Is augmentation index a good measure of vascular stiffness in the elderly?

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Abstract

Objectives: we investigated the exact relationship between age and gender on augmentation pressure (AG) and augmentation index (AI) measured over the radial (muscular) and carotid (elastic) arteries.

Design and Methods: AG is the contribution that wave reflection makes to systolic arterial pressure. AI is an indirect measure of arterial stiffness and is calculated as AG divided by pulse pressure (PP) × 100. AG and AI both increase with age. AG and AI were measured in 458 subjects using SphygmoCor. A total of 755 readings were obtained (302 carotid, 453 radial). The mean age was 57.5 ± 13.7 years. Diabetic subjects were excluded. Among the subjects, 13.5% were hypertensive.

Results: statistically, women had mean values of AI significantly higher than men in both radial and carotid arteries. These differences were less marked with AG. Quadratic equations better described the relationship between AI and age but not AG and age. Thus, AI increased with age up to our median age of 55 years but plateaued thereafter, whereas the AG continued to increase steadily with age. A multiple regression analysis demonstrated that both AI and AG were negatively related to height and positively related to diastolic blood pressure (DBP).

Conclusions: AG continues to increase in the elderly over the age of 55, but not AI. AI is higher in women and higher when measured over the carotid than the radial. AI is positively related to DBP and negatively to height. AG is proposed as a more suitable measure of arterial stiffness than AI.

Keywords: ageing, arteries, blood pressure, augmentation index, arterial compliance, elderly

Introduction

Arterial compliance is defined as the change in arterial volume divided by associated distending pressure (when applied to closed chambers) [1]. It is known that the compliance of arteries in the human body declines with age [2, 3], and it decreases from childhood [4] even in the absence of cardiovascular diseases. It is reduced in hypertensive subjects and patients with end-stage renal disease or diabetes [5–7].

Increased arterial stiffness is an independent marker of cardiovascular disease in hypertensive subjects and is linked to ventricular hypertrophy and atherosclerosis [8–10].

Arterial compliance can be modified by antihypertensive drugs, exercise and oestrogen replacement in women and androgen suppression in men [5, 11, 12]. The arterial tree is composed of different arterial segments. These segments differ in the proportions of elastin-collagen to smooth muscle. Most studies show that the ageing process is greater in the more proximal, more elastic and less muscular arteries [13].

The augmentation pressure (AG) is the measure of contribution that the wave reflection makes to the systolic arterial pressure, and it is obtained by measuring the reflected wave coming from the periphery to the centre. Reduced compliance of the elastic arteries causes an earlier return of the ‘reflected wave’, which arrives in systole rather than in diastole, causing a disproportionate rise in systolic pressure and an increase in pulse pressure (PP), with a consequent increase in left ventricular afterload and a decrease in diastolic blood pressure (BP) and impaired coronary perfusion.

The augmentation index (AI) is an indirect measure of arterial stiffness and increases with age, and it is calculated as AG (augmentation pressure) divided by PP × 100 to give a percentage. With an increase in stiffness there is a faster propagation of the forward pulse wave as well as a more rapid reflected wave.
The aim of this study is to assess the effect of age and gender on AI and AG in the two different types of arteries, one predominantly elastic and the other predominantly muscular.

**Methods**

**Subjects**

This was a cross-sectional study that recruited volunteers, 40% of whom were involved in a population study in west London. The remaining 60% were healthy volunteers for various studies involving arterial compliance. They were all free of acute illness; people with diabetes were excluded; 13.5% of the study population were hypertensive.

A total of 755 readings were obtained from 458 subjects. Two hundred and sixty-two volunteers were male (57.2%) and 196 volunteers were female (42.8%). Only 297 subjects had both carotid and radial augmentations measured, but both recordings were not attempted in all participants. The mean age of those with carotid augmentation measurement was 58.5 ± 11.0 years (mean ± SD), range 30.6–89.0. The mean age of the group with radial augmentation measurement was 56.8 ± 15.2 (mean ± SD), range 13.1–90.3. The study was approved by the Hammersmith Hospital ethics committee.

**Blood pressure measurements**

Non-invasive brachial BP was measured thrice using an Omron automatic oscillometric digital BP monitor (HEM-705CP) in the left arm of the subject, in the supine position. The mean of the three readings was used in the SphygmoCor® as the BP of the subjects. The BP was recorded immediately prior to every tonometric recording.

**Arterial stiffness measures**

Applanation tonometry was performed using SphygmoCor® (AtCor, Australia) over the right radial artery and/or over the right carotid artery, with the subject in the supine position, to calculate AI. This was done by applying a tonometer (Miller inc., Houston, TX, USA) to the carotid and radial artery and compressing the vessel wall sufficiently to record the pulse trace. All readings recorded met the manufacturer's quality control standards integrated into the software package. In view of doubts about the usefulness of central pressure measurement with a transfer function, we do not report central pressure here.

**Statistical analysis**

SPSS for Windows, version 11.0, was used for all analyses (SPSS Inc., Chicago, IL, USA). The association of AG and AI with age was investigated by univariate and quadratic linear regression. Multiple linear regressions were performed using two age groups. The subjects were split into two stiffness parameters. The data were additionally analysed by performing a simple regression between age, AG and AI within the whole group, the group under 55 years as well as the group above 55 years.

**Results**

Characteristics of the study population are shown in Table 1. The mean HR for the whole group was 61.6 ± 9.7. The mean HR for the group of subjects under 55 years was 61.6 ± 9.7, the mean HR for the group above 55 years was 61.8 ± 9.4 years. There was no statistically significant difference between the two groups (P = 0.62).

Table 2 shows the values of AG (mmHg) and AI (%) according to age, gender and site of measurement, and the statistical differences between men and women.

The values of augmentation derived from the carotid artery (AGC) were higher than the values of augmentation derived from the radial artery (AGR) in all groups and for both genders. The AGC was 4.17 mmHg higher than AGR (paired t-test P<0.001).

The values of Alc (augmentation index derived from the carotid artery) were higher than the values of augmentation index derived from the radial artery (Alr) in all groups and for both genders. The Alc was 7.2% higher than Alr. Performing a paired t-test between Alc and Alr showed a significant difference between the two measurements (P<0.001).

AI in women was higher than in men (50% versus 24.9% when measured over the radial artery P<0.001). Similar findings were found when AI was measured over the carotid artery (39.2% versus 34.3% P<0.001 Table 2). AG also tended to be higher in women than in men, but the difference was less marked.

Considering the results of the whole group, the relationship between age and both AG and AI was positive for both radial and carotid arteries and for both men and women.

The regression of AI on those aged below 55 years showed the following values: female radial regression coefficient, 0.75 (P<0.001); female carotid regression coefficient, 0.73 (P<0.001); male radial regression coefficient, 0.61 (P<0.001); and male carotid regression coefficient, −0.03 (P = 0.93) (data not shown in the table).

<table>
<thead>
<tr>
<th>Table 1. Characteristics of the study population</th>
</tr>
</thead>
<tbody>
<tr>
<td>Demographic and haemodynamic variables</td>
</tr>
<tr>
<td>Age (years)</td>
</tr>
<tr>
<td>Females</td>
</tr>
<tr>
<td>Males</td>
</tr>
<tr>
<td>Hypertension</td>
</tr>
<tr>
<td>Weight (kg)</td>
</tr>
<tr>
<td>Height (cm)</td>
</tr>
<tr>
<td>Heart rate (bpm)</td>
</tr>
<tr>
<td>Mean brachial systolic BP (mmHg)</td>
</tr>
<tr>
<td>Mean brachial diastolic BP (mmHg)</td>
</tr>
</tbody>
</table>

SD, standard deviation; BP, blood pressure.
There were no significant regressions of AI on those aged above 55 years and showed the following values: female radial regression coefficient, $-0.10 (P = 0.60)$; female carotid regression coefficient, $-0.32 (P = 0.54)$; male radial regression coefficient, $0.11 (P = 0.29)$; and male carotid regression coefficient, $-0.06 (P = 0.62)$.

Excluding people on treatment for hypertension did not materially alter these results except for male radial AI over 55, which showed a regression coefficient of $\beta = 0.21; P = 0.04$ (data not shown in the table).

The relationship between age and AI was better explained using a quadratic equation. The model was significantly improved for both the radial ($P < 0.001$) and carotid ($P < 0.05$) in men, and for the radial in women ($P < 0.001$). When we considered the same model for AG, the variation was not further explained by a quadratic equation except for the carotid in men ($P < 0.05$).

Figure 1 gives the regression lines for the equations between AI and age with a peak male radial AI at 85 years, male carotid AI at 70 years, female radial AI at 65 years and female carotid AI at 70 years. Figure 1 also gives the corresponding linear results for AG.

Table 3 gives a multiple regression of AI on age, age$^2$, diastolic blood pressure (DBP), height and gender in the whole group. When gender is included in the model, the AI depended on age (positively — a quadratic association), DBP (positively) and height (negatively), explaining 15–22% of the variance. The corresponding results for AG, excluding age$^2$, are also given and confirm the results for gender, height and DBP.

Excluding the subjects with uncontrolled systolic hypertension (SBP $\geq 160$) and those on treatment for hypertension did not materially alter these results (data not shown in the table).

**Discussion**

The current study demonstrates an independent relationship between AI and age in a group of males and females with a wide range in age (13.1–90.3 years). The AI (AG/PP)

### Table 2. Augmentation pressure and augmentation index according to age, gender and site of measurement

<table>
<thead>
<tr>
<th>Variable</th>
<th>Whole group</th>
<th>$\leq$55 years</th>
<th>$\geq$55 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>N obs</td>
<td>Mean ± SD</td>
<td>$P$ value</td>
</tr>
<tr>
<td>AG Car M (mmHg)</td>
<td>101</td>
<td>20.2 ± 11.0</td>
<td>0.45</td>
</tr>
<tr>
<td>AG Rad F (mmHg)</td>
<td>195</td>
<td>16.5 ± 10.4</td>
<td>0.07</td>
</tr>
<tr>
<td>AG Rad M (mmHg)</td>
<td>258</td>
<td>14.8 ± 8.9</td>
<td>0.07</td>
</tr>
<tr>
<td>AI Car F (%)</td>
<td>101</td>
<td>39.2 ± 9.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AI Car M (%)</td>
<td>201</td>
<td>34.3 ± 10.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AI Rad F (%)</td>
<td>195</td>
<td>30.0 ± 10.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AI Rad M (%)</td>
<td>258</td>
<td>24.9 ± 12.2</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

AG Car F, aortic augmentation pressure derived from carotid in females; AG Car M, aortic augmentation pressure derived from carotid in males; AG Rad F, aortic augmentation pressure derived from radial in females; AG Rad M, aortic augmentation pressure derived from radial in males.

**Table 3. Multiple regression between AI, age, age$^2$, gender, height and DBP and between AG, age, gender, height and DBP in the whole group**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient</th>
<th>$P$ value</th>
<th>R square</th>
</tr>
</thead>
<tbody>
<tr>
<td>AG Carotid ($n = 287$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>1.22</td>
<td>0.01</td>
<td>–</td>
</tr>
<tr>
<td>$Age^2$</td>
<td>-0.01</td>
<td>0.04</td>
<td>–</td>
</tr>
<tr>
<td>Gender</td>
<td>4.23</td>
<td>0.01</td>
<td>–</td>
</tr>
<tr>
<td>Height</td>
<td>-0.26</td>
<td>0.003</td>
<td>–</td>
</tr>
<tr>
<td>DBP</td>
<td>0.23</td>
<td>&lt;0.001</td>
<td>0.22</td>
</tr>
<tr>
<td>Age (n = 334)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.59</td>
<td>0.20</td>
<td>–</td>
</tr>
<tr>
<td>$Age^2$</td>
<td>-0.004</td>
<td>0.34</td>
<td>–</td>
</tr>
<tr>
<td>Gender</td>
<td>3.82</td>
<td>0.02</td>
<td>–</td>
</tr>
<tr>
<td>Height</td>
<td>-0.17</td>
<td>0.04</td>
<td>–</td>
</tr>
<tr>
<td>DBP</td>
<td>0.30</td>
<td>&lt;0.001</td>
<td>0.15</td>
</tr>
<tr>
<td>AG Carotid ($n = 287$)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.38</td>
<td>&lt;0.001</td>
<td>–</td>
</tr>
<tr>
<td>Gender</td>
<td>3.09</td>
<td>0.02</td>
<td>–</td>
</tr>
<tr>
<td>Height</td>
<td>-0.13</td>
<td>0.05</td>
<td>–</td>
</tr>
<tr>
<td>DBP</td>
<td>0.32</td>
<td>&lt;0.001</td>
<td>0.33</td>
</tr>
<tr>
<td>AG Radial (n = 334)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>0.29</td>
<td>&lt;0.001</td>
<td>–</td>
</tr>
<tr>
<td>Gender</td>
<td>2.19</td>
<td>0.05</td>
<td>–</td>
</tr>
<tr>
<td>Height</td>
<td>-0.14</td>
<td>0.02</td>
<td>–</td>
</tr>
<tr>
<td>DBP</td>
<td>0.33</td>
<td>&lt;0.001</td>
<td>0.31</td>
</tr>
</tbody>
</table>

AI, augmentation index; AG, augmentation pressure; DBP, diastolic blood pressure. Coefficient, regression coefficients (%/year, %/year$^2$, %/female, %/cm, %/mmHg).
of arterial compliance in the elderly. It has already been observed that augmentation index (AI) and augmentation pressure (AG) increase with age, but do not increase at higher ages. AG, however, continues to decline in the elderly individuals as the aorta stiffens and the impedance mismatch between the central and peripheral arteries diminishes, and the major reflection locates more towards the periphery. Augmentation pressure, however, continues to increase as the central arteries stiffen and the magnitude of the forward wave increases.

It is known that cardiovascular risk is greater in men. The incidence of cardiovascular diseases is higher in men compared with women [18, 19], and this affects longevity. However, AI and AG were higher in women than in men.

This increase in AI and AG in women, as shown by multiple regression model in Table 3, is in part due to their shorter height and a closer physical proximity between the heart and reflecting sites, as already described in other studies [14, 20]. Nevertheless, the gender difference remained statistically significant. Men appear to be at a higher level of cardiovascular risk despite a lower AI and AG.

Mitchell et al. found that the carotid-femoral pulse wave velocity was lower than the carotid-brachial artery pulse wave velocity before the age of 50, but it was higher after 50 years, with a reversal of the normal central to peripheral arterial stiffness gradient [14]. Van der Heijden et al. showed that the distensibility of the aorta, an elastic artery, decreases with age in both sexes, but they did not find any significant changes in the distensibility of the aorta in men and women [13]. However, we did not have any information about muscular arteries because AI in men was higher than in women (60.8 mmHg in men versus 63.4 mmHg in women, P<0.001). AG was 3–4 mmHg higher in women of this age (P = 0.03 for the carotid and P = 0.08 for the radial measurements).

When looking at the effect of age, however, the results for AI were ‘misleading’ but understandable. As PP increased with age, AI did not increase at higher ages. AG, however, did continue to increase with age, especially for the carotid AG in women (Figure 1).

It appears that the AI would not to be a valid surrogate of arterial compliance in the elderly. It has already been suggested that the inappropriateness of correction for PP explains the modest validity of AI in a diabetic population [17]. Considering our results, we propose the utilisation of the AG instead of AI in the elderly, taking into consideration the well-known increase in PP in this population.

A possible explanation for the increase of AG and lack of increase in AI in the elderly could be as follows. The sites of major wave reflection are determined by the impedance mismatch between the large central arteries, such as the aorta, and the smaller conduit arteries branching from the aorta (muscular arteries). AI continues to decline in the elderly individuals as the aorta stiffens and the impedance mismatch between the central and peripheral arteries diminish, and the major reflection locates more towards the periphery. Augmentation pressure, however, continues to increase as the central arteries stiffen and the magnitude of the forward wave increases.

There are a number of limitations to this study. This is a cross-sectional study and not a longitudinal one. In the study population, people with diabetes were excluded, but 13.5% of the population was hypertensive. However, the exclusion of these subjects did not materially alter the results.

The study did not include data on glucose tolerance, insulin levels, or accurate measures of visceral adiposity or details of exercise capacity. These are all factors that may affect the arterial compliance and could influence augmentation. We did not determine the life-style of the study population. For example, smoking, alcohol and salt intake could influence arterial compliance, both acutely and chronically [21–24], and in this study we did not assess the presence of smokers, regular drinkers or the quantity of daily salt intake.
The population was mainly Caucasian, but it also included Africans and Asians who represented the west London population. It is known that there are differences in the arterial stiffness among different ethnic groups [25–29]. Racial details were not recorded for all our subjects. The incidence of some diseases that affect arterial compliance is higher in the Afro-American population than in the Caucasian population [28].

Finally, we did not have the measures of both carotid and radial AI for all subjects. Height was available for only 69% of the subjects.

In conclusion, this study shows that AI, an important measurement of arterial compliance, increases with age till about 55 years and slows down to a plateau thereafter. However, AG continues to increase steadily with age, suggesting that AG should be measured in the elderly and not AI.

In women, the value of this measurement of arterial stiffness is always higher than that in men, which is in part due to their lower height and a closer physical proximity between the heart and the reflecting sites.

AG should therefore be reported instead of AI when assessing the effect of age, gender and other factors on augmentation.

Key points
- Augmentation pressure (AG) is a more suitable measure of arterial stiffness than Augmentation Index (AI).

Conflict of interest
None.

Previous presentation
Abstract ESH 2005, Milan

References
26. Pinto E, Mensan R, Meenan K et al. Peripheral arterial compliance differs between races-comparison between Asians,
Minimal-preparation CT colon in detection of colonic cancer, the Oxford experience

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Abstract

Background: the main colonic imaging modalities, including barium enema, colonoscopy and computed tomography colonography, require bowel preparation. Performing these imaging procedures in the elderly can be difficult due to immobility, incontinence and poor tolerance of bowel cleansing. Minimal preparation CT (MPCT) colon was introduced in the early 1990s in the UK. Much of the published literature on MPCT colon is limited by small patient numbers and short duration of follow-up.

Objective: the aim of this study is to review our experience with the MPCT technique involving a large consecutive cohort of patients with long follow-up.

Methods: all studies of MPCT performed in a 1-year period between July 2000 and July 2001 at our institution were reviewed retrospectively. MPCT reports were cross-referenced with the cancer registry to allow for an average period of 30 months follow-up. A definite diagnosis of cancer was only given following the appearance on the cancer registry. Those patients who had negative MPCT colon were assumed to be true negatives if no corresponding name was identified on the cancer registry. In the event of data mismatch, patient notes were reviewed to ascertain a diagnosis.

Results: 391 MPCT examinations were performed during the period of the study (209 males, median age 82: age range 56–91 years). Thirty-four patients who had MPCT colon during the study period appeared on the cancer registry. A further three patients with disseminated colorectal malignancy identified on MPCT colon died without histological confirmation (tumour prevalence = 9.5%). Thirty-two of the registry confirmed 34 cases were detected on MPCT colon, giving a sensitivity of 0.94 (95% confidence interval 0.86–1.00). Including the three cases without histological confirmation gives a slightly higher sensitivity of 0.95. There were seven patients with definitely abnormal MPCT colons, who did not appear on the registry, resulting in specificity for definite abnormality of 0.98 (confidence interval 0.97–1.0). However, three of these seven are those who died of disseminated colorectal malignancy as above, raising the specificity to 0.99. Fourteen cases (3.5%) of extra-colonic malignancies were observed in this study.

Conclusion: even with the longer follow-up of this large cohort of patients the sensitivity and specificity in our study for the diagnosis of colorectal cancer with MPCT remains comparable with that of other studies and this technique competes well with other common colonic imaging modalities.

Keywords: colonic cancer, elderly patients, MPCT