An anatomical investigation of rare upper limb neuropathies due to the Struthers’ ligament or arcade: a meta-analysis

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Background: The Struthers’ ligament (SL) is a fibrous band that originates from the supracondylar humeral process and inserts into the medial humeral epicondyle, potentially compressing both the median nerve and brachial artery. The controversial Struthers’ arcade (SA) is a musculotendinous band found in the distal end of the arm that might compress the ulnar nerve. This study aimed to evaluate the pooled prevalence estimate of the SL and SA, and their anatomical features.

Materials and methods: A meticulous search of major electronic medical databases was carried out regarding both structures. Applicable articles (and all relevant references) were analysed. Data from the eligible articles was extracted and evaluated. The quality and the potential risk of bias in the included studies were assessed using the AQUA tool.

Results: The arcade was reported in 13 studies (510 arms), whereas the ligament in 6 studies (513 arms). The overall pooled prevalence estimate of the ligament was 1.8%, and 52.6% for the arcade. Most frequently, the ulnar nerve was covered by a tendinous arcade (42.2%). In all cases, the ligament inserted into the medial humeral epicondyle, but had various origins. Only 1 study reported compression of the median nerve by the ligament, whilst another contradicted this view.

Conclusions: Although the SL is rare, and the SA is a valid anatomical entity (though with a variable presentation), clinically meaningful neurovascular entrapments caused by these structures are infrequent. Nonetheless, a better understanding of each may be beneficial for the best patient outcomes. (Folia Morphol 2021; 80, 2: 255–266)

Key words: Struthers’ arcade, Struthers’ ligament, meta-analysis

INTRODUCTION

Neurovascular compressions of the upper limb may have highly variable clinical manifestations including pain, numbness, weakness and muscular atrophy [1]. Fortunately, the entrapment site is often easily localised with careful physical examination and/or radiographic imaging [1]. Rare instances of such syndromes have been attributed to two anatomical structures: the Struthers’ ligament (SL) and the Struthers’ arcade (SA). These two structures are frequently confused, and some contention exists pertaining to their prevalence. Sir John Struthers described 9 arcades (a series of mus-
culotendinous and fibrous arches) in the arm — eight associated with the median nerve, and one with the ulnar nerve [2]. The eighth of this series was a fibrous structure known as the SL, and attached to a bony spur on the humerus. The ninth, known nowadays as the SA, was a fibrous band at the brachial fascia, and not anchored to any bony elements [2].

The SL typically begins at a bony projection approximately two inches above the medial epicondyle on the anteromedial aspect of the humerus, labelled the supracondylar process (or spur), which can usually be identified on X-ray imaging [33]. The ligament itself extends from this process, and attaches to the medial humeral epicondyle. The brachial artery, the median nerve, or both can run beneath this fibrous band. Initial descriptions suggested a prevalence of 1% in the human population [3]. Although it is an uncommon feature, its existence is undisputed. However, it has been implicated in causing a rare compression of the neurovascular entities, causing paraesthesia and numbness associated with forearm claudication or median nerve dysfunction [5]. A surgical procedure involving release of the entrapped element, in combination with excision of the SL and its bony spur, effectively eliminates all the clinical symptoms permanently [1].

The SA is a more disputed anatomical structure, with highly variable descriptions and classifications [12]. Kane et al. [24] were the first to apply Struthers’ work and define the fibrous canal (definition as applied herein) with a roof formed by a deep fascial thickening, an anterior border at the medial intermuscular septum, and a lateral border at the humerus and the muscular fibre covering of the triceps brachii. Several subsequent reports have supported the existence of this structure to various degrees; however, the discrepancies in findings may be attributable to the differences in definition [15, 20, 34]. Alternatively, other authors [7, 32, 40] debate its existence altogether, suggesting that the previous findings are only anatomical variations of the intermuscular septum and the forearm fascia. As such, it is important not only to assess the prevalence of this structure, but also the clinical presentation, and its possible variations [37]. The disagreements regarding the SA extend to its role as a possible site for entrapment. Although it is unlikely as a primary site for entrapment, most tend to agree that it is a factor in recurrent ulnar neuropathy after an anterior transposition of the nerve at the elbow [14, 25, 31].

This study seeks to evaluate the differences from an anatomical perspective, establish the pooled prevalence estimate (PPE) of both the SL and the SA, assess their involvement in the median/ulnar nerve entrapments, respectively, and provide the answer as to whether the disputed SA is a valid anatomical structure. Becoming acquainted with the said variants is of immense importance to physicians encountering unusual upper limb neural entrapments that cannot be explained by more commonly existing pathologies.

**MATERIALS AND METHODS**

This study is a systematic review and meta-analysis, level of evidence: II.

**Search strategy**

An extensive search on the SA and the SL, as well as their anatomy, was conducted on PubMed, Embase, ScienceDirect and Web of Knowledge databases. The following search terms applied: “Struthers’ ligament” OR “Ligament of Struthers” OR “supracondylar canal” OR “supracondylar spur” OR “supracondylar process” OR “supratrochlear spur” OR “avian spur” OR “Arcade of Struthers” OR “Struthers’ Arcade.” No restrictions were set to date or language of the original publication. Additionally, all references in the included articles were assessed to identify any other potentially eligible studies.

**Study selection criteria**

Eligibility for inclusion was governed by the following criteria: cadaveric or imaging studies containing information about the SL or the SA — both anatomically and clinically. Case reports, conference abstracts, letters to editors, reviews, or studies containing irrelevant or incomplete data about the SL or the SA were not considered.

**Eligibility assessment**

The authors (E.M., M.P.Z., J.R.P., L.N.K., M.G.) completed an independent review of all the included studies. Any disagreements were settled by consensus, where necessary also involving a consultation with the authors of the original study. Any studies published in a language not fluently spoken by the reviewing authors were translated by medical professionals fluent in both the original language of the manuscript and English.

**Data extraction**

The extraction of data from the included studies was performed separately by independent reviewers.
The following data was extracted: country of study origin, method, total number of patients/specimens with the SL/SA, as well as characteristics of modality. Elements of interest included laterality, typical vs. atypical presentation and type, morphology, relation to associated nerve, extent of compression, and insertion (the SL).

Quality assessment
The quality assessment was completed by independent reviewers by utilising the Anatomical Quality Assurance tool (the AQUA Tool), a versatile instrument capable of appraising anatomical studies [23]. This method employed a “risk of bias” table assessing the five domains: (1) Aim and subject characteristics; (2) Study design; (3) Characterisation of methods; (4) Descriptive anatomy; and (5) Results reporting. Each criterion level of bias was deemed “High”, “Low,” or “Unclear” in accordance to “Yes” or “No” answers to specific determining questions. Conditions where “Yes” was selected identified a “Low” risk of bias, whereas a “No” answer suggested a “High” risk. Any disagreements were resolved with discussions, or by involving an additional reviewer.

Statistical analysis
All the extracted data was processed using MetaXL version 5.3 (EpiGear International, Australia) as a meta-analysis with random-effects model. The PPE of the SL and the SA, respectively, was the primary measure of this study, with subsequent analysis by subgroups.

Heterogeneity was tested for using the $\chi^2$ and Higgins I$^2$ tests. A significant heterogeneity was identified from a p-value of $< 0.10$ in the $\chi^2$ test [22]. Heterogeneity was determined from the I$^2$ test according to the following scheme: 0% to 40% may not be present; 30% to 60% possible indications of moderate heterogeneity; 50% to 90% likely meaningful heterogeneity; and 75% to 100% suggests considerable heterogeneity [22].

In order to investigate possible sources of heterogeneity, subgroup analyses were completed to consider the effect of geographical distribution and modality. Confidence intervals were utilised to illustrate any determined statistical differences between two or more subgroups. Conclusions regarding statistical insignificance could be drawn if any such intervals overlapped [22].

Results
Study identification
The study selection process is illustrated in Figure 1. Initially, 891 articles were identified according to the specified parameters across all major electronic databases. Additional 31 articles were included when the cited articles of the previous group were checked. Of all the articles, 124 were identified as potentially meeting the inclusion criteria, from which 106 were deemed ineligible, for reasons such as being case reports/series, containing irrelevant/incomplete/no original data or were letters/commentaries to the editor. Therefore, 18 studies were utilised for this meta-analysis (5 pertaining to the SL, 12 to the SA and 1 study to both the SA and the SL).

Characteristics of the included studies
The tables outline the characteristics of the included studies in this meta-analysis. The 6 studies pertaining to the SL (n = 513 upper limbs) were conducted from 1983 to 2017. The 13 studies reporting on the SA (n = 510 upper extremities) were published from 1991 to 2016. The prevalence rates of the relevant structures are reported in (Tables 1, 2).

Prevalence of the SL
A complete assessment of the SL was completed according to a subgroup analysis by geography,
laterality, reported median nerve compression, and insertion, the results of which can be found, respectively. The geographical analysis differentiated all the studies (PPE 1.8%; 95% confidence interval [CI] 0.1–5.2%) from cadaveric studies (PPE 2.3%; 95% CI 0.0–7.4%), as well as those reporting from North America (PPE 0.8%; 95% CI 0.0–2.6%) (Table 3).

Four studies (Table 4) included which side the SL was present on. The SL appears slightly more often on the right side (55.8%; 95% CI 24.7–84.8) than the left (44.2%; 95% CI 15.2–75.3).

Gessini’s surgical study [19] from Italy supported the SL as a contributor to median nerve compression, whereas Gunther’s cadaveric study [21]...
from the United States did not support this finding (Table 5).

Lastly, 3 studies (Table 6) outlined the distal insertion point of the SL. In all cases, the SL was found to terminate at the medial humeral epicondyle. Notwithstanding, the origin of the SL varied in all those three instances, as in 1 case it was attached to the supracondylar humeral process, in another into the anteromedial surface of the humerus (with no bony spur present) and into the brachialis muscle in the last case (Fig. 2).

Prevalence of the SA

Similarly to the analysis of the SL, assessment of the SA was divided by subgroups — geographical prevalence, atypical prevalence, atypical type, morphology, relation to the ulnar nerve, and ulnar nerve compression.
Table 7. The modality and geographical distribution of the Struthers arcade (SA) studies

| Subgroup          | Number of studies (no. of subjects) | Pooled prevalence of SA; % (95% CI) | I²; % (95% CI) | Cochran's Q, p-value |
|-------------------|-------------------------------------|-------------------------------------|----------------|---------------------|
| Overall           | 13 (510)                            | 52.6 (27.1–77.5)                    | 96.9 (95.8–97.7)| 381.8, p < 0.001    |
| Cadaveric         | 11 (388)                            | 59.6 (35.0–82.1)                    | 95.4 (93.4–96.8)| 218.4, p < 0.001    |
| North America     | 5 (134)                             | 69.4 (32.3–97.1)                    | 93.7 (88.3–96.7)| 64.0, p < 0.001     |
| Asia              | 4 (248)                             | 45.2 (0.0–94.2)                     | 98.2 (97.1–98.9)| 169.7, p < 0.001    |
| South America     | 2 (100)                             | 68.0 (0.0–100.0)                    | 99.1 (98.1–99.5)| 105.6, p < 0.001    |
| Europe            | 2 (28)                              | 15.1 (0.0–59.1)                     | 83.2 (30.0–96.0)| 6.0, p = 0.015      |

CI — confidence interval

Table 8. The definitions of the Struthers arcade applied in this meta-analysis.

| Author                  | Year | Definition                                                                                     |
|-------------------------|------|----------------------------------------------------------------------------------------------|
| Kane et al. [24]        | 1973 | Fibrous canal with roof formed by a deep fascial thickening, an anterior border at the medial  |
|                         |      | intermuscular septum, and a lateral border at the humerus and the muscular fibre covering      |
|                         |      | of the triceps brachii. (Considered as the “classical” in this study.)                         |
| Al-Qattan and Murray [2]| 1991 | “Classical” definition provided by Kane et al. OR                                               |
|                         |      | Multiple ligaments of the thickened deep fascia and medial intermuscular septum passing       |
|                         |      | superficial and deep to the ulnar nerve OR                                                     |
|                         |      | Roof formed by the triceps muscular fibres alone. (Both considered as the “atypical” in this |
|                         |      | study.)                                                                                       |
| Tubbs et al. [38]       | 2011 | Thickening of the brachial fascia OR                                                             |
|                         |      | Thickening of the internal brachial ligament OR                                                  |
|                         |      | Thickening of the medial intermuscular septum. (All three considered as the “atypical” in this |
|                         |      | study.)                                                                                       |

Table 9. Prevalence of the classical and atypical Struthers arcade

| Type        | Number of studies (no. of subjects) | Pooled prevalence; % (95% CI) | I²; % (95% CI) | Cochran’s Q, p-value |
|-------------|-------------------------------------|--------------------------------|----------------|---------------------|
| Classical   | 8 (142)                             | 72.8 (30.0–100.0)              | 95.8 (93.5–97.2)| 165.6, p < 0.001    |
| Atypical    | 8 (142)                             | 27.2 (0.0–70.0)                | 95.8 (93.5–97.2)| 165.6, p < 0.001    |

CI — confidence interval

The geographical analysis separated all the studies (PPE 52.6%; 95% CI 27.1–77.5%) from cadaveric studies (PPE 59.6%; 95% CI 35.0–82.1%), as well as those reporting from North America (PPE 69.4%; 95% CI 32.3–97.1%), Asia (PPE 45.2; 95% CI 0.0–94.2%), South America (PPE 68.0%; 95% CI 0.0–100.0%), and Europe (PPE 15.1%; 95% CI 0.0–59.1%) (Fig. 3, Table 7).

The formal description of the SA by Kane et al. [24] (a fibrous canal with a roof formed by a deep fascial thickening, an anterior border at the medial intermuscular septum, and a lateral border at the humerus and the muscular fibre covering of the triceps brachii) did not always apply to the findings of certain included studies due to its variability in presentation [2, 27, 32, 38]. The variant definitions of the SA can be found in Table 8. Since most of the authors described their SA in accordance with the definition stated by Kane et al. [24], we applied this term as the classical type in opposition to the atypical SAs, found and described less commonly. All the typical and unusual SAs encountered in analysed studies fit into one of the definitions from Table 8. Table 9 presents the PPE of the typical and atypical SAs, and Table 10 reports the types of the atypical SAs (when reported in the respective studies). The most common of the atypical types is a thickening of the brachial fascia, found in 39.3% (95% CI 0.0–89.0%) of the reported 38 structures.

The morphology of the SA was found to be mostly musculotendinous (PPE 54.2; 95% CI 12.6–89.1%), or
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Examples of the SAs found during our own routine cadaveric examinations are presented on Figure 4 (a tendinous arcade) and Figure 5 (a musculotendinous arcade).

The various relations of the SA to the ulnar nerve are shown in Table 12, where it most typically presented as a tendinous arcade passing over the ulnar nerve (PPE 42.2; 95% CI 2.5–77.9%). Table 13 presents the findings of the 3 studies concerning the prevalence of ulnar nerve compression. Forty limbs from 1 study [12] were reported to show no compression, whereas all the SAs of Mirza et al. [27] and Yoshida et al. [41] were associated with the ulnar nerve compression.

**Risk of bias analysis**

The complete appraisal of the included studies in terms of the risk of bias they pose is presented in Table 14. All in all, the vast majority of the studies were assessed as having a “High” risk of bias in Domains 1 and 3, due to the lack of complete information about the patients’ baseline characteristics and demographics, as well as the specialty and experience of the scientists in charge of a particular part of the study. Domains 2 and 5 were evaluated as being at “Low” risk of bias for all the included studies. Nonetheless,
Domain 4 had two studies at “High” risk of bias due to them not specifying their definition of the SA (Table 14).

**DISCUSSION**

This study aims to clarify the differences between the SL and the SA, and investigate their respective

| Study                          | Country | Type of study | Number of limbs with SA | Prevalence of ulnar nerve compression (%) |
|-------------------------------|---------|---------------|-------------------------|------------------------------------------|
| Caetano et al., 2017 [12]     | Brazil  | Cadaveric     | 40                      | 0.0                                      |
| Mirza et al., 2014 [27]       | USA     | Cadaveric     | 2                       | 100.0                                    |
| Yoshida et al., 2014 [41]     | Japan   | Surgery       | 1                       | 100.0                                    |

**Table 12.** Relation of the Struthers arcade to the ulnar nerve

| Type                                               | Number of studies (no. of subjects) | Pooled prevalence; % (95% CI) | I²; % (95% CI) | Cochran’s Q, p-value |
|----------------------------------------------------|------------------------------------|--------------------------------|----------------|---------------------|
| Musculotendinous arcade covers the nerve            | 9 (193)                            | 34.2 (0.0–71.6)                | 96.5 (94.9–97.6)| 227.2, p < 0.001    |
| Tendinous arcade passing over the ulnar nerve       | 9 (193)                            | 42.2 (2.5–77.9)                | 96.5 (94.9–97.6)| 227.2, p < 0.001    |
| Triceps muscle covers the nerve                     | 9 (193)                            | 13.8 (0.0–43.9)                | 96.5 (94.9–97.6)| 227.2, p < 0.001    |
| The ulnar nerve passing anteriorly to the arcade    | 9 (193)                            | 2.5 (0.0–21.9)                 | 96.5 (94.9–97.6)| 227.2, p < 0.001    |
| Triceps aponeurosis covers the nerve                | 9 (193)                            | 3.6 (0.0–24.9)                 | 96.5 (94.9–97.6)| 227.2, p < 0.001    |
| Multiple ligaments of thickened deep fascia and medial intermuscular septum pass superficially and deeply to the nerve | 9 (193) | 3.7 (0.0–25.2) | 96.5 (94.9–97.6) | 227.2, p < 0.001    |

CI — confidence interval

**Table 13.** The ulnar nerve compression by the Struthers arcade (SA)

| Study                          | Country | Type of study | Number of limbs with SA | Prevalence of ulnar nerve compression (%) |
|-------------------------------|---------|---------------|-------------------------|------------------------------------------|
| Caetano et al., 2017 [12]     | Brazil  | Cadaveric     | 40                      | 0.0                                      |
| Mirza et al., 2014 [27]       | USA     | Cadaveric     | 2                       | 100.0                                    |
| Yoshida et al., 2014 [41]     | Japan   | Surgery       | 1                       | 100.0                                    |

**Table 14.** The risk of bias analysis

| Study                          | Objective(s) and study characteristics | Study design | Methodology characterisation | Descriptive anatomy | Reporting of results |
|-------------------------------|----------------------------------------|--------------|------------------------------|---------------------|----------------------|
| Al-Qattan and Murray, 1991 [2] | High                                   | Low          | High                         | Low                 | Low                  |
| Bartels et al., 2003 [7]      | High                                   | Low          | High                         | Low                 | Low                  |
| Bilecenoglu et al., 2005 [8]  | High                                   | Low          | High                         | Low                 | Low                  |
| Caetano et al., 2017 [11]     | High                                   | Low          | High                         | Low                 | Low                  |
| Caetano et al., 2017 [12]     | High                                   | Low          | High                         | Low                 | Low                  |
| Dellon et al., 1987 [17]      | High                                   | Low          | High                         | Low                 | Low                  |
| Gessini et al., 1983 [19]     | High                                   | Low          | High                         | Low                 | Low                  |
| Gonzalez et al., 2001 [20]    | High                                   | Low          | High                         | Low                 | Low                  |
| Gunther et al., 1993 [21]     | High                                   | Low          | High                         | Low                 | Low                  |
| Dellon 1986 [16]              | High                                   | Low          | High                         | Low                 | Low                  |
| Mirza et al., 2014 [27]       | High                                   | Low          | High                         | Low                 | Low                  |
| Poujade et al., 2014 [30]     | High                                   | Low          | High                         | Low                 | Low                  |
| Siqueira and Martins, 2005 [32]| Low                                    | Low          | High                         | Low                 | Low                  |
| Tsiavvoranan et al., 2010 [37]| High                                   | Low          | High                         | High                | Low                  |
| Tubbs et al., 2011 [38]       | High                                   | Low          | High                         | Low                 | Low                  |
| Von Schroeder and Scheker, 2003 [39]| Low       | Low          | High                         | Low                 | Low                  |
| Yoshida et al., 2014 [41]     | High                                   | Low          | High                         | High                | Low                  |
| Zhong et al., 2016 [42]       | High                                   | Low          | High                         | Low                 | Low                  |
properties in a clinically relevant manner. Disagree-
ments in prior publications exist, promoting a poor
understanding of these structures and their implica-
tions in the treatment of upper limb neuropathies
[11]. In order to improve patient outcomes during the
associated procedures, this meta-analysis attempted
to evaluate the SL and the SA in terms of their PPE,
anatomical features (such as e.g. morphology), and
relation to the median and ulnar nerves, respectively.

Clinicians must consider the possible involvement
of these two structures in their practice — especially
in surgeries. The SL is rare, and is typically associated
with the brachial artery and/or the median nerve.
Due to its low prevalence, it is infrequently being
considered in the differential diagnosis as a cause of
entrapment [21]. Also, even if present (when
identified by radiographs) it may not necessarily be
the origin of the symptoms [21]. The SA is a valid
structure, most typically presenting as a musculo-
tendinous band associated with the ulnar nerve, but
has extensive variability. Primary entrapment has not
been described, but it has been largely implicated
in failed cubital tunnel surgery, or otherwise during
the anterior transposition of the ulnar nerve, which
may be the result of unsuccessful decompression or
formation of a new site of compression [15, 18, 32].
Since both structures have been suggested to be in-
volved in neuropathies, it is of immense importance
for medical professionals to get acquainted with
their variants and consider them in the differential
diagnoses. This recommendation is especially valid in
cases which cannot be explained by more commonly
prevalent conditions, e.g. cubital tunnel syndrome in
the ulnar nerve entrapment.

Henceforth, patients presenting with unusual cas-
es (such as with the SL or the SA involvement) may
be treated more accurately by medical professionals
acquainted with their infrequent causes, possibly
mitigating the risk of permanent nerve injuries.
Compressions to the median, radial, or ulnar nerve, which
occur especially when such bands of fibrous or mus-
cular tissue traverse them, may lead to upper limb
entrapment peripheral neuropathies [8]. Ulnar nerve
neuropathies at the elbow are important in particular,
as they are the second most common entrapment
neuropathy in adults [10].

The SL is a consistently reported structure, and our
PPE findings (1.8%) are in line with that of previous
descriptions [3, 5, 29, 35]. These results support that
this structure is vestigial, and is likely analogous to the
latissimocondyloides muscle found in climbing ani-
mal conditions is poorly described [4, 13]. Palpation of
the bony process may or may not be possible, so it cannot
be used as an indication for investigative imaging [9].
In addition, rare instances have been reported where
the SL was found associated with only a minimal pro-
trusion, or none whatsoever [35]. Gunther et al. [21]
report that the supracondylar spur is most typically an
incidental finding on radiography, and that no surgi-
cal corrections should be made without any clinical
complaints present. Furthermore, a clinician should
not automatically assume that the SL (if present) is re-
sponsible for any neuropathies prior to investigation.
However, the surgeon should be conscious of these
structures during surgical exploration. Also, it may be
benevolent to recognize that the SL likely inserts into
the medial humeral epicondyle (possibly lending aid
to the identification of this rare anatomical variant)
as per all the reports evaluated herein [8, 19, 21].

Since the overall PPE of the SL is very low, it will
ultimately be a rare cause of entrapment. Importantly,
Laha et al. [26] define a simple differentiation of the
median nerve entrapment by the SL and its bony liga-
ment from the more common pronator syndrome be-
cause of pronator teres weakness found in the latter.

Lastly, Taylor et al. [36] discuss the possibility of
using the SL (associated with the coracobrachialis
tendinous arches during their nerve entrapment. Dellon [16] claim to have never observed any such
ments passing deep to the ulnar nerve should also
have resulted in further entrapment, and that liga-
an atypical SA with a roof of multiple ligaments may
forming a procedure to release an entrapped nerve,
in differentiating the possible interactions of the SA.
of this structure may be likely, suggesting a difficulty
0%. Therefore, an extensively variable presentation
suggested a 100% association with compression or
compression from the SA, the results of which either
cpecifically outlined the prevalence of the ulnar nerve
over the ulnar nerve. Only 3 included studies spe-
tendinous or tendinous arcade covering or passing
and descriptions of their division [34].
and it most frequently presents as either a musculo-
tendinous band. Even more inconsistency
lies in the morphology of the SA, where most tend-
ed to be musculotendinous, but a large proportion
was still found to be solely tendinous or muscular.
Another factor may be, as Bartels et al. [7] suggest,
where any dissection can be conducted to replicate
a fibrous structure depending on the stepwise tech-
nique. However, earlier reports on the arcade clearly
differentiate the absence or presence with images
and descriptions of their division [34].
The course of the SA was described in 9 studies,
and it most frequently presents as either a musculo-
tendinous or tendinous arcade covering or passing
over the ulnar nerve. Only 3 included studies spe-
cifically outlined the prevalence of the ulnar nerve
compression from the SA, the results of which either
suggested a 100% association with compression or
0%. Therefore, an extensively variable presentation
of this structure may be likely, suggesting a difficulty
in differentiating the possible interactions of the SA.
Firstly, inaccurate reporting may be the result of
confusion between the SL and the SA [11] as the two
similarly named structures appear in the same region.
This clarification is imperative for future consistency.
Al-Qattan and Murray [2] report that when per-
forming a procedure to release an entrapped nerve,
an atypical SA with a roof of multiple ligaments may
have resulted in further entrapment, and that liga-
ments passing deep to the ulnar nerve should also
be released at their insertions. Bartels et al. [7] and
Dellon [16] claim to have never observed any such
tendinous arches during their nerve entrapment
release surgeries, and suggest that any observed
bands are likely the result of improper release of the
brachial fascial sheath during the previously under-
taken anterior transposition of the ulnar nerve that
now became the fibrotic point of compression of the
nerve. Bartels et al. [7] also suggest that the edge of
the sheath that was cut might become more fibrotic
and hence resemble the structure known as the SA.
Dellon [16] continues to suggest that an appropriate
incision for the cubital tunnel release should be into
the brachium, but end more proximally to the me-
dial humeral epicondyle. Attempts to further study
the causes of secondary entrapments have not been
successful [28, 29, 38].
The disparity in the frequencies of surgeons find-
ing the SA may be in part due to the differences in
dissection methodologies; Bartels et al. [7] suggest
that in order to standardise the procedure, a step
by step dissection focusing on the fascial coverings
should be demonstrated. Otherwise, Bartels et al. [7]
report that the findings may be in part by the cut edge
becoming more fibrotic, and therefore appearing as
a tendinous band, however this secondary observa-
tion was not the case for most of the included cases.
Overall, the findings of this analysis suggest that
the SA is a common structure, albeit with great var-
iability in terms of morphology, relation to the ulnar
nerve, or otherwise atypical. It is not likely to be the
primary site for nerve entrapment, but it is largely
implicated in post transposition syndrome [14]. To
reduce the likeliness of a secondary compression,
the ulnar nerve must be adequately mobilized from
the SA or otherwise any soft tissue attachments that
may cause compression [37].
Although a thorough risk of bias assessment was
completed, and the quality of the analysed data was
evaluated, this study is still subject to the limitations
of the availability of the previously published studies.
Since the SL is not frequently found, large scale stud-
ies cannot be realistically executed, thus potentially
reducing the effects of bias altogether. Fortunately,
the statistical power of this meta-analysis enabled
appropriate conclusions to be drawn. The disagree-
ments regarding the SA (such as its involvement in
entrapment, or its existence altogether) added to the
difficulty in the investigation, as the findings tended
to be bimodal — either largely present, or completely
absent. However, with a comprehensive study, and
efforts to explicate the discrepancies, statistically
significant values could be obtained.
CONCLUSIONS
Since some of the upper limb entrapment peripheral neuropathies have been reported in association with the presence of the SL or the SA, a clinical picture of these two structures must be established in practice. Although the presence of the SL is infrequent, and the manifestation of the SA is highly variable, they are still important considerations in treatment of the aforementioned condition. The SL had a PPE of 1.8% overall, and may be found minimally more likely on the right side (55.8%) than the left (44.2%), but seems to always insert into the medial humeral epicondyle. It was associated with median nerve compression in one of the two studies on the matter. The SA is a valid anatomical structure, and has an overall PPE of 52.6%. Although most typically presenting as a musculotendinous band, it has extensive variability, and may be problematic in procedures involving the anterior transposition of the ulnar nerve. In view of the foregoing, a better understanding of each may be beneficial for the best patient outcomes.

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