Causes, symptoms, and treatments of nerve entrapments around the elbow: Current concepts

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ABSTRACT
The elbow is a joint extremely susceptible to stiffness, even after a trivial trauma. As for other joints, several factors can generate stiffness such as immobilisation, joint incongruity, heterotopic ossification, adhesions, or pain. Prolonged joint immobilisation, pursued to assure bony and ligamentous healing, represents the most acknowledged risk factor for joint stiffness.

The elbow is a common site of nerve entrapment syndromes. The reasons are multifactorial, but peculiar elbow anatomy and biomechanics play a role. Passing from the arm into the forearm, the ulnar, median, and radial nerves run at the elbow in close rapport with the joint, fibrous arches and through narrow fibro-osseous tunnel. The elbow joint, in fact, has a large range of flexion which exposes nerves lying posterior to the axis of rotation to traction and those anterior to compression.

Current concepts
- The elbow is a common site of nerve entrapment syndromes.
- The reasons are multifactorial but peculiar elbow anatomy and biomechanics play a role.
- Passing from the arm into the forearm, the ulnar, median, and radial nerves run at the elbow in close relationship with the joint, fibrous arches and through narrow fibro-osseous tunnel.
- Soft tissue and bone surgical release for elbow stiffness can have significant consequences on nerve conduction especially in severe contracture.

Future perspectives
- In spite of a normal preoperative neurological condition, preventive nerve release and tunnel decompression must be considered when addressing surgically an elbow contracture.
- The significant motion recover can indeed decrease nerves paths volume turning symptomatic neuropathy that was subclinical.
- Neuropathies can be the upstream events which lead to secondary tissue remodeling and following joint stiffness.
- Surgical management for elbow stiffness should evaluate the potential neurological role as root of joint contracture.

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Introduction

The elbow is a joint extremely susceptible to stiffness, even after a trivial trauma [1]. Up to 5% of patients with elbow injury can develop an ankylosis [1], whereas 10 to 15% of patients may need an additional surgery to manage joint contracture [2]. As for other joints, several factors can generate stiffness such as immobilisation, joint incongruity, heterotopic ossification, adhesions, or pain. Prolonged joint immobilisation, pursued to assure bony and ligamentous healing, represents the most acknowledged risk factor for joint stiffness. Consequently, increasing efforts have been channelling to achieve elbow stability as much as possible, thereby avoiding unfavourable prolonged immobilisation. Nonetheless, immobilisation time as well as energy of incipit trauma cannot predict the degree of joint stiffness, suggesting that other elements may play a significant role in contracture development.

The elbow is a common site of nerve entrapment syndromes. The reasons are multifactorial, but peculiar elbow anatomy and biomechanics play a role. Passing from the arm into the forearm, the ulnar, median, and radial nerves (RNs) run at the elbow in close rapport with the joint, fibrous arches and through narrow fibro-osseous tunnel. Here, pulleys and sheaths promote a smooth gliding throughout the range of motion. The elbow joint, in fact, has a large range of flexion, which exposes nerves lying posterior to the axis of rotation to traction and those anterior to compression. The ulnar nerve (UN), which is subjected to traction during elbow flexion, has an excursion of 5.1 mm from 10° to 90° of elbow motion [3]. In the same range of motion, 8.8 mm of nerve excursion is needed for the RN [4]. Even though not sufficient to compromise nerve function, these anatomical features when coupled with factors restricting excursion can result in repetitive traction and friction of the nerve. In this way, capsule and ligament thickening as well as adhesions and heterotopic ossifications following a trauma can play a role in the pathophysiology of a mechanical neuropathy.

Nerve irritation can induce connective tissue fibrosis and heterotopic ossification due to chronic inflammation or irritation that limits motion of the elbow [1]. However, pre operative EMG may not always show a gross abnormality even though the affected nerve may appear discolored, compressed and edematous [5]. Despite this, surgical treatment including releases of bony and soft tissue contractures can have a negative impact on nerve function if significant contractures are released allowing for more extensive nerve excursion. Nonetheless, in the setting of normal nerve function, nerve decompression should be considered while addressing elbow contractures. This is because more nerve excursion may be present after release of the joint contracture and a subclinical nerve impairment may become clinically significant with more nerve mobility [6–10]. Neuropathies can be the upstream events which lead to secondary tissue remodelling and following joint stiffness. Consequently, surgical management of elbow stiffness should evaluate the potential neurological role as a root of joint contracture. Scar tissues and heterotopic ossification surgical release could be otherwise unsuccessful.

Ulnar nerve

The UN arises as an extension of the medial cord of brachial plexus lying medial to the brachial artery. The UN travels in the medial aspect of the arm between the coracobrachialis laterally and the medial head of the triceps posteriorly. Then, the nerve pierces the intermuscular septum at the level of distal insertion of coracobrachialis, at approximately 10 cm above the medial epicondyle, to enter the posterior compartment of the arm. Here, the nerve can travel inside the arcade of Struthers, which is an occasional fascia enveloping the UN at approximately 8 cm proximal to the medial epicondyle [11]. This arcade is a thickening of the deep fascia found 8 to 10 cm proximal to the medial epicondyle, and it is an infrequent structure since it is present in about 70% of anatomical specimens [12]. The UN then crosses the elbow posteriorly entering the cubital tunnel, which is defined anteriorly by the posterior surface of medial epicondyle and posteriorly by the retinaculum. At this point, as the nerve travels more distally, it is in close relationship with the medial collateral ligament (MCL), and in particular its posterior band (pMCL). Here, a sensitive branch arises for the capsule and the first motor branch for the flexor carpi ulnaris (FCU) (Fig. 1). As a matter of fact, multiple branches can arise, with some degree of anatomical variation, from 4 cm proximal to 10 cm distal to the medial epicondyle [11]. Muscular branches for flexor digitorum profundus (FDP) usually arise at 4 to 5 cm distal to the medial epicondyle [11].

Sites of compression

The arcade of Struthers

The arcade of Struthers is the most proximal side of potential primary UN entrapment in the elbow as well as secondary when it is not released during an anterior transposition. This should not be confused with the ligament of Struthers which is a facial band of variable thickness and width observed in less than 1% of specimens (Fig. 2) [12–14]. This ligament extends from the humeral supracondylar process to the medial epicondyle, and the proximal insertion of pronator teres (PT) muscle and may compress the median nerve (MN) [13,14]. The arcade of Struthers is a thickening of the deep fascia of the arm observed in about 70% of anatomical specimens, 8 to 10 cm proximal to the medial epicondyle [12]. However, the studies on the anatomical features are limited and their finding contrasting. If in some studies this has been described as a fibrous canal with an average length of 5.7 cm through which the UN passes 8 to 10 cm proximal to the medial epicondyle [15], in others it has been described as a fascial condensation spanning from the medial head of triceps to the medial intermuscular septum [16].

Medial intermuscular septum

This is an area of special interest for the surgeon approaching to the UN during the treatment of a stiff elbow. Dividing the anterior from the posterior compartment, this septum is remarkably thicker in the inferior supracondylar region, whereas its superior portion is thinner and with the less distinct attachment to the UN. The UN is firmly bounded to the underlying triceps and then to the posterior aspect of the tick medial septum and the dorsal surface of the medial epicondyle. A complete resection of the distal 3.5 cm of septum is mandatory when an anterior nerve transposition is performed since it may otherwise represent a tenacious and predictable site of nerve kinking and compression (Fig. 3).

Anconeus epitrochlearis muscle

The anconeus epitrochlearis a rudimental muscle present in about 4 to 34% of population [17]. Although it is a rare finding, this muscle is a well-established cause of ulnar neuritis secondary to static compression. The muscle arises from the medial border of the olecranon to insert into the medial epicondyle, and it can vary in shape from a very small band to

Fig. 1. The ulnar nerve and possible compression points around the elbow.
a thick muscle to be even appreciated on physical examination (Fig. 4). However, an anconeus epitrochlearis ligament is more often observed instead of the muscle in about 70% of elbows. In case of an anconeus epitrochlearis muscle, the management suggested is an excision and a nerve anterior subcutaneous transposition.

Cubital tunnel

The cubital tunnel is a fibro-osseous structure posterior to the medial epicondyle. The posterior portion of MCL and the overall medial aspect of the humeroulnar capsule form the floor of this tunnel, whereas a thick fibrous layer between the epicondyle and the olecranon forms its roof (Fig. 5). In 1991, O’Driscoll et al., studying 27 specimens, defined the roof of the cubital tunnel formed by the cubital tunnel retinaculum (CTR) and the FCU aponeurosis. The CTR, likely a remnant of the anconeus epitrochlearis muscle, is a 4-mm-wide fibrous band that forms the entrance to the cubital tunnel. This band tightens during elbow flexion, preventing UN dislocation. The FCU deep aponeurosis also known as Osborne’s band is a fibrous arcade formed from the fusion of the deep FCU fascia and the anti-brachial fascia. As the UN enters the anterior forearm compartment through the interval between the humeral and ulnar origin of the FCU muscle, it can be compressed by this fascial arcade of varying thickness.

The first portion or entrance of the cubital tunnel is composed of the medial epicondyle, the medial border of the olecranon, and the CTR. The latter, as described by O’Driscoll, presents four times of anatomical variance according to their potential of UN compression [18]: when the CTR is absent, it is defined as type 0; type 1A is considered the normal type: it is thin and becomes taut with elbow flexion; when the band is taut inducing nerve compression and chronic neuropathy, then it is defined as 1B type; type 2 is defined as the presence of the anconeus epitrochlearis muscle [18]. The second portion of the capital tunnel is characterised by the presence of the Osborne’s band which connects the ulnar and humeral head of the FCU muscle.

Deep flexor pronator aponeurosis

The compression of the UN at the common flexor pronator aponeurosis (CFA) is uncommon, but it can be a secondary point of compression when it is transposed anteriorly. The CFA lies between the flexor pronator region and the flexor digitorum superficialis (FDS) muscle (Fig. 6). When the UN enters the forearm between the two heads of FCU, it runs into an elliptical tunnel on the anterior medial edge of the CFA. In a cadaveric study, CFA was observed in all elbows and the average length was 2.9 cm [19]. In the surgical nerve release, the UN should be thoroughly exposed removing all the anatomical structures potentially a cause of compression till the deep flexor pronator fascia aponeurosis.

The cubital syndrome represents the second most frequent peripheral neuropathy of upper limb. UN can be compressed in several points around the elbow. However, cubital tunnel is the most frequent site for the UN compression. The nerve crosses a narrow cubital tunnel with a diameter of $6 \pm 1.9$ mm, whereas the UN diameter is $4.9 \pm 1.3$ mm [20]. In addition, the cubital tunnel is subjected to a dynamic variation during elbow motion. As the flexion increases, the distance between the humeral and ulnar insertions of the CTR increases with flexion, leading to tightening of the band [18,21] and a decrease of the volume of the cubital tunnel [22]. In particular, with elbow flexion, the cross-sectional contour of cubital tunnel narrows by about 55%, changing from smooth and
The deep flexor pronator aponeurosis.

**Clinical findings**

The compression on the nerve in a stiff elbow can lead to different clinical scenarios on the prevalence of which kind of fibres are compressed, if sensitive or motor. However, the topography and intra-neural anatomy of the fibres composing the UN in the cubital tunnel can in some way predict the most frequent clinical manifestations. Sunderland et al., have studied the intra-neural topography and the branches that take off from the nerve into the cubital tunnel [25]. Sensory fibres and intrinsic motor axons of the hand lay superficial under the CTR, while in the deeper central area, the motor fibres to the FCU and the FDP can be found. In the cubital tunnel, the UN usually gives off branches to the two heads of FCU within or at approximately 3 cm from the medial epicondyle. The deep branches for the FDP take off the nerve at 5 cm distal to the medial epicondyle.

The clinical presentation of UN compression in a stiff elbow can be variable. The initial phase is usually characterised by a reported ‘cotton wool’ feeling involving the last two fingers and pain along the medial side of the proximal forearm. With the same cutaneous distribution, intermittent paresthesia and progressive sensory loss are thereafter observed. Symptoms are usually worse at the night likely due to the loss of muscle pump and following pooling of interstitial fluids. The motor symptoms usually reveal later and after the sensory deficit even though sometimes they can be observed in the initial phases.

**Surgical technique**

Several techniques have been described to manage UN neuropathy due to an entrapment at the elbow, in particular in stiff joints. These techniques aim to release and free the nerve from any kind of compression or restraint along all its course and throughout the elbow range of motion. The methods so far known are the following: 1. in-situ nerve decompression, 2. medial epicondylectomy, 3. anterior subcutaneous transposition, 4. anterior intramuscular transposition, 5. submuscular transposition.

These five methods can be divided into two groups on the basis of the rationale used to manage the nerve compression. The in-situ decompression and the medial epicondylectomy decompress the nerve suffering while preserving the deep attachment without affecting its blood supply and general local environment. Conversely, the transposition techniques involve a more aggressive approach necessarily needed to release all the potential secondary nerve compression sites and create a new pathway for the UN. These techniques are suggested when a plain or subtle instability in the epicondylar groove is detected at the end of nerve release throughout the range of motion. Even though instability with subsequent neuropathy can be in this way prevented, a more aggressive approach on the nerve can determine a tissue damage known as “double crush phenomenon” [26–30].

**Median nerve**

The MN travels in the arm antero-medially to the humerus and laterally to the brachial artery without any muscular branches. Approaching the elbow, the rapport with the brachial artery changes, becoming medial to it. At the cubital fossa, it is covered by the lacertus fibrosis and then proceeds between the two heads of PT. The anterior interosseous nerve (AIN) arises from the postero-lateral aspect of MN immediately after the superior border of FDS about 4 cm distal to the median epicondyle. The AIN is a pure motor nerve to FDP of the index and middle finger, flexor pollicis longus (FPL), and pronator quadratus (PQ). The MN may be compressed in predictable anatomical sites at elbow and proximal forearm leading to two pathological conditions (Fig. 7): the pronator tunnel syndrome and the AIN compression. The potential anatomical sites from proximal to distal are the following: the supracondylar process and the ligament of Struthers; a fibrous thickening of the bicipital aponeurosis; hypertrophic PT muscle with or without an abnormal fibrous band between the two heads; the fibrous arch of the FDS; and the Gantzer's muscle which is an accessory head of the FPL.

**Sites of compression**

**Supracondylar process and ligament of struthers**

Described in 1848 by Struthers, the supracondylar process is an abnormal congenital bone structure arising from the medial aspect of the humerus at approximately 3.5 cm above the median epicondyle (Fig. 8) [13,14]. This process gives attachment to a band of connective tissue, the ligament of Struthers, which inserts distally to the medial epicondyle. When present, the process and the ligament create, with the medial aspect of the humerus, a tunnel where the brachial artery and the MN travel. Sometimes these structures received insertion of the brachialis muscle or an anomalous superficial head of the PT. These are usually easy to diagnose both with the palpation that reveals the process and thereafter with an X-ray confirming its presence. Clinical findings are similar to pronator syndrome except for elbow flexion and forearm pronation that are often painful and are associated with paresthesia.

**Fibrous thickening of lacertus fibrosus**

The lacertus fibrosus (LF) is normally a thin fibrous band, but...
sometimes it is prominent, becoming a substantial structure that may cause an entrapment of MN, known by some authors as “lacertus fibrosus syndrome” (Fig. 9) [31]. The symptoms are usually triggered by elbow flexion and supination [32]. The MN compression can be found in the presence of anatomical variation such as anomalous accessory bicipital aponeurosis [33], abnormal flexor muscle origin, or an anomalous PT that uncovers the nerve and exposes it directly to LF compression [34]. This space, dubbed by Hager “lacertus tunnel”, is bounded medially by the PT and laterally by the brachialis muscle, while the roof is formed by the LF and the floor by the medial humeral trochlea [31]. At this point, it is evident that the close relationship MN has with the anterior capsule and significant consequences that abnormal bony or fibrous connective tissue can have on it. Another condition that can increase the pressure of the MN in this narrow space is the distal biceps pathology. In particular, an acute compression has been described as a complication of partial complete rupture of the distal biceps tendon insertion with or without formation of any haematoma or compressive bursa [35]. In patients with the distal biceps tendon rupture and an intact LF, the muscle retraction displaces the LF proximally and laterally. This situation creates a secondary compression site when the elbow is actively flexed or passively extended with the forearm supinated. When the brachial artery is compressed as well, the nerve entrapment syndrome is associated with the intermittent radial and UN pulse loss during passive elbow hyperextension and forced forearm supination [36]. This aspect represents a crucial condition to be considerate in the management of the elbow stiffness. In patients with the long-standing elbow stiffness with the extension contracture (>60°), the LF becomes fibrotic and retracted, reducing the tunnel space and placing the MN and the brachial artery at risk of compression in patient undergoing surgical release to increase elbow extension. For this reason, to avoid an acute compression on the MN, the LF release and the PT insertion tenotomy are mandatory in this scenario and when a significant lengthening of the biceps tendon and the brachialis muscle is performed in cases with long-standing elbow flexion contracture without posterior joint impingement.

**PT muscle**

The most common site of MN compression is the narrow muscle space between the two heads of the PT. Here, the humeral head may vary extremely in size and sometimes also in type of origin (Fig. 10). There are
several conditions linked to the PT muscle that can generate entrapment of the MN. Direct compression may occur when the nerve passes through the humeral and ulnar head of the PT, for example, for a hypertrophic muscle [32]. Repetitive pronation and supination with elbow flexed can induce increased muscle volume and strengthening with the subsequent myostatic contracture. The nerve compression can also be secondary to the presence of abnormal structures that hamper nerve mobility during joint motion and medial epicondylar muscle contraction. For example, intramuscular fibrous structure or fascial bands between the superficial and deep head of PT or vascular leashes can predispose to dynamic muscle compression [37]. The MN runs between the two heads of the PT in more than 80% of patients, whereas in 8–22% the ulnar head is absent [38]. Other causes of nerve compression are represented by anatomical variants of the PT ulnar head, which may arise from the FDS so that the MN may pierce the PT humeral head [37]. At the same time, an abnormal insertion of the humeral head could also represent a potential source of nerve compression as a very proximal fibrous insertion can push the nerve anterior to the medial epicondyle favouring nerve entrapment [39]. Moreover, an accessory muscle, that is the accessory PT humeral head, can be predisposed to MN neuropathy. As described in a case report [40], this accessory muscle starting from this medial intermuscular septum and reaching the humeral head of the PT covers the brachial artery and the MN. The relationship of the MN branches with the humeral and ulnar heads varies in relation to their location and number. The MN provides to the PT one to three or more muscular branchings originating from the medial aspect of the nerve with the most proximal branch originating about 1 to 7 cm above the inter-epicondylar line and the distal 2 to 3 cm below the epicondyle in 90% of cases [41].

**Fibrous arcade of the FDS**

The MN and the ulnar artery enter the forearm passing under a fibrous arch formed by the radial and the humeroulnar head of the FDS. This is a common site of MN entrapment, which is also particularly predisposed by anatomical variants (Fig. 11) [39]. Anatomical variability can interest the origin of the heads of the FDS with the ulnar head that can be found in not more than 32% of specimens [42], but more importantly the variation can be observed in the arch of the FDS. This can be found as a tendinous arcade, called “sublime bridge” by Tubbs et al., or as muscular arcade [43]. The prevalence of the fibrous arcade is extremely variable in the studies thus far published, with 75% described by Tubbs et al. and 36% by Dellon and Mackinnon [16].

**Gantzer’s muscle**

Described in 1813 by Gantzer, this is an accessory head of the FPL or the FDP, and it is considered one of the causes of AIN entrapment and paralysis. Observed in 45 to 74% of the dissected forearms [42], its origin is variable, with the most frequent site being the coronoid process of the ulna and the medial epicondyle of the humerus [44]. Then assuming a spindle shape, the muscle inserts distally with a tendon into the ulnar border of the FPL and sometimes in the FDP muscle. Despite a certain variability, this accessory muscle runs usually anteriorly to the AIN and posteriorly to the MN. However, the mechanism determining the AIN or MN syndrome is not well known. Among the different authors describing cases of these rare syndromes, the most plausible mechanism is the direct compression of a hypertrophic muscle as the cause of neuropathy. Nonetheless, the majority of the Gantzer’s muscles are asymptomatic [42, 44].

**Clinical findings**

The MN neuropathy at the elbow can show up with the two clinical presentations that are the “pronator tunnel syndrome”, with the similar but not identical findings to the carpal tunnel syndrome, and the “AIN syndrome” or Kiloh-Nevin syndrome with specific impairment in the ability to pinch with the thumb and the index finger due to palsy of the FPL muscle, FDP to the index finger, and PQ muscle.

Described first in the year 1951 by Seyffarth and Henry [45], “pronator tunnel syndrome” is an MN neuropathy due to the entrapment usually between the two heads of the PT or less commonly by the arch of the FDS. In patients with PT muscle overuse, that is those involved with the manufacturing jobs, weightlifting, or sport like tennis, the nerve can be dynamically compressed when it passes between the two heads of the PT. The symptoms can be triggered by prolonged or repetitive forearm pronation associated with forced elbow and finger flexion. The other abovementioned causes of nerve compressions, such as beneath the LF or the Gantzer’s muscle, are markedly less common.

Patients with pronator tunnel syndrome typically report an aching pain with subtle onset localised in the volar region of proximal forearm and elbow and proximal irradiation, prompted by forceful pronation. The pain is usually associated with the weakness in grip and pinching small objects during manual tasks. Using the comparison with the asymptomatic hand, clinical examination specifically shows weakness of pronation, in the FPL, abductor pollicis brevis, and opponens pollicis muscles. Patients can complain of forearm muscle cramping spasms associated with the repetitive pinching movements, such as writing that are reported as “writer’s cramp”.

Several provocative tests or manoeuvres can be useful to confirm the diagnosis on the basis of the specific site of nerve compression. For instance, when the MN is compressed at the level of the bicipital aponeurosis, lacertus fibrosis test which consists in elbow flexion and forearm supination against the resistance would exacerbate the symptoms of the neuropathy. Similarly, PT test and the FCS test trigger pain or paresthesia. The former is reproduced by pronation against resistance in full elbow extension or with the elbow flexion and progressive elbow extension. The latter is performed with the flexion only of the proximal interphalangeal joint against resistance of the middle to isolate the FDS activity. To diagnose the site of the MN compression at the elbow, other tests such as the MN stretch test and the scratch collapse test can be used. However, diagnosis is often delayed because of the vague and poorly defined findings.

The AIN is a terminal branch of the MN exiting from it on the lateral or anterolateral aspect running on the interosseous membrane between and below the FDP and the FPL. The aetiology of the AIN syndrome is unclear and includes both inflammatory and mechanical causes. As described by Spinner [46], the compression neuropathy can be determined by both spontaneous palsy and trauma. A fibrous band originating from the deep head of the PT and the fibrous band from the arch of the FDS are detected in the most cases of spontaneous palsy [46]. Among the trauma-related causes of the AIN palsy, supracondylar fracture and the iatrogenic injury due to open reduction internal fixation of radius fracture have to be reported [47]. The AIN is more susceptible to the MN to suffer palsy after supracondylar fracture being less mobile in the
proximal forearm. This nerve supplies motor innervation to the FDP of the index finger and the FPL and the PQ, whereas the medial portion of the FDP is supplied by the UN.

Accordingly, the AIN compression is a pure motor neuropathy resulting in weakness and paralysis of the FPL and FDP to the index combined with PQ involvement. The patients cannot typically make the pincer movement, and they present a characteristic pinch deformity where the interphalangeal joint of the thumb and the distal interphalangeal joint of the index collapsed in extension [46]. Thus, the “OK sign test” results positive since the patients cannot make the OK sign but rather a narrow, oval pinch grip (Fig. 12). To evaluate the PQ function, Spinner proposed the PQ test which evaluates the pronation resistance with the elbow completely flexed and the arm stabilised by the examiner to avoid shoulder rotation. With the elbow flexion, the effect of the humoral head of PT is removed, leaving only the pronating strength to the ulnar head that is approximately 25% (Fig. 13) [46].

A thorough knowledge of the course of the MN is the utmost importance to perform correct diagnosis and definition of the site of compression to avoid muscle and nerve injuries during surgery. For instance, physical examination is crucial to understand if the MN compression is between the two PT heads. In this situation, weakness can be found in all the muscle innervated by the MN except for the PT which receives its innervation proximal to the humeral head.

**Surgical technique**

The surgical nerve decompression can be performed with both the limited and the extensive approach. If clinical diagnosis suggests a compression in the proximal part of the MN at the elbow, a limited approach can be performed to release the MN within the LF and the PT. Conversely, a traditional approach with an extensive S-shaped or zigzag incision is recommended when all the potential compression sites are to be released [46,47].

**Radial nerve**

If the nerve compression syndromes are generally well-described conditions with usually specific motor and sensory deficits, the RN syndrome does not show itself with an easy pattern. The RN compression syndrome represents up to 2% of the upper limb peripheral syndromes [48,49]. This significant difference, if compared to other conditions like carpal or cubital tunnel, can reflect the real incidence of these conditions but also the difficulty in making an accurate diagnosis in particular if it is
related to a stiff elbow. Despite a usually more straightforward differential diagnosis with median and UL compression syndromes, the RN entrapment is less clear because a severe compression causing major nerve and motor dysfunction is rare. The close relationship with the radiocapitellar joint capsule and other fibrous and muscle structures bounding the narrow radial tunnel make the RN significantly vulnerable to be compressed or kinked after a trauma or surgical approaches for the treatment of post-traumatic conditions.

The most frequent site of nerve compression is the arcade of Frohse [50–52]. In an anatomical study, Clavert et al. found a tendinous arcade in approximately 80% of cases [53]. At this site, superficial head of supinator muscle exerts a pressure of 40 to 50 mmHg over the posterior interosseous nerve (PIN), which increases almost fourfold with active muscle contraction [54]. Since reduction in nerve venous blood flow can occur at 20 to 30 mmHg and ischaemia at 60 to 80 mmHg, Werner et al. hypothesised that chronic and dynamic compression can lead to perineural abnormal changes [54]. With epineural vessels compressions, epineural oedema followed by fibrosis can induce mechanical sensitivity of nerve fibres resulting in typical symptoms. The RN represents the termination of the posterior cord of the brachial plexus and carries the fibres for the nerve at C6, C7, and C8. As it takes origin in the axilla, it descends behind the brachial artery and anterior to the subscapularis muscle and the insertion of the teres major and the latissimus dorsi tendon. As the nerve reaches the lateral border of the medial head of the triceps, the RN pierces the intermuscular septum at around 10 to 12 cm proximal to the lateral epicondyle. Then, it runs distally in the flexor compartment between the brachioradialis and the brachial muscle. Antero-medially to the lateral epicondyle and under the brachioradialis and the extensor muscles within 3 cm of the elbow joint proximally or distally, the RN bifurcates into a superficial sensory and a deep motor (PIN) branch. The superficial RN then runs superficial to that supinator muscle and continues distally under the brachioradialis to pierce the fascia and became superficial in the subcutaneous tissue at approximately 7 cm above the wrist. The PIN, instead, runs directly over the radiocapitellar joint before entering into the radial tunnel (Fig. 14). The radial tunnel extends for about 5 to 10 cm from the radiocapitellar joint to the distal edge to supinator muscle. It is delimited medially by the brachialis and distal biceps tendon; anterolaterally by the mobile wad of the brachioradialis, extensor carpi radialis longus (ECRL), and extensor carpi radialis brevis (ECRB); posteriorly by the caput of the humerus, the capsule of the radiocapitellar joint, and the deep head of supinator distally; and anteriorly by the radial recurrent vessels and superficial supinator head [55]. Even though it is not a real tunnel, the PIN can be compressed in four different points as follows: 1. at the entrance of the tunnel where the narrow is in close relationship with the radiocapitellar joint, which can become more significant and associated with risky adhesions after thickening or fibrous reaction; 2. anterior to the radial neck, a fan-shaped leach of vessels, both arteries and veins (the radial recurrent arteries and veins) described by Henry crosses anterior to the PIN; 3. at the level of tendinous edge of the ECRB medial proximal border, which may be abnormally thickened and compress the nerve during wrist flexion and forearm rotation [50]; 4. at the fibrous proximal edge of the superficial head of supinator (arcade of Frohse) which forms a ligamentous band on the anterior aspect of the supinator, as the PIN runs between the two heads of the muscle [56] (Fig. 15). The PIN compression in the radial tunnel may present with or without loss of motor function. Most of the time, the compression syndrome shows up without sensory deficits but mainly with persistent pain over the lateral aspect of the elbow typically distal to the lateral epicondyle [55, 57, 58]. A vague wrist pain may be infrequently associated. This condition defined “radial tunnel syndrome” (RTS) can be considered as the consequence of a neurapraxia leading to pain due to intermittent and mild compression. Conversely, a most severe compression so as to induce a clear and evident motor dysfunction is known as PIN syndrome, and it is characterised by various degrees of weakness in wrist and digits extensors [59, 60].

Clinical findings

On the clinical examination, patients with RTS report a dull pain in the lateral forearm which usually worsens with forearm rotation and activities. In the details, the pain is elicited by firm compression at approximately 3 to 5 cm distal to the elbow with supination. This specific point where the pain may be elicited can be at the level of the arcade of Frohse in the proximal edge of the supinator that is at approximately 5 cm distal to the epicondyle or when the nerve emerges from the two heads of supinator muscle at the junction of the proximal middle third of the forearm [55]. The pronation should release the pain since the nerve moves away from the pressure point. According to the rule-of-nine test in which the volar surface of proximal forearm is divided into nine squares, the pain is classically located in squares one and two that overlap the course of the RN at the most radial and proximal portions of the forearm [61]. Moreover, these patients report pain and grip weakness without motor or sensory loss since weakness is likely elicited by the pain rather than a real motor nerve dysfunction [62]. The pain typically increases with certain activities and diminishes with the rest because the pain is caused by a dynamic nerve compression that is induced by both the passive pronation and wrist.
flexion or active supination and resisted wrist dorsiflexion [63]. In these situations, both ECRB and the supinator muscle are placed under tension eliciting pain that is increased when the elbow is extended.

The most frequent differential diagnosis for the RTS is the lateral epicondyritis (LE) and the radiocapitellar disease which usually are different for pain location and clinical findings. RTS and LE may coexist, but the pain site location is generally different. Lister has compared the pain elicited by digital pressure on three areas lying at the same distance from one another in the proximal forearm: the lateral epicondyle with the ECRB insertion, the radial capital joint, and the PIN course [55]. However, it is always useful to compare the physical findings with the uninvolved side to establish whether the tenderness is significant. Hagert et al. defined three elements that suggest a PIN entrapment at the arcade of Frohse, rather than a lateral epicondyritis: night pain, tenderness at about 5 cm distal to the lateral epicondyle, and pain elicited by supination against resistance [52]. Lister, instead, described three pathognomonic signs: severe tenderness in the mobile wad just distal to the radial head, which is elicited by resisted middle finger extension (Maudsley’s test), and similarly by supination against resistance with elbow extended [55]. For the latter sign, some controversies can be observed among different authors. Although some authors sustain elbow extension to neutralise supination force of biceps brachii [64], others suggest performing resisted supination with elbow flexion of 90° and full pronation to trigger the pain with the compression of PIN by proximal edge of supinator [65].

When the nerve compression becomes worse, motor deficiencies may show up with specific losses based on the location of the entrapment. Thus, a nerve compression in the radial tunnel, that is distal to the RN bifurcation, can reveal with lateral arm pain, weakness of finger extension, no weakness of wrist extension, and potential dorsal wrist pain.

Electrodiagnostic studies are usually normal and without any specific finding in patients with RTS on both nerve conduction velocity and EMG studies. With a conductive slowing, the motor symptoms are likely observed then excluding the diagnosis of RTS. Even though most of the electrodiagnostic studies result negative even with forced supination, resisted supination can significantly increase the occurrence of latency prolongation in the PIN in patients with RTS when compared with control patients [51,64]. However, electrodiagnostic studies can help to rule out other conditions such as cervical radiocarpalities when clinical findings are not diriment. Selective and ultrasound-guided injection of a local anaesthetic can be helpful and sometimes extremely conclusive in the diagnosis process.

**Surgical technique**

Surgical exploration and décompression are the same for both the PIN entrapment in RTS and nerve palsy in PIN syndrome. Carried out along the course of the nerve, the surgical release of the PIN consists in freeing the nerve at the level of the arcade of Frohse or along its entire course between the two heads of the muscle, from the tendinous edge of the ECRB insertion including the fibrous tissue anterior to the radiocapitellar joint and from ectatic vessels which may develop and compress the nerve. Even though most of nerve compressions are due to the arcade of Frohse, the tendinous edge of the ECRB must be considered in the surgical treatment of the neuropathic compression of the RN. While Nayak et al. described the presence of tendinous edge in 29.1% of its specimens [56], Vergara-Amador and Ramírez found a fibrous arch in the anteromedial border of the muscle in 95.1% of cases [67]. This represents the medial thickened edge of the aponeurosis found in the undersurface of the ECRB of all the specimens analysed [67]. The nerve can be exposed through several surgical approaches within the radial tunnel, including extensive anterolateral, limited anterior, and limited anterolateral approaches [57,68-71].

**Declaration of competing interest**

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