Review paper

# Diagnosis of compression syndromes in neurological practice

## *Diagnostyka zespołów z ucisku w praktyce neurologicznej*

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### Abstract

Compression syndromes are a multidisciplinary issue. Many specialists are involved in the problem of peripheral nerve injury, but it is the neurologist's task to identify the nerve and locate the level of its damage. The nerve damage occurs in the anatomic isthmus and in the area of the nerves' course under the surface of the skin. Compression syndromes manifest themselves with muscle weakness and sensory ailments such as paraesthesia, numbness, and pain. Symptoms occur in the sensory and motor supply of a given nerve. In diagnostics, we use clinical evaluation, provocative tests, electrophysiological diagnostics, and imaging examinations. Treatment is divided into conservative and operational. The increased availability of electrophysiological research in recent years allows for precise differential diagnosis of individual compression syndromes and the selection of optimal treatment. In this study, a clinical picture of the most frequent syndromes of compression syndromes and the role of electrophysiological research in their diagnosis and treatment are presented.

### Streszczenie

Zespoły z ucisku są zagadnieniem wielodyscyplinarnym. Z problemem uszkodzenia nerwów obwodowych spotyka się wielu specjalistów, jednak to zadaniem neurologa jest identyfikacja nerwu i lokalizacja poziomu jego uszkodzenia. Do uszkodzenia nerwów dochodzi w cieśniach anatomicznych oraz w miejscach przebiegu nerwów pod powierzchnią skóry. Zespoły z ucisku objawiają się niedowładami mięśni oraz dolegliwościami czuciowymi, takimi jak parestezje, drętwienia i bóle. Objawy występują w zakresie zaopatrzenia czuciowego i ruchowego danego nerwu. Diagnostyka obejmuje ocenę kliniczną, testy prowokacyjne, diagnostykę elektrofizjologiczną i badania obrazowe. Leczenie dzieli się na zachowawcze i operacyjne. Większa dostępność badań elektrofizjologicznych w ostatnich latach umożliwia precyzyjną diagnostykę różnicową poszczególnych zespołów i wybór optymalnego leczenia. W pracy przedstawiono obraz kliniczny najczęstszych zespołów z ucisku i rolę badań elektrofizjologicznych w ich rozpoznawaniu i leczeniu.

Compression neuropathy is a group of peripheral nervous system diseases that damage individual peripheral nerves as a result of their compression. Due to the mechanism of damage, these syndromes can be divided into two groups: mononeuropathies caused by damage of the nerve located superficially by external compression, trauma, or multiplied micro-injuries (e.g. neuropathy of the superficial nerve in football players); and mononeuropathies caused by compression at the nerve passage through the anatomic fibro-osseous tunnels (e.g. carpal tunnel syndrome). Compression syndromes occur with a higher incidence in conditions predisposed to their development, such as endocrine disorders (diabetes type 2, acromegaly, hypothyroidism, parathyroidism), connective tissue diseases (CTD), obesity, gout, local inflammation, and in physiological conditions such as pregnancy. It is estimated that in 7–43% of pregnant women, electrophysiological tests have found features of carpal tunnel syndrome, and 7% of pregnant women report symptoms

of irritation of the ulnar nerve [1, 2]. A patient reporting to a neurologist complains of severe pain in the sensory supply of a given nerve, intensifying at night, poorly responsive to non-steroidal anti-inflammatory drugs, significantly worsening the functioning and possibly leading to mood disorders [3]. Motor impairments in the form of paresis usually occur later.

The identification of the characteristic pain syndrome allows for appropriate profiling of the interview, including the listed systemic diseases and conditions predisposing to the development of compression syndromes. The next step is to conduct clinical provocation tests. Abnormalities in the neurological examination in the form of muscular atrophies, paresis, weakness of reflexes, and disturbances in the somatosensory system are often the symptoms occurring at the later stage. They also do not allow unambiguous determination of the level of peripheral nerve injury. The most objective, sensitive, and specific method of identifying peripheral nerve injury is nerve conduction test and electromyography. It also allows us to determine the degree of advancement in electrophysiological changes in the nerve, which translates into the selection of optimal treatment and assessment of the prognosis of the return of the function of the damaged nerve.

The doctor referring to the electrophysiological study should determine what disease is suspected and, if possible, determine the scope of the examination. The standard examination is divided into a study of the conductivity in the peripheral nerves (sensory and motor fibres) and electromyography, which is the study of muscle functioning. Electroneurography is performed by stimulation of peripheral nerves by means of low currents and analysis of conduction parameters by using electrodes located in the muscle or on the skin. Electromyographic study consists of puncturing the muscle with a thin needle in order to test the bioelectrical activity of this muscle. The test is performed at rest, in minimal and maximal contraction, which gives a full range of information on the functioning of the muscle.

The basic abnormalities in the nerve conduction study will be the partial decrease of the conduction velocity at the location of compression. In advanced cases, the demyelination process may progress towards a partial or complete conduction block. In the case of a protracted process, secondary axonal damage may occur, resulting in a decrease in the amplitude of the response.

In some of the compression syndromes, and in particular, in the diagnosis of carpal tunnel syndrome, imaging examinations such as ultrasound and magnetic resonance imaging (MRI) may be helpful.

Radiological diagnostics show the features of nerve oedema and surrounding tissues, as well as degenerative changes, post-traumatic changes, and the presence of pathological masses affecting the nerve.

The MR study uses the T1 and T2 sequences. They enable the assessment of the nerve, the muscles it innervates, and the pathologies causing its compression. Damage is indicated by an increase in the T2 signal of the nerve, enlargement of its diameter, and a change of its position. Early symptoms of muscle denervation are seen as an increase in the T2 signal, and their disappearance is demonstrated by high T1 and T2 signals [4].

Ultrasound examination enables the assessment of morphology and continuity of nerves, and of pathological states and structures that may cause their compression, such as tendinitis, cysts, soft tissue bumps, and bone, muscle, and joint anomalies [4]. Despite lower sensitivity in the detection of early oedema changes, compared to the MR scan, ultrasound is very useful due to the short examination time, high availability, and low cost.

In chronic compression neuropathies, a nerve swelling occurring proximally to the location of compression is visible as a reduction in echogenicity and a change in the shape of the nerve to be more round. In the longitudinal cross-section, pressure is visible as an hourglass narrowing. As a result of chronic denervation, the supplied muscles lose mass and undergo retroactive changes, which are manifested by increased echogenicity and loss of the filamentous structure [5].

In connection with the occurrence of mononeuropathy with compression in the course of systemic diseases, it is advisable to extend the diagnosis with laboratory tests, especially toward disorders of carbohydrate metabolism, lipid, thyroid, parathyroid, and gout diseases. In the case of doubts indicating connective tissue diseases, diagnostics should also be carried out in this direction.

When writing about compression syndromes, one cannot forget about genetic syndromes predisposing to their formation. Particular attention should be paid to the hereditary neuropathy with pressure palsies (HNPP).

This neuropathy usually manifests itself in the second and third decade of life but can occur at any age. Its genetic basis is the deletion or less frequent point mutation of the PMP22 gene in chromosome 17p11. HNPP is characterised by an episodic, recurrent demyelinating neuropathy manifested by paresis and sensory disorders without pain. The most common clinical syndromes occurring in the course of the disease are paresis and sensory disturbances in the area of the median, ulnar, radial, and sagittal nerves, and paralysis of the shoulder plexus. In the nerve biopsy, which nowadays rarely is performed, occure structures called tomaculas which are focal myelin thickening. In diagnostics, the key role is played by genetic and electrophysiological studies revealing segmental demyelination in the affected nerves, most expressed in anatomic aneurysms. These changes may lead to con-

duction blocks. After recognising the syndrome, it is important to properly rehabilitate and avoid situations in which there may be pressure on peripheral nerves. The most common and best-studied compression syndrome is carpal tunnel syndrome

## Median nerve

The median nerve is made up of fibres derived from C6-C7-C8-Th1 ventral roots. It originates from the combination of the lateral and medial cords of the brachial plexus. By means of the recurrent branches, it innervates within the hand, the abductor pollicis brevis muscle, the flexor pollicis brevis muscle, the opponens pollicis muscle, and lumbricals I and II. Sensory innervation on the palmar side includes fingers I, II, and III, 2/3 of the radial surface of finger IV, and on the dorsal side the surface of two further finger phalanges I, II, III, and the radial side of finger IV.

Complete damage to the nerve trunk causes the hand to be in the position of a "benediction sign".

### Carpal tunnel syndrome (CTS)

Carpal tunnel syndrome is the most common compression mononeuropathy. It was first described in 1845 by James Paget [6]. It occurs more often in women than men, in a 4 : 1 ratio [7]. In half of cases, it is idiopathic. Among the diseases predisposing to development of the syndrome are diabetes type 2 (5–60%), hypothyroidism (33%), rheumatoid arthritis, connective tissue diseases (20–42%), pregnancy, use of oral hormones, and obesity [8, 9]. A common cause is occupational exposure when performing manual work requiring alternate movements in the wrist.

Among the symptoms reported by the patient, pain, numbness, and tingling of the thumb (and later fingers II–IV) predominate. Pain intensifies at night, awakens the patient from sleep, and can radiate along the limb up to the shoulder. Mitigating factors are heating of the limb and trembling of the hand. Sensory disturbances affect fingertips I–III. Motor disturbances initially manifest themselves in the form of difficulties in performing precise movements (button closure, lifting a coin lying flat on the table). In advanced stages, muscular atrophy of the thenar occurs, particularly the abductor pollicis brevis muscle, which is manifested by the impossibility of touching fingers I and V.

During the neurological examination, provocative tests are used to diagnose CTS.

- Phalen test – the most sensitive, consisting of the increase of pain and paraesthesia while holding the wrist in flex for at least 30 s [10, 11].
- Tinel test – the most specific, consisting of tapping the wrist on the median nerve, which causes pain.

Other tests, such as the compression test, band test, clenched fist test, and elevation test, are less frequently used and have less clinical value.

In addition to provocation tests, sensory tests are applied, such as:

- test of discrimination between two stimuli – the test is positive if the patient does not distinguish between two pricks of two points at a distance of more than 6 mm;
- vibration sensation test.

In the nerve conduction study, only the decrease of the conduction velocity in the sensory nerves of the median nerve at the level of the tunnel is initially recorded. Gradually, as the disease progresses, the final latency increases to the abductor pollicis brevis muscle, the loss of active sensory axons, and then, in the advanced stage, motor axonal loss. The electromyography (EMG) test shows denervation in the thenar muscles.

A ultrasonography (USG) examination of the median nerve shows enlargement of the surface area and nerve perimeter at the level of the pisiform bone. An MRI scan shows a flattening of the nerve and its swelling, which is expressed by an increase in signal intensity in T2.

In the mild stages of the disease, conservative treatment is possible (local and oral steroid therapy, laser, ultrasound, immobilization) [12]. In the case of disease progression, surgical treatment is necessary.

### Pronator teres syndrome

A very rare syndrome in which the median nerve is compressed in the area of the elbow and on the forearm by the pronator teres. It manifests itself in numbness and muscle weakness in the medial nerve supply, as well as pain in the pronator teres when the forearm is turned. The conductivity study is not useful in diagnostics [9]. The MRI scan reveals the denervation of the muscles of the anterior forearm group in the form of an increase in their signal in T2 images. The ultrasound shows nerve oedema proximal to the place of compression. During the supination of the forearm in the longitudinal cross-section, the degree of bending of the nerve is increased.

### Anterior interosseous syndrome (Kiloh-Nevin syndrome)

This manifests as a palsy in the bending of distal finger phalanges I and II with associated forearm pain. The Flexor pollicis longus muscle, flexor digitorum profundus muscle, and pronator quadratus muscle are damaged. The paresis of the latter can be confirmed upon asking the patient to perform the recurrence of the forearm with total flexion in the elbow joint, which eliminates movement from the pronator teres muscle. It is confirmed by an EMG examination, finding denervation in the muscles in question. The conductivity study is not useful [13]. The MR scan reveals the denervation of the muscles of the anterior

forearm group in the form of an increase in their signal in T2 images.

## The ulnar nerve

The ulnar nerve originates from roots C8 and Th1, which form the medial cord of the brachial plexus. Sensorially, the nerve affects finger V, the elbow half of finger IV, and the skin above the hypothenar from the dorsal and palmar sides. Regarding motor functions, it innervates the hypothenar muscles, the interosseous muscles, lumbricals IV and V, the adductor pollicis muscle, the deep head of the flexor pollicis brevis muscle, and the palmaris brevis muscle.

Ulnar nerve mononeuropathy occurs twice as often in men, most often in the fifth and six decades of life [14]. Regardless of the level of damage, patients with mononeuropathy of the ulnar nerve complain about numbness of fingers IV and V, paraesthesia and hyperesthesia on the skin supplied by the nerve, and impediments to the precise movements of fingers. In the later stages of neuropathy, the muscles of the hypothenar disappear [15]. The clinical examination is characterised by the weakening of the conductivity of finger V and the thumb, and the impossibility of spreading fingers II–V.

Complete damage to the trunk nerve causes the hand to be in the position of an "ulnar claw".

### Compression at arm level

Compression of the ulnar nerve at arm level is very rare. It can be caused by injuries, a local disease process that takes place around the nerve, and by the hypertrophy of connective tissues stretched between the septum and the medial epicondyle (arcade of Struthers) [15]. The conductivity study shows a decrease of the conduction velocity at the level of the compression.

### Cubital tunnel syndrome

Compression at the elbow level can occur in the cubital tunnel through the compression of the flexor carpi ulnaris muscle and lateral epicondylitis through repetitive compression, e.g. when leaning on elbows.

In the conduction study, there is a conduction decrease, and sometimes conduction block, at elbow level. The test can be extended by the short-sections method (inching). Conservative treatment can be used in the early stages of the disease. In later stages, surgery is necessary [12].

### Guyon's canal syndrome (ulnar tunnel syndrome) – compression at the wrist level

The compression in Guyon's canal is usually caused by a wrist injury (fractures) or the presence of a pathological mass in the canal (usually ganglions). The conductivity test is supplemented by an ultrasound examination of the wrist, which helps determine the cause of the compression [14]. The examination of the conductivity is supplemented by ultrasound and MR scans of the wrist, which suggest the cause of compression and help the assessment of the morphology of the nerve itself [14]. Surgery can be performed as a treatment by choice [12].

## Radial nerve

The radial nerve originates from the C5-Th1 root fibres forming the posterior cord of the brachial plexus. The nerve innervates all the extensors of the elbow joint, wrist, fingers, the supinator muscle, the abductor pollicis longus, and brachioradialis. Sensory innervation includes the dorsolateral part of the arm, the forearm ridge, the radial part of the dorsal hand, the dorsal side of the thumb, the index finger, and the radial part of the middle finger.

Radial nerve injuries most often have traumatic origin (humerus fracture, fracture within the wrist).

The most characteristic symptom of damage to the trunk of the radial nerve is the "wrist drop", i.e. paralysis of the straightening of the hand in the wrist and the straightening of the fingers. Radial nerve neuropathy, especially after waking up, should be differentiated from central paresis, e.g. as a result of a small ischaemic cortical focus [13].

### Compression in the axillary cavity

The compression of the radial nerve in the axillary cavity causes muscle weakness within the entire motor supply. The patient cannot perform an extension in the elbow joint, or in the wrist and finger joints. The most common causes of compression include the use of axillary crutches and prolonged elevation of the limb above the head.

A nerve conduction study shows a decrease of the conduction velocity upon stimulation at the Erb point (supraclavicular fossa) [16, 17].

### Damage at shoulder level

Damage at shoulder level, known as "Saturday night palsy" or "park bench paralysis", consists of compressing the radial nerve by the head of an unconscious person or in a state of alcoholic intoxication. Another common cause of nerve injury on this level is fracture or adjustment of the humerus. The clinical picture is characterised by the image of a "wrist drop". Sometimes swelling of unknown cause on the dorsal hand side – Gubler's syndrome – may occur [18]. The triceps muscle of the arm is saved. Electrophysiological examinations show a velocity decrease or block of conduction at the level of a spiral groove and a preservation of the triceps muscle [15].

### Damage to the posterior interosseous nerve

The posterior interosseous nerve is the distal branch of the radial nerve. Its compression is reached by ganglions, lipomas, radial nerve canal structures, and radial head fracture (Monteggia fracture). Symptoms are purely motor based; no sensory disturbances occur. Finger straightening is compromised. When straightening the hand at the wrist, it deviates in the radial direction due to the preservation of the radial wrist extensor muscle with the elbow rectifier paresis [15].

A nerve conduction study reveals a decrease in conduction velocity at the level of the forearm with the correct response from sensory fibres.

Neuropathy from the posterior interosseous nerve should be differentiated from tennis elbow syndrome.

### Supinator muscle syndrome

Supinator muscle syndrome is manifested by the paresis of the muscles supplied by the radial nerve distally from the supinator muscle, which is manifested by the dropping of the hand and fingers without accompanying sensory disturbances [9, 13].

Dropping of fingers II and III with the correct straightening of the rest of the fingers, called the longhorn sign, is caused by compression of the radial nerve through the distal part of the supinator muscle [7, 9].

### Wartenberg syndrome (cheiralgia parestetica)

Damage to the sensory superficial radial nerve branch is caused by compression in the distal part of the forearm, most often by an overly tight watchstrap (watchstrap nerve compression) or handcuffs (handcuff neuropathy) [18]. It is manifested by impaired sensation and paraesthesia of the dorsal side of the hand and pain of the distal part of the forearm [9, 15]. The syndrome should be differentiated from de Quervain syndrome, i.e. inflammation of two tendons that control the movement of the thumb and their tendon sheath.

### Compression of the dorsal nerve of the thumb

This is caused by the pressure of the nerve during work with scissors. It manifests as a weakening of sensation on the radial side of the thumb [15].

## The sciatic nerve

The sciatic nerve originates from root fibres L4-S3 and is the final branch of the sacral plexus. It supplies the rear thigh muscle group through motor functions, and the whole leg drum and foot through motor and sensory functions.

### Piriformis syndrome

This is a rare neuropathy caused by a conflict between the sciatic nerve and the piriformis muscle. It is estimated that it occurs in 6% of patients complaining of backaches. It occurs six times more often in women than in men. The most common mechanism of its origin is the nerve compression passing through the piriformis muscle or in the infrapiriform foramen, through which it leaves the lesser pelvis. The first reports on the possibility of such a conflict were described by Yeoman in 1928.

The most common cause of the syndrome is muscle abrasion and enlargement of its mass due to falling on the buttocks. In terms of the causes and location of the disease process, the syndrome is divided into the following:

– primary – caused by pathology in the area of the muscle, nerve, and accompanying vessels in the form of anatomical anomalies, calcifications, post-traumatic haematoma, endometriosis, hip fractures, femur, and fibrosis after intramuscular injection;
– secondary – caused by changes outside the piriformis muscle, such as degeneration or inflammation of the lumbo-sacral spine, sacroiliac joints, deep lumbar lordosis, and complications of surgical treatment of intervertebral disc herniation.

The syndrome is manifested by acute pain in the buttock, especially in a sitting position, which increases during walking. Pain radiates to the sacrum and along the posterior surface of the lower limb. The severity of pain during defecation is also characteristic. The physical examination shows muscle soreness, decreased tension and strength of the piriformis muscle, relative shortening of the limb due to flexion in the hip joint, and, in the case of prolonged symptoms, atrophy of the buttock muscles. Laseque's symptom is not present, and deep reflexes are preserved.

Specific provocation tests, such as the following, aid in diagnosis:

– Freiberg test – passive internal rotation straight into the knee joint and the lower limb, bent at the hip, causes pain in the affected buttock,
– Pace's test – the abduction of the thighs in a sitting position while a doctor provides resistance causes pain on the affected muscle,
– Beatty's test – pain in the buttocks of the patient lying on the "healthy side" during the abduction of the limb bent at the knee and hip joint, initially lying freely on the ground.

In additional studies, computed tomography (CT) and MRI show focal lesions in the muscle associated with haematoma, calcification, abscess, scarring, and anatomical changes of nerve and vascular bundles [9].

## Femoral nerve

The femoral nerve originates from roots L2-L3-L4 and exits the lumbar plexus as its largest branch. Sensorially, it innervates the antero-medial part of the thigh, knee, crus, medial ankle, and the medial border of the foot. Kinetically, it innervates all of the thigh muscles.

### Compression neuropathy of the femoral nerve

Injury is the most common cause of femoral nerve damage. Other causes include hip joint prosthesis [19], iliac artery aneurysm, and ilium and iliopsoas tumour [15]. A rare cause of neuropathy is nerve stenosis under the inguinal ligament as a result of intense physical exercise, e.g. gymnastics or karate [20]. Sometimes, femoral nerve neuropathy may be the first symptom of diabetic polyradiculoneuropathy [18].

Typical symptoms are weakness and atrophy of the quadriceps muscle, weakness of the knee reflex, and impaired skin sensation on the anterior medial part of the thigh.

Weakening of the iliopsoas muscle, which is innervated by the branch extending above the inguinal ligament, indicates fibre damage at the level of the plexus or roots L2, L3, L4 [21].

## The lateral cutaneous nerve of the thigh

The lateral cutaneous nerve of the thigh is formed from the root fibres L2-L3 and exits from the lumbar plexus.

### Meralgia paresthetica

Meralgia paresthetica is compression neuropathy of the lateral cutaneous nerve. The compression occurs at the nerve passage under the inguinal ligament as a result of predisposing factors such as abdominal obesity, enlarged lymph nodes, tumours, pregnancy, postoperative scars, spinal curvature, forcing abdominal muscles exercise, wearing tight clothing, or using belts in hernia treatment. The syndrome manifests as pain and paraesthesia on the anterolateral surface of the thigh. Electroneurographic examination has little usefulness in confirming the compression [13].

## The saphenous nerve

The saphenous nerve is the largest branch of the femoral nerve. It supplies the antero-medial part of the knee and crus, and the medial part of the foot.

### Neuropathy of the saphenous nerve

In most cases, nerve damage occurs during invasive procedures on the crus. Compression syndromes rarely occur at the nerve exit from the adductor canal (Hunter's canal) and between the tendon of the sartorius muscle and the edge of the medial condyle of the femur. The syndrome manifests itself with pain in the anteroposterior part of the knee, crus, and medial part of the foot. Clinical tests confirming neuropathy include palpation pain at the nerve exit from the canal (10 cm above the knee) and Tinel's symptom (pain when tapping the nerve) [15].

## Peroneal nerve

It forms in the popliteal fossa from the division of the sciatic nerve into the common peroneal nerve and tibial nerve. It contains the root fibres L4-S2. At the level of the fibula bone, it is divided into superficial peroneal nerve (containing mainly sensory fibres) and deep peroneal nerve (containing mainly motor fibres). It sensitively innervates the anterolateral part of the crus and the dorsum of the foot and toes. Kinetically, it innervates the muscles of the front and side of the crus, as well as the dorsum muscles of the foot and toes.

### Peroneal tunnel syndrome

This is the most common neuropathy of the peroneal nerve. The compression occurs at the level of the nerve wrapping around the head and neck of the fibula bone in the space bounded by the insertion of the peroneus longus [15]. The most common causes of compression include prolonged squatting, prolonged holding of a leg on a foot, tight bandages, improper leg positioning during surgery, sleeping in an uncomfortable position, and fibula fracture [15].

The syndrome manifests with pain in the area of the head of the fibula arrow radiating along the crus, weakening of the dorsal flexion of the toes and the foot, and increasing weakening of the foot. Pain may intensify during compression (Hoffman's symptom) or when tapping the head of the fibula (Tinnel's symptom). The patient's walk is referred to as a "waddling gait". In the neurophysiological examination, the decrease or conduction block is characteristic in the section between the popliteal side and the neck of the fibula. If a fibula injury is suspected, an imaging test should be performed.

### Front tarsal canal syndrome

The frontal tarsal canal is formed by the tarsal bones, the transverse tarsal joint, and the lower extensor retinaculum. Compression occurs in the case of tendon inflammation, dislocation or sprains in the ankle, or walking in tightly laced shoes. The main symptom is pain located on the back of the foot radiating to the space between the big toe and toe II. During the test, one finds a positive Tinel symptom or an increase in pain at the dorsal flexion of the foot. The weakness of the dorsiflexion of the toes is poorly expressed.

A nerve conduction examination shows elongation of the terminal latency and reduction of the response from the muscle of the short extensor of the toes with the correct response from the tibialis anterior muscle.

### External popliteal syndrome

The compression occurs at the nerve passage area through the fascia (10–12 cm above the lateral mal-

leolus) in athletes (especially footballers) as a result of multiple crus microdamages [22]. Neuropathy manifests itself as pain on the anterolateral surface of the crus with radiation to the dorsum of the foot. The syndrome is diagnosed clinically, identifying the characteristic symptoms and performing provocative tests, such as compressing or tapping the nerve exit site at the dorsiflexed foot. The nerve conduction test shows a reduction of conduction velocity, reduced amplitude, or lack of response from the nerve. Sometimes, despite the symptoms, the conductivity study shows no abnormalities.

### Sural nerve entrapment syndrome

The sural nerve originates from the combination of the sagittal and tibial nerve branches (lateral and medial calf nerves). The compression occurs at the nerve passage site through the fascia. The syndrome manifests itself in pain and paraesthesia in the heel-lateral surface of the heel and lateral surface of the foot margin. Electrophysiological examination shows conduction with decreased response amplitude in the nerve [23].

## The tibial nerve

The tibial nerve is formed from the division of the sciatic nerve in the popliteal fossa. It contains root fibres L4-S1. It innervates the flexor muscles of the crus and toes. Sensorially, it supplies the skin of the sole of the foot and the medial part of the heel. It is divided into medial and lateral plantar nerves.

### Tarsal tunnel syndrome

Compression of the tibial nerve extends between the malleolus and the heel, in the tarsal canal, and in the space limited by the tarsal bones and the fascia of the fibrous band of the flexor retinaculum of the foot. The syndrome manifests as a burning and tingling sensation in the plantar part of the foot with pain radiating up along the nerve. In the neurological examination, pain increases with tapping of the nerve or bending of the dorsal foot [15]. Regarding motor functions, there is a weakening of the abduction of the big toe and toe V [13].

An electrophysiological examination shows an extension of the terminal latency to the abductor of the big toe and toe V. A hundred per cent sensitivity in the diagnosis of the syndrome is conduction in the sensory fibres of the distal part of the tibial nerve (between the malleolus and toe V) showing slowness of conduction [24].

An MRI scan helps to visualise the pathological masses that press against the nerve, such as tenosynovitis, fasciitis, fasciated cysts, or schwannomas. These changes are visible in the form of a low homogeneous T1 signal and a high T2 signal. In the ultrasound examination, as well as pathological changes, structural changes of the nerve in the form of its thickening and loss of the bundle echostructure can be observed.

## Summary

Widely understood pain syndromes are the most common reason for reporting to a neurologist. Carefully collected and targeted interviews and neurological examinations are still the basic diagnostic tools. A valuable supplement is the possibility of electrophysiological examination. It is particularly important in the diagnosis of compression syndromes, differentiating the level of nerve damage. An accurate and precise diagnosis gives the possibility of optimal treatment, often operational, meeting the expectations of orthopaedists and neurosurgeons.

## Conflict of interest

The authors declare no conflict of interest.

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