

category [male or female]) score (i.e., myocardial infarction, complex aortic plaque, and PAD), advocated by the European Society of Cardiology guidelines on the management of atrial fibrillation, to assess the risk of stroke (3). However, we think that prior to proposing the measurement of ABI in patients with nonvalvular atrial fibrillation, several issues should be discussed.

First, the main interest of the CHADS₂-VASc score is to determine whether oral anticoagulant therapy (OAC) is necessary. The European Society of Cardiology guidelines recommend OAC when CHADS₂-VAsC is ≥ 2 and prefer OAC to aspirin when the score is 1 (3). Hence, the practical interest of ABI measurement would be limited to those with a CHADS₂-VAsC score of 0 or 1 in order to detect “missed” cases of PAD, which would lead to increasing the score by 1 point and ultimately to revising the anticoagulation strategy. In their study, Violi et al. (1) report high rates of diabetes, hypertension, and history of myocardial infarction or stroke among those with an ABI ≤ 0.90 . All those variables lead to a higher CHADS₂-VAsC score, so that the prevalence of ABI ≤ 0.90 among those with a current CHADS₂-VAsC score at 0 or 1 should be reported to clarify its incremental value to change anticoagulation strategy in these low-risk patients.

Second, 10% of the study population had an ABI > 1.40 , and these patients have apparently not been adequately taken into consideration, because almost one-half of these patients do have underlying PAD, although the definite diagnosis would need further tests as the ABI measurement is impeded by calcified arteries (4).

Finally, the accuracy of the ABI measurement in case of irregular rhythm is unknown. It has been shown that the measurement of arm blood pressure in this situation is associated with considerable intra- and interobserver variability, and it is plausible to wonder at similar poor results when making the ratio of pressures measured in several limbs (5). The only way to moderate the level of inaccuracy is to advocate systematically repeated measurements and to avoid taking the crucial decision of OAC on the basis of a sole measurement of the ABI.

Once these issues are addressed, we agree with Violi et al. that a prospective study is necessary to assess the ability of the ABI to reclassify low-risk patients and increase the CHADS₂-VAsC score discrimination index to predict stroke events in case of nonvalvular atrial fibrillation. Ultimately, a trial would be necessary to clarify the interest of OAC in patients with both low CHADS₂-VAsC score and ABI.

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<http://dx.doi.org/10.1016/j.jacc.2013.09.071>

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Reply

Ankle-Brachial Index in Patients With Nonvalvular Atrial Fibrillation



We thank Dr. Aboyans and colleagues for their comments on our paper (1) in which we reported a 21% prevalence of low (≤ 0.90) ankle-brachial index (ABI) in a population suffering from non-valvular atrial fibrillation. The inclusion of low ABI in the definition of vascular disease within the CHA₂DS₂-VAsC (congestive heart failure [or left ventricular systolic dysfunction]; hypertension [blood pressure consistently $> 140/90$ mm Hg or on hypertension medication]; age ≥ 75 years; diabetes mellitus; previous stroke, transient ischemic attack, or thromboembolism; vascular disease [e.g., peripheral artery disease, myocardial infarction, aortic plaque]; age 65 to 74 years; sex category [i.e., female]) score substantially modified the prevalence of vascular disease, which increased from 17.3% to 33%. This had a particular impact on the CHA₂DS₂-VAsC score subclasses between 0 and 1, whereby the inclusion of a low ABI in the score resulted in a potential risk upgrading.

Thus, 9.5% of patients in classes 0 and 1 had a low ABI; of these, 20% and 80% were CHA₂DS₂-VAsC classes 0 and 1, respectively. Among patients classified as CHA₂DS₂-VAsC score 0, smoking was the only risk factor in 29% of patients; among those classified as CHA₂DS₂-VAsC score 1, 43 (18%) were women, 34 (14%) aged from 65 to 74 years, 149 (63%) were hypertensive, 5 (2%) were diabetic, and 6 (3%) had vascular components of the “classic” CHA₂DS₂-VAsC score.

The presence of an ABI ≥ 1.40 can be detected in atherosclerotic patients, particularly in those with diabetes and this usually reflects tibial artery calcification and is a predictor of cardiovascular disease and total mortality (2). An ABI ≥ 1.40 was detected in 10% of our nonvalvular atrial fibrillation patients. When patients with ABI ≥ 1.40 were compared with those with ABI > 0.90 to 1.39 (Table 1), we found that women were more prevalent in the group of patients with normal ABI; conversely arterial hypertension and diabetes mellitus were more frequent in patients with ABI ≥ 1.40 . Although the definite diagnosis of PAD would need further tests as the ABI measurement is confounded by calcified arteries, we will consider the possibility to evaluate the predictive power of this parameter in future survival analyses.

We planned this study in 2010 using the Doppler method for the determination of the ABI and organized a training meeting to reduce inaccuracy level. Training included demonstration of performance of an ABI in nonvalvular atrial

Table 1

Clinical Characteristics and Pharmacologic Treatments According to ABI Ranging From 0.91 to 1.39 and ≥ 1.40

	ABI 0.91–1.39 (n = 1,381)	p Value	ABI ≥ 1.40 (n = 204)
Age 65–74 yrs	347 (25)	NS	41 (20)
Age ≥ 75 yrs	603 (44)		94 (46)
Female	642 (47)	<0.002	71 (35)
Paroxysmal NVAf	600 (44)	NS	87 (42)
Persistent NVAf	196 (14)		26 (13)
Permanent NVAf	585 (42)		91 (45)
AH	1,115 (81)	<0.04	177 (87)
Diabetes	265 (19)	<0.03	53 (26)
Smoking habit	194 (14)	NS	19 (9)
Previous TIA/stroke	136 (10)	NS	21 (10)
Vascular disease	215 (16)	NS	26 (13)
CHA ₂ DS ₂ -VASc*			
0	66 (5)	NS	7 (4)
1	181 (13)		31 (15)
≥ 2	1,134 (82)		166 (81)
Antithrombotic		NS	
None	217 (16)		30 (15)
OAC	836 (60)		138 (67)
APs	270 (20)		28 (14)
OAC + APs	58 (4)		8 (4)
Statins	496 (36)	NS	62 (31)
Oral hypoglycemic agents	166 (12)	NS	35 (17)

Values are n (%). *Vascular disease includes previous myocardial infarction, peripheral arterial disease, or aortic plaque.

ABI = ankle-brachial index; AH = arterial hypertension; APs = antiplatelet drugs; NVAf = non-valvular atrial fibrillation; OAC = oral anticoagulants; TIA = transient ischemic attack; CHA₂DS₂-VASc = congestive heart failure [or left ventricular systolic dysfunction]; hypertension [blood pressure consistently $>140/90$ mm Hg or on hypertension medication]; age ≥ 75 years; diabetes mellitus; previous stroke, transient ischemic attack, or thromboembolism; vascular disease [e.g., peripheral artery disease, myocardial infarction, aortic plaque]; age 65 to 74 years; sex category [i.e., female].

fibrillation patients with clear delineation of each step. In addition, the follow-up study required a repeat measurement of ABI every 12 months.

We agree with Dr. Aboyans and colleagues that our findings are particularly relevant in patients classified as CHA₂DS₂-VASc score 0 and 1 as the inclusion of low ABI may upgrade the risk and eventually change the therapeutic approach in patients considered at low-to-moderate risk. Inclusion of ABI ≤ 0.90 in the CHA₂DS₂-VASc might have important therapeutic implications only if incorporation of ABI into the CHA₂DS₂-VASc score is prospectively tested. Thus, the results of our ongoing prospective study will validate this and clarify its incremental value.

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New Insights on Plaque Erosion and Calcified Nodules



“Seeing Is Believing”

Plaque erosion (PE) and calcified nodules (CN) have been classically described in pathological studies as causes of acute coronary syndromes (ACS) (1). However, until very recently, the diagnosis of these entities in the clinical setting has remained largely elusive (1). In this regard, the study of Jia et al. (2), using optical coherence tomography (OCT) for the diagnosis of PE and CN, is of major clinical interest and raises several important issues. First, the investigators considered that OCT might provide a “definitive” diagnosis of PE when fibrous cap disruption is excluded and a thrombus overlying an “intact” plaque is visualized. However, considering that OCT lacks the resolution required to visualize mild superficial endothelial erosions and that coronary thrombi may displace along the vessel (either spontaneously or following instrumentation) and actually originate from a remote source (3), we believe that these OCT findings should be considered as diagnostic of “probable” PE. Likewise, lumen surface irregularities without associated thrombus and plaque attenuation by red thrombus (2) are probably better classified as “possible” PE. In our experience, in some patients, a repeated OCT study after several days of intense antithrombotic therapy may unravel the true characteristics of the underlying plaque, once the overlying thrombus has disappeared or drastically reduced in size (3). Indeed, in some of these patients, a previously hidden small plaque rupture—rather than an intact plaque—may be eventually visualized. As this strategy may help to better identify patients with PE, it will be of interest to know if similar “evolving” findings were found in some patients in this study. Second, until now, the value of OCT to detect CN has not been established. Nevertheless, anecdotal patients presenting with large “superficial” calcified plates and associated intracoronary thrombosis have been recently reported (4,5). However, we believe that visualization of a “rupture” should not be required for the diagnosis of CN (2), although this finding might be critical in the diagnosis of complicated CN. Notably, previous studies using virtual histology have demonstrated that silent, uncomplicated, protruding CN may be detected in nonculprit vessels of ACS patients and also that the prognosis of these plaques is rather benign (6). Furthermore,