Males with abdominal aortic aneurysm have reduced left ventricular systolic and diastolic function

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Abstract

Background: Abdominal aortic aneurysm (AAA) increases the risk of chronic heart failure and other major cardiovascular events. Knowledge about left ventricular function in patients with AAA is lacking. This echocardiographic study aimed to investigate whether AAA is associated with left ventricular systolic and diastolic dysfunction.

Methods: Echocardiography was performed in 307 males (199 AAA and 108 controls) recruited from a regional ultrasound surveillance programme of known AAA, or from an ongoing ultrasound screening programme, during 2011–2016.

Results: Subjects with AAA had thicker septal and posterior walls and a reduced left ventricular function compared to controls. Left ventricular ejection fraction (AAA 55 ± 8%, controls 57 ± 7%) and global longitudinal strain (AAA 19 ± 3%, controls 20 ± 3%) were lower in the group with AAA (both p < 0.05). Moreover, decreased mitral annular plane systolic excursion (12 ± 2 mm versus 13 ± 2 mm) and higher E/e’ (13 ± 5 versus 11 ± 4) were observed in subjects with AAA (both p < 0.05). The aortic sinus (38 ± 4 mm versus 35 ± 2 mm) and ascending aorta (36 ± 4 mm versus 34 ± 5 mm) were also wider in the AAA group compared to controls (both p < 0.01).

Conclusion: AAAs are associated with reduced left ventricular systolic and diastolic function in males. The larger diameter of the aortic sinus and ascending aorta among AAA patients suggests that AAA is a general aortic disease.

Keywords
AAA, arteries, cardiovascular risk assessment, echocardiography, left ventricular dysfunction

1 | INTRODUCTION

Abdominal aortic aneurysm (AAA) is a disease that predominantly affects elderly males. In developed countries, AAA is estimated to cause 1-3% of deaths in males aged 65–85 years (Sakalihasan et al., 2005). AAA is usually asymptomatic, only rarely producing clear symptoms. Rupture of the aneurysm is a life-threatening event, with a survival rate of only 10%-20% (Hultgren et al., 2016). However, even without rupture, AAA increases the risk of other major cardiovascular events, including chronic heart failure, but the mechanisms underlying this are unclear (Bath et al., 2015). Males with AAA may have decreased aortic wall distensibility and enhanced systolic wave reflection (Åström Malm et al., 2020). Moreover, Yoshida et al. (2020) demonstrated an association between increased arterial stiffness and subclinical left ventricular dysfunction in a healthy population. Both arterial load and arterial stiffness are strongly related to left ventricular function. Aortic
systolic blood pressure determines the workload of the heart and the left ventricle (Antonini-Canterin et al., 2013; Ikonomidis et al., 2019; Nichols et al., 2011). Thus, increased arterial stiffness may promote left ventricular hypertrophy, and systolic as well as diastolic heart failure (Mottram et al., 2005).

Increased arterial stiffness as well as generalized arteriomegaly have been demonstrated in AAA patients (Åström Malm et al., 2020; Johnsen et al., 2009). This indicates that AAA may be a systemic disease rather than a local disease, and moreover that its pathological process is not confined to the abdominal aorta. However, there is a lack of knowledge about left ventricle function and cardiac comorbidity in patients with AAA. To our knowledge, no study has previously described left ventricular function in AAA patients. This echocardiographic study aimed to investigate whether AAA is associated with left ventricular systolic and diastolic dysfunction.

2 | METHODS

2.1 | Study population

During 2011–2016, males were recruited consecutively from a regional ultrasound surveillance programme of known AAA or from an ongoing ultrasound AAA screening programme in two neighbouring regions in the southern part of Sweden. In Sweden, 75%–85% of all 65-year-old males are screened annually for AAA, and the prevalence of AAA in this group is just below 2% (National Board of Health & Welfare, 2016). This corresponds to approximately 3900 patients screened per year in the two regions, with 98 newly detected AAA cases. Criteria for exclusion from this study were cardiac arrhythmia, severe disability, advanced cancer and language barriers. Our study included 307 males, 199 patients with AAA and 108 controls, ranging in age from 55 to 80 years. Males with AAA had a maximum infrarenal aortic diameter, measured according to the leading edge-to-leading edge (LELE) method, of at least 30 mm during their most recent clinical ultrasound examination. Patients from the screening programme with an AAA diameter exceeding 55 mm were immediately referred for surgical intervention and never included in the study. During the study examination one patient was identified with an AAA diameter exceeding 55 mm. AAA <55 mm is classified as a small AAA. The controls had an absolute infrarenal aortic diameter within the reference range (<30 mm), and no sign of ectasia, at their screening examinations during the past 5 years (Åström Malm et al., 2020).

All subjects participating in the study gave written informed consent. The study was approved by the regional ethical review board in Linköping, Sweden, and was conducted in accordance with principles stated in the Declaration of Helsinki, Dnr 2016/143-32.

2.2 | Study protocol

All subjects were instructed to refrain from consuming alcohol for 12 h, and from caffeinated beverages or tobacco for 4 h prior to their visit. A questionnaire regarding smoking status, cardiovascular diseases (i.e. myocardial infarction, effort related chest pain or previous coronary bypass surgery) and current medications were completed by the examiner based solely on participant responses. The examinations were performed at the outpatient Departments of Clinical Physiology at Linköping University Hospital and at the County Hospital Ryhov, Jönköping.

Body weight and height was measured and rounded to the nearest 0·5 kg or cm. Body mass index (BMI) and body surface area (BSA) (Du BOIS & Du BOIS, 1916) was calculated using weight in kg and height in metres. The systolic and diastolic blood pressures (SBP, DBP) were measured bilaterally with an oscillometric device (Dinamap model PRO 200 Monitor, Critikon, Tampa), in supine position after 10 min of rest.

2.3 | Echocardiography

Echocardiography was performed using a Vivid 9 ultrasound scanner (GE Vingmed Ultrasound; General Electric, Milwaukee). Apical four-chamber, two-chamber and long axis views were acquired at a frame rate of >40 frames/sec, and analysed off-line by one experienced echocardiographer using 2D speckle tracking (EchoPAC PC version 202, GE Ultrasound). Peak global longitudinal strain (GLS) was calculated as the mean value from 18 segments of the left ventricle (three in each level, base-mid-apex). Left ventricular ejection fraction (LVEF) was measured using the modified Simpson method (Lang et al., 2015). Reproducibility in our lab has been reported previously (Blomstrand et al., (2018)). LVEF < 52% and GLS < 18% were classified as abnormally decreased (Lang et al., 2015; Nagueh et al., 2016; Ommen et al., 2000).

Mitril annular plane systolic excursion (MAPSE) was measured using the tissue tracking algorithm, which uses colour tissue Doppler measurement of systolic tissue velocity, integrated over time (Brodin et al., 1998). MAPSE was calculated by averaging the total amplitude values measured for three consecutive heartbeats at the septal, lateral, inferior and anterior aspects of the two- and four-chamber views. MAPSE < 13 mm was classified as an abnormally low value (Grue et al., 2019).

Diastolic transmitral inflow profiles were obtained at the level of the tip of the mitral leaflets using pulsed Doppler, and the peak of the E-wave was evaluated. Peak early diastolic myocardial velocity (e’) was measured from colour tissue Doppler recordings at the base of the septum and lateral wall, and was presented as the mean of three consecutive heartbeats. The E/e’ was calculated and was considered to reflect the filling pressure to the left ventricle. E/e’ > 14 was classified as increased (Lang et al., 2015; Nagueh et al., 2016; Ommen et al., 2000).

Aortic diameter was measured by the LELE method in the aortic arch and in the ascending aorta. The diameter in the aortic sinus was measured using M-mode.

2.4 | Statistics

Data were analysed using SPSS 25·0 for Windows (IBM SPSS Statistics for Windows, Version 25·0). Continuous variables were...
expressed as mean ± standard deviation (SD), and categoric variables were expressed as number of participants and per cent. Comparisons between AAA and control were performed using the unpaired Student’s t-test, a non-parametric means test (Mann–Whitney U-test) and Fisher’s exact test. Univariate linear regression analyses were used to investigate the relationships between baseline characteristics and left ventricular function in subjects with AAA. Non-normally distributed variables were transformed using a logarithmic transformation. Values of $p < 0.05$ were considered statistically significant.

3 | RESULTS

3.1 | Demographic data

The demographics and medical history of the study subjects are shown in Table 1. There was no difference in age between the AAA and the control groups. The AAA group had higher mean BMI ($p < 0.01$) and weight ($p < 0.05$), however, BSA was similar between the groups. No difference was apparent in SBP, but DBP was significantly higher in the AAA group ($p < 0.05$).

Based on the questionnaire, the AAA group had greater incidence than controls of symptoms of ischaemic heart disease ($p < 0.01$), hypertension ($p < 0.001$) and cerebrovascular events ($p < 0.001$), as well as hyperlipidaemia ($p < 0.001$). In addition, a greater proportion of AAA patients were current or former smokers ($p < 0.001$). There was no difference in the reported prevalence of diabetes mellitus.

3.2 | Echocardiographic parameters

The aortic diameters and other echocardiographic parameters of the subjects are shown in Table 2. The mean abdominal aortic diameter in AAA patients was 42 cm, with a range of 30 to 58 mm. The AAA group had a wider ascending aorta (35-9 mm versus 33-7 mm) and aortic sinus (37-7 mm versus 35-4 mm) compared to controls (both $p < 0.01$). However, there was no difference between the groups in the diameter of the aortic arch, nor in the prevalence of aortic regurgitation.

No differences were observed between groups in heart rate (HR), left ventricular end systolic volume (LVESV), left ventricular end diastolic volume (LVEDV), stroke volume (SV) or E/A ratio. Subjects with AAA had thicker septal and posterior walls compared to controls ($p < 0.05$, $p < 0.001$, respectively). Left ventricular ejection fraction (LVEF) and global longitudinal strain (GLS) were lower in the group with AAA ($p < 0.05$ for both). Moreover, mitral annular plane systolic excursion (MAPSE) was reduced, and E/e’ was higher, in subjects with AAA ($p < 0.05$ for both). However, there was no difference in the number of participants with abnormally decreased LVEF, GLS or increased E/e’ between subjects with AAA and controls. In the AAA group 29% had an abnormally decreased LVEF compared to 21% in controls, 29% in the AAA group had a decreased GLS compared to 21% in controls and moreover, 30% in the AAA group had an increased E/e’ compared to 22% in the controls.

Univariate linear regression analysis of echocardiographic parameters (Table 3) indicated that age was negatively associated with LVEF and with MAPSE. In addition, BSA was associated with the diameter of the aortic sinus, with left posterior wall thickness (LPWT)
and with left septal wall thickness (LSWT). Furthermore, BMI was associated with the diameter of the ascending aorta, with E/e’, with LPWT and with LSWT. DBP was associated with the diameter of the aortic sinus, while current or former smoking was associated with the diameter of the ascending aorta. Diabetes was negatively associated with LVEF. Ischaemic heart disease showed a negative association with MAPSE.

### Discussion

This echocardiographic study shows that males with small AAA have increased left ventricular wall thickness, reduced left ventricular systolic function (assessed as LVEF, GLS and MAPSE), and higher filling pressures to the left ventricle (E/e’), compared to a control group without AAA. So far as we know, this is the first study to assess left ventricular function and cardiac comorbidity in patients with AAA.

About 29 per cent of participants in the AAA group had reduced systolic and/or diastolic function compared with 21–22 per cent in the control group depending on the measure used, EF-GLS-E/e. Even though ischaemic heart disease was reported four times more often and hypertension two times as frequently in participants with AAA as in controls, there were only mild differences in left ventricular morphology and function. However, the AAA group in our study had a mean AAA diameter of 42 mm, which is classified as a small AAA (JCS, 2013). A patient with an AAA diameter exceeding 55 mm should be sent to surgery, except when patient characteristics make such intervention too risky (Wanhainen et al., 2019). Thus, several patients with AAA over 55 mm had to be surgically treated and did not participate in the study. The exclusion criteria might also have influenced our results, because we excluded participants with more advanced AAA disease who in addition might have had more severe left ventricular dysfunction.

We observed larger diameters of the aortic sinus and ascending aorta in subjects with AAA. Studies have shown the association of higher DBP with increased aortic root diameter (Lam et al., 2010; Palmieri et al., 2001), in accordance with our findings of both higher DBP and increased aortic root diameter in males with AAA compared to controls. Univariate linear regression analysis of our data likewise demonstrated an association between DBP and the diameter of the aortic sinus. The occurrence of a widened aortic root and ascending aorta in AAA patients may indicate that AAA is not a local disease but affects the arterial system in general. Aortic dimensions at the level of the aortic sinus and the ascending aorta are strongly

### Table 2: Echocardiographic parameters

<table>
<thead>
<tr>
<th></th>
<th>AAA</th>
<th>Control</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aorta abdominalis (mm)</td>
<td>42 ± 7</td>
<td>≤29</td>
<td></td>
</tr>
<tr>
<td>Sinus aorta (mm)</td>
<td>38 ± 4</td>
<td>35 ± 2</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ascendens aorta (mm)</td>
<td>36 ± 4</td>
<td>34 ± 5</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Arcus aorta (mm)</td>
<td>27 ± 4</td>
<td>27 ± 4</td>
<td>ns</td>
</tr>
<tr>
<td>GLS (%)</td>
<td>18.5 ± 3.4</td>
<td>19.6 ± 2.8</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LVEF (%)</td>
<td>54.8 ± 8.4</td>
<td>57.0 ± 7.4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LVEDV (ml/m²)</td>
<td>49.0 ± 11.3</td>
<td>48.6 ± 10.4</td>
<td>ns</td>
</tr>
<tr>
<td>LVESV (ml/m²)</td>
<td>22.3 ± 7.4</td>
<td>20.9 ± 5.8</td>
<td>ns</td>
</tr>
<tr>
<td>SV (ml)</td>
<td>54 ± 14</td>
<td>56 ± 13</td>
<td></td>
</tr>
<tr>
<td>MAPSE (mm)</td>
<td>11.8 ± 2.0</td>
<td>12.6 ± 2.1</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>E/e’</td>
<td>13.0 ± 5</td>
<td>11.0 ± 4.0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>E/A</td>
<td>0.89 ± 0.29</td>
<td>0.94 ± 0.27</td>
<td>ns</td>
</tr>
<tr>
<td>LPWT (mm)</td>
<td>10.9 ± 1.4</td>
<td>10.4 ± 1.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LSWT (mm)</td>
<td>11.7 ± 1.9</td>
<td>11.0 ± 1.7</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>LVEF &lt; 52, n (%)</td>
<td>58 (29-1)</td>
<td>23 (21-3)</td>
<td>ns</td>
</tr>
<tr>
<td>GLS &lt; 18, n (%)</td>
<td>58 (29-1)</td>
<td>22 (20-6)</td>
<td>ns</td>
</tr>
<tr>
<td>MAPSE &lt; 13, n (%)</td>
<td>141 (70-8)</td>
<td>69 (63-9)</td>
<td>ns</td>
</tr>
<tr>
<td>E/e &gt; 14, n (%)</td>
<td>59 (29-6)</td>
<td>24 (22-2)</td>
<td>ns</td>
</tr>
<tr>
<td>AR, n (%)</td>
<td>102 (51-3)</td>
<td>48 (44-4)</td>
<td>ns</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD for continuous variables and numbers of participants and per cent for categorical variables, n (%).

Abbreviations: e’, early diastolic mitral annulus velocity; E, transmitral E-wave velocity; GLS %, global longitudinal strain; LPWT, Left posterior wall thickness; LSWT, left septal wall thickness. AR, aortic regurgitation; LVEDV, left ventricle end diastolic volume; LVEF, left ventricle ejection fraction; LVESV, left ventricle end systolic volume; MAPSE, Mitral annular plane systolic excursion; ns, not statistically significant; SV, stroke volume.
TABLE 3  Relationships between echocardiographic variables and baseline characteristics in AAA patients: results of univariate regression analysis

<table>
<thead>
<tr>
<th>Explanatory variable</th>
<th>Sinus Aorta $\beta_1$, $R^2$</th>
<th>Ascending Aorta $\beta_1$, $R^2$</th>
<th>GLS $\beta_1$, $R^2$</th>
<th>LVEF $\beta_1$, $R^2$</th>
<th>LVESV $\beta_1$, $R^2$</th>
<th>MAPSE $\beta_1$, $R^2$</th>
<th>E/e' $\beta_1$, $R^2$</th>
<th>LPWT $\beta_1$, $R^2$</th>
<th>LSWT $\beta_1$, $R^2$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>$-0.07, 0.05$</td>
<td>$0.04, 0.001$</td>
<td>$-0.06, 0.003$</td>
<td>$-0.02*, 0.04$</td>
<td>$0.10, 0.01$</td>
<td>$-0.25***, 0.06$</td>
<td>$0.13, 0.02$</td>
<td>$0.03, 0.001$</td>
<td>$0.05, 0.01$</td>
</tr>
<tr>
<td>BSA</td>
<td>$0.24***, 0.06$</td>
<td>$0.13, 0.02$</td>
<td>$-0.11, 0.01$</td>
<td>$-0.05, 0.003$</td>
<td>$0.06, 0.003$</td>
<td>$0.06, 0.004$</td>
<td>$0.06, 0.003$</td>
<td>$0.22**, 0.05$</td>
<td>$0.18*, 0.03$</td>
</tr>
<tr>
<td>BMI</td>
<td>$0.05, 0.003$</td>
<td>$0.15*, 0.02$</td>
<td>$-0.12, 0.01$</td>
<td>$-0.13, 0.02$</td>
<td>$0.09, 0.01$</td>
<td>$-0.01, 0.000$</td>
<td>$0.18*, 0.03$</td>
<td>$0.31***, 0.10$</td>
<td>$0.29***, 0.09$</td>
</tr>
<tr>
<td>SBP</td>
<td>$-0.002, 0.000$</td>
<td>$0.04, 0.002$</td>
<td>$-0.02, 0.00$</td>
<td>$0.00, 0.00$</td>
<td>$0.05, 0.002$</td>
<td>$0.09, 0.009$</td>
<td>$-0.14, 0.02$</td>
<td>$0.12, 0.01$</td>
<td>$0.07, 0.004$</td>
</tr>
<tr>
<td>DBP</td>
<td>$0.21**, 0.04$</td>
<td>$0.14, 0.02$</td>
<td>$0.08, 0.006$</td>
<td>$0.06, 0.004$</td>
<td>$-0.07, 0.005$</td>
<td>$0.06, 0.004$</td>
<td>$-0.13, 0.02$</td>
<td>$0.09, 0.01$</td>
<td>$-0.01, 0.00$</td>
</tr>
<tr>
<td>Current or former smoker</td>
<td>$-0.04, 0.001$</td>
<td>$-0.20*, 0.04$</td>
<td>$-0.07, 0.001$</td>
<td>$-0.05, 0.003$</td>
<td>$-0.001, 0.00$</td>
<td>$-0.04, 0.002$</td>
<td>$0.14, 0.02$</td>
<td>$0.03, 0.001$</td>
<td>$0.001, 0.00$</td>
</tr>
<tr>
<td>Diabetes</td>
<td>$0.07, 0.01$</td>
<td>$-0.001, 0.00$</td>
<td>$-0.13, 0.02$</td>
<td>$-0.22**, 0.05$</td>
<td>$0.22**, 0.05$</td>
<td>$-0.11, 0.01$</td>
<td>$-0.001, 0.00$</td>
<td>$0.10, 0.01$</td>
<td>$0.10, 0.01$</td>
</tr>
<tr>
<td>Family AAA</td>
<td>$0.06, 0.004$</td>
<td>$0.10, 0.01$</td>
<td>$0.05, 0.003$</td>
<td>$0.15, 0.02$</td>
<td>$-0.10, 0.01$</td>
<td>$0.04, 0.002$</td>
<td>$-0.05, 0.003$</td>
<td>$0.15, 0.02$</td>
<td>$0.07, 0.01$</td>
</tr>
<tr>
<td>HL</td>
<td>$-0.07, 0.01$</td>
<td>$-0.12, 0.02$</td>
<td>$0.012, 0.00$</td>
<td>$0.02, 0.00$</td>
<td>$-0.002, 0.00$</td>
<td>$-0.01, 0.00$</td>
<td>$0.03, 0.001$</td>
<td>$-0.03, 0.001$</td>
<td>$0.11, 0.01$</td>
</tr>
<tr>
<td>IHD</td>
<td>$-0.08, 0.01$</td>
<td>$0.05, 0.002$</td>
<td>$-0.11, 0.01$</td>
<td>$-0.11, 0.01$</td>
<td>$0.04, 0.001$</td>
<td>$-0.20**, 0.04$</td>
<td>$0.14, 0.02$</td>
<td>$0.05, 0.003$</td>
<td>$0.14, 0.02$</td>
</tr>
<tr>
<td>HT</td>
<td>$0.02, 0.00$</td>
<td>$-0.05, 0.003$</td>
<td>$-0.14, 0.02$</td>
<td>$-0.05, 0.002$</td>
<td>$0.12, 0.01$</td>
<td>$0.09, 0.01$</td>
<td>$0.01, 0.01$</td>
<td>$0.06, 0.003$</td>
<td>$0.04, 0.002$</td>
</tr>
<tr>
<td>CVD</td>
<td>$0.04, 0.002$</td>
<td>$-0.01, 0.00$</td>
<td>$-0.04, 0.002$</td>
<td>$-0.02, 0.00$</td>
<td>$0.01, 0.00$</td>
<td>$0.01, 0.00$</td>
<td>$0.02, 0.001$</td>
<td>$-0.003, 0.00$</td>
<td>$0.12, 0.02$</td>
</tr>
</tbody>
</table>

Results are presented as estimated regression parameters $\beta_1$ and $R^2$.

*p < 0.05, **p < 0.01, ***p < 0.001

Abbreviations: BMI, body mass index; BSA, body surface area; CVD, history of symptomatic cerebrovascular disease; DBP, diastolic blood pressure; e', early diastolic mitral annulus velocity; E, transmitral E-wave velocity; Family AAA, aortic aneurysm in any close relative; GLS, global longitudinal strain; HL, history of hyperlipidaemia; HT, hypertension or taking blood pressure-lowering drugs; IHD, history of ischaemic heart disease; LPWT, left posterior wall thickness; LSWT, left septal wall thickness; LVEF, left ventricle ejection fraction; LVESV, left ventricle end systolic volume; MAPSE, Mitral annular plane systolic excursion; SBP, systolic blood pressure.
and-effect associations can be derived. Study was cross-sectional in design, and therefore, no direct cause-pants was based solely on self-reported data. Further, the present dominantly classified as small. The medical history of the partici-
pation having been recommended. Twenty-four per cent of males in our study with AAA con-
considered to be 85% among subjects with AAA, compared with only 37% in 2000). Our study found the proportion of current or former smokers re-
ered to be due to smoking (Jacomelli et al., 2016; Lederle et al., 2011). Moreover, an estimated 75% of all AAA cases were consid-
ered to have a small AAA (Wanhainen et al., 2019).

Elevated SBP is associated with an increased risk of cardiovascu-
lar disease. Hypertension causes more than 40% of cardiovascular deaths (Zhou et al., 2017). The males in our AAA cohort reported hypertension to a greater extent than controls, but SBP did not differ significantly between the groups. Not surprisingly, the AAA group received more treatment with blood pressure-lowering agents, as well as statins and antiplatelet agents. The European Society for Vascular Surgery recommends that blood pressure control, statins and antiplatelet therapy should be considered for all patients with a small AAA (Wanhainen et al., 2019).

Results of the questionnaire revealed that the AAA group was not only affected by aneurysm but was also more affected by other cardiovascular diseases, including ischaemic heart disease, cerebral vascular diseases, hyperlipidaemia and hypertension. This may not be remarkable, because AAA shares several risk factors in common with these diseases. One of the best-known risk factors for cardio-
From a lifestyle change in all cardiovascular diseases (Piepoli et al., 2016). While the underlying mechanisms of AAA development are unclear, smoking is strongly associated with development of the an-
erysm and is known to be its strongest risk factor (Nordon et al., 2011). Moreover, an estimated 75% of all AAA cases were consid-
ered to be due to smoking (Jacomeilli et al., 2016; Lederle et al., 2000). Our study found the proportion of current or former smokers to be 85% among subjects with AAA, compared with only 37% in the control group, confirming the association between smoking and AAA. Twenty-four per cent of males in our study with AAA con-
tinued smoking after being diagnosed with AAA, despite smoking cessation having been recommended.

### 4.1 Study limitations

This study is not without limitations. The study included only male subjects in an elderly age range, from 55 to 80 years, with AAA pre-
dominantly classified as small. The medical history of the particip-
ants was based solely on self-reported data. Further, the present study was cross-sectional in design, and therefore, no direct cause-
and-effect associations can be derived.

### 5 CONCLUSION

In conclusion, the presence of a small AAA is associated with slightly reduced left ventricular systolic and diastolic function in males. Larger diameters of the aortic sinus and ascending aorta observed in the AAA group suggest that AAA is connected to general vascular effects. Previous studies have shown a strong association between smoking and AAA, which we also observed in our study, in which nearly one in four males with AAA continued to smoke after the diagnosis.

More studies are needed to investigate the connection between AAA and cardiovascular disease, especially in females and in sub-
jects with AAA surpassing 55 mm.

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inations; to Helene Carlsson for administrative assistance; and to Martin Welander for recruitment of the participants. Prof. Toste Länne, who passed away before this manuscript was completed, contributed greatly to the project.

### CONFLICT OF INTEREST

The authors have no conflicts of interest.

### DATA AVAILABILITY STATEMENT

Data will be available at reasonable request.

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