Evidence of increased axillary blood flow velocity without increased handgrip strength and endurance in persons with a fibromuscular axillary arch

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\textbf{Background:} The purpose of this in vivo study was to compare axillary artery blood flow velocity, and maximal handgrip strength and endurance performance in young subjects with and without an axillary arch (AA).

\textbf{Materials and methods:} One hundred and fifty-six young adults were screened for the presence of an AA on their dominant arm side. After physical examination subjects were checked using diagnostic echography for the presence of an AA. Sixteen subjects with an AA and 15 without an AA had their axillary artery peak systolic velocity quantified in 3 different arm positions using Doppler ultrasound. Maximal handgrip strength and endurance performance was quantified in the same positions using a functional rehabilitation system.

\textbf{Results:} Mean peak systolic velocity was significantly higher in the AA group compared to controls in abduction/external rotation of the arm during muscle relaxation ($p = 0.003$) and contraction ($p = 0.01$). No significant differences between groups were found for maximal handgrip strength and endurance performance.

\textbf{Conclusions:} This study provides evidence for a transient axillary artery compression by the AA in a throwing position. This is not reinforced by additional contraction of the shoulder muscles along with the AA. Axillary artery compression does not influence maximal handgrip strength and endurance performance in symptom-free young adults. (Folia Morphol 2015; 74, 4: 486–492)

\textbf{Key words:} axillary arch, compression, ultrasound, peak systolic velocity, maximal grip strength, endurance

\textbf{INTRODUCTION}

The axillary arch (AA) muscle is a (fibro)muscular ‘bridge’ crossing transversally the axillary fossa. This supernumerary muscle or tendon extends from the lateral border of the latissimus dorsi muscle and inserts mostly close to the insertion of pectoralis major tendon overlying the axilla’s neurovascular bundle [39, 40]. Review of the literature reveals that the AA may have a variable incidence ranging from 0.3\% to 37.5\% (Table 1) [1–3, 9–11, 15–18, 20, 22, 23, 26–30, 34, 39, 41–43, 46]. In most cases it ends with a humeral insertion close to the tendon or fascia.
of the pectoralis major muscle, but it may also insert between the pectoralis major tendon and the intertubercular groove, on the tunica vaginalis in particular [4]. Other insertions reported in literature are the coracoid process, the fascia of the coracobrachialis and pectoralis minor muscle [13, 36].

By the fact that the AA always runs superficially and perpendicularly to the vessels and nerves of the brachial plexus its presence has been of interest to surgeons dealing with lymph node dissection and/or neurovascular compression [22, 33]. Although its structure (muscular or fibrous), its course and insertion site can be visualised using medical imaging such as computed tomography and magnetic resonance imaging, detailed ultrasonographic descriptions are scarce [12]. Clinically and physiologically, the AA has been implicated in thoracic outlet syndrome, and associated sensory and motor strength changes [3, 21, 25]. The neurovascular bundle is supposed to be most strongly compressed by stretching the AA with the arm in abduction and external rotation. In a recent consecutive series of 148 patients diagnosed with neurogenic thoracic outlet syndrome it was found that 51% had definite arterial occlusion [24]. Since hypertrophied muscles of the arm-shoulder complex may compress the axillary veins and artery, it is of paramount importance to gain a better insight in the role of the AA with regard to (intermittent) circulatory deficiency [19].

To the knowledge of the authors, only 1 study investigated the influence of an AA on the blood flow of the axillary artery in a group of 9 subjects. The authors concluded that the artery showed no changes in haemodynamic parameters in different positions [29]. However, without ultrasound-based diagnosis and a low relative number of subjects results might be biased. Furthermore there is very limited evidence that having an AA also may act upon physiologic muscle

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**Table 1. Incidence of the axillary arch muscle in different population groups**

<table>
<thead>
<tr>
<th>Authors, year</th>
<th>Approach</th>
<th>N</th>
<th>Incidence</th>
<th>Country</th>
</tr>
</thead>
<tbody>
<tr>
<td>Meckel, 1825</td>
<td>Dissection</td>
<td>30</td>
<td>3.3%</td>
<td>–</td>
</tr>
<tr>
<td>Langer, 1846</td>
<td>Dissection</td>
<td>40</td>
<td>25.0%</td>
<td>Austria</td>
</tr>
<tr>
<td>Wood, 1865</td>
<td>Dissection</td>
<td>102</td>
<td>5.8%</td>
<td>–</td>
</tr>
<tr>
<td>Calori, 1866</td>
<td>Dissection</td>
<td>144</td>
<td>1.5%</td>
<td>–</td>
</tr>
<tr>
<td>Perrin, 1871</td>
<td>Dissection</td>
<td>29</td>
<td>24.1%</td>
<td>–</td>
</tr>
<tr>
<td>Princeteau, 1892</td>
<td>Dissection</td>
<td>+100</td>
<td>12.0%</td>
<td>–</td>
</tr>
<tr>
<td>La Double, 1897</td>
<td>Dissection</td>
<td>95</td>
<td>6.3%</td>
<td>France</td>
</tr>
<tr>
<td>Pichler, 1916</td>
<td>In vivo palpation</td>
<td>4200</td>
<td>4.0%</td>
<td>–</td>
</tr>
<tr>
<td>Wagenseil, 1927</td>
<td>Dissection</td>
<td>32</td>
<td>37.5%</td>
<td>China</td>
</tr>
<tr>
<td>Kasai and Chiba, 1977</td>
<td>Dissection</td>
<td>+100</td>
<td>M: 9%, T: 17%</td>
<td>–</td>
</tr>
<tr>
<td>Haagensen, 1986</td>
<td>In vivo surgery</td>
<td>?</td>
<td>7.7%</td>
<td>Europe</td>
</tr>
<tr>
<td>Serpell and Baum, 1991</td>
<td>In vivo surgery</td>
<td>2000</td>
<td>0.3%</td>
<td>New Zealand</td>
</tr>
<tr>
<td>Clarys et al., 1996</td>
<td>Echography</td>
<td>1321</td>
<td>8.5%</td>
<td>Belgium</td>
</tr>
<tr>
<td>Kalaycioglu et al., 1998</td>
<td>Dissection</td>
<td>60</td>
<td>1.7%</td>
<td>Turkey</td>
</tr>
<tr>
<td>Miguel et al., 2001</td>
<td>Dissection</td>
<td>100</td>
<td>3.3%</td>
<td>Spain</td>
</tr>
<tr>
<td>Mérida Valasco et al., 2003</td>
<td>Dissection</td>
<td>32</td>
<td>6.3%</td>
<td>Spain</td>
</tr>
<tr>
<td>Turgut et al., 2005</td>
<td>Dissection</td>
<td>26</td>
<td>3.8%</td>
<td>Turkey</td>
</tr>
<tr>
<td>Georgiev et al., 2007</td>
<td>Dissection</td>
<td>56</td>
<td>3.6%</td>
<td>Bulgaria</td>
</tr>
<tr>
<td>Risk and Harbaugh, 2008</td>
<td>Dissection</td>
<td>35</td>
<td>8.6%</td>
<td>United States</td>
</tr>
<tr>
<td>Bartone et al., 2008</td>
<td>Dissection</td>
<td>78</td>
<td>11.5%</td>
<td>Argentina</td>
</tr>
<tr>
<td>Van Hoof et al., 2008</td>
<td>Ultrasound</td>
<td>640</td>
<td>3.0%</td>
<td>Belgium</td>
</tr>
<tr>
<td>Uzansel et al., 2010</td>
<td>Dissection</td>
<td>50</td>
<td>M: 7.0%, T: 7.0%</td>
<td>Turkey</td>
</tr>
<tr>
<td>Guy et al., 2011</td>
<td>MRI</td>
<td>1054</td>
<td>6.0%</td>
<td>United States</td>
</tr>
<tr>
<td>Provyn et al., 2011</td>
<td>In vivo palpation</td>
<td>239</td>
<td>8.4%</td>
<td>Belgium</td>
</tr>
</tbody>
</table>

MRI — magnetic resonance imaging; M — muscular; T — tendinous
parameters such as isometric strength, endurance and motor control but findings have been inconsistent [4]. Since it is still unclear whether an AA may lead to an arterial compression syndrome and/or associated motor strength changes, the purpose of this study was to compare axillary artery blood flow velocity and maximal handgrip strength and endurance in a group of subjects with and without AA diagnosed using ultrasound imaging.

**MATERIALS AND METHODS**

**Subject recruitment**

Clinically, the AA can be revealed by physical examination through observation and palpation of the axilla, notably while performing an internal rotation resistance test in 90° abduction [9]. One hundred and fifty-six students of the Vrije Universiteit Brussel were independently screened by two manual therapists for the presence of an AA on their dominant arm side. Only those students that were found to be positive for an AA by at least one of the investigators were enrolled. In order to exclude type I errors (false positives) an additional ultrasound examination was performed by two musculoskeletal radiologists on a ‘Toshiba Apio XG’ echographic device (Toshiba Medical Systems Europe, linear probe: 12 MHz). A detailed description of the echographic diagnosis of the arch has been described elsewhere [3]. Based on the ultrasound image AA’s were categorised into two types: muscular or fibrous. A fibrous AA was defined as a clear solid white strand that made a connection between the pectoralis major muscle and the latissimus dorsi muscle. A schematic overview of the regional anatomy of the axilla with corresponding echographic imaging is presented in Figure 1.

A control group matched for number of subjects, age, gender and arm dominance (Table 2) was equally examined by echography by the two radiologists in order to rule out type II errors (false negatives). The study was conducted in accordance to the World Medical Association’s Declaration of Helsinki (1964). The protocol was approved by the local ethical committee of the University Hospital Brussels and all participants provided written informed consent.

**Peak systolic velocity**

Because of its anatomical location — and according to the position of the arm — the AA may hinder the axillary artery and thus affect the blood flow through the vessel [3, 4]. This may result in a mechanical obstruction (stenosis). Doppler examination of an artery distal to a stenosis typically shows an increase in peak systolic velocity (PSV). The criteria used to define stenosis vary between laboratories, but overall, a doubling of the PSV-values distally to the narrowing compared with the velocity in the proximal segment is usually defined as a stenosis of 50% or more [6]. Using PSV as a parameter to determine stenosis has also proved to be highly sensitive and specific [35]. In the present study PSV-values were determined by a vascular radiologist using a continuous wave Ultrasound Doppler device (Toshiba Apio XG, Toshiba Medical Systems Europe, linear probe: 7 MHz).

Measurements were carried out in the following three arm positions: neutral, abduction/external rotation without resistance and abduction/external rotation with resistance. Flow analysis in neutral position through the vessel [3, 4]. This may result in a mechanical obstruction (stenosis). Doppler examination of an artery distal to a stenosis typically shows an increase in peak systolic velocity (PSV). The criteria used to define stenosis vary between laboratories, but overall, a doubling of the PSV-values distally to the narrowing compared with the velocity in the proximal segment is usually defined as a stenosis of 50% or more [6]. Using PSV as a parameter to determine stenosis has also proved to be highly sensitive and specific [35]. In the present study PSV-values were determined by a vascular radiologist using a continuous wave Ultrasound Doppler device (Toshiba Apio XG, Toshiba Medical Systems Europe, linear probe: 7 MHz). Measurements were carried out in the following three arm positions: neutral, abduction/external rotation without resistance and abduction/external rotation with resistance. Flow analysis in neutral position

**Table 2. General characteristics of the subjects according to study group**

<table>
<thead>
<tr>
<th>With AA (n = 16)</th>
<th>Without AA (n = 16)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age [years]</td>
<td>22.0 ± 2.3</td>
</tr>
<tr>
<td>Height [cm]</td>
<td>171.0 ± 5.5</td>
</tr>
<tr>
<td>Weight [kg]</td>
<td>68.3 ± 6.2</td>
</tr>
<tr>
<td>Gender</td>
<td>M: 8, F: 8</td>
</tr>
<tr>
<td>Arm dominance</td>
<td>R: 14, L: 2</td>
</tr>
</tbody>
</table>

Data are represented as mean ± standard deviation; AA — axillary arch; F — female; L — left; M — male; R — right.
was performed in supine position with the dominant arm (unsupported on the table) hanging comfortably in external rotation of 70–80°. The abduction/external rotation position was standardised by asking the subjects to make a fist and hold it exactly above the ear. For the abduction/external rotation position with resistance subjects had to counteract an external rotation momentum created by a 3 kg weight hanging from their elbow.

Handgrip strength and endurance

Strength and endurance of the forearm flexors was measured using a BTE® primus work simulator (Baltimore Therapeutic Equipment Co., Hanover, MD, USA). Subjects were positioned with their back on a physiotherapy table with their dominant arm in an abduction/external rotation position. This position was standardised by means of a plastic splint that formed a 90° angle between the proximal and distal segments of the upper limb. Additionally the arm was fixed with straps in order to avoid muscle compensation.

Maximal grip strength was measured 3 times consecutively while encouraging the subjects verbally. Grip strength consistency was evaluated using the coefficient of variation score automatically produced by the BTE® system. The highest peak value of the 3 test sessions was used as maximal handgrip strength.

For the grip endurance tests, the resistance was set at half the maximal grip strength value (individualised). Subjects performed 2 dynamic endurance tests both at a pace of 30 repetitions per minute. Both tests, one performed without weight the other with a weight of 3 kg acting on the upper limb, were carried out at a set time interval of 10 min (subjects were detached from splint and straps). During this period weight and height were measured.

Handgrip tests and Doppler measurements were recorded 1 month apart. During this period 1 subject without an AA dropped out due to a radius fracture.

Statistical analysis

Normality of all data was verified using Kolmogorov-Smirnov goodness of fit tests. Differences between groups were examined using unpaired t-tests. Within group differences were assessed using repeated measures ANOVA with Bonferroni correction. Comparison of endurance tests within groups was done using paired t-tests. All data were analysed by using IBM® SPSS® Statistics version 20 (2011, SPSS Inc., New York, USA). Group data are expressed as mean ± standard deviation and statistical significance was set at p < 0.05.

RESULTS

After physical screening, 31 out of 156 students were identified as possibly having an AA on their dominant arm side. After echography 16 (10.3%) subjects were confirmed with an AA in the axillary fossa of their dominant arm. Nine of them presented with an AA bilaterally. Five individuals had a clear muscular AA (3.2%) (4 bilateral and 1 unilateral). The cross-sectional diameters of the muscular type AA’s varied 40–80 mm in relaxed state with a maximum of 150 mm in a tense state. Eleven (7.1%) subjects showed evidence of a fibromuscular slip connecting the latissimus dorsi muscle with the pectoralis major muscle.

The AA group showed significantly higher PSV-values compared to the group without an AA in the abduction/external rotation positions of the arm (Table 3). In the AA group the latter positions increased the PSV-values with 50% compared to those in neutral position.

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No group differences for maximal handgrip strength and endurance of the forearm flexors were found (Table 4). Contraction of the shoulder muscles did not significantly influence grip endurance.

Figure 2. Ultrasound image of the axilla in a 20-year-old man with a muscular axillary arch (AAM). The axillary arch joins the superior portion of the latissimus dorsi muscle (left) and the inferior portion of the pectoralis major muscle (right). The axillary neurovascular bundle is noted below the AAM; P — pectoralis major muscle; LD — latissimus dorsi muscle; V — axillary vein(s); A — axillary artery.
DISCUSSION

The AA is undoubtedly one of the most common and most discussed anatomical variants of the human body and has attracted the interest of many for its possible clinical implications related to the compression of the axillary neurovascular bundle [9, 14, 41]. To our knowledge, this is the first report comparing blood flow velocity between subjects with and without AA using diagnostic ultrasound.

The incidence found in the present study by ultrasoundography is in line with previously reported ranges determined by medical imaging [10]. It has previously been suggested that clinical examination of the axilla is considered reliable for detecting an AA [4, 9, 32]. However, to the knowledge of the authors, the sensitivity and specificity of a particular test for the detection of a (muscular or fibrous) AA has not yet been reported in literature. It is therefore suggested that ultrasonography might be clinically important as this evaluation makes it possible to distinguish muscular from fibrous type AA’s in relation to underlying neurovascular structures.

The main finding of the present study is that persons with an AA exhibit a substantial increase of the blood flow velocity in the axillary artery in an abduction/external rotation position of the arm. This finding is in contrast with that of Provyn et al. [29] who found no functional vascular implication in their sample, but in agreement with a number of others who suggested that the axillary artery could be compressed by an AA [22, 31, 34, 36]. The increase in PSV-values suggests a haemodynamically significant reduction (≥ 50%) in axillary artery diameter similar to what is found in stenosis. It is postulated that the compression of the structures in the axillary fossa (i.e. between the skin and the humeral head) in subjects with an AA has 2 causes (Fig. 1). First, the AA divides the axillary fossa anatomically in a superficial part consisting essentially of subcutaneous adipose tissue...
and a deep part containing the neurovascular structures and internal rotator muscles of the shoulder. Abduction of the arm reduces the space between the AA and the humeral head by stretching the structures in the axilla. Second, externally rotation of the arm translates the humeral head forward further narrowing the axillary fossa space. Furthermore, our results suggest that the functional occlusion of the axillary artery is position dependent and not influenced by the additional contraction of the shoulder muscles (along with the AA). With regard to this observation it has previously been shown that the AA and the latissimus dorsi muscle share common innervation [37]. Although latissimus dorsi muscle is considered to generate a posterior force generated to the humeral head that helps resist anterior humeral head translation [8], it is suggested that it cannot significantly expand the axillary space in subjects with an AA. Finally and in contrast to fibrous AA’s, the muscular type AA can actively narrow the axillary space. Therefore, further research should elucidate whether the haemodynamic occlusion of the axillary artery is type dependent, as two-thirds of the subjects in our sample had a fibrous AA type.

Restriction of blood flow distal to a mechanical stenosis may reduce the endurance of distal muscles due to oxygen depletion [44, 45]. It has been reported that non-traumatic vascular complications (including stenosis) in athletes may lead to impaired performance [7]. Our results did not indicate any difference in maximal strength nor endurance between subjects with and without an AA. It might be argued that the level of difficulty for the endurance tests (50% of 1 RM) was not high enough to detect differences between groups as our subjects were clinically symptom-free. Moreover given the young age of our test subjects it can be assumed that the hand flexors have sufficient anastomotic support in order to compensate for the intermittent stenosis of the axillary artery in abduction/external rotation. Therefore future investigations should preferably focus on elucidating the relation between the compression of the artery and endurance with and without neurogenic signs.

This study is not without limitations. For practical reasons the test positions of the Doppler and strength measurements were similar but not identical. Since it has been shown that changes in arm position may influence axillary blood flow [38], we cannot completely rule out haemodynamic differences between positions.

Our primary outcome measure, handgrip strength, is a well-validated and reliable surrogate measurement for more complicated measures of muscle strength in the arm [5]. It must be remembered that the anterior compartment of the arm is mainly irrigated by the ulnar artery, a direct branch of the brachial artery. Since the forearm flexors are not directly perfused by the axillary artery mechanical obstructions distal from the AA might influence results. Therefore, and under ideal circumstances, strength measurements of muscles directly perfused by the axillary artery might be more sensitive. Since we did not assess the blood supply of shoulder muscles including the variations in the branching pattern of the axillary artery this approach was not feasible in the present study.

CONCLUSIONS

In conclusion, the incidence of an AA (10%) determined by echography in a homogeneous Belgian sample is consistent with previous data found in similar groups. Our results suggest that the axillary artery is significantly compressed in the abduction/external rotation position of the arm in subjects with an AA. This compression is not necessarily reinforced by additional contraction of the shoulder muscles. Finally, the presence of an AA does not alter the strength and endurance of the forearm flexors in symptom-free subjects.

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REFERENCES


