

Ankle to brachial systolic pressure index at rest increases with age in asymptomatic physically active participants

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ABSTRACT

Background: It is commonly acknowledged that the ability to use the ankle-brachial index (ABI), a reliable way to diagnose atherosclerosis, decreases with age in the general population. The aim of this study was to determine the relationship between resting ABI and age in different populations.

Methods: 674 physically active participants with (active high risk, ACT_{HR}) or without (active low risk, ACT_{LR}) cardiovascular risk factors or/and sedentary (SED) subjects, aged 20–70 years. Systolic arterial pressure was recorded at rest and simultaneously with automatic sphygmomanometers at the arms and ankles. ABI was calculated as the ratio of the lowest, highest or mean ankle pressure to the highest arm pressure.

Results: Proportion of ABI_{min}<0.90 was 10.3% in SED_{HR} subjects versus 0.5% and 1.2%, respectively, in ACT_{HR} and ACT_{LR} groups. The averaged ABI value of each group was in the normal range in all groups (ABI>0.90) but was significantly lower in SED_{HR} compared with all active participants ($p<0.001$). Regression lines from ABI_{mean} versus age could lead to approximately +0.05 every 15 years of age in apparently healthy active participants (ACT_{LR}).

Conclusion: ABI at rest increases with the increase in age in the groups of low-risk asymptomatic middle-aged trained adults. The previously reported decrease in ABI with age is found only in SED_{HR} subjects, and is very likely to rely on the increased prevalence of asymptomatic arterial disease in this group. The increase of ABI with age is consistent with the ‘physiological’ stiffness observed in ageing arteries even in the absence of ‘pathological’ atherosclerotic lesions.

Trial registration number: NIH clinicaltrials.gov: NCT01812343.

INTRODUCTION

Atherosclerosis is a widely prevalent disease, especially in Western countries. Numerous risk factors are known to increase the risk of atherosclerosis, including age, diabetes,

Key messages

- This is the first study to report that resting ankle-brachial index (ABI) increases with age in asymptomatic and physically active participants without risk factors.
- The decrease of ABI value with age could be due to the increase in the prevalence of asymptomatic peripheral artery disease in older participants.
- This study suggests that the so-called borderline or low normal ABI (in the range 0.90–0.99) remains to be studied by considering the age of the participant.

hypertension, dyslipidaemia, smoking status and history of familial cardiovascular events.^{1 2} Ankle to brachial systolic arterial pressure index (ABI) has largely been validated as an accurate way of detecting peripheral artery disease (PAD), even in asymptomatic patients.³ It is generally acknowledged that ABI decreases with age in the general population,⁴ although conflicting results can be found.⁵ This inverse ABI-to-age relationship observed in population studies is likely to result from an increased prevalence of asymptomatic lower limb arterial stenosis in the elderly (‘pathological’ ageing). On the contrary, remodelling of the arterial wall with age (‘physiological’ ageing) results in arterial stiffness,^{6 7} and arterial stiffness is known to increase ABI or even lead to non-compressible ankle arteries.⁸ Therefore, in participants without stenosis, ABI should increase with age.

One way of excluding most potential cases of stenosis and getting an idea of the ABI-to-age relationship resulting from the ‘physiological’ ageing of the arterial wall, is by focusing on physically active and

asymptomatic participants. Indeed, during exercise, even very mild to moderate stenosis becomes symptomatic whereas these lesions remain asymptomatic in sedentary subjects.⁹ Hence, physically active and asymptomatic participants are very unlikely to present even mild stenosis. We hypothesised that: (1) on the one hand, in such physically active and asymptomatic participants without cardiovascular risk factors, instead of the usually reported negative relationship between resting ABI and age, we would find a positive relationship as a result of arterial wall remodelling with age, (2) on the other hand, using the same technique, the usual inverse relationship between ABI and age would be found in asymptomatic but sedentary subjects with cardiovascular risk factor, as a result of significant stenosis that remains asymptomatic due to low-activity level.

METHODS

This multicentre retrospective study was performed among patients with no history of cardiovascular disease and reporting no lower limb symptoms. All participants were referred to departments of sports medicine for the systematic evaluation of exercise performance or departments of vascular investigations to evaluate the presence or absence of PAD from ABI screening in patients with cardiovascular risk factors. In all participants, medical examination reports were used to bring out the presence or absence of cardiovascular risk factors (smoking status, hypertension, diabetes mellitus, dyslipidaemia, familial history of cardiovascular events). Although the gender of a participant could be considered a risk factor, each group consisted of males as well as females. Characteristics of the participants were recorded from a standard questionnaire, interview and physical evaluation. Only patients not reporting the use of cardiovascular drugs, lipid-lowering agent, any kind of limb pain at rest or exercise, or a history of cardiovascular disease, were included in the present study.

Procedure

Anthropometric data were recorded and ABI measured with automatic devices. Indeed, in the past few years, multiple devices have become available to automatically measure arm and ankle systolic pressures and determine ABI, in order to facilitate routine screening for PAD.¹⁰ These automatic devices are rapidly spreading^{11–17} and have been extensively used in previous studies.^{9 18 19} One of the major interests of automatic determination is to homogenise the measurement between various operators. Ankle and arm pressures were measured automatically using four Dynamap Critikon V100 15 cm large cuff sphygmomanometers (Johnson and Johnson, France), with the participant resting supine for at least 5 min. ABI was calculated for each leg using the highest arm pressure as a denominator. The numerator was either: the maximal (ABI_{max}), minimal (ABI_{min}) or mean (ABI_{mean}) of the two ankle pressures.

Subjects

From our database, we retrieved 674 participants fulfilling the inclusion criteria. Patients were divided into three groups. The first group (sedentary high risk, SED_{HR}), comprised of sedentary subjects referred to the vascular unit for systematic vascular investigations due to their risk factors. All these patients had one or multiple cardiovascular risk factors and reported no significant leisure physical activity. The other two groups consisted of asymptomatic physically active participants referred to sports medicine departments for the evaluation of their leisure physical activity. Among these asymptomatic and active participants, the second group included participants with one or more cardiovascular risk factors (active high risk, ACT_{HR}) and the third group included those reporting no cardiovascular risk factors (active low risk, ACT_{LR}). The study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the institution's human research committee. The study was registered in the NIH clinicaltrial.gov database under reference NCT01812343.

Statistical analysis

Analyses were performed using SPSS software, V.17. Quantitative variables are presented as mean \pm SD. Analysis of variance was used to compare continuous variables in the three groups. Linear regression was employed to describe the ABI-to-age relationship and its underlying hypotheses were verified. General linear model was used to compare the slopes of regression lines.

RESULTS

In brief, 674 participants aged 51.7 ± 10.4 years; weight 76.7 ± 14.6 kg; stature 1.72 ± 0.08 m; body mass index (BMI) 26.0 ± 4.8 , were included in this study. Anthropometric characteristics and cardiovascular risk factors for each group are shown in tables 1 and 2.

SED_{HR} subjects logically had a mean weight and BMI significantly higher than active participants, either ACT_{HR} or ACT_{LR} ($p<0.001$). There were significant differences between the mean ages of the three groups ($p<0.001$).

Regarding cardiovascular risk factors (table 2), of interest to note is that in the ACT_{HR} subjects, the proportion of participants having one or two cardiovascular risk factors, was higher than for SED_{HR} . However, SED_{HR} subjects were more likely to accumulate at least three cardiovascular risk factors. The major risk factor observed in ACT_{HR} subjects was smoking status and a history of familial cardiovascular events. In contrast, in SED_{HR} subjects, a large proportion of participants with diabetes was found.

Few participants had an $ABI<0.90$. Taking the ABI_{min} as a reference, proportion of $ABI_{min}<0.90$ was 10.3%, 0.5% and 1.2%, in SED_{HR} , ACT_{HR} and ACT_{LR} subjects,

Table 1 Characteristics of included participants at rest

	Active low risk (ACT _{LR})	Active high risk (ACT _{HR})	Sedentary high risk (SED _{HR})	p Value
n	252	218	204	–
Males/females	223/29*	175/43	149/55	<0.001
Age (years)	48.5±10.5*†	50.4±9.5*	56.9±9.1	<0.001
Weight (kg)	74.1±11.4*	75.9±14.0*	80.9±17.8	<0.001
Height (m)	1.74±0.07	1.73±0.09	1.68±0.08	0.151
Body mass index (kg/m ²)	24.4±3.2*	25.2±3.8*	28.7±6.1	<0.001
Power output (W)	249±55	221±59	Not available	–
Minimal ankle–brachial index (ABI _{min})	1.15±0.10*	1.14±0.10*	1.08±0.14	<0.001
Maximal ankle–brachial index (ABI _{max})	1.20±0.10*	1.20±0.09*	1.16±0.14	<0.001
Mean ankle–brachial index (ABI _{mean})	1.17±0.09*	1.17±0.09*	1.12±0.13	<0.001

Data are presented as mean±SD.

*p Value <0.05, significantly different from SED_{HR}.

†p Value <0.05, significantly different from ACT_{HR}. p Value is a tendency of overall comparison.

respectively. Comparable results were observed using ABI_{mean} as reference—the proportion of ABI_{mean}<0.90 was 4.4%, 0.0% and 0.8%, in SED_{HR}, ACT_{HR} and ACT_{LR} subjects, respectively. Lastly, only six SED_{HR} subjects (2.4%) had an ABI_{max}<0.90. On average, as shown in table 1, the ABI observed in SED_{HR} subjects was lower than that in the other two groups, regardless of the method of calculation. In asymptomatic active participants, no significant differences were observed between the average ABI of high-risk or low-risk participants, regardless of the calculation mode (p=0.456, 0.931 and 0.663, respectively, for ABI_{min}, ABI_{max} and ABI_{mean}).

Finally, regression lines were ABI_{mean}=−0.02 age/10 +1.23 for SED_{HR}, ABI_{mean}=+0.01 age/10+1.14 for ACT_{HR} and ABI_{mean}=+0.03 age/10+1.04 for ACT_{LR} (figure 1). Slopes were significantly different between groups: p=0.01 for ACT_{LR} versus ACT_{HR}, p<0.001 for ACT_{LR} versus SED_{HR} and p=0.04 for ACT_{HR} versus SED_{HR}.

Comparable results were observed for relationships of ABI_{min} versus age and ABI_{max} versus age, as shown in figure 1.

DISCUSSION

To the best of our knowledge, this is the first study reporting resting ABI in asymptomatic and physically active participants without risk factors (ie, very unlikely to suffer even mild stenosis), in asymptomatic and physically active participants with risk factors, and in asymptomatic sedentary subjects with risk factors (ie, where mild to moderate PAD is likely to remain asymptomatic due to inactivity). Note that the average ABI value of this population was generally in the normal range (1.30>ABI_{rest}>0.90). The finding that ABI at rest does not decrease (but increases) with age in ACT_{LR} is consistent with our initial hypothesis, and appears logical

Table 2 Occurrence frequencies of various risk factors of overall participants

	Active low risk (ACT _{LR})	Active high risk (ACT _{HR})	Sedentary high risk (SED _{HR})
Number of risk factors (%)			
0	100	0	0
1	0	78.9	69.6
2	0	18.8	16.2
3	0	2.3	12.2
4	0	0	2.0
Smoking status,	0/252	183/35	24/180
Smoker (active+former)/no smoker, n (%)	(0)/100	(83.9)/(16.1)	(11.8)/(88.2)
Hypertension, n (%)	0 (0)	19 (8.7)	62 (30.4)
Diabetes mellitus, n (%)	0 (0)	2 (0.9)	157 (77.0)
Dyslipidaemia, n (%)	0 (0)	41 (18.8%)	47 (23.0)
Familial history of cardiovascular (CV) events, n (%)	0 (0)	66 (30.3)	9 (4.4)
Average of CV risk factors, n	0	1.2	1.5

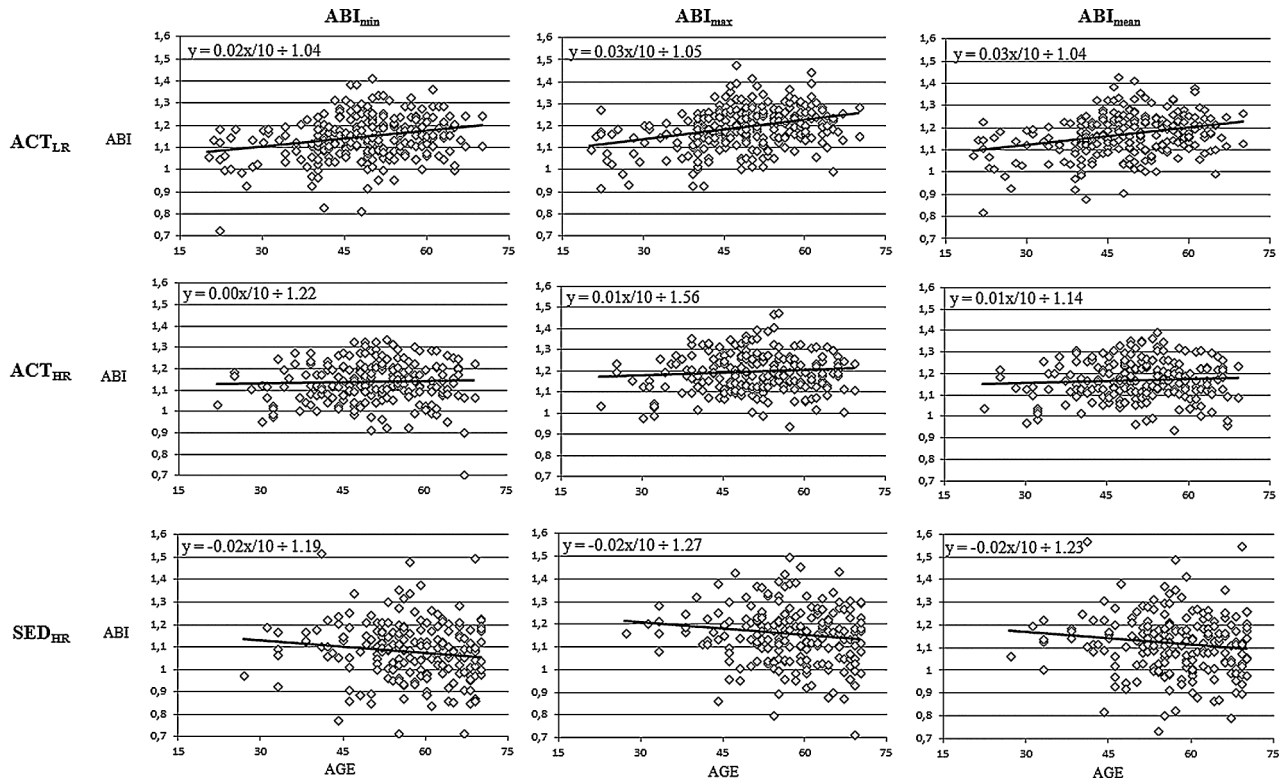


Figure 1 ABI_{min}, ABI_{max} or ABI_{mean}-to-age scatterplots and regression lines for ACT_{HR}, ACT_{LR} and SED_{HR} (ABI, ankle-brachial index; ACT_{HR}, active high risk; ACT_{LR}, active low risk; SED_{HR}, sedentary high risk).

with the expected ‘physiological’ ageing of the arterial wall. On the contrary, we found the expected decrease of ABI with age in the SED_{HR} population. Interestingly, the slope of the relationship of ABI to age in the ACT_{HR} subjects is half way between the two other regression analyses. We contend that these observations objectively support the assumption that the decrease of ABI value with age is due to the increase in the prevalence of asymptomatic PAD in older participants, whereas ABI ‘physiologically’ increases with age.

It is commonly accepted that ABI<0.90 at rest is the most common and agreed on value, having reported acceptable levels of sensitivity and specificity^{4, 20} to detect PAD. Nevertheless, this cut-off was determined on populations with proven PAD or in advanced age populations.²¹ The results of our study present a ‘physiological’ trend of resting ABI to increase by approximately +0.05 every 15 years of age in apparently healthy subjects. Thus, the use of a cut-off point of ABI<0.90, could overestimate the prevalence of PAD in the young, while this could underestimate the prevalence of PAD in the elderly. This could also be an explanation for the fact that the sensitivity of ABI is reportedly decreased in elderly patients.²²

There are limitations to the present study. First, it may be that our observation results from a technical issue. Whether automatic devices are equally accurate in young or old participants has never been studied. The fact that automatically determined pressures overestimate the pressure that would be observed in manual

recordings, especially for low-pressure values, cannot consistently be excluded.^{10, 23} This potential overestimation should explain the difference between groups. Second, it could be argued that physically active patients are more likely to have higher ABI values than participants with low physical activity levels.²⁴ This is also true, and we found a similar trend in our group in terms of average ABI value within each group. Nevertheless, the ABI to age relationship was analysed in groups with a relatively homogeneous activity level (Power Output ACT_{LR} vs ACT_{HR}: p=0.433). Third, we provide no argument that the increased trend of ABI with ageing in ACT_{LR} is related to arterial stiffening. Previous studies have reported a weak but significant association between a high ABI and high pulse wave velocity.²⁵ Further investigations are needed in ACT_{LR} subjects to correlate ABI measurements to measurements of arterial stiffness. Fourth, no ultrasound imaging was carried out to exclude those patients who might have had mild to moderate arterial ultrasound lesions. We advocate that this would have been of little interest because, in sports-related claudication, even extremely localised and mild lesions may become symptomatic, as illustrated by endofibrosis in athletes.^{26, 27} The sensitivity of ultrasound to detect these extremely moderate lesions would be relatively low. Inversely, we think that it is unlikely that the absence of decrease of ABI resulted from undetected severe stenoses, since ABI decreases with the severity of endoluminal lesions. Lastly, on the one hand, our groups show a very high proportion of males and

Caucasian participants. Thereby, our results would require confirmation due to the differences in ABI that are reported between males and females or between black and Caucasian participants.²⁸ On the other hand, extrapolation to other automatic devices is needed as well as confirmation with manual recordings. It should be kept in mind that the variability of manual recording is high and depends on the observer experience. Using automatic devices allowed us to get rid of interindividual variability²⁹ and to reduce the time of measurement.³⁰

In conclusion, the present study provides, for the first time, objective evidence that the ‘physiological’ change in ABI increases with age. This reinforces the belief that the decrease in ABI at rest with age observed in general population studies likely results from the increased prevalence of participants with subclinical PAD. This may also question whether the same resting ABI value should be used as a cut-off point in the youngest and oldest participants. Specifically, the present study suggests that the so-called borderline or low normal ABI (in the range of 0.90–0.99) is more likely to predict the presence of PAD in older than in younger patients.

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Competing interests None declared.

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Provenance and peer review Not commissioned; internally peer reviewed.

Data sharing statement No additional data are available.

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