Snapping of the Upper Limb: a Clinical Overview

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INTRODUCTION

Snapping is an audible, palpable and often, a visible phenomenon, resulting from the sudden and abnormal displacement of an anatomic or pathologic structure during the movement of a neighboring joint (1-4). Usually, snapping causes mild symptoms and may not require medical treatment. When it is associated with severe pain and functional impairments, leading to significant reduction in sports and professional activities, surgical management may be necessary (4-6). Snapping phenomena have been reported in various regions of the body, especially in close proximity to joints characterized by a wide range of motion, which allows an anatomical or heterotypic structure to interact with its surrounding environment (1, 5, 6). Snapping phenomena may affect bony structures resulting in the so-called “joint instability”. Repeated snaps in the field of joint instability may result from torn ligaments, a situation that is not uncommon in the knee and the wrist (1, 2). In other cases, snaps involve a large range of soft tissue structures, which can be ligamentous, tendinous or fibrocartilaginous (1, 4). Therefore, snaps can occur at intra-articular or extra-articular locations (1, 4). Snapping in the shoulder region has previously been reported secondary to “grating” of the scapula caused by impingement between the medial border of the scapula and the adjacent ribs (3, 5). Extra-articular forms of elbow snapping are commonly related to anterior dislocation of the ulnar nerve or the distal end of the radial triceps above the medial epicondyle. When intra-articular, snapping of the elbow may be related to the synovial fringe capsule-synovial layer at the junction between the radial collateral ligament and the annular ligament (4). Snapping sensation in the wrist has been traditionally thought to be due to underlying carpal instability or tendon instability such as snapping of the extensor carpi ulnaris secondary to a subsheath tear (2, 6).

Identifying the structures responsible for the snap and their underlying pathology could be clinically challenging considering that different sites and multiple causes may contribute to the snapping phenomenon. Diagnosis

SUMMARY

Snapping results from an abrupt displacement of an anatomic or pathologic structure during the movement of a closely related joint. Snaps are audible, palpable, and often, visible. Snapping phenomena are common within the general population and, in most cases, are associated with mild symptoms. However, especially in athletes, snapping could determine pain and functional impairment that may severely affect their sport activities. We focus on three major types of snapping occurring at specific joints of the upper limbs: the shoulder, the elbow, and the wrist. Here, we provide a comprehensive overview of major advances in the aetiology, pathophysiology, diagnostic imaging, and treatments of these specific snapping phenomena.

KEY WORDS

Snapping; upper limb; shoulder; elbow; wrist; pathophysiology; diagnosis; treatment.
is based on both clinical assessment and imaging. As we shall see later, imaging may require different exams, due to the functional characteristics of this condition. Indeed, advanced imaging techniques can provide a better understanding of the snapping mechanism together with an image-guided diagnostic and therapeutic algorithm to improve its management.

SNAPPING OF THE SHOULDER

Definition and clinical signs
Chronic shoulder instability is often accompanied by joint noises. The snapping shoulder includes intra- and extra-articular forms. The intra-articular forms associated with loose bodies are poorly documented while several different extra-articular causes of snapping are reported. In particular, the definition “snapping scapula” is extremely frequent. This condition is related to the scapula-thoracic joint, following the impingement between the medial edge of the scapula and the poster-medial region of the rib cage (3).

The snapping scapula syndrome is usually characterized by a loud popping or cracking sound, known as crepitus, which occurs when the arm is raised up overhead. The sound is due to the rubbing of soft tissue between the scapula and the thoracic wall. The tissue caught between these two structures could be a bursa, tendon, or muscle (3). Medial scapular border tenderness is also a commonly reported clinical sign (7). Patients may or may not experience pain while moving the arm (5). Asymptomatic patients commonly reporting scapulothoracic crepitus alone do not necessitate any treatment at all (8).

Aetiology
The most common causes are related to the process of exostoses (figure 1), soft tissue sarcomas, chronic bursitis of the scapula-thoracic joint, congenital bone anomalies (i.e., Sprengel deformity or congenital high scapula), or the presence of the Luschka tubercle (pathological hypertrophy of the super-medial angle of the scapula) (9). An anatomical variation, which is one of the main causes of the syndrome, is an anomalous anterior curvature of the superomedial angle of the scapula. The measurement of the scapula superomedial angle ranges from 124° to 162° (mean 144.34° ± 9.09) in normal anatomical specimens. When this angle is lower than 142°, the chance of developing scapular snapping increases (10, 11).

Scapular snapping can also be associated with scapular dyskinesia, which can have articular, musculoskeletal, and neurological causes (12). Regardless of the original condition causing dyskinesia, the snapping of the scapula is generated when abnormal movement brings the extremities of the scapula into closer proximity to the rib cage, leading to a scapula in pronation, which is not conducive to optimal shoulder function and results in subacromial space reduction with symptoms of impingement (9). Other common causes include incorrect posture and incorrect training techniques during sport activities (overtraining or training before strengthening). Recurrent fractures and injuries of the scapula and rib cage can also cause bone deformities, which can increase the friction among the structures of the scapulothoracic joints (13).

Diagnosis
Clinical diagnosis heavily relies on physical examination, which is also crucial in informing following steps in the diagnostic process. Physical examination is supplemented by advanced imaging such as magnetic resonance imaging (MRI) and/or computed tomography (CT) to assess for potential bony or soft tissue aetiologies of snapping scapula (9, 14).

Physical examination should evaluate for spinal deformities, palpable crepitus, point tenderness, and scapular winging. It is worth mentioning that kyphoscoliosis, which decreases scapulothoracic congruity, can cause snapping scapula. Symmetry should be assessed to rule out periscapular muscle atrophy. Neurological assessment is also essential to rule out referred pain. Scapular winging is a common presentation in patients with scapulothoracic bursitis or snapping scapula, which can occur from long thoracic nerve injury and dysfunction of the serratus anterior muscles (5, 8, 15, 16).

Although the diagnosis of snapping scapula based on appropriate clinical assessment is reliable, the determination of the underlying aetiology may require further imaging. While plain film radiographs are the traditional first choice because of their ease of access and low associated morbidi-

Figure 1. Right shoulder snapping in patient with scapula exostosis. (A) An axial CT scan of the right shoulder joint showing the exostosis circled in red. (B) Enlarged detail of the exostosis.
ity, Mozes et al. (11) reported them unreliable for definite diagnosis with only 26.9% detection of scapular bony incongruity compared with 70% detection using CT and 100% detection achieved by 3D-CT. CT, with or without 3D optimization, appears to be beneficial in further characterizing space-occupying skeletal incongruity after plain film detection. However, 3D-CT has demonstrated poor correlation to clinical findings in the setting of non-skeletal aetiologies of snapping scapula, such as scapulothoracic bursitis (8). This, together with additional limitations such as radiation exposure and costs, make CT imaging unsuitable for routine diagnoses of snapping scapula. MRI, by providing an accurate outline of the nature and heterogeneity of soft tissue lesions, remains the most useful diagnostic method in detecting soft tissue aetiologies of snapping scapula (5). In particular, the use of MRI is recommended in investigating scapulothoracic soft tissue and space-occupying lesions as potential aetiologies of snapping scapula when nonoperative treatment fails after clinical diagnosis (see below).

Management

Treatment of patients with this syndrome begins with nonoperative methods and when nonoperative treatments fail, several surgical options exist. Nonoperative treatments include rehabilitation exercises, activity modification and pain management. Physiotherapy and rehabilitation are recommended when the offending cause of snapping scapula are altered posture, scapular winging, or scapulothoracic dyskinesia (17). Scapular malposition can lead to abnormal force distribution throughout the shoulder joint resulting in abnormal shoulder kinematics and problems with motion (18). Controlled scapular position on the thorax is essential for optimal shoulder function, providing maximum force to the rotator cuff muscles while contracting (19). The direction of the rehabilitation plan will depend on factors causing the snapping scapula. Several studies reported improvements in clinical parameters and improved rotator cuff muscle strength after restoration of scapular muscle balance (5). Corticosteroid injections (CSI) have also proven to be effective as an initial nonoperative treatment, which can be particularly useful as a diagnostic tool differentiating between scapular superomedial angle pathology and scapulothoracic bursitis in patients with superomedial angle pain (20). Extracorporeal shockwave therapy (ESWT) is another nonoperative modality, which has been successfully used in the treatment of snapping scapula bursitis (21). Both ESWT and CSI can be utilized as adjuncts to the rehabilitation program (21). If nonoperative management is proven ineffective, open or arthroscopic scapular superomedial resection and scapulothoracic bursectomy are the most frequently performed surgical procedures. One study managed snapping scapula with pectoralis minor tendon release, though the long-term effect of such treatment remains to be established (22). Previous studies suggest that surgical treatment should be reserved to patients with symptomatic snapping scapula after a 3-to-6-month period of unsuccessful nonoperative management (22-24).

A recent study compared the effectiveness of open superomedial scapular resection to nonoperative management for milder snapping scapula presentations (25). No significant difference in the management of snapping scapula outcomes between operative and nonoperative modalities was reported; with patients subjected to the operative intervention presenting with more pain at baseline (25). However, this study failed to provide any conclusive evidence regarding a potential superiority of nonoperative versus operative management because of its non-randomized nature and possible pre-operative differences in symptom severity between the two groups (25).

Arthroscopic or open scapulothoracic bursectomy is recommended for refractory patients who are symptomatic but do not present scapular skeletal abnormalities on imaging (20). Although arthroscopy offers improved cosmesis and earlier rehabilitation, potential risks associated with this approach include the possibility of injury to neurovascular structures when penetrating the rhomboids, the intraoperative swelling, and the inability to evaluate the potentially pathologic trapezoid bursa (26). The choice of arthroscopic versus open or combined procedures largely depends on the surgeon experience and so far, there are no studies directly comparing the two procedures.

In summary, patients presenting with medial scapular border tenderness, palpable crepitus, and audible snapping should prompt high clinical suspicion of snapping scapula. Focused history and physical examination in conjunction with imaging procedures to assess structural aetiologies, when nonoperative management fails, are essential initial steps towards the diagnosis. Nonoperative management of snapping scapula in the form of analgesia, physiotherapy, local CSI and/or ESWT should be initiated for 3 to 6 months before considering surgical management. Open or arthroscopic bursectomy with or without superomedial angle resection, can then be carried out for refractory patients depending on the musculoskeletal pathology presented.

**SNAPPING OF THE ELBOW**

**Definition and classification**

Snapping elbow is a rare condition, which is largely associated with more frequent pathologies such as epicondylitis or intra-
cellular free bodies (4, 27). Patients usually seek attention when the snapping becomes associated with pain and/or limited function (4). In some patients, the condition may be triggered by athletic performances (28). However, it is important to emphasize that for most cases, the condition can be unrelated to any sport activities (4). Snapping of the elbow joint is rightly divided into lateral (intra-articular) and medial (extra-articular) snapping as pathology, diagnostic strategy, and treatment of these two conditions are different (table I) (4, 29).

Briefly, snapping over the medial humeral epicondyle is caused by dislocation of the ulnar nerve or a part of the triceps tendon and is demonstrated by dynamic ultrasound and by an accurate physical examination (4, 27, 28, 29). The treatment is based on open surgery (30). Lateral snapping over the radial head has an intra-articular pathology: a synovial plica, a torn annular ligament, intra-articular tumor pathologies or a meniscus-like remnant from the fetal elbow (4, 31, 32). Pathology can be visualized by conventional arthrography, magnetic resonance arthrography, MRI and arthroscopy while conventional MRI and radiographs often appear normal (33, 34). Treatments for lateral snapping usually consist in arthroscopic or eventual open resection (4, 35, 36) (table I).

**Extra-articular (medial) snapping**

Cases of snapping more frequently involve extra-articular structures, in particular the ulnar nerve that displaces anteriorly or the tricipital tendon that snaps ventrally to the medial epicondyle during complete elbow flexion. The ulnar nerve usually snaps in the 70°-90° flexion range, while the triceps tendon at 115° of flexion. Usually, the shot is easily audible and visible. It is not uncommon for the two conditions to coexist as the snap of the triceps favors that of the nerve, producing a double snap on the medial side of the elbow (27-29). Watts and Bain (37) found that out of 17 patients with compression of the ulnar nerve at the elbow, 14 had a pathology associated with snapping of the triceps tendon. Triceps snapping has various causes and is generally attributed to a morphologically anomaly at the distal attachment of the medial head of triceps brachii (38). Some authors (38) attribute this medial dislocation to cubitus varus, either congenital or post-traumatic (39) or an abnormal insertion of the medial head of the triceps brachii (40), or a fourth muscle belly of the triceps brachii that inserts on the medial portion of the olecranon, more distally than the standard triceps brachii insertion (41). Finally, hypertrophied medial head due to working or sport activities can also cause triceps snapping (28).

The diagnosis starts with physical examination. Triceps dislocation can be palpated during elbow flexion-extension movements and, in some cases, can even be visible. The clinical diagnosis is confirmed by a careful selection of imaging modalities. X-ray images are usually normal unless there is a previous trauma or a congenital osseous anomaly. Conventional MRI has limited diagnostic value when performed with extended elbow as in routine investigations. MRI of the elbow in various flexion positions may effectively demonstrate the dislocation of the anatomical structures involved in snapping. Dynamic ultrasound is often used as it allows real-time visualization of the underlying pathology (42).

Conservative treatment is initiated at first and consists in stopping at risk-activities, a course of non-steroidal anti-inflammatory drugs, a brace limiting elbow flexion to 70° and

![Table I. Snapping elbow subtypes (medial and lateral) are caused by different pathologies and are clinically different entities.](image-url)

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<thead>
<tr>
<th>Snapping of the Elbow</th>
<th>Snapping sites</th>
<th>Diagnosis</th>
<th>Treatment</th>
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<td><strong>Medial</strong></td>
<td>Ulnar nerve skipping</td>
<td>Clinical evaluation</td>
<td>Reduction of activity</td>
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<td></td>
<td>Medial displacement of the distal triceps</td>
<td>Ultrasonography</td>
<td>Transposition/stabilization of the nerve</td>
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<td>Snapping of the brachial muscle</td>
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<td>Resection/suture of tendon</td>
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<td>Postero-lateral rotator instability</td>
<td>High resolution MRI</td>
<td>Arthroscopic/open resection</td>
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<td>Lateral displacement of the distal triceps</td>
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corticosteroid injections. If the patient continues to experience symptoms despite the treatment, then a surgical solution is attempted (30). Surgical treatment consists of laterally transposing the dislocated medial head of the triceps brachii or, if small, in its removal. If the snapping triceps is due to cubitus varus, a rather rare event, humeral osteotomy must also be performed to realign the elbow (30). Once the snapping triceps has been corrected and depending on the ulnar nerve intraoperative stability and on the results of the electrophysiology examination, possible interventions on this nerve may be necessary. They consist in neurolysis and in subcutaneous or submuscular transposition of the nerve (28). After the surgery, the elbow is immobilized in 90° flexion for one month to control pain while passive motion between 0° and 90° is allowed. After 1 month, active and passive movements are allowed. Outcomes are reported to be very good with a satisfactory return to physical activities (28).

Recently, a fixing and redirection tendon technique has been used to treat a case of snapping lateral triceps. This technique, previously described for repairing rotator cuff tears, is the two-strand-overhand locking (TSOL) knot, which in combination with the double pulley technique provides promising results in the treatment of this type of snapping (43). Isolated ulnar nerve instability is not a common condition and is usually accompanied by the snapping of the medial head of the triceps muscle over the medial epicondyle. Dislocation usually occurs in the dominant extremity (27). Because of recurring events in the ulnar nerve, instability during elbow flexion and extension is felt like a snap. Repeated trauma can lead to ulnar neuritis (29). The isolated snapping ulnar nerve may be palpated. MRI in the flexion position can show that the ulnar nerve has come out of its groove (44). Dynamic ultrasound can reveal the snapping of the ulnar nerve during elbow movements (44). A recent study using dynamic ultrasound visualization identified a significant prevalence of ulnar nerve displacement in young baseball players, although some forms of nerve subluxation may also be observed in asymptomatic patients (45). In case of ulnar neuropathy, nerve conduction studies and electromyography (EMG) can assess nerve damage but are not useful to identify the underlying aetiology. Patients with mild pain, without recurrent snapping and not much discomfort in every day-life, may receive conservative treatment (NSAIDs, posterior elbow splints, rest, and physical therapy). However, we should emphasize that non-surgical treatments reduce symptoms but do not eliminate the underlying snapping (46, 47). If there are recurrent snapping and ulnar neuropathy, surgical treatment should be considered. The surgical procedure usually involves ulnar nerve dissection and transposition to the anterior medial epicondyle. Although the nerve can be transferred to either the submuscular or the subcutaneous fixation, submuscular fixation makes the surgery safer (46).

Dincer et al. (29) transposed the ulnar nerve from its natural location posterior to the medial epicondylye to an anterior site, under the fascia of the flexor carpi ulnaris muscle. The medial head of the triceps muscle was also elevated and moved laterally. Intraoperative examination revealed the disappearance of the pathology. Surgical procedures such as excision of the triceps medial head, lateral lubrication of the triceps and partial medial epicondylectomy may be necessary if there is also snapping of the triceps (30, 48-52). Dincer et al. (29) advised to accompany the transposition of the ulnar nerve with the release or excision of the medial head of the triceps independently of the involvement of the triceps muscle in the snapping. In some cases, after isolated ulnar nerve procedures, snapping persisted and a secondary surgery was made necessary to intervene on the triceps muscle (39, 48, 53). An adequate intraoperative examination may avoid these repeated surgeries.

Rare causes of extra articular pathology include snapping of the triceps on the lateral epicondyle and snapping of the brachialis muscle, a powerful forearm flexor. Snapping brachialis is a rare condition and only a handful number of cases have been reported in the literature (54). Patients with this condition, usually, present with anteromedial elbow swelling, pain and snapping on elbow extension and/or supination (54). Normally, the very medial portion of the brachialis muscle is located medial to the trochlear border during elbow flexion and extension. Snapping brachialis occurs if the medial portion of the muscle dislocates anterolaterally, to lie outside the medial border of the trochlea during elbow flexion. On elbow extension, the tendon returns to its normal position often with a visible and/or audible snap (54). Surgical release or excision of the medial portion of the brachialis muscle responsible for the snapping results in resolution of symptoms (54). Another, rather rare, occurrence of medial snapping at the elbow joint concerns the medial cutaneous nerve of the forearm, which runs medially to the medial epicondyle. In a case series by Cesmebasi et al. (51) dynamic ultrasound demonstrated snapping of the medial cutaneous nerve of the forearm in all four patients included. Patients were surgically treated with nerve transposition, with prompt resolution of symptoms.

**Intra-articular (lateral) snapping**

The forms of intra-articular snapping are mainly due to the presence of intra-articular loose bodies (figure 2), as in synovial chondromatosis or in post-traumatic capsular calcifications (4). Snapping of the annular ligament is an uncommon cause of lateral elbow pain. Slipping of the annular ligament
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Figure 2. Intra-articular snapping of the elbow due to intra-articular free bodies. Lateral (A) elbow radiographs and CT axial images in (B) and (C) respectively, showing intra-articular free bodies. (D) Arthroscopic image of intra-articular free bodies.

...into the radiohumeral joint, while flexing and extending the elbow can cause lateral snapping and pain in the elbow (31). A positive history of fractures of the distal humerus or proximal radius or a dislocation of the radial head are possible risk factors for snapping annular ligament (31, 33, 34, 55, 56). Aoki et al. (55) also suggested that multiple microtrauma could cause degeneration of the ligament. However, in most patients, elbow symptoms start after an acute event; suggesting that, besides chronic degeneration, snapping annular ligament can be caused by trauma (35). Diagnosis of snapping annular ligament is usually confirmed by MRI or dynamic ultrasound of the elbow (33, 34). Treatments have included arthroscopy or an open procedure with removal of the interpositioning part of the annular ligament (31, 33, 34, 55, 56).

Being snapping annular ligament an unusual condition, only a limited number of cases have been reported in the literature. In these studies, symptoms and treatments are limited to one or several patients making difficult to determine the most common clinical features associated with diagnosis and to evaluate effectiveness of treatment options (31, 34, 55, 57, 58). However, a recent paper reported a case series study in which patients with annular ligament snapping were treated with either resection (through arthroscopic or open procedure) or open annular ligament reconstruction (54). Interestingly, both surgical procedures led to a significant improvement in pain and daily functioning. Hence, these data indicate that either resection or reconstruction of the annular ligament can be performed in patients affected by this type of snapping with a substantial and clinically relevant amelioration of symptoms (35).

...syndrome is mostly manifested with lateral-sided elbow pain commonly accompanied by local tenderness, painful limitation of movements and snapping. The general consensus is that this condition should be initially treated with conservative therapy. Patients are advised to decrease the amount of physical activity, restore a range of motion with a guided physiotherapy and use non-steroidal anti-inflammatory drugs (36, 64, 65). In case of symptomatic impinging plica, confirmed by dynamic ultrasonography and no resolution after conservative measures, arthroscopic removal should be considered without any delay to avoid possible secondary cartilage degenerative changes (36, 64, 66).

In a recent case series study, 64 patients with a history of elbow pain, snapping and unsuccessful non-operative treatment were subjected to arthroscopic resection of the synovial plicae in the radiohumeral joint (64). At final follow-up, 47% of these patients reported to have a satisfactory joint function while only 19% had a function similar to that of the healthy population (64). In striking contrast with these results, Kim et al. (36), reported that arthroscopic resection of symptomatic synovial plicae on a cohort of 12 throwing athletes led 75% of the patients to have an excellent outcome and 17% to have a good outcome. Reasons underlying these differences remain to be seen. However, it has been postulated that the different composition of the two study groups could explain the different outcomes. The study by Kim et al. (36) included healthy and young athletes (mean age: 22 years) while the cohort of patients in the study by Pedersen et al. (64) were nonathletic and had a mean age of 44 years. Concisely, snapping of the ulnar nerve is extremely rare and an associated snapping triceps tendon should always be suspected. The primary diagnostic method is dynamic ultrasonography. Surgical treatment is the elective method for both a final diagnosis and a definitive treatment. Since snapping of the ulnar nerve and of the triceps are frequently associated, it is important to evaluate intraoperatively the possibility to intervene on both to avoid repeated surgeries.
Symptomatic plicae, at the elbow, causing pain and snapping are at first clinically evaluated. Clinical suspicion may be supported by using MRI or ultrasound scan. If symptoms persist despite initially non-operative management, surgical treatment with arthroscopic resection appears to be effective and safe.

**SNAPPING OF THE WRIST**

**Definition and clinical signs**

Snapping wrist is a relatively rare condition with few reported cases in the literature. The terms “snapping wrist” and “trigger wrist” are interchangeably used to define a pathology presenting with painful sensation and a clicking sound during finger and wrist movements (2, 6). The definition of trigger wrist remains controversial. Historically, the snapping wrist has been considered a condition in which movement of the wrist or fingers leads to the triggering of the wrist. However, in 1986, Desai et al. (67) proposed to use the term trigger wrist to indicate triggering which occurs following the movement of the wrist and not that of the fingers (68). This is true trigger wrist while the condition in which triggering at the wrist is associated with finger movements would be more appropriately described as trigger finger at the wrist (6). Recently, it has been proposed that both true trigger wrist and the trigger finger at the wrist are combined under the unifying denomination of trigger wrist (6). These differences are dictated by the causative pathology and its location.

**Aetiology**

Trigger wrist is caused by various conditions (table II). The pathology may generate from volar structures, such as the flexor tendons, from dorsal structures such as the extensor tendons and carpal bone, and from tumors (6). Suematsu et al. (69) proposed to classify trigger wrist into three different categories. In class A, several causative factors have been identified. The most common causes are benign tumors affecting the tendon, synovial tissue, and nerve sheath, namely ganglion, fibroma, lipoma, fibro lipoma, angiolipofibroma, leiomyoma, tenosynovitis and pigmented villonodular synovitis (PVNS) (70-79).

Class B in Suematsu’s classification is due to anomalous muscle belly including an abnormal lumbrical muscle or abnormal muscle belly of the flexor digitorum superficialis (70). In class C, there is a combination of both pathologies. For instance, anomalous muscle belly of the lumbrical muscle with tenosynovitis and anomalous muscle belly of flexor digitorum superficialis with fibroma (74).

In the true trigger wrist, the pathology is related to the extensor compartment and intracarpal pathology (68). Lemon and

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<th>Table II. Leading causes of triggering at wrist at specific sites.</th>
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Engber (80) reported a case presented with true trigger wrist due to a nodule in the extensor carpi radialis longus tendon. The triggering occurred each time the nodule passed through the second compartment. The triggering was treated by reduction tenoplasty and releasing the second compartment. Koob and Steffens (81) reported a synovial mass around the extensor carpi radialis brevis and extensor pollicis long tendon at the level of extensor retinacular. True trigger wrist can also be due to recurrent subluxation of extensor carpi ulnas tendon (82).

However, some of these patients may also present with carpal tunnel syndrome (83). This is due to the mass effect within the carpal tunnel that compresses the medial nerve and needs to be addressed during carpal tunnel release (72). Leading causes of trigger wrist accompanied by carpal tunnel syndrome reported so far, include soft tissue tumors such as giant cell tumor (figure 3) and intramuscular lipoma (70), flexor tenosynovitis and tendon adhesions, wrist ganglion cysts, anomalies of the flexor tendons, localized amyloidosis and gouty tophus deposit (6, 84). Recently, Enayati et al. (84) reported muscle belly hypertrophy and extension to the carpal tunnel as a common underlying cause of both trigger wrist and carpal tunnel syndrome at the volar side of the wrist.

More particularly, snapping wrist can be secondary to ligament injuries, a condition known as mid-carpal instability, due to the rupture of the radiotriquetral ligament and the ulnar bundle of the arched palmar ligament (85). Additional causes include navicular-lunate instability, capitate-lunate instability and cartilaginous free bodies (85). Swann et al. (86) reported two cases of young patients with dorsal snapping wrist after traumatic injury. In both cases, the patients had hypertrophy of the articular surface of the radio-triquetal ligament, with an abundance of scar tissue, without associated inflammatory reaction. One of the most frequent sites of snapping is the ulnar extensor of the carpus as its partial rupture can lead to its medial dislocation during wrist motion (87).

As previously stated, snapping wrist can be caused by tumor formation near the carpus. In particular, fibromas or other less frequent neformations (lipomas, lipofibromas, leiomyomas, giant cell tumor and hemangiomas) could originate from the superficial or deep flexor tendon at the crossing with flexor retinaculum (83). Unfrequently, neformations could originate from the lumbrical muscle or from the respective tendon: fibromas are the most common cause, followed by lipomas, giant cell tumor, leiomyoma and hamartoma. Abnormalities of the muscle belly affecting the superficial flexor of the fingers, the deep flexor of the fingers, the extensor indicis proprius and the lumbrical muscles are described. In these patients, the enlarged muscle belly has been identified as the cause of the snapping (88). A case report described a patient with trigger at the wrist due to the median nerve (89). The patient had a lipofibromatous hamartoma affecting the carpal nerve and carpal tunnel syndrome (89).

Nodular neformations of extensor tendons could determine snapping by impingement with the capsule or retinaculum of the extensors. Usually, it is secondary to partial rupture of the tendon that generates mucoid cysts, synovial cysts or masses in soft tissues. The most affected tendons are the long radial extensor of the carpus and the short extensor of the carpus, both with the long extensor of the thumb or isolated lesions of the superficial flexor of the fingers. Chronic tenosynovitis could also be associated with exudative nodules that could generate the impingement with relative click, as in rheumatoid arthritis or in gout (90, 91).

De Quervain’s stenosing tenosynovitis can cause snapping on the radial side of the wrist (92) while tenonitis with the formation of calcific bodies similar to rice grains is common in LES (79). Idiopathic synovial cysts, ganglions and iatrogenic cases were also described. Itsubo et al. (93) reported a case of snapping elicited by the release of the flexor tendon of the fifth finger causing its subluxation to the hook of the hook bone. Iwasaki et al. (94) reported a case of a patient, in which harvesting of the palmaris longus tendon produced a snapping wrist, possibly due to fibrous scar tissue between the flexor longus of the thumb and the superficial flexor of the fingers. Recently, Subramanyiam et al. (95) described a case of snapping wrist due to an accessory tendon in the first extensor channel. Another cause of snapping involves the common extensor of the fingers, near the methacarlo-phalangeal joint. Typical phenomenon in boxers, it is due to repeated trauma on the knuckles of the 3rd and 4th ray, with rupture of the reti-
naculum. Ultrasound easily shows the tendon to displace ulnarly during the handshake (96). A non-traumatic form of snapping of the common extensor, affects the 5th ray and it is a condition due to the presence of a supernumerary fibrous band that causes snap (97). De Quervain’s tenosynovitis is rarely associated to tendon click in the first extensor compartment. Impingement between the flexor tendon of the fingers and the flexor retinaculum is a less frequent cause of snapping wrist. This variant may be distinguished from that due to carpal instability as in this case the snapping occurs with the movement of the fingers, regardless of the movement of the radiocarpic. Trigger wrist is a relatively uncommon condition and there are no major studies reporting on its incidence. Sometimes is also underreported as patients may have been diagnosed at first with carpal tunnel syndrome or with trigger finger (2).

The stenosing tenosynovitis, commonly known as trigger finger, is thought to be caused by inflammation and is characterized by the painful popping or clicking sound elicited by flexion and extension of the affected digit (98). Inflammation and hypertrophy of the retinacular sheath progressively of the flexor tendons restricts the motion of the flexor tendons. This sheath normally forms a pulley system in each digit that serves to maximize the flexor tendon’s force production and efficiency of the motion. Possible causes include local trauma and overuse but also pathologies such as arthritis rheumatoid, diabetes and gout may act as contributing factors (98). Diagnosis may require clinical and ultrasound evaluation. Initial management of trigger finger is conservative and involves non-steroidal anti-inflammatory drugs, metacarpophalangeal joint immobilization, and corticosteroid injections (99). If non-operative treatments are unsuccessful, then surgery is recommended (99).

Physical examination and diagnosis

A detailed patient history may help in the diagnosis. Patients may complain about triggering, snapping, clicking, or catching sensation at the wrist and some may even experience pain in the palm on gripping objects. Triggering at wrist is caused by finger movements, wrist movements, or forearm supination or pronation. It is necessary to differentiate whether it is a trigger finger, true trigger wrist or trigger finger at wrist. For this, an accurate clinical examination is performed to avoid unnecessary surgery, releasing of A1 pulley, or steroid injections (6).

Information about the precise nature of the triggering must be obtained from patients. If a patient does experience triggering with the movement of the fingers, and discomfort is perceived around the wrist, especially during flexion or extension of the fingers, this is highly suggestive of a trigger finger at wrist. However, it should be taken into account that triggering of the fingers may occur several weeks before the appearance of the trigger wrist phenomenon (6). In addition, this analysis may be made difficult by the fact that patients may present with discomfort or pain caused by finger movements and not by wrist movements (2). Finally, paresthesia may be brought about by the flexion of the fingers (6).

Patients can present with trigger wrist occurring during movements of the fingers or with wrist movements. Presence of tenderness around A1 pulley is suggestive of trigger finger while its absence may suggest trigger wrist. For trigger wrist, any swelling at the wrist or malunion around the wrist joint should also be examined. Palpation for any bony prominence, clicking or crepitus during wrist movements is also indicative of trigger wrist. Finally, examination for the presence of the carpal tunnel syndrome should be performed (6). A simple radiograph of the wrist joint is needed to see any possible bony pathology such as malunion, instability or arthritis of the carpals bone (6, 89). For soft tissue assessment, ultrasound under dynamic modality would allow the assessment of the triggering pathophysiology (78). MRI or ultrasound may be necessary to further assess the space-occupying lesion within the carpal tunnel and determine characteristics of the mass such as its extension and its possible origin. Nerve conduction studies are indicated for patients with median nerve compression symptoms. Once this information is acquired, surgical intervention can be planned and successfully executed through adequate and complete excision (78).

Management

Conservative management is initially advised. The patient will be advised for activity modification to reduce the wrist and finger movements. If the pain during triggering is troublesome, a wrist splint is prescribed together with analgesics, as necessary. Patients with symptoms of carpal tunnel syndrome will need the routine conservative management before definitive surgical intervention.

Surgical treatment will depend on the causative factor. After radiological investigation to delineate the mass, surgery will be planned under general anesthesia (78). However, the advantage of doing the surgery under local anesthesia is that, during the surgery the patient can be asked to actively move the fingers or the wrist to confirm the resolution of the symptoms (100). This is advisable for patients with trigger wrist and carpal tunnel syndrome without definitive lesion detected by MRI or even CT scan. Inappropriate management may lead to worsening of the symptoms such as severe tenosynovitis, flexor tendon adhesion, or advanced carpal tunnel syndrome with tenar muscle atrophy, which requires a more extensive exploration and reconstructive surgery (2). The prognosis is good if it is done at the early stages of the disease. In chronic
cases, with irreversible pathology to the surrounding tendon or even the median nerve, the prognosis is guarded. Briefly, due to its uncommon presentation and various possible pathologies, diagnosis of trigger wrist requires detailed history taking and examination. Cardinal symptoms of trigger wrist are more than two fingers triggering at the wrist with mild to moderate carpal tunnel syndrome and palpable mass or crepitus felt over the wrist. Subsequent investigation such as plain radiograph or ultrasound will guide detailed radiological investigation such as MRI or CT scan to delineate the extension and origin of the lesion before definitive surgery.

CONCLUSIONS
Symptomatic snapping syndrome may severely affect the every-day life of individuals and in athletes, may force them to quit the agonistic activity. Most clinicians have limited experience with the snapping syndrome, as it is a rare condition. Therefore, it is important to raise awareness about this condition to avoid the risk of misdiagnosis, delays in relevant treatment and even, inappropriate surgical procedures. Here, we summarize current evidence related to the aetiology, diagnosis and treatments of snapping phenomena occurring at the level of the shoulder, the elbow and the wrist. Multiple and various in nature can be the causes of snapping, making the diagnosis of this condition quite challenging, at times. Although the diagnosis relies, at first, on focused history and physical examination, advanced imaging technologies may provide an excellent assist in the diagnostic process and even the treatment of these diseases. Nonoperative management consisting in reduced activity, physiotherapy, local CSI and analgesia is attempted at first; however surgical intervention still appears to be, in many cases, the most effective and safe treatment.

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The authors declare that they have no conflict of interests.

REFERENCES
M. Moscagiuri, A. Frizziero, D. Bigliardi, et al.