Peripheral nerve lesions in the neck, shoulder girdle and upper limb can occur anywhere along the extraspinal extent of the nerve – between the intervertebral foramen and the most distal nerve endings in the extremities. The function of the nerve becomes impaired either as the result of an entrapment phenomenon, or as the outcome of an injury causing bruising or elongation of the nerve tissue.

Entrapment phenomena occur typically at four different sites giving rise to four different mechanisms (see Chapter 2):

- Pressure on a distal nerve causes mainly analgesia as well as some paraesthesia in the territory of the nerve.
- When a nerve trunk or plexus becomes compressed, the release phenomenon – paraesthesia when the pressure ceases – is found.
- Nerve root compression is characterized by pain and paraesthesia, felt in the corresponding dermatome, and often followed by sensory and motor deficit in the same segment.
- Pressure on the cervical spinal cord is painless. An early symptom is paraesthesia with multisegmental distribution. When the compression becomes more severe, numbness, incoordination, spasticity and hyperreflexia may occur.

Disorders of the spinal cord and nerve roots are discussed in Chapters 2 and 8.

Disorders of the spinal accessory nerve

Anatomy

The accessory nerve is a cranial nerve (XI) and consists of two parts. Its main part is the spinal root; the other is the cranial root.

The spinal root takes origin in the spinal cord from a small pillar of nuclei in the anterolateral part of the anterior horn of the levels C1–C5/C6. The fibres leave the cord between the anterior and posterior rami of the nerve root. They join and form a strand that ascends parallel to the spinal cord and enters the skull through the foramen magnum.
Nerve Lesions and Entrapment Neuropathies of the Upper Limb

The cranial root takes its origin in the caudal part of the nucleus ambiguus in the medulla oblongata.

Both parts accompany the glossopharyngeal (IX) and vagus (X) nerves in their exit through the jugular foramen. The fibres originating from the nucleus ambiguus then join the vagus nerve and the other fibres – the real spinal accessory nerve – descend towards the muscles they innervate (Fig. 1).

Innervation

The spinal accessory nerve is a pure motor nerve and innervates the sternocleidomastoid and the trapezius muscles (Fig. 2).

Disorders

A lesion of the spinal accessory nerve may be either idiopathic or result from a compression along its course. Idiopathic spinal accessory neuropathy may occur in isolation or in combination with a disorder of other nerves (glossopharyngeal, vagus, long thoracic or dorsal scapular).1

Mechanical lesions of the spinal accessory nerve can occur at different levels:

- Within the skull where the cause is usually tumourous.2,3 This is uncommon.

- At the level of the exit through the jugular foramen where again, rarely, metastases or schwannomas may affect the nerve.4,5

- At the level of the neck where iatrogenic trauma, for example biopsy of lymph nodes in the posterior triangle, forms the commonest cause of isolated paralysis.6–12 Injury rates from these procedures are reportedly 3–8%.13,14 External traumas may also damage the nerve.

Mononeuropathy of the spinal accessory nerve

The patient initially complains of intermittent pain in the shoulder girdle area, which soon may become permanent.15,16 At the same time, the arm starts to feel weak and heavy, which leads to some functional loss.17 Exceptionally, pain is absent. Pain normally lasts for about 3 weeks, after which it disappears spontaneously.

Inspection elicits an asymmetrical neckline with drooping of the effected shoulder. This may be accompanied with lateral displacement and winging of the scapula.18 Typically, winging is minimal and is accentuated during arm elevation, with the scapula moving upwards with the superior angle more lateral to the midline than the inferior angle. Limitation of active elevation of the arm is a consistent finding, and in one series of patients, the majority could only elevate to 80–90°.19 Passive neck, scapular and arm movements do not influence the pain. Resisted elevation of the shoulder girdle is weak. Resisted external rotation of the arm makes the scapula more prominent medially.20 In severe cases the trapezius muscle may be wasted. The diagnosis is confirmed by asking the patient to adduct both scapulae while the therapist applies counter-pressure at the medial border of the inferior scapular angle (Fig. 3). In neuritis of the accessory nerve, the scapula on the affected side can easily be pushed away at the side.

Spontaneous cure of motor function of the trapezius is the rule and usually takes about 4–8 months.21,22 If inadequate functional recovery is seen after a year, additional conservative treatment is unlikely to be beneficial and surgery is indicated. In a recent review of the literature, authors have reported good...
Nerve lesions and entrapment neuropathies of the upper limb

Peripheral nerve

Disorders of the brachial plexus

Anatomy

The ventral rami of the spinal nerves C5, C6, C7, C8 and T1 unite to form the brachial plexus. Occasionally a prefixed (C4) or postfixed (T2) ramus takes part in the formation of the plexus. Several interconnections lead to the formation of trunks, divisions, cords and branches.

Trunks

There are three trunks: superior, middle and inferior. The superior trunk is created by the fusion of the ventral rami of C5 and C6. The middle trunk is the continuation of the ventral ramus of C7. The inferior trunk is formed by the ventral rami of C8 and T1.

Divisions

The three trunks divide into an anterior and a posterior part. The posterior parts form the posterior cord. The anterior parts form the other cords: the superior trunk continues in the lateral cord and the inferior trunk in the medial cord. The superior and inferior trunks also give off branches for the middle trunk, thus forming interconnections.

Cords

The cords are lateral, posterior and medial according to their relation to the subclavian/axillary artery. The lateral cord is formed from fibres of the superior trunk, together with fibres from the middle trunk. The posterior cord results from the fusion of fibres originating from the three trunks. The medial cord is the continuation of the inferior trunk.

Innervation

The brachial plexus is responsible for the complete motor and sensory innervation of the shoulder girdle and upper limb.

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Nerve Lesions and Entrapment Neuropathies of the Upper Limb

**Traumatic disorders**

The brachial plexus lies quite superficially within a very mobile shoulder girdle and is closely related to the different bony structures of neck, shoulder girdle, shoulder and thorax. This situation makes it very vulnerable. Traumatic disorders are therefore one of the commonest causes of brachial plexus dysfunction. As the result of traction injuries (e.g. motorcycle accidents), compression by dislocated (e.g. shoulder luxation) or fractured bones (e.g. fracture of the clavicle) or by haematomas, and intraoperative or birth injuries, larger or smaller parts of the plexus may become damaged, leading to total or partial syndromes.

**Upper brachial plexus palsy**

This is called Erb–Duchenne’s paralysis and is defined as a palsy of C5 and C6 and sometimes of C7. There is a motor deficit of the muscles innervated by the nerves originating from these fibres and possibly a sensory deficit in the C5 and C6 dermatomes (lateral and anterior aspects of arm and forearm and radial aspect of hand and fingers) (Table 1).

The patient cannot bring the arm up and has difficulty in bending the elbow; there is a visible atrophy of the deltoid, supraspinatus and infraspinatus muscles.

**Middle brachial plexus palsy**

If the middle part of the brachial plexus becomes damaged by trauma, the serratus anterior and rhomboid muscles remain unaffected. There is slight weakness of the deltoid and supraspinatus muscles, which results in the patient not being able to elevate the arm above the horizontal. Elbow flexion is weak because of paresis of the biceps muscle. Sensation remains normal.

**Lower brachial plexus palsy**

This is known as Dejerine–Klumpke paralysis and is not so common but it causes severe disability. The lesion affects the C8 and T1 segments, and quite often also C7. As a result, there

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Long thoracic</td>
<td>Serratus anterior</td>
</tr>
<tr>
<td>Dorsal scapular</td>
<td>Rhomboids</td>
</tr>
<tr>
<td>Suprascapular</td>
<td>Supraspinatus</td>
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<tr>
<td></td>
<td>Infraspinatus</td>
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<tr>
<td>Axillary</td>
<td>Deltoid</td>
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<td>Teres minor</td>
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<td>Musculocutaneous</td>
<td>Coracobrachialis</td>
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<td></td>
<td>Biceps</td>
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<tr>
<td>Radial</td>
<td>Brachioradialis</td>
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<tr>
<td></td>
<td>Supinator</td>
</tr>
</tbody>
</table>

**Anatomy**

The thoracic outlet is the space bounded by the upper part of the sternum, clavicle, first rib and the first thoracic vertebra. Towards the centre, it is limited by the trachea and oesophagus. It forms the communicating area at the base of the neck for the passage of blood vessels and nerves from mediastinum and neck to the axilla and into which the dome of the pleura rises upward. The vagus, phrenic nerves, sympathetic trunk and thoracic duct also pass through the same openings.

The first rib has a flat upper surface. In its anterior portion there are two grooves, which are separated by the tubercle at which the anterior scalene muscle inserts. The more medial groove accommodates the subclavian vein. Behind the muscle, in the posterior groove, both subclavian artery and brachial plexus are found (Fig. 5).

The clavicle overlies the first rib just cranial to the posterior groove. At this site, compression of the neurovascular structures between first rib and clavicle is possible; this mainly happens to the most medially localized trunks, which contain fibres of C8 and T1, so causing symptoms in the territory of the median and the ulnar nerve.

**Space-occupying lesions**

Metastatic tumours – usually originating from the breast, the lung or the lymphatic system – may invade the brachial plexus.

The superior pulmonary sulcus tumour (Pancoast) typically invades the lower trunk of the plexus as well as the sympathetic ganglia at the base of the neck. A lower brachial plexus dysfunction is then accompanied by Horner’s syndrome (see p. 3 of online chapter Disorders of the thoracic cage and abdomen).

Aneurysm of the subclavian artery and pseudoaneurysm of the axillary artery are other possible causes of compression of the brachial plexus.

**Thoracic outlet syndrome**

Thoracic outlet syndrome (TOS) is a vague term, only suggesting the presence of a disorder within the area of the thoracic outlet. Although it is generally accepted that the aetiology is compression of the plexus and vascular bundle in the thoracic outlet, different opinions exist about the pathogenesis. This is expressed in the various names that have been given to the syndrome (see Box 1). The consequence of disagreement on the aetiology is that numerous methods of treatment are advocated.
Nerve lesions and entrapment neuropathies of the upper limb

Types

According to Cyriax, the syndrome is the outcome of a compression of the most medial branches of the brachial plexus, usually occurring between the clavicle and first rib, seldom as the result of a cervical rib. The compression is usually bilateral, intermittent or continuous and may or may not involve the subclavian artery and vein. 32 It gives rise to a set of neurovascular symptoms, which are rarely present all together. Symptoms of neurological disturbances are usually found but sometimes only features of vascular compression. 33 The brachial plexus is involved in 98% of cases, the subclavian vein in 1.5%, and the artery in 0.5%, 34, 35

Cyriax recognized two main groups of thoracic outlet syndromes based on anatomical and clinical grounds: the cervical rib syndrome and the first rib syndrome. We prefer to substitute for these terms. We make a distinction between the thoracic outlet syndrome caused by anatomical changes and that from postural factors. This approaches the growing agreement about the use of four terms to indicate the presence of the thoracic outlet syndrome; 36, 37 true neurologic, arterial and venous TOS – those syndromes that result from compression by a cervical rib (anatomical variety); and non-specific neurologic TOS 38, 39 – the postural variety.

Anatomical variety

This is caused by structural changes – the presence of a bony cervical rib or a band of fibrous tissue which is found in 0.5% of a normal population. Only 5% of them will ever suffer from

Box 1

**Synonyms for thoracic outlet syndrome**

Shoulder–hand syndrome  
First thoracic rib syndrome  
Cervical rib syndrome  
Brachiocephalic syndrome  
Scalenus anticus syndrome  
Humeral head syndrome  
Costoclavicular syndrome  
Nocturnal paraesthetic brachialgia  
Adson’s syndrome  
Hyperabduction syndrome  
Cervicobrahcial neurovascular compression syndrome  
Fractured clavicle syndrome  
Pneumatic hammer syndrome  
‘Rucksack’ paralysis  
Effort vein thrombosis  
Cervicothoracic outlet syndrome  
Pectoralis minor syndrome  
Subcoracoid syndrome  
Syndrome of the scalenus medius band  
Brachial plexus syndrome  
Paget–Schroetter syndrome  
Naffziger’s syndrome  
Shoulder girdle syndrome  
Haven’s syndrome  
Sympathetic algodystrophy

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Fig 5 • A slightly schematic representation of the relationship of neurovascular structures in the thoracic outlet.
In more severe cases, chronic compressive stenosis of the artery may give rise to claudication of the upper extremity. It may further lead to a poststenotic dilation, sometimes to the formation of an aneurysm. Atheromata arising from these aneurysms or from chronic compression injury of the artery can result in peripheral embolization, which may lead to irreversible damage to the hand and even to the entire arm.\(^34,43–45\)

The presence of an acute ischaemic syndrome of the upper extremity, usually in a young female, suggests the possibility of a thrombosis in a poststenotic aneurysm.

**Venous**

If the subclavian vein is impaired, cyanosis, swelling and oedema of the hand and forearm are the indirect signs of this process. Thrombosis of the subclavian vein may occur.\(^46–48\)

**Postural variety**

This category comprises cases that have an almost identical clinical pattern but in which there is no cervical rib or fibrous band. It is characterized by neurological symptoms typically present after lying down for, say, 2–3 hours. It is a benign disorder seldom resulting in vascular disturbance or in muscular atrophy.

There are two types, depending on the clinical features.

**Acute onset**

This is unusual, but the diagnosis is important although difficult to make. Frequently the patient is admitted to the hospital suspected of having had a heart attack.\(^49\)

Young patients with acute onset of symptoms after carrying a heavy load are typical. They complain of a sudden and severe thoracic pain, radiating down the arm, feel faint and have pain on breathing. All such features draw attention to serious visceral disorders (heart attack, pneumothorax). A short time later, the arm and hand blanche. This situation resolves after a few hours, so that by the time investigations, such as electrocardiography, radiography of the thorax and possibly laboratory tests, have been done, the symptoms have disappeared and the patient is perfectly normal again.

**Slow onset**

This is the more common type. It is very slow in progression and has a benign evolution, seldom giving rise to neurological deficit. The diagnosis is often missed, although not difficult to establish.

It affects the middle aged or elderly, more frequently females. It sometimes occurs in pregnancy. The onset is with pins and needles in the hand and fingers, mainly at night and usually after 2–3 hours of sleep. The process is often bilateral, although worse on one side. Paraesthesia may be felt in all digits but may predominate in the median or ulnar distribution. It wakes the patient, who finds that she has to sit up or walk around for a short period of time, rubbing and moving the hands and fingers, to make the symptoms go. The symptoms disappear after a few minutes, allowing sleep to be continued, although recurrence may take place in the early morning hours. The more physical activity during the preceding day, the worse the symptoms at night.\(^50\) In periods of rest or sickness, when
the patient lies down for the whole day, no pins and needles are felt. Some patients also experience symptoms during the day, on activities such as knitting, holding a newspaper in front of the eyes or bicycling, all of which require some degree of shoulder elevation. Augmenting the pressure by carrying a heavy object may exceptionally provoke the symptoms as well, but normally only to a mild degree.

Cyriax explained this pattern as being the consequence of a diminishing tone in the shoulder muscles, starting in middle age. As a result, the shoulder girdle droops down during the day, resulting in compression of the most medial trunks of the brachial plexus, between the first rib and clavicle. Compression occurs during the day but the symptoms come on mainly at night after the pressure on the nerve has disappeared. He called this the ‘release phenomenon’ (see p. 26). The process seldom leads to damage of the nerve parenchyma with subsequent muscular atrophy because the brachial plexus can recover every night when the pressure is released.

**Functional examination**

The diagnosis is based mainly on the typical history, all passive movements of neck, shoulder and shoulder girdle being normal. The resisted movements are of normal strength and painless, except for C8 or T1 structures in the hand, which may be weakened when the compression is the outcome of a cervical rib. This type of characteristic history should always be followed up with the following additional tests.

**Sustained elevation of the shoulders**

The patient sits in a comfortable position and is asked to shrug the shoulders for about 3 minutes (Fig. 7). This causes maximum release of pressure and therefore may bring on the pins and needles and abolish vascular symptoms if present. However, this test is not always positive when thoracic outlet syndrome is present; in this case, release of pressure must be tried in different positions, either fully raising the arms above the head and maintaining this position for 3 minutes (Fig. 8) or lying supine with both hands on the head for the same length of time (Fig. 9).

**Auscultation, pulse and blood pressure**

The subclavian area should always be auscultated for a bruit, the radial pulse must be checked and blood pressure must be measured. The diagnostic significance, however, is not certain.

**Other tests**

Other tests for thoracic outlet syndrome have been advocated classically. Although we regard them as less specific and reliable, they are mentioned here for completeness. Adson’s test, the modified Adson’s test and Roos’ test (elevated arm stress test) are regarded by some as totally unreliable, because about 50–60% of the normal population have positive findings.

**Adson’s test**

The patient stands with the arm resting at the side. In this position the examiner feels the patient’s radial pulse. Then the patient is asked to take a deep breath and to turn the head towards the involved side. Any change in pulse degree or in blood pressure, preferably measured by a Doppler probe, is noted. If there is a change, it means that the subclavian artery...
Examination of the cervical spine

If pins and needles are present in the upper extremities, a full examination of the cervical spine must always be carried out. In thoracic outlet syndrome, the passive movements of the neck are painless and of full range. When a cervical rib is present, weakness and atrophy of the thenar, hypothenar and interosseous muscles may be found.

Tests for carpal tunnel syndrome

All the specific tests for a carpal tunnel syndrome must be carried out (see below, Lesions in the carpal tunnel).

Technical investigations

It should be emphasized that thoracic outlet syndrome is primarily a clinical diagnosis, based on a full history and a complete clinical examination.

Electromyography (EMG) and conduction studies are of little value, for two reasons. First, the range is quite variable in normal patients. Second, because the stimulating electrode can not be placed proximal to the level of the compression, the compound action potential which is measured does not cross the site of the nerve compression. But EMG is helpful for differential diagnosis, in excluding nerve compression at other levels, such as ulnar nerve impingement at the elbow or carpal tunnel syndrome.

A radiograph of both the cervical spine and thorax can help to detect a cervical rib, a hypertrophic transverse process of C7 (suggesting a fibrous band) or the formation of a clavicular callus. It also helps to exclude a Pancoast’s tumour. A CT scan may demonstrate an abnormal fibrous band.

Angiography (arteriography and/or phlebography) must be considered but is only indicated when the vascular symptoms are so severe that surgery is contemplated. Such can be the case when signs and symptoms of arterial embolism or arterial and/or venous occlusion are present.

Differential diagnosis

Cervical disc protrusions

When a posterolateral cervical disc compresses a nerve root, the cervicobrachial pain is very severe, often worse at night, coming and going without apparent reason. Some articular movements of the cervical spine increase the pain, although, surprisingly enough, their influence may be only very slight.

In posteroentral cervical protrusion with cord compression, pins and needles are felt in both hands and feet and are brought on or increased by neck flexion.

Compression of the ulnar nerve

A lesion of the ulnar nerve provokes pins and needles felt only in the fifth finger and at the ulnar half of the fourth. In compression at the cubital tunnel, some local pain around the elbow may also occur (see p. e138 of this chapter).
Compression of the radial nerve
Pins and needles may be felt at the dorsal aspect of the lower arm, and of the $3\frac{1}{2}$ radial digits. Resisted extension of the hand is weak but painless. Sometimes a drop hand is found (see p. e132 of this chapter).

Carpal tunnel syndrome
Compression of the median nerve in the carpal tunnel causes paraesthesia felt on the palmar aspect of the thumb, index and middle finger and the radial half of the ring finger. Carpal tunnel tests may be positive, although in 50% they remain negative (see p. e155 of this chapter).

Carcinoma of the superior sulcus of the lung (Pancoast’s tumour)
The coexistence of pins and needles and Pancoast’s tumour implies invasion of the brachial plexus, which may give rise to a palsy of the interosseous muscles at the hand. Very often a palsy of the recurrent nerve, causing hoarseness, is also present. Clinical inspection of the face may reveal Horner’s syndrome (ptosis of the upper eyelid, enophthalmia and myosis; see p. 3 of online chapter Disorders of the thoracic cage and abdomen).

Raynaud’s syndrome
This provokes only pain, and no pins and needles. Typically the hand becomes white and then blue in cold conditions.

Treatment

Anatomical variety
Thoracic outlet syndrome due to a cervical rib or a fibrous band can only be treated surgically. Paraesthesia and pain mostly disappear but wasting and weakness seldom resolve completely.

Postural variety
Because of the lack of consensus about the aetiology of this syndrome, various forms of treatment have been described (see Box 2). In the light of the mechanism that we consider responsible for the symptoms, the following approach is proposed.

Posture and exercise
Cases caused by the first rib can be helped by conservative management. However, the first step in the treatment is a clear explanation to the patient of the pressure and release mechanism of the disorder. He or she should understand that the pins and needles at night are the result of compression during daytime and that they come on when the nerve is liberated from the pressure. He or she also should realize that, to get rid of the complaints, pressure on the nerve during the day must always be avoided.

To achieve the latter, the patient is asked to keep the shoulders slightly shrugged all day. Carrying loads and wearing heavy coats must be avoided.

For some weeks, the following daily exercise must be done in the evening. Seated in an armchair, elbows resting on the arms, both shoulders are kept shrugged passively (Fig. 11). This brings the pins and needles on after a while but the position is maintained, even if symptoms become more severe. Once they diminish and disappear spontaneously, usually in half an hour, the shoulders are let down.

If the exercise is repeated daily, the patient soon finds that the paraesthesia comes on later and later at night and then appears only in the early morning hours and, after some more weeks of exercising at night, finally disappears completely. Exercises can then be stopped. But the patient must remember to continue indefinitely to keep the shoulders slightly shrugged during the day.

As mentioned previously, it is important to explain the release phenomenon in clear terms to the patient, so that he/she understands that the pins and needles are due to the nerve being fully liberated from the pressure. Lacking such understanding, the patient will mistakenly regard the exercise as harmful and discontinue it.
Examination in the early stage may show that the neck movements influence the cervicoscapular pain even though the lesion is not articular, but a clear pattern is not found. Once the acute stage of an attack is over and the initial pain has disappeared, a patchy paresis and atrophy will become evident. There is visible atrophy in shoulder and shoulder girdle and isometric testing reveals gross weakness in several muscles. All kinds of combinations of weakness may be possible. A typical feature of neuralgic amyotrophy is the patchiness of the motor and sensory symptoms. Histological studies have already shown that the pathologic process can cause very focal damage to one or a few of the fascicles that make up a brachial plexus trunk or cord, while simultaneously affecting several parts of the plexus as a whole. This is clinically reflected by a wide variety in the possible distribution – and severity – of paresis. Any part of the brachial plexus, and clinically any muscle or skin area can be involved, in all sorts of combinations. It is precisely the recognition of this patchiness that is a very important clue to the diagnosis of neuralgic amyotrophy. The muscles most often involved are: serratus anterior, deltoid, supraspinatus and infraspinatus, followed in frequency by biceps and triceps. The infraspinatus muscle seems to be always affected. Sensory abnormalities are much less pronounced than pain and weakness.

Analgesics may be necessary during the pain period but the other symptoms and signs recover spontaneously. In the majority of patients, the weakness abates in the next few months after the disappearance of the pain.

### Disorders of the long thoracic nerve

**Anatomy**

The long thoracic nerve takes origin in the upper trunk of the brachial plexus from the ventral rami C5, C6 and often C7. It courses behind the brachial plexus and follows the lateral wall of the thorax where it divides into several branches (Fig. 12).

**Innervation**

The long thoracic nerve is a pure motor nerve and innervates the serratus anterior muscle. The serratus anterior is a broad flattened sheet of muscle originating from the first nine ribs and passes posteriorly around the thoracic wall before inserting into the costal surface of the medial border of the scapula (Fig. 13). The main function of the serratus anterior is to protract and rotate the scapula, keeping it closely opposed to the thoracic wall and optimizing the position of the glenoid for maximum efficiency for upper extremity motion.

**Disorders**

The nerve can become affected:

- As the result of iatrogenic causes, such as axillary or first rib surgery

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Table 2 provides a summary and differential diagnosis of the anatomical and postural varieties of thoracic outlet syndrome.

<table>
<thead>
<tr>
<th>Anatomical</th>
<th>Postural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>20–30 years</td>
</tr>
<tr>
<td>Release phenomenon?</td>
<td>At first</td>
</tr>
<tr>
<td>Pins and needles?</td>
<td>All day</td>
</tr>
<tr>
<td>Atrophy and weakness?</td>
<td>Thenar/hypothenar/interosseous</td>
</tr>
<tr>
<td>Cold hands?</td>
<td>Possible</td>
</tr>
<tr>
<td>Cynosis and swelling?</td>
<td>Possible</td>
</tr>
<tr>
<td>Treatment</td>
<td>Surgery</td>
</tr>
</tbody>
</table>

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An orthosis designed to elevate the shoulder has been described. It has good results in 77% of patients.

Surgery

In cases refractory to conservative treatment, resection of the normal first rib can be performed. However, this is major surgery and not always successful in relieving symptoms. This approach also carries potential dangers, because of the close relationship to the brachial plexus and subclavian/axillary artery.

Table 2 provides a summary and differential diagnosis of the anatomical and postural varieties of thoracic outlet syndrome.
As the result of direct compression (‘backpack injuries’) because of its long course along the thorax.

As the result of excessive shoulder activity. Although the association is well established, there is no consensus of exactly how trauma injures the long thoracic nerve. Work on cadavers suggests that injury may take place as the nerve exits the fascial sheath that encompasses it, either in the form of a traction injury or in a ‘bow-stringing’ effect.

As the result of idiopathic neuritis.

**Mononeuropathy of the long thoracic nerve**

The patient presents with pain around the affected shoulder, which either arises spontaneously or is linked to some traumatic event. This pain may radiate down the arm and to the scapula. In addition, he typically complains of shoulder weakness. The pain usually resolves spontaneously over the next several weeks, but the patient is left with weakness and a winged scapula.

Upon inspection, medial scapular winging is evident, with the medial and inferior borders closer to the spine and lifted superiorly when compared to the normal side.

Examination of the shoulder shows a limitation on active elevation of the arm of about 45–90°. Passive movements are of full range; resisted movements are normal. The diagnostic manoeuvre is to ask the patient to push against a wall with the arms stretched out horizontally in front of the body (Fig. 14). In this position, the vertebral border of the scapula lifts further from the thoracic wall due to the loss of serratus anterior scapular protraction.
Nerve Lesions and Entrapment Neuropathies of the Upper Limb

Disorders of the suprascapular nerve

Anatomy

The suprascapular nerve takes origin from the upper trunk of the brachial plexus with fibres from the C4 to C6 ventral rami. It courses through the posterior triangle of the neck, underneath the trapezius muscle. The nerve passes through the suprascapular notch of the scapula, which is bridged by the thick transverse scapular ligament (Fig. 15). After entering the supraspinatus fossa the nerve gives off two motor branches to the supraspinatus muscle and then passes laterally within the fossa, providing sensory branches to the posterior capsule of the glenohumeral joint and acromioclavicular joint. It then goes around the lateral border of the base of the spinous process to the infraspinatus fossa, where the nerve terminates by supplying motor branches to the infraspinatus muscle.

Innervation

The nerve innervates the supraspinatus and infraspinatus muscles and receives sensory and proprioceptive branches from the glenohumeral and acromioclavicular joints, as well as the subacromial bursa and posterior aspect of the capsule.

Disorders

The suprascapular nerve has a short course and several sites of relative fixation, making it vulnerable to both traction and compression forces. The nerve is fixed at both its origin at the Erb point on the brachial plexus and at its terminal insertion on the infraspinatus. The nerve is relatively fixed at the suprascapular notch, and anatomical studies have shown that motion does not occur at this point. 

Entrapment can be caused by:

- Entrapment of the nerve by a ganglion, or a tight ligament at the level of the suprascapular notch
- Entrapment at the spinoglenoid notch
- Acute brachial plexitis (the suprascapular nerve is involved in 97% of the cases)
- Trauma, e.g. shoulder luxation or scapular fracture
- Idiopathic neuritis.

A lesion at the suprascapular notch is either the result of a ganglion cyst or a traction trauma. Downward traction of the scapula can result in opposition of the suprascapular nerve against the sharp inferior border of the transverse scapular ligament. Cross-body abduction or protraction with forward flexion, as seen in fencing, throwing sports, racquet sports, and weight lifting, have also been found to maximally stretch the suprascapular nerve.

Entrapment can also occur more distally at the spinoglenoid notch, which is more commonly seen in athletes whose sports require rapid forceful external rotation movements, such as volleyball. The cocking motion for the smash results in rapid external rotation of the shoulder; this rapid motion of the infraspinatus muscle is thought to pull the suprascapular nerve against the base of the scapular spine, resulting in nerve injury at this level. Injury to nerves in the spinoglenoid area has also been noted secondary to ganglion cysts.

Patients complain of a fatigue-like posterior shoulder pain which may be aggravated by activities that stretch or mobilize the nerve, such as combing one’s hair or moving the scapula.

Fig 14 • Test to confirm long thoracic nerve palsy.

Fig 15 • Anatomy of the suprascapular nerve.
Patients with pathology at the spinoglenoid notch may also present with painless and isolated wasting of the infraspinatus muscle as the nerve fibres at the notch contain no sensory afferents.

Inspection shows atrophy of the supraspinatus and/or infraspinatus muscles. On clinical examination, full passive lateral rotation of the arm may provoke some pain, as may all active and passive scapular movements. Passive horizontal adduction of the arm is also painful. Depending on the level of nerve involvement, there is weakness in abduction or external rotation, or both. Proximal injury of the suprascapular nerve at the level of the suprascapular notch causes the weakness of both abduction and external rotation, while nerve injury at the level of the spinoglenoid notch affects only the infraspinatus and results in isolated weakness of external rotation.

An EMG shows a denervation of the infra- and suprascapularis muscles which, together with the history of an injury, confirms the diagnosis. Ultrasonography can be used to show a ganglion. Magnetic resonance imaging is appropriate to diagnose entrapment.

The management for suprascapular nerve entrapment includes a conservative regimen of observation, rest, analgesics, and cortisone injections into the suprascapular notch. If this treatment is unsuccessful, surgical decompression of the nerve at the suprascapular ligament is necessary. In most patients it leads to full relief.

**Disorders of the axillary nerve**

**Anatomy**

The axillary nerve takes origin in the posterior cord of the brachial plexus from fibres derived from C5–C6. It curves around the neck of the humerus and passes through the quadrilateral space close to the inferior shoulder joint capsule. This quadrilateral space is bounded by the teres minor superiorly, the long head of the triceps medially, the teres major inferiorly, and the humeral neck laterally and forms a potential site of compression for the axillary nerve as it passes from the anterior to the posterior aspects of the shoulder. After passing through the quadrilateral space, the axillary nerve divides into anterior and posterior branches, which supply the anterior and posterior portions of the deltoid muscle. The posterior branch of the axillary nerve also supplies the teres minor and supplies cutaneous sensation overlying the deltoid (the upper lateral cutaneous nerve of the arm). The short length of the axillary nerve renders it vulnerable to stretch injuries, especially during shoulder dislocation (Fig. 17).

**Innervation**

The axillary nerve is mixed. The motor branches innervate the deltoid muscle and the teres minor muscle. The sensory branch innervates the skin over the deltoid region and the upper and lateral part of the arm.
Cyriax, (his p. 153) some patients tend to relax the axillary nerve as much as possible by elevating the scapula at the same side during the first few weeks after the injury. The process is a consequence of spasm of the trapezius and leads to pain on passive side flexion of the neck towards the contralateral side. Deltoid weakness is often masked by the activity of supraspinatus and pectoralis major. 120 Because the supraspinatus muscle is not involved, active elevation of the arm remains possible, and weakness is only found on resisted abduction. The diagnostic test is to ask the patient to abduct the arm to 90° and to bring it further backwards into horizontal extension (Fig. 18). This is impossible with an axillary nerve lesion. Spontaneous cure is possible, but takes about 6 months. Care should be taken to mobilize the shoulder during the recovery so as to avoid an immobilizational arthritis. If no improvement has occurred after 6 months, surgical decompression is needed via a release of the teres minor and major tendinous insertions.121

Disorders of the radial nerve

Anatomy

The radial nerve takes origin in the posterior trunk of the brachial plexus and thus contains fibres from C5–T1. It reaches the lateral wall of the axilla and winds around the posterior aspect of the humerus in the groove for the radial nerve. It then pierces the lateral intermuscular septum to enter the
The nerve tissue. The symptoms and signs depend on where, along the nerve, the lesion lies. The radial nerve may become affected at five different sites: the proximal and middle part of the upper arm, the distal part of the upper arm, the proximal part of the forearm and the distal part of the forearm (Table 3).

**Lesions at the proximal and middle part of the upper arm**

When the lesion results from an injury – fracture or dislocation of the humerus – it is usually combined with an axillary palsy. Space-occupying lesions in the axilla or the use of old-fashioned axillary crutches are other (rare) causes.

**Innervation**

The motor part of the radial nerve innervates mainly the extensors of the arm and forearm: triceps and anconeus muscles, brachioradialis, extensors of the wrist, supinator and extensors of thumb and fingers.

The sensory part supplies the skin of the lateral arm, the posterior part of the forearm, the radial dorsum of the hand and the skin of the dorsal aspect of the proximal and middle phalanges of 3½ radial digits.

**Disorders**

The radial nerve is quite frequently affected by pathological conditions. This happens in more generalized diseases, such as poisoning by heavy metals (e.g. lead), but also in more localized lesions, either traumatic or following entrapment of
Table 3  The radial nerve and its branches

<table>
<thead>
<tr>
<th>Branching off</th>
<th>Nerve</th>
<th>Supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper third of upper arm</td>
<td>Posterior cutaneous nerve of arm (sensory)</td>
<td>Skin of lateral arm</td>
</tr>
<tr>
<td>Middle third of upper arm</td>
<td>Muscular rami (motor)</td>
<td>Triceps, Anconeus, Skin of posterior forearm</td>
</tr>
<tr>
<td></td>
<td>Posterior cutaneous nerve of forearm (sensory)</td>
<td></td>
</tr>
<tr>
<td>Lower third of upper arm</td>
<td>Muscular rami (motor)</td>
<td>Brachioradialis, Extensor carpi radialis longus, Extensor carpi radialis brevis (Brachialis)</td>
</tr>
<tr>
<td>Proximal forearm (elbow)</td>
<td>Superficial terminal branch (sensory)</td>
<td>Skin of radial dorsum of hand, Supinator</td>
</tr>
<tr>
<td></td>
<td>Deep terminal branch (motor)</td>
<td>Extensor digitorum, Extensor digiti minimi, Extensor carpi ulnaris</td>
</tr>
<tr>
<td></td>
<td>Posterior interosseous nerve (motor)</td>
<td>Abductor pollicis longus, Extensor pollicis longus, Extensor pollicis brevis, Extensor indicis proprius</td>
</tr>
<tr>
<td>Distal forearm (wrist)</td>
<td>Superficial terminal branch dorsal digital nerves (sensory)</td>
<td>Skin of dorsal aspect of proximal and middle phalanges of 3½ radial digits</td>
</tr>
</tbody>
</table>

**Fig 20**  Sensory supply in the hand by the radial nerve.

Examination reveals weakness of extension and supination of the elbow, together with weakness of extension of wrist, fingers and thumb. This results in a characteristic position of elbow and hand – *porter’s hand* or *drop hand*. Because of the anastomoses between the posterior cutaneous nerve of the arm – first branch of the radial nerve – and other neighbouring nerves, sensory deficit is not common but when it occurs it is found at the lateral aspect of the arm and posterior aspect of the forearm as far as the dorsum of the wrist.

Differential diagnosis must be made from spinal cord, intraspinal and radicular lesions (C7), as well as conditions affecting the brachial plexus, neuritises or myopathies.

**Lesions at the distal part of the upper arm**

Lesions are more common at the distal than the middle and proximal part of the arm. The palsy may be traumatic – fracture of the humerus,124,125 fracture or dislocation of the elbow – or may result from a sustained pressure just proximal to the elbow. This is typically the case in a patient who has fallen asleep with the arm over the edge of a chair or has lain all night with the arm resting against the hard edge of a bunk – ‘Saturday night paralysis’. The full radial syndrome develops, except that the triceps and anconeus muscles are unaffected, as is the brachioradialis muscle. The patient awakes with a painless dropped wrist.

Sensory disturbances may occur at the dorsal aspect of the forearm when the posterior cutaneous nerve of the forearm is involved, although this area may have overlapping supply from neighbouring sensory nerves – the lateral and medial cutaneous nerves of the forearm. The superficial sensory branch of the radial nerve is responsible for cutaneous deficit at the radial and dorsal aspect of the hand, except when anastomoses with either the lateral cutaneous nerve of the forearm – a sensory branch of the musculocutaneous nerve – or the dorsal branch of the ulnar nerve at the hand provide an alternative pathway.

**Lesions at the upper part of the forearm**

Level with the head of the radius, the radial nerve divides in its two terminal branches – the superficial (sensory) and deep
has become fibrous, this opening forms the arcade of Fröhse,127
(1) muscular rami running towards the supinator, extensor digitorum, extensor digiti minimi and extensor carpi ulnaris muscles, and (2) the posterior interosseous nerve of the forearm, which supplies the long abductor and extensors of the thumb as well as the extensor indicis proprius. When a lesion occurs proximal to this division, symptoms of both are combined. As the branches supplying the brachioradialis, extensor carpi radialis longus and brevis, and brachialis escape the compression, there is only weakness of supination, extension of the fingers, ulnar deviation of the wrist and extension and abduction of the thumb.

Deep branch of the radial nerve

Conditions such as fracture and/or luxation of the head of the radius or local inflammatory processes, for example chronic bicipitoradial bursitis, may cause compression of the deep branch of the radial nerve where it turns around the head of the radius. The result is weakness of supination, ulnar deviation of the wrist and finger extension.

Posterior interosseous nerve of the forearm

Just below the point where the deep radial nerve comes to lie at the dorsal aspect of the forearm, the posterior interosseous nerve of the forearm branches off and passes through the deep and superficial heads of the supinator brevis muscle126 (Fig. 21). When the edge of the upper border of the superficial head has become fibrous, this opening forms the arcade of Fröhse,127 also called the radial tunnel. The nerve then passes further down along the interosseous membrane and innervates the abductor pollicis longus, extensor pollicis longus and brevis, and extensor indicis proprius muscles.

The posterior interosseous nerve can be compressed as the result of: (1) an injury, usually a fracture (or the hardware used to fix fractures),128,129 or elbow joint dislocation;130 (2) space-occupying lesions, such as synovial proliferations from the elbow joint in rheumatoid arthritis131,132 or soft tissue tumours,133,134 for example lipomas;135 or (3) fibrous bands, which can be traumatic in origin (e.g. Volkmann’s contracture) or developmental (arcade of Fröhse).136–140

Many authors believe that compression of the posterior interosseous nerve at the point where it passes through the arcade of Fröhse is a cause of lateral elbow pain and they therefore consider it as a type of tennis elbow. We do not agree that this condition should be considered to be a (resistant) type of tennis elbow, because the lesion does not lie in the extensors of the wrist. Hagert et al141 also regarded epicondylitis and posterior interosseous nerve entrapment as ‘two different disorders, which have nothing to do with each other, and which should therefore not be mixed up’.

Radial tunnel syndrome

In 1883, Winckworth stated that the posterior interosseous nerve could become compressed where it passes through the supinator muscle.142 Since Roles and Maudsley described the radial tunnel syndrome in 1972,143 this pathology has become recognized as a cause of resistant tennis elbow.133,136,144–146 This idea is based on the reports of good results after surgical decompression of the posterior interosseous nerve at the radial tunnel.147,148

Nerve compression by the edge of the superficial supinator muscle seems to occur on passive pronation of the forearm. On active supination the increase in pressure is much greater, which has led to the conclusion that dynamic compression of the posterior interosseous nerve by the edge of the superficial supinator muscle is probably a cause of local nerve irritation and pain.

In compression of a predominantly motor nerve, such as the posterior interosseous nerve, the main symptom would be paralysis. Kopell and Thompson, however, state that entrapment of a motor nerve may cause diffusely localized dull aching pain.149 The pain would originate from the nociceptive thin or non-myelinated afferent nerve fibres of muscular and extramuscular origin.150

However, the symptoms described by different authors are very similar to those found in tennis elbow: pain at the lateral side of the elbow, radiating distally along the posterior aspect of the forearm. The pain may be constant and can be brought on or aggravated by exertion, especially rotation movements, and the symptoms continue for some time after the causative strain has ceased. There is also diffusely localized pain on resisted supination and/or pronation as well as on resisted extension of the middle finger. Local tenderness is present over the proximal and posterior aspect of the forearm, at the suspected entrapment site. These symptoms and signs correspond with the clinical picture of what we have described as type IV (muscular) tennis elbow (see p. 313).

Treatment consists of surgical decompression. After surgery, the pain seems to disappear gradually, in the course of 6–12 months.

Werner analysed the hypothesis that posterior interosseous nerve entrapment can be a cause of lateral elbow pain.151 The following investigations were carried out: patients with suspected posterior interosseous nerve entrapment were operated on by decompression of the nerve and then subjected to a follow-up for 2 years; the topographical anatomy was

**Fig 21** • Course of the posterior interosseous nerve through the supinator muscle.

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compared with observations at dissections; the epidemiology and symptomatology were compared with that in a series of cases of lateral epicondylitis. He concluded that lateral elbow pain may indeed be caused by dynamic compression of the posterior interosseous nerve and that it can be relieved by decompression of the nerve where it enters through the supinator muscle. The diagnosis is based solely on palpation and on positive resisted supination. Pain on resisted extension of the middle finger seems to be an unreliable test. Electrophysiological examinations (EMG) are of limited diagnostic value.

Using Cyriax’s methods to examine lateral elbow pain, we have some difficulties in accepting the existence of a radial tunnel syndrome, giving rise to pain. If the main symptom is pain at the lateral aspect of the elbow and the pain is clearly aggravated by a resisted movement, one should think of a lesion of a contractile structure and try to find out what structure is at fault:

- Pain on resisted supination draws attention to the supinator brevis muscle.
- Pain on resisted extension of the middle finger does not exclude a tennis elbow, because this test also implicates the extensors of the wrist.

Differential diagnostic tests should be included (see p. 313). Pain on palpation is not reliable: the lateral aspect of the elbow is always tender to the touch and therefore the response to the palpatory manoeuvres may be false-positive.

We conclude that the diagnosis of radial tunnel syndrome is probably made too often. A clear distinction should be made between the two different conditions: (1) tennis elbow, a lesion in the radial extensors of the wrist, and (2) radial tunnel syndrome (Table 4). Other authors share this view.\textsuperscript{152,153}

We believe that what has been described in the literature as radial tunnel syndrome very often has nothing to do with the posterior interosseous nerve at all, but is simply a lesion of an extensor of the wrist, the symptoms of which have erroneously been attributed to that nerve. We speculate that the good results claimed for surgery may be the outcome of either spontaneous recovery or incidental permanent lengthening of some muscular fibres during operation. In the latter instance, the operation, ostensibly for radial tunnel syndrome, unintentionally becomes an operation for tennis elbow.

When entrapment of the posterior interosseous nerve at the elbow occurs it should give rise to a dull ache over the lateral aspect of the elbow, eventually radiating down the posterior aspect of the forearm. In due course, weakness will be found of abduction and extension of the thumb and of extension of the index finger. Paraesthesiae are absent.

Stewart confirms our view.\textsuperscript{154} His ideas are also based on clinical findings and have been reinforced following the publication of three studies showing that, in this group of patients in whom pain is the main symptom, there is little evidence for a focal nerve lesion.\textsuperscript{155–157}

\textbf{Lesions at the distal part of the forearm}

The superficial sensory branch of the radial nerve runs under the brachioradialis muscle along the radial artery and further towards the hand. At the level of the wrist it divides into five dorsal digital nerves (Fig. 22) and supplies the skin at the dorsal aspect of the thumb, index, middle and radial half of the fourth finger but not the terminal phalanges, which are supplied by the median nerve.

In the distal part of the forearm the nerve may rarely become affected as the result of external pressure (e.g. damage by handcuffs);\textsuperscript{158,159} the result is paraesthesia and sensory deficit over the radial and dorsal aspect of the hand. If the pressure involves the medial and dorsal branch to the thumb, for example after the protracted use of small scissors, the ulnar aspect of the thumb becomes numb.

\begin{table}[h]
\centering
\begin{tabular}{|c|c|}
\hline
\textbf{Tennis elbow type IV} & \textbf{Radial tunnel syndrome} \\
\hline
Elbow pain & Lateral pain on hand movements & Ache at elbow, especially at rest or after repetitive pronation and supination \\
\hline
Resisted test & Pain on wrist extension and radial deviation & Negative \\
\hline
Tenderness & In deep muscles overlying neck of radius & In supinator \\
\hline
Weakness & No muscular weakness & Possible weakness of thumb and index finger \\
\hline
\end{tabular}
\caption{Differential diagnosis: type IV tennis elbow and radial tunnel syndrome}
\end{table}

\begin{figure}[h]
\centering
\includegraphics[width=0.8\textwidth]{image}
\caption{Branching of the superficial sensory branch of the radial nerve.}
\end{figure}
Disorders of the ulnar nerve

Anatomy

The ulnar nerve takes origin in the lower trunk of the brachial plexus from the spinal nerves C8–T1. It runs from the axilla down the medial aspect of the upper arm. At mid-humerus it pierces the medial intermuscular septum towards the posterior compartment. It follows the medial head of the triceps onto the retrocondylar groove at the elbow. It then courses under the aponeurotic arch joining the two heads of the flexor carpi ulnaris muscle – the cubital tunnel – and follows its course towards the wrist, under this muscle (Fig. 23). At the wrist it runs through Guyon’s tunnel before it divides into its terminal branches.

Innervation

There are no branches in the upper arm. In the forearm motor branches supply the flexor carpi ulnaris muscle and the ulnar half of the deep flexor digitorum. Sensory branches supply the skin of the ulnar half of the hand. At the wrist the ulnar nerve, as it leaves Guyon’s tunnel, divides into a superficial and a deep branch. The deep branch is motor and innervates the hypothenar muscles, the interossei, the two ulnar lumbricals and, on the thenar side of the hand, the adductor pollicis and the deep head of the flexor pollicis brevis. The superficial branch is sensory and supplies the skin of the little finger and the ulnar half of the ring finger.

Disorders

Ulnar nerve entrapment is one of the most frequent peripheral neuropathies, especially compression of the nerve at the level of the elbow. As it is a combined motor and sensory structure (Table 5) entrapment leads to a gamut of symptoms and signs. The two most frequent localizations of compression are pressure in the cubital tunnel at the inner side of the elbow, and pressure in the area of Guyon’s tunnel at the wrist.

Lesions at the elbow

In 1877, Panas was the first to describe ulnar nerve palsy, in a paper to the Académie de Médecine. Since then, several

The full ulnar nerve syndrome leads to weakness of the ulnar flexors of wrist and fingers (flexor carpi ulnaris, ulnar half of flexor digitorum superficialis and profundus), the hypothenar muscles (abductor digiti minimi, flexor digiti minimi brevis, opponens digiti minimi, palmaris brevis), the intrinsic muscles of the hand (dorsal and palmar interossei, ulnar two lumbricals) and part of the thenar muscles (adductor pollicis and deep head of flexor pollicis brevis). Sensory deficit is found in the ulnar half of the hand and, in the ulnar two fingers, the entire fifth finger and the ulnar half of the fourth finger.

Fig 23 • Course of the ulnar nerve and the possible locations of compression:
1. Inner side of the upper arm
2. Cubital tunnel
3. Guyon’s tunnel.
Table 5  The ulnar nerve and its branches

<table>
<thead>
<tr>
<th>Branching off</th>
<th>Nerve</th>
<th>Supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Upper arm</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Elbow</td>
<td>Muscular rami (motor)</td>
<td>Flexor carpi ulnaris</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Deep flexor digitorum – ulnar half</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(Superficial flexor digitorum – ulnar half)</td>
</tr>
<tr>
<td>Lower forearm</td>
<td>Palmar cutaneous (sensory)</td>
<td>Skin of ulnar proximal palm of hand</td>
</tr>
<tr>
<td>Proximal to Guyon’s tunnel</td>
<td>Dorsal digital (sensory)</td>
<td>Skin of ulnar dorsum of hand</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Skin of dorsal aspect of ulnar 1½ fingers</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Palmaris brevis</td>
</tr>
<tr>
<td>Distal to Guyon’s tunnel</td>
<td>Muscular rami (motor)</td>
<td>Abductor digiti minimi</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Opponens digiti minimi</td>
</tr>
<tr>
<td>Hand</td>
<td>Superficial terminal branch (sensory)</td>
<td>Skin of ulnar distal palm of hand</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Skin of palmar (and dorsal) aspect of ulnar 1½ fingers</td>
</tr>
<tr>
<td></td>
<td>Deep terminal branch (motor)</td>
<td>Interossei</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Ulnar two lumbricals</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adductor pollicis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flexor pollicis brevis (deep head)</td>
</tr>
</tbody>
</table>

Box 3

Summary of the aetiology of ulnar nerve palsy

Friction
Traumatic
Structural
Postural
Loose body
Space-occupying lesions
Unknown

Aetiology

Entrapment of the ulnar nerve – cubital tunnel syndrome – is the second most common compressive neuropathy of the upper extremity, with only carpal tunnel syndrome presenting more frequently. Entrapment of the ulnar nerve at the elbow has several different causes, summarized in Box 3:

- **Friction** may be the result of recurrent dislocation of the nerve. The patient notices that from time to time the nerve comes out of its groove and then replaces spontaneously. This phenomenon is sometimes accompanied by a paraesthetic twinge felt in the ulnar aspect of the hand and the ulnar one and a half fingers. Recurrent dislocation may in the end lead to ulnar palsy.
- **A fall on the elbow or a direct blow** to the medial aspect of the elbow may bruise the nerve sheath which becomes irritated. The condition is self-perpetuating, as flexion movements continue the trauma.
Diagnosis

The history is obvious. The patient describes paraesthesia and/or numbness in the ulnar half of the fourth and the entire fifth finger and eventually weakness or clumsiness of the hand. A slight ache may also be felt at the elbow. A history of trauma may be elicited or merely that the symptoms came on spontaneously. In spontaneous onset, further questioning may determine whether the cause is postural.

On inspection there may be signs of joint abnormality. Furthermore, there may be wasting of the intrinsic muscles of the hand, the hypothenar muscles or some forearm muscles – flexor carpi ulnaris and deep flexors of the fingers. In severe cases, the patient may present with an ulnar palsy leading to weakness of the ulnar half of the deep flexor digitorum and of the flexor carpi ulnaris muscles, with weakness of the intrinsic hand muscles resulting in a claw hand.

On examination, signs are found that immediately draw attention to the ulnar nerve. A few accessory tests may give some further confirmatory information:

- The elbow is brought into maximal flexion and maintained there for a few minutes, which may bring on the pins and needles and indicates a possible postural cause.

A differential diagnosis must be made with other possible proximal causes: cervical myelopathies, anterior horn lesions, polyneuropathies, myopathies, cervical nerve root problems (C7, C8) and thoracic outlet syndrome, or a more distal cause, which is compression in Guyon’s tunnel.

Electromyography can be useful, and alternatively, an infiltration with a local anaesthetic may prove diagnostic.

Treatment

A diagnosis of cubital tunnel syndrome does not in itself necessitate surgery. In mild cases, patient education and avoidance of strains often leads to spontaneous cure.

Avoidance of postural strains or pressure

The patient’s daily activities should be studied to see what causes or influences symptoms. Precipitating or aggravating postures or movements which stretch or compress the ulnar nerve should be avoided. It may be necessary to explain this in some detail to the patient. Some patients can be helped by the use of a night splint, worn for several months.
Nerve Lesions and Entrapment Neuropathies of the Upper Limb

Infiltration

An injection of 1 ml of triamcinolone suspension about the nerve, not into it, will desensitize the nerve sheath and lead to lasting relief in those cases in which the only symptom is paraesthesia and in which conduction has not yet become impaired (Fig. 25).

Surgery

Surgical treatment is offered for more severe cases and where conservative management is deemed to have failed. Simple decompression involves incising longitudinally over the cubital tunnel to release the surrounding retinacular fibres. This procedure must be performed with some care, as damage to small branches of the nerve may lead to painful neuroma. Some authors believe that a release should be supplemented by medial epicondylectomy. This eliminates the medial epicondyly as a source of compression. The remaining options involve transposition of the ulnar nerve, in which the surgeon moves the nerve anteriorly to a subcutaneous position. In the past decade, various authors have described endoscopic release of the ulnar nerve a safe and reliable treatment for the condition.

Lesions at the wrist

Three different types of compression of the ulnar nerve at the wrist have been described: purely motor, purely sensory and a mixed form, dependent on the site of the compression. Consequently, the diagnosis may not be easy. Furthermore, there is the possibility of a Martin-Grüber anastomosis (see p. e96 of online chapter Applied anatomy of the elbow) which again makes the diagnosis more difficult.

The ulnar nerve, together with the ulnar artery, passes through the tunnel of Guyon. This tunnel lies between two dynamic structures, the pisiform and hamate bones, and is covered by the pisohamate ligament (Fig. 26), which is a continuation of the flexor carpi ulnaris tendon.

Proximal to the wrist, the palmar cutaneous branch arises and runs over the palmar aspect of the forearm and wrist outside the tunnel of Guyon to supply the proximal part of the ulnar side of the palm. A few centimetres more distally the dorsal cutaneous branch arises and supplies the ulnar side of the dorsum of the hand, the dorsal aspect of the fifth finger and the ulnar half of the fourth finger (see Fig. 26).

As it leaves the tunnel of Guyon, the nerve divides into a mainly sensory superficial terminal branch, which supplies the distal ulnar border of the palm of the hand and the palmar surfaces of the fifth and ulnar half of the fourth finger (Fig. 27), and a deep terminal branch, which is entirely motor and innervates nearly all of the small muscles of the hand: palmaris brevis, abductor digitii minimi, opponens digitii minimi, flexor digitii minimi brevis, dorsal interossei, palmar interossei, third and fourth lumbricals, adductor pollicis and the deep head of the flexor pollicis brevis.

Aetiology

The cause may be either intrinsic or extrinsic. Intrinsic causes are a ganglion, the most common cause; a lipoma; an abnormal position of the abductor digitii minimi muscle; anatomical variation in the flexor carpi ulnaris tendon. Extrinsic conditions are an injury with or without fractures of the pisiform or hamate bones and professional or sporting overuse (handlebar palsy).

Symptoms

The symptoms may be compared with those resulting after compression of the nerve at the elbow. Because the ulnar nerve divides into a superficial and a deep branch at the wrist, the
Nerve lesions and entrapment neuropathies of the upper limb

Distal to Guyon’s tunnel, at the hook of the hamate bone; as a result of compression of the deep palmar branch the intrinsic hand muscles are weak.

In the palmaris brevis muscle at the distal part of Guyon’s tunnel; the superficial branch is compressed and gives rise to a purely sensory deficit.

A fifth type of compression with motor deficit of the first dorsal interosseous and the adductor pollicis muscles has been reported by Yu-Sung et al.206

Treatment

This depends on the cause and severity of the lesion. Very often rest and avoidance of the causative strain can be sufficient. In more severe cases, local infiltration with a steroid suspension or surgical decompression may be necessary.

Disorders of the median nerve

Anatomy

The median nerve arises from the junction of the medial and lateral cords of the brachial plexus and thus from the segments C5–T1. In the upper arm the nerve lies superficial to the brachial muscle and arches around the brachial artery. In the proximal part of the forearm the nerve leaves the artery and innervates the pronator teres, flexor carpi radialis, palmaris longus, and flexor digitorum superficialis muscles. It then passes under the bicipital aponeurosis and between the two heads of the pronator teres to enter the anterior compartment of the lower arm. Just distal to that point, it gives off the anterior interosseous (ante-brachial) nerve branch that supplies the flexor digitorum profundus, flexor pollicis longus, and pronator quadratus muscles. The nerve then courses between the flexor digitorum superficialis and profundus muscles. As the muscles change to the tendons in the lower half of the arm, the median nerve runs with the tendons of index and middle fingers to the carpal tunnel. Before passing under the flexor retinaculum and into the carpal tunnel, it gives off the

Fig 26 • The ulnar nerve passes through the tunnel of Guyon (a); but the dorsal cutaneous branch (b) does not.

symptoms may be purely motor (deep branch) or purely sensory (superficial branch). Sensation over the dorsal aspect of the fingers remains unaltered, because the dorsal sensory branch has an origin proximal to the wrist (see Fig. 26).

Four localizations are possible:205

• Proximal to Guyon’s tunnel, at the pisiform bone. This causes compression of the superficial and deep branches with sensory and motor deficit (hypothenar and intrinsic hand muscles).

• Within Guyon’s tunnel, with compression of the deep motor branch, which results in a motor deficit of the hypothenar and intrinsic hand muscles. There is no sensory deficit.

• Distal to Guyon’s tunnel, at the hook of the hamate bone; as a result of compression of the deep palmar branch the intrinsic hand muscles are weak.

• In the palmaris brevis muscle at the distal part of Guyon’s tunnel; the superficial branch is compressed and gives rise to a purely sensory deficit.

A fifth type of compression with motor deficit of the first dorsal interosseous and the adductor pollicis muscles has been reported by Yu-Sung et al.206

Fig 27 • Sensory supply in the hand by the ulnar nerve.

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superficial palmar branch. Distal to the carpal tunnel, it divides into its terminal branches to: the thenar, the radial lumbricals and the palmar aspect and the radial side of the hand and the 3½ radial fingers (Fig. 28).

**Innervation**

There are no branches in the upper arm. Just distal to the elbow the first branches arise for the pronator teres, the flexor carpi radialis, the palmaris longus and the superficial flexor digitorum. The anterior interosseous nerve of the forearm innervates the pronator quadratus, the flexor pollicis longus and the radial half of the deep flexor digitorum. A sensory branch innervates the skin over the thenar and the radial half of the palm of the hand. After passing the carpal tunnel the digital nerves divide to supply the skin over the 3½ radial digits and the muscles of the thenar eminence, except those innervated by the ulnar nerve (see Fig. 28).

**Disorders**

The median nerve (Table 6, Fig. 29) is the most important nerve of the hand, because it renders opposition of the thumb possible, combined with a circular pronation movement, as well as flexion of the radial fingers. Palsy of this nerve leads to total incapacity of the hand.

The full median nerve syndrome affects the pronator teres, the flexor carpi radialis, the radial half of the flexor digitorum superficialis and profundus, the flexor pollicis longus and the pronator quadratus, the thenar (abductor pollicis brevis, superficial head of the flexor pollicis brevis, opponens pollicis) and the radial two lumbricals. The sensory deficit is detected in the radial half of the palm of the hand, the palmar aspect of the thumb, index and middle fingers and the radial half of the ring finger, as well as the dorsal aspect of the distal phalanges of the same fingers.

**Lesions at the lower part of the arm and around the elbow**

The median nerve can become damaged as the result of supracondylar fractures or elbow dislocation. It can also become compressed above the elbow by a supracondylar process and the ligament of Struthers, if the latter is present. Thickening or fibrosis of the bicipital aponeurosis may also cause

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**Table 6 The median nerve and its branches**

<table>
<thead>
<tr>
<th>Branching off</th>
<th>Nerve</th>
<th>Supply</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbow</td>
<td>Muscular rami</td>
<td>Pronator teres</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Flexor carpi radialis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Palmaris longus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Superficial flexor digitorum</td>
</tr>
<tr>
<td>Upper forearm</td>
<td>Anterior interosseous</td>
<td>Flexor pollicis longus</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Deep flexor digitorum (radial half)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pronator quadratus</td>
</tr>
<tr>
<td>Wrist</td>
<td>Palmar cutaneous</td>
<td>Skin of radial palmar aspect of hand</td>
</tr>
<tr>
<td>Hand</td>
<td>Common palmar digital</td>
<td>Abductor pollicis brevis</td>
</tr>
<tr>
<td></td>
<td>Motor palmar digital branch I</td>
<td>Flexor pollicis brevis (superficial head)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Opponens pollicis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lumbral I</td>
</tr>
<tr>
<td></td>
<td>Motor palmar digital branches II–III</td>
<td>Lumbral II</td>
</tr>
<tr>
<td></td>
<td>Sensory palmar digital branches I–III</td>
<td>Skin of palmar aspect of 3½ radial digits</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Skin of dorsal aspects of terminal phalanges of 3½ radial digits</td>
</tr>
</tbody>
</table>
The median nerve innervates the following structures:

1. Pronator teres
2. Palmaris longus
3. Palmaris brevis
4. Flexor digitorum superficialis
5. Flexor digitorum profundus II and III
6. Flexor pollicis longus
7. Pronator quadratus
8. Cutaneous branch for palm
9. Abductor pollicis brevis
10. Flexor pollicis brevis
11. Opponens pollicis
12. Lumbricalis I
13. Lumbricalis II
14. Cutaneous branches for the palmar aspect of radial 3½ digits

Compression. Below the elbow, the problem is most common at the point where the median nerve and its anterior interosseous branch dip between the two heads of the pronator teres. Thickening of the fascia that holds these two heads together can cause compression – the pronator teres syndrome.207,208

Pronator syndrome results from entrapment or compression of the median nerve between the humeral (superficial) and the ulnar (deep) heads of the pronator teres muscle, at the bicipital aponeurosis (lacertus fibrosus), or at the arch of the origin of the flexor digitorum superficialis (Fig. 30). Compression and entrapment may result from anatomic constraints due to congenital abnormalities in the involved tendons or muscles, such as hypertrophy of the pronator teres muscle bellies or aponeurotic prolongation of the biceps brachii muscle.209 These conditions may be clinically silent for years and then suddenly become evident after repetitive pronation–supination stress. Less common causes of pronator syndrome include post-traumatic haematoma, soft-tissue masses and prolonged external compression.

The clinical picture includes weakness of the median innervated muscles distal to the pronator teres muscle – the flexor pollicis longus and the thenar muscles. Sensory deficit is often present. Pain is certainly not the main complaint.

The following accessory tests have been described:

• A resisted pronation during thirty seconds may provoke the symptoms.
• Sustained isometric flexion of the middle finger causes the symptoms
• Tinel’s sign: percussion in the area of the pronator teres muscle.

Infiltration of steroid solution in the area of compression is usually curative. If not, surgical release is performed.

The pronator teres syndrome is probably a tunnel syndrome that is easily overestimated. The picture should be confirmed objectively by sensory and electrophysiological examination.210

When the tentative diagnosis cannot be confirmed, possible compartment syndrome or lesion of the pronator teres muscle should be considered.
Nerve Lesions and Entrapment Neuropathies of the Upper Limb

Lesions at the forearm: anterior interosseous nerve

Compression of the anterior interosseous nerve – a branch of the median nerve – is known as the Kiloh–Nevin syndrome. The most frequent causes of anterior interosseous nerve syndrome are direct traumatic damage and external compression. Traumatic nerve damage may be the result of surgery, venous puncture and injection. External compression of the anterior interosseous nerve may be caused by a soft-tissue mass such as lipoma or ganglion, an accessory muscle, a vascular abnormality or cast pressure.

Clinical findings

Typically, patients with anterior interosseous nerve syndrome experience a dull pain in the volar aspect of the proximal forearm, which sometimes worsens at night, combined with an acute onset of muscle weakness. The muscle weakness affects the thumb, the index finger, and occasionally the middle finger because the deep flexor muscles of these fingers are innervated by the anterior interosseous nerve. Since the anterior interosseous nerve does not innervate the skin, numbness is not associated with the syndrome.

Patients with anterior interosseous nerve syndrome have difficulties in writing and are not able to form an ‘O’ with the thumb and index finger. This characteristic finding, called the circle sign, is due to a lack of innervation of the flexor pollicis longus muscle and the flexor digitorum profundus muscles: the distal interphalangeal joints stay in extension during the pinch.

Lesions in the carpal tunnel

The palmar aspect of the carpus is concave and covered with the transverse carpal ligament. An osteofibrous canal, the carpal tunnel, is thus formed, of which the boundaries are the scaphoid and pisiform bones proximally and the trapezium and hamate bones distally. The canal thus lies more distally and more towards the ulnar aspect than is often thought (Fig. 31).

Within the carpal tunnel are found the median nerve, the tendons of the flexor carpi radialis and the flexor pollicis longus, in separate sheets, and the superficial and deep flexors of the digits within a common tendon sheath (Fig. 32).

Carpal tunnel syndrome is the most common peripheral entrapment neuropathy with an incidence between 1.5 and 3.5 per 1000 subjects per year. It affects mostly women (68%) between 40 and 60 years. While first recognized by Sir James Paget in 1854, it was not until 1942 that Cyriax described irritation of the median nerve under the heading ‘median perineuritis’. The condition remained further unrecognized until 1946, when more detailed description was given. Full appreciation of how common the lesion is followed Phalen’s article.

Aetiology

Clinical carpal tunnel syndrome is caused by a raised intracarpal pressure which results from an increase in content or a
decrease in size of the carpal canal, or some combination of the two. The cause may either be local or systemic. On the basis of observation, two types are distinguished: a full syndrome (symptoms in the entire territory of the nerve) and a partial syndrome (symptoms in only a part of the nerve’s territory) (Box 4).

**Full syndrome**

*Idiopathic*

Half of all cases are idiopathic. Recent research suggests a multi-factorial cause. Ultrasound imaging of the carpal tunnel during dynamic stress testing manoeuvres demonstrates notching, flattening, or compression of the nerve. The roof of the tunnel tightens and lowers at the same time the floor (flexor tendons) of the tunnel tightens and raises, thereby compressing the nerve.

*Traumatic*

Subluxation of the lunate bone following a (possibly minor) trauma suddenly fixes the wrist in flexion, immediately
Space occupying lesions

These can also cause median nerve compression. There are reports of tumorous conditions, hypertrophy of tenosynoviums, tophaceous gout and ganglia that raise the intra-carpal pressure and cause a carpal tunnel syndrome.

Vascular conditions

These include conditions such as spontaneous bleeding in haemophiliacs and in patients on anticoagulant therapy; thrombosis of a congenital median artery may also damage the median nerve.

Partial syndromes

These occur when pressure is exerted on one or several of the most distal terminal branches of the nerve. This can be the result of an injury, incorrect use of a walking stick or a trigger finger (see p. e149 of this chapter).

History and symptoms

The condition starts in one hand, very often the dominant one, but becomes bilateral in 30% of cases. The first symptoms are pins and needles felt in the territory of the median nerve (see Figs 28 and 33): the palmar aspect of the thumb, index, middle and radial half of the ring finger, as well as the dorsal aspect of the distal phalanges of these fingers. Symptoms often are worse at night and are exacerbated by repetitive flexion and extension of the wrist, strenuous gripping, or exposure to vibration. Nocturnal paraesthesiae rarely occur as the only symptom but are mentioned more often in combination with the other complaints (see below). At onset, the symptoms may be vague and diffuse and may involve only one finger – usually the middle one. After a while, the typical distribution becomes clear.

Box 4

Aetiology of carpal tunnel syndrome

Full syndrome
- Idiopathic
- Traumatic
- Subluxation of lunate bone
- Occupational
- Friction
- Rheumatoid arthritis
- Gout/pseudogout
- Amyloidosis
- Haematoma
- Rubellar polyarthritis
- Endocrine
- Hypothyroidism
- Acromegaly
- Pregnancy
- Menopause
- Generalized peripheral neuropathy
- Diabetes
- Uraemia
- Tumours
- Ganglia
- Benign tumours: lipofibroma; neuroma
- Malignant tumours
- Vascular
- Haemorrhage: haemophiliacs; anticoagulants
- Thrombosis of median artery

Partial syndromes
- Traumatic
- ‘Stick palsy’
- Trigger finger

Fig 33 • The median nerve passes through the carpal tunnel to supply the palm of 3½ digits.
After some months a burning wrist pain develops, which may radiate distally into the fingers and proximally to the forearm. The pins and needles, however, do not have this proximal reference but remain distal to the point of compression.

Slight numbness in the radial fingers is sometimes present and, therefore, mentioned by the patient but it is often only detected when sensory deficit is sought during the examination.

In some long-standing or severe cases, the patient may complain of weakness and inability to use the hand: things slip from his or her fingers without noticing (loss of grip, dropping things).

In the assessment of carpal tunnel syndrome the history is most relevant. The precision of the patient’s account very strongly implies the possibility of the presence of the syndrome. The area of paraesthesia described is exactly that of the median nerve and mention is made of the intermittent appearance and activity dependence of the symptoms.

**Findings during inspection and examination**

Inspection may reveal structural changes – post-fracture, deformities in rheumatoid arthritis, ganglia, bony subluxations – conditions that could be relevant when the overall clinical picture is considered.

In long-standing cases, and very occasionally early in the course of the condition, muscular atrophy of the thenar eminence may occur, involving the opponens pollicis and abductor pollicis brevis muscles. The lateral aspect of the thenar eminence becomes flattened and gives rise to a so-called ape hand.

The standard examination of the wrist is usually negative and some accessory tests may then be performed:

- **The carpal compression test:** the wrist is held in supination and extension. The examiner presses at the carpal tunnel while the patient actively flexes and extends his fingers. A positive test is indicated by paraesthesia in the 3½ fingers.
- **Phalen’s test:** the wrist is kept passively flexed for a minute. A positive test is indicated by pins and needles in thumb, index finger, middle finger and radial half of the ring finger (Fig. 34).
- **Modified Phalen’s test:** the wrist is held passively flexed and the thumb, index and long fingers are forcefully flexed as well.
- **Tinel’s test:** percussion of the carpal tunnel gives rise to pins and needles felt in the median nerve distribution (Fig. 35).

In longstanding and/or severe compression, some weakness of the thumb can be detected: abduction (abductor pollicis brevis), flexion (flexor pollicis brevis, superficial head) and opposition (opponens pollicis) may be weak.

The two main physical examination signs, Tinel sign and Phalen sign, have only a moderate sensitivity (20 to 70%) and specificity (70 to 83%). However, in combination with the typical history and localization of the symptoms, they give a useful clinical indication of the probability of a carpal tunnel syndrome. The best way to confirm the tentative diagnosis is a trial injection with triamcinolone which should ease the symptoms for at least 2 weeks.

**Technical investigations**

During the last decades, nerve conduction studies have been considered the gold standard test for the confirmation of diagnosis of carpal tunnel syndrome with sensitivities between 49% and 84%, and specificities between 90% and 95%. Thus, approximately 10% of patients suspected of having clinical carpal tunnel syndrome have normal electrodiagnostic studies. Moreover, abnormal results in a working asymptomatic population occur between 9 and 16%. Therefore, some authors advise the use of the clinical diagnosis for carpal tunnel syndrome as the criterion standard. For the majority of patients who are considered to have carpal tunnel syndrome on the basis of their history and physical examination alone, electrodiagnostic tests do not change the probability of diagnosing this condition to an extent that is clinically relevant.

Attempts have been made with cross-sectional imaging of the carpal tunnel, both with CT and MRI. However, the
sensitivity and specificity of both techniques are low (sensitivity, 23%–96%; specificity, 39%–87%), and for this reason they do not play a role in the clinical assessment of carpal tunnel syndrome. Nevertheless, MR imaging does have clinical utility when the cause of carpal tunnel syndrome is a neoplasm (e.g. neurofibroma), arthritis, or a congenital anomaly and in evaluating the postoperative wrist.

High-resolution sonography is a low-cost alternative to MRI, and has gained increasing popularity. Sonography measures the cross-sectional area of the median nerve and bowing/flatting of the flexor retinaculum. There seems to be a high degree of correlation between the US findings and the nerve conduction studies in diagnosing carpal tunnel syndrome.

Diagnostic infiltration

A much simpler, and very reliable, diagnostic approach is the infiltration of 20 mg of triamcinolone suspension into the carpal tunnel. If diagnosis is right, all symptoms should disappear for at least a few weeks. When symptoms have not disappeared some days after the injection, the diagnosis must be reconsidered.

Differential diagnosis

Carpal tunnel syndrome must be differentiated from a cervical disc protrusion that compresses a nerve root and from thoracic outlet syndrome with compression of a part of the brachial plexus. Both are often difficult.

When distal paraesthesiae are the result of a cervical disc lesion, the symptoms usually come and go in an erratic fashion during the day as well as at night. They are transient and not particularly activity related. The distribution within the fingers is related to a dermatome, but the patient cannot localize the paraesthesia exactly. They are preceded and accompanied by severe root pain and often followed by segmental motor and/or sensory deficit. On examination of the cervical spine, a partial articular pattern is found. The symptoms disappear spontaneously in the course of a few weeks to a few months (see pp. 152–157).

In the early stage of thoracic outlet syndrome, pins and needles are strictly nocturnal and wake the patient – usually a middle-aged woman – after some hours’ sleep as a result of the decompression of the nerve. The paraesthesia are felt in all the fingers and do not have any particular distribution. Decompression tests of the thoracic outlet are positive (see p. e122 of this chapter and Ch. 2).

Other differential diagnoses are cervical myelopathy, anterior horn lesions, polyneuropathies, myopathies and arthropathy of the trapezium–first metacarpal joint.

Treatment

Treatment is conservative and consists of one or more injections into the carpal tunnel. Surgery is called for in refractory or recurrent cases.

Wrist splint

Splinting with the wrist in the neutral position, especially at night, leads to good but possibly only temporary results in about 70% of patients.

More recent studies show best results with a full-time splint.

Injection

As a diagnosis of carpal tunnel syndrome can be made in spite of a negative examination, it remains provisional until therapeutic confirmation has been obtained. Relief, even if it is only temporary, after injection with triamcinolone acetonide is diagnostic. If paraesthesia is not relieved by injection, the diagnosis must be wrong. If the symptoms do not reappear for several months or years, a later recurrence can be treated with a further injection.

Technique: injection

The patient sits next to the couch. The physician stands medial to the affected hand, that lies palm upwards on the couch. The wrist is held in 45° of extension. The proximal border of the carpal tunnel (pisiform and scaphoid) and the palmar tendons of the wrist are easily detectable in this position. A 2 ml syringe is filled with 20 mg triamcinolone and fitted to a 5 cm needle. The tip of the needle is inserted between the tendons (usually ulnar of the palmaris longus) at a point 2 cm proximal to the proximal border of the carpal tunnel and inserted in the direction of the base of the fourth metacarpal bone. It slips, without resistance, between the tendons and under the transverse ligament into the carpal tunnel (Fig. 36). At 4 cm depth, the needle lies at the distal aspect of the carpal tunnel. Some fluid is injected and no resistance or painful sensation should be felt. The needle is then slowly withdrawn while the syringe is further emptied.

Results

All cases of carpal tunnel syndrome respond immediately, but 60% recur within 3 to 6 months and at 2 years only 8% remain cured. Several studies have demonstrated that for the first 3 months, local injections with steroids are equally good or better than surgical decompression for the symptomatic relief of mild carpal tunnel syndrome. However, recurrences after injections are frequent and, although steroid injections are safe and effective for temporary relief, most patients will eventually require surgery for long-term control of their symptoms. We advise that if the symptoms recur more than 6 weeks after the injection, one or two further attempts may be made. When the result is still not sufficient or the recurrence is too rapid – within 6 weeks, or more than twice in 6 months – surgical treatment is required (Fig. 37).

Cases with severe symptoms and evidence of motor atrophy respond poorly.

Surgery

Carpal tunnel release is one of the most common surgical procedures with an annual incidence in the US of 134 per 100000. Although carpal tunnel release is an effective treatment, supported by high-quality evidence, it has several disadvantages, including surgery-related pain and hand
Nerve lesions and entrapment neuropathies of the upper limb

assumed that preservation of the superficial fascia and adipose tissue over the flexor retinaculum allows faster recovery of grip strength, less scar tenderness and pillar pain, and earlier return to work. However, the decision to perform ECTR is influenced by the surgeon’s experience and patient factors, including occupation, socioeconomic status, and preference.

Partial syndrome

Distal to the carpal tunnel the common palmar digital nerve divides into terminal motor and sensory palmar digital branches. In the hand one of these can become compressed, with the result that only part of the territory of the median nerve is involved. There are a number of possible causes.

Traumatic

As the result of a direct blow, usually a fall on the hand, the sensory palmar digital branch to the thumb at the level of its carpometacarpal joint may be bruised. The symptoms are painful pins and needles in the thumb only and there is little tendency to spontaneous cure.

‘Stick palsy’

In elderly people, the sensory palmar digital branches to the index and middle fingers may become compressed in the palm of the hand, just distal to the carpal tunnel, by incorrect use of a walking stick. This results in pins and needles, felt in these two fingers. Instruction in how to use a walking stick correctly is given.

Swelling on a digital flexor tendon

A trigger lesion that has formed on the proximal part of a flexor tendon in the palm of the hand, just distal to the carpal tunnel, may rarely cause compression of the branch of the median nerve that supplies the middle and ring fingers and give rise to pins and needles at the adjacent surfaces of these two digits. Infiltration or surgery are the possible therapeutic measures.
Disorders of the musculocutaneous nerve

Anatomy

The musculocutaneous nerve takes origin in the lateral cord of the brachial plexus and contains fibres from C5 to C7. It pierces the coracobrachialis muscle and descends in the anterior compartment of the arm between the biceps and brachialis muscles. It runs lateral to the distal biceps tendon and then continues as the lateral cutaneous nerve of the forearm (Fig. 38).

Innervation

The musculocutaneous nerve supplies the coracobrachialis, biceps and brachialis muscles. The lateral cutaneous nerve of the forearm innervates the skin at the anterolateral aspect of the forearm.

A lesion of the musculocutaneous nerve is rare and occurs as the result of an injury in the upper arm. This causes motor deficit with weakness of the flexors of the elbow and sensory deficit over the radial aspect of the forearm. When the lesion is at the forearm, it involves the sensory branch – the lateral cutaneous nerve of the forearm – and leads to a similar sensory deficit. This sometimes occurs after a paravenous injection.

Fig 38 • The musculocutaneous nerve.

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