTNERS (PAD Awareness, Risk and Treatment: New Resources for Survival) program, 296 patients had an ABI of  $\geq$  1.40, and 4,420 patients had an ABI between 0.90 and 1.40 [5]. Patients with an increased ABI had higher odds for foot ulcers and neuropathy and 2.0 lower scores on the physical component scale on the Medical Outcomes Study Standard Form-36 and 5.5 points lower scores on the Walking Impairment Questionnaire walking distance domain [5].

In 118 patients, mean age 73 years, with a decreased ABI, the

prevalence of Coronary Artery Disease (CAD) was 75%, whereas in 118 age-matched and gender-matched patients with a normal ABI, the prevalence of CAD was 29% [6]. The prevalence of aortic valve calcium or mitral annular calcium was also higher in the patients with a decreased ABI (69%) than in the patients with a normal ABI (36%) [6].

In 279 men and women, mean age 71 years, with documented PAD with a low ABI and in 218 men and women, mean age 70 years, without PAD with a normal ABI undergoing coronary angiography for suspected CAD, the prevalence of obstructive PAD was significantly higher in patients with PAD (98%) than in patients with a normal ABI (81%) [7]. The prevalence of 3 or 4-vessel obstructive CAD was also significantly higher in patients with PAD (63%) than in patients with a normal ABI (81%) [7]. In 273 patients, mean age 71 years, with CAD, the lower the ABI, the higher the prevalence of 3-vessel or 4-vessel CAD [8]. Patients with PAD and CAD have more extensive and calcified coronary atherosclerosis, constrictive arterial remodeling, and greater disease progression [9].

The prevalence of PAD increases with age. The prevalence of PAD diagnosed by ABI was 16% in 360 men and 13% in 306 women aged 60 years [10]. Criqui et al. [11] showed that the prevalence of PAD diagnosed by ABI was 5.6% in persons aged 38 to 59 years, 15.9% in persons aged 60 to 69 years old, and 33.8% in persons aged 70 to 82 years old. In the Cardiovascular Health Study, PAD diagnosed by a low ABI was present in 13.9% of 2,214 men aged  $\geq$  65 years and in 11.4% of 2,870 women aged  $\geq$  65 years without cardiovascular disease [12]. In the Rotterdam Study, PAD diagnosed by ABI was present in 16.9% of 2,589 men aged  $\geq$  55 years and in 20.5% of 3,861 women aged  $\geq$  55 years [13]. The prevalence of PAD in 6,979 men and women, mean age 69 years, screened for PAD by an ABI because they were aged 70 years or older or because they were aged 50-69 years with a history of cigarette smoking or diabetes mellitus was 29% [14]. Among these patients with PAD, classic intermittent claudication was present in only 11% [14]. In the MESA (Multi-Ethnic Study of Atherosclerosis) study, of 6,674 men and women aged 45 to 84 years, 806 patients (12.1%) had an ABI <1.0, and 110 patients (1.7%) had an ABI of  $\geq$  1.4 [4].

An abnormal ABI is associated with an increased incidence of cardiovascular events, cardiovascular mortality, and all-cause mortality. At 10-year follow-up of 565 men and women, mean age 66 years, PAD significantly increased the risk of all-cause mortality (relative risk = 3.1), of mortality from cardiovascular disease (relative risk = 5.9), and of mortality from CAD (relative risk = 6.6) [15]. At 4-year follow-up of 1,492 women, mean age 71 years, an ABI of 0.9 or less was associated

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with a relative risk of 3.1 for all-cause mortality after adjustment for age, smoking, and other risk factors [16]. Of 1,537 patients in the Systolic Hypertension in the Elderly program (SHEP) without clinical cardiovascular disease screened with an ABI, the prevalence of an ABI less than 0.90 was 19.7% [17]. At 4-year follow-up, the presence of an ABI less than 0.9 was associated with an age-adjusted relative risk of 3.00 for all-cause mortality in men and of 2.67 in women [17]. Results were similar for cardiovascular mortality and persisted after adjustment for cardiovascular risk factors including an abnormal electrocardiogram [17]. At 5.3-year follow-up of 6,647 persons living in thecommunity, an ABI less than 1.0 increased cardiovascular events 1.77 times, and an ABI of 1.40 or higher increased cardiovascular events 1.85 times [4].

At 7.5-year follow-up of persons in the Cardiovascular Health study in a propensity-matchedstudy of community dwelling older adults, matched hazard ratios for PAD diagnosed by a low ABIfor all-cause mortality,incident heart failure, andsymptomatic PAD were 1.57, 1.32, and 3.92, respectively[18]. In a well-balanced propensity-matched population of 2689 patients with advanced chronic systolic heart failure, during 4.1 years of follow-up, PAD diagnosed by a low ABIwas significantly associated with increased mortality and hospitalization [19].

Of 6,880 patients aged 65 years and older, 836 (12.2%) had asymptomatic PAD with an ABI less than 0.90, and 593 (8.6%) had symptomatic PAD [20]. At 5-year follow-up, all-cause mortality was significantly increased 66% in patients with asymptomatic PAD and 89% in patients with symptomatic PAD [20]. Compared with no PAD, all-cause mortality or severe cardiovascular event was significantly increasedin patients with asymptomatic PAD (hazard ratio = 1.81) and in patients with symptomatic PAD (hazard ratio = 2.66) [20].

Of 414 patients seen in a vascular surgery clinic, 259 (63%), mean age 72 years, had PAD diagnosedby an ABI of either less than 0.90 or  $\geq$ 1.40 [21]. At 33-month follow-up of patients with PAD and 48-month follow-up of patients without PAD, stepwise Cox regression analysis for the time to death showed that PAD was a significant independent risk factor for all-cause mortality with a hazard ratio of 2.2 [21]. In 508 older patients followed with PAD, a decrease in ABI greater than 0.15 between 2 visits to a vascular laboratory was significantly associated with an increased risk of all-cause mortality (risk ratio = 2.4) and of cardiovascular mortality (risk ratio of 2.8) at 3-year follow-up and of cardiovascular morbidity/mortality (risk ratio = 1.9) at 6-year followup independent of visit 2 ABI and other risk factors [22].

The post exercise ABI is also a strong independent predictor of all-cause mortality and provides additional risk stratification beyond the ABI obtained at rest [23]. An alternative ABI method using the lower of the 2 ankle pressures has been shown to identify additional persons at increased risk for mortality [24]. However, these data need confirmation.

Despite the data discussed, the U.S Preventive Services Task Force recommended against screening for PAD with the ABI in asymptomatic adults because most prognostic studies did not allow for calculation of a bias-corrected net reclassification improvementand because treatment benefits for asymptomatic persons with screening detection of ABI have not been established [25]. However, the American College of Cardiology Foundation /American Heart Association guidelines for PAD provide a Class 1b recommendation for ABI screening in patients at risk for PAD [26]. A limitation of the ABI is that its usefulness has not been formally tested in a randomized controlled trial with a design that uses ABI as the entry point to assess outcomes [27].

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