Pressure on nerves

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Anatomy

Peripheral nerves contain both neural and supportive elements. A large multifascicular nerve is composed of a number of different bundles of nerve fibres or fasciculi (Fig. 2.1). These are bound together by the epineurium, a condensation of areolar connective tissue derived from the mesoderm. In humans, the epineurium normally constitutes 30–50% of the total cross-sectional area of the nerve bundle: it contains fibroblasts; collagen (types I and III); variable amounts of fat (possibly to cushion the nerve fibres it surrounds); lymphatic; blood vessels (vasa vasorum); and free nerve endings. In a monofascicular nerve, the epineurium only surrounds the fasciculus and is fused with the perineurium.

The perineurium surrounds and protects one fascicle. It has two different layers; an outer collagen-rich connective one and an inner epithelial layer of contiguous cells. The perineurium has an important role in maintaining the osmotic milieu and fluid pressure within the endoneurium and also acts as a barrier against chemical and bacterial invasion.

The connective tissue of peri- and epineurium possesses blood and lymph vessels – the so-called vasa vasorum. Also free nociceptive nerve endings which come from the related multifascicular nerve trunks are embedded in the perineurium and epineurium.

Enclosed in the perineurium is the fasciculus – a bundle of nerve fibres bound together and protected by the endoneurium. The latter consists of long collagen fibres running with the nerve fibres. The fibrous and cellular components of the endoneurium are bathed in endoneural fluid.

The nerve fibres are axons – the distal offshoots of nerve cells (Fig. 2.2). Most axons are surrounded by a myelin sheath formed from the compressed and concentric Schwann cell membranes. Axons range in diameter from 0.2 µm (small non-myelinated nociceptive axons) to 20 µm (large and myelinated efferent motor axons) and in length from 1 to 100 cm. They contain most of the cell volume.

From central to peripheral, the nervous system can be clinically divided into four zones (Fig. 2.3):

- The spinal cord
- The spinal nerve, which contains fibres belonging to one segment
- In the brachial and sacral area, and distal from the spinal ganglion, the different spinal nerves form a nerve plexus, from which originate the large multifascicular nerve trunks
- Further distally the trunks split into peripheral nerves, with motor, sensory or combined function.
Although the fasciculi, from their exit from the central nervous system to the distal extremity of the nerve, exhibit the general morphology summarized above, the structure and behaviour of the connective supportive elements differ considerably. This may explain the different clinical behaviour of a compressed cord, a compressed nerve root (within or without its dural sleeve), a compressed nerve trunk and a compressed small peripheral nerve.

**Roots**

The course of the spinal nerve within the spinal canal, from the emergence of the rootlets at the anterior and posterior aspect of the spinal cord to the outer border of the foramen, is called the intraspinal root (Fig. 2.4). Being inside the meningeal membranes of the spinal cord, the posterior and anterior roots are devoid of the elaborate epineural and perineural membranes that are characteristic of peripheral nerves. Proximally, the rootlets float freely within the cerebrospinal fluid which is the main source of their metabolic needs. In this intrathecal part of the intraspinal root, the rootlets are held together by the endoneurium which is more loosely arranged than is typically seen in peripheral nerves. Further distally, the nerve root becomes enclosed in the dural sheath – a tubular prolongation of the dura. In this dural investment the nerves do not lie freely but are bound by the arachnoid membrane (Fig. 2.5). This area is known as the extrathecal part of the intraspinal nerve root – that length of the root and the dural sleeve between the main dural sac and the exit from the foramen. The extrathecal portion is short in the cervical region but becomes longer with the increasing obliquity of the intraspinal roots in the thoracolumbar and lumbar regions.

Distal to the posterior root ganglion, at the level of the foramen, the posterior and anterior roots are fused into one single bundle. Here the nerve root becomes extraspinal. The tissue of the epidural pouch becomes more condensed and blends with the epineurium of the extraspinal nerve root. The segment of the spinal root which is liable to compression, whether by a disc protrusion, an osteophytic outgrowth or a narrow lateral recess, is the extrathecal part of the intraspinal root. To understand the symptom of a compression or inflammation of this part of the peripheral nerve system, it is necessary to recognize the importance of the dural investment.
The fasciculi of plexus and trunks do not differ significantly from those of the roots or the peripheral nerves. The connective support tissue, however, has some anatomical particularities. Because the monofascicular spinal nerve changes into a multifascicular structure, there is an increased amount of epineurial tissue, forming a protective packing for the nerve tissue. The perineurium is also reinforced by elastin fibres. The fasciculi have an undulating course, whereas the collagen fibres run more longitudinally. This structure ensures that the nerve fibres are protected from mechanical deformation (compression and elongation) during normal movements of the limbs.  

Although the epi- and perineurium contain nociceptive nerve endings, these seem to be relatively insensitive.

Small peripheral nerve

A small peripheral nerve is the distal termination of a branching nerve trunk. The nerve ending is often monofascicular. The epineurium is then fused with the perineurium. Peripheral nerves can either have only a motor or a sensory function, or both can be combined. They all have nociceptive nerve endings in their supporting connective tissue.

Terminology

Lesions of the peripheral nervous system are characterized by a pathognomonic sensation: paraesthesia (‘pins and needles’). Although all tissues in the human body which contain nociceptive structures can be a source of pain, pins and needles will only arise when some part of the peripheral nervous system is at fault. Hence, the medical world tends to use the term ‘neuritis’ when pain is accompanied by pins and needles. Strictly, however, the suffix ‘-itis’ implies inflammation. Therefore the word neuritis should only be used when the peripheral nerve is affected by infectious or toxic irritation – i.e. there is an intrinsic disorder of the nervous parenchyma. Classically, these lesions are classified into mono- and polyneuritis. They are not discussed in this book, except in the shoulder region, where the clinical appearance of three mononeurites and neuralgic amyotrophy of the shoulder girdle is reviewed (see online chapter Nerve lesions and entrapment neuropathies of the upper limb).

When external pressure is applied to a normal nerve, pins and needles arise, although the nerve tissue is initially not inflamed. If the compression is not severe, the nerve continues to conduct normally. This situation of extrinsic pressure on a normal nerve is not a ‘neuritis’ and requires a totally different therapeutic approach. In this situation, the terms ‘pressure on nerve’ or ‘entrapment neuropathy’ are preferred.

Pain originating from the peripheral nerve system

Nociceptive pain

Peripheral nociceptors in the connective tissue of the peripheral nerves are stimulated, and via Ad and C fibres of the nervi...
nervorum conducted to the spinal cord and thence to the pain projection areas in the cortex. There are indications that most of the pain that stems from direct irritation of the peripheral nervous system is of nociceptive origin. ‘Nerve pain’ thus behaves identically to other peripheral pain, and obeys exactly the rules of referred pain and is not to be distinguished from pain of ligamentous, tendinous or arthrogenic origin (see Ch. 1).

**Neuropathic pain**

This type of pain, also called ‘de-afferentation or neuralgic pain’ is less common than nociceptive pain and results from prolonged damage to peripheral nerve tissue, such as avulsion, dissection or amputation. Research on experimental neumomata has shown that regenerating axons have a spontaneous excitability and an increased sensitivity to mechanical stimuli. An action potential in one axon probably leads to an impulse in a nearby axon. This mechanism of ‘cross-talking fibres’ accounts for the repetitive train of action potentials in a bundle of regenerating axons. A small stimulus thus leads to a self-perpetuating series of action potentials, and excessive and long-standing pain.

Chronic damage or formation of scar tissue seems to provoke pain mechanisms without involvement of peripheral nociception. Also, the formation of a neumoma leads to increased sensitivity and spontaneous pain. Research on experimental neumomata has shown that regenerating axons have a spontaneous excitability and an increased sensitivity to mechanical stimuli. An action potential in one axon probably leads to an impulse in a nearby axon. This mechanism of ‘cross-talking fibres’ accounts for the repetitive train of action potentials in a bundle of regenerating axons. A small stimulus thus leads to a self-perpetuating series of action potentials, and excessive and long-standing pain.

Another mechanism that may account for neuropathic pain is the loss of inhibitory effects of the large diameter mechano-receptor afferents in a traumatized nerve. This leads to a relative increase of the activity from the small nociceptive afferents, and thus an opening of the gate at the dorsal horn.

**Superficial dysaesthetic pain**

This type of pain is also rare, and is typical of diffuse polyneuritis, for example in diabetes, vitamin B1 deficiency or chemical irritation. Damage to small C fibres leads to sprouting of small offshoots in the regenerating axons. This leads to increased excitability, which results in unpleasant painful sensations during normal stroking of the skin (alldynia). The patient also complains of a burning feeling and ‘electrical sensations’ when the skin is gently touched (dysaesthesia), and there is also some analgesia (see Box 2.1 for an overview of neurogenic pain).

**Behaviour of nervous tissue during pressure**

Entrapment of peripheral nerve tissue is defined as mechanical compression of the nerve, which includes the reduction of radial dimensions in the neural cells, the neural support elements or any combination of these. Depending on the degree and the duration of compression, the effects can be subtle or can lead to displacement, deformity and morphological changes in the compressed tissue (neural tissue or neural support tissue). The clinical effects of nerve compression are pain, paraesthesia and loss of function (see Box 2.2 for an overview of pressure on nerves).

**Pain**

The pain mechanism in entrapment phenomena is usually nociceptive: free nerve endings in the connective tissue of the nerve or in the dural investment of the nerve root are depolarized by application of mechanical forces or after exposure to irritating chemical substances, released from inflamed tissues. The pain stems from irritation of the support tissue enclosing the nerve fibres and only exceptionally does it result from pathological processes in the nerve tissue itself (neuropathic and dysaesthetic pain). This has the following clinical consequences.

The pain will depend largely on the density of the nociceptive receptors in the support elements. It follows that the intensity of the pain depends not only on the intensity of compression but also on the localization along the course of the peripheral nerve. Pressure on a nerve root, for instance, will be more painful than an equal degree of force applied on a nerve plexus.

Because an external force acts first on the outer supporting structures of the nerve, pain will usually be the first symptom and it sometimes appears before involvement of the parenchyma is present. A chronic but moderate pressure that is insufficient to impair conduction solely influences the outer structures and results in pain only. It is thus possible to have a completely normal examination of the peripheral nervous system, even though the patient does have nerve compression.
Paraesthesia

Pins and needles are pathognomonic of involvement of the peripheral nervous system in that the sensation cannot be produced in any way other than compression or inflammation of nerve tissue. Paraesthesiae are always felt in the cutaneous area supplied by the nerve tissue involved and distal to the site of the lesion. It is therefore extremely important to ascertain the precise site of the symptom, in that this helps to determine the site of compression.

Provocation of pins and needles by movements (distant movements or local pressure) or by stroking over the affected skin demonstrates an external origin for the symptoms. In primary afflictions of the peripheral nerve (neuritis), the pins and needles come and go spontaneously and movements do not influence them.

Loss of function

The epineurium and perineurium initially buffer the fasciculi from constrictive effects, but with a greater amount of compression, structural changes of the elements within the endoneurium follow. Recent research has demonstrated that the intraradicular oedema caused by alteration of the blood–nerve barrier is the most important factor in the nerve root dysfunction of chronic compression.

Sometimes only the Schwann cells are affected, without damage to the axons. Destruction of the myelin sheath then results in loss of conduction. This type of lesion (lesion of Schwann cells without lesion of the axon fibre) is termed ‘neuropraxis’. If the compression has been only temporary, recovery of the Schwann cells will not take more than 2 weeks. This is the type of lesion responsible for the ‘Saturday night palsy’ seen after prolonged pressure on the radial nerve, or the ‘gardener’s palsy’ seen after prolonged traction on the peroneal nerve.

If considerable compression is maintained for a longer period, atrophy of the nerve tissue occurs and is followed by Wallerian degeneration of the distal part of the axon. Oedema, cellular proliferation and ingrowth of connective tissue also follows. If the compression is maintained for long periods, fibrotic degeneration appears at the site of the lesion, which makes recovery most unlikely.

Clinical syndromes

Cyriax (see his pp. 37–39) distinguished four different syndromes in entrapment phenomena, corresponding to the site of compression along the peripheral nerve: at the small peripheral sensory nerve, at the nerve trunk/plexus, at the nerve root and at the spinal cord (see Fig. 2.3).

Depending on the localization of compression, the peripheral nervous system behaves differently. These differences are a major help to the clinician trying to ascertain the precise site of the lesion.

Small peripheral nerves

Pressure on a small peripheral sensory nerve results in pain, paraesthesia and numbness. Pain is usually moderate and the main symptom is numbness. Together with some paraesthesia, these symptoms occupy the appropriate area of supply, which is usually well defined with clear-cut borders. The patient can tell precisely where the cutaneous analgesia is felt and where sensation is still normal. The centre of the region is often completely anaesthetic. A typical example is ‘meralgia paraestheticca’, which results from compression of the lateral cutaneous femoral nerve of the thigh.

Nerve trunk/plexus

Minor and intermittent pressure on a nerve trunk or a plexus causes paraesthesia and numbness. Sudden and serious tissue damage may provoke neuropathic pain. Constant pressure on a nerve trunk leading to parenchymatous damage does not usually provoke pain nor paraesthesia but only loss of motor and sensory function.

If the pressure is intermittent, a neurological deficit does not appear, even after many years. Paraesthesiae do not appear during the time of compression but only when the pressure on the nerve trunk has been released. It is common knowledge that pressure on the sciatic nerve while sitting causes only vague analgesia in the affected area or no symptoms at all. The shower of pins and needles then only appears when the subject relieves the pressure by standing up. The interval between the cessation of the pressure and the onset of the tingling depends on the duration of the compression: the longer the pressure is applied, the longer is the interval between the relief of pressure and onset of symptoms.

There is also a relation between the duration of compression and the duration of paraesthesia. Thus, after 15 minutes of pressure, the pins and needles appear 20–60 seconds after the release and last only 1 or 2 minutes. After release from 15 hours’ compression, paraesthesiae will probably appear only after an interval of some hours, then persist for 1 to 2 hours before recovering spontaneously. Cyriax calls this strange and hitherto unexplained phenomenon the ‘release phenomenon’ (see his p. 37). Lundburg and Rydevik have demonstrated that fluctuations in membrane permeability of the structures within the endoneurium are more noticeable when compression on the nerve trunk is released and oedema appears, than during the compression of the nerve and its supplying blood vessels. This might explain the release phenomenon.

Another characteristic of paraesthesia induced by compression at the level of the plexus is that active movements of the limb or the digits, or stroking over the analgesic area of skin usually brings on or increases the pins and needles. It is a common experience that the paraesthesia in the feet that comes on after relieving pressure on the sciatic nerve increases when the subject walks around or stamps the feet on the ground.

Paraesthesia and numbness are usually felt in the distal part of the cutaneous area supplied by the compressed plexus or nerve, no matter at what point in its course the compression...
Deficit

The absence of the protective packing by epineurial tissue renders the nerve roots more susceptible to direct compression than nerve trunks. Compression disturbs nerve conduction by interfering with the blood supply of the nerve fibres. Loss of function of the nerve fibres results in sensory and motor deficit. Paraesthesiae usually disappear with the onset of cutaneous analgesia.

Progressive compression of a nerve root within its dural sleeve causes a typical sequence of symptoms: pain, paraesthesia and numbness will follow each other, rather than coincide. This is typically the case in a progressively increasing pressure exerted by an evolving disc lesion: slight compression on the epidural sheath of the nerve root causes pain only (Fig. 2.6a). As the pressure increases, paraesthesia and muscle fasciculations – symptoms of parenchymatous hyperexcitability – appear (Fig. 2.6b). In the final stage, pressure has induced such ischaemic damage to the nerve root that function is completely lost, including the conduction of pain (Fig. 2.6c). The patient then complains of weakness and numbness, but pain and paraesthesia have disappeared.

Spinal cord

Pressure on the anterior aspect of the spinal cord results, inter alia, in bilateral paraesthesia. Pain is absent if the compression is slowly progressive, but dural pain may accompany the paraesthesia if the compression is sudden. The main cause

Compression of the nerve root

Pressure on the extrathecal intraspinal nerve root results in a typical set of symptoms (pain and paraesthesia) and signs (motor and sensory deficit) strictly related to the segment involved. Contrary to the pins and needles brought on by the release of pressure on a nerve trunk, the paraesthesias only appear during the period of compression, and cease immediately thereafter.

The sequence of appearance of pain, paraesthesia and deficit and their mutual interrelation have important clinical significance and are often of considerable help in the diagnosis of nerve root compression.

Pain

The nerve root has a dural sheath, which is innervated by the sinuvertebral nerve. The latter is derived from the corresponding nerve root. Therefore pain originating from the dural sheath is strictly segmental and follows the rules of segmental reference of pain. Compression applied to the dural sleeve of the nerve root thus results in pain occupying all or any part of the dermatome. Pain felt in a particular dermatome in combination with other symptoms of nerve compression, immediately draws attention to an impingement on the nerve root.

Paraesthesia

Pins and needles stem from pressure on the parenchyma itself. In root pressure, they accompany the segmental pain, or appear at a later date. Pins and needles indicate that the parenchyma itself is irritated, whereas radicular pain is a symptom of compression of the dural sheath.

In nerve root compressions, paraesthesiae are felt in the distal extremities of the dermatomes, which are areas often not supplied by a particular nerve trunk or nerve. As in compression of a nerve trunk, stroking the skin may provoke or increase the pins and needles, but moving the digits does not influence them.

Fig 2.6 • Progressive compression of a nerve root.
of spinal cord compression is spinal stenosis at the cervical or thoracic level. When the cord is compressed over the thoracic region, the paraesthesiae are felt only in the limbs. In compression at the cervical level, pins and needles will be present in all limbs or in the lower limbs only. The paraesthesiae are usually bilateral and extend beyond the borders of the areas of the cutaneous innervation of any spinal nerve, nerve trunk or peripheral nerve. For instance, the patient may complain of pins and needles in both hands and forearms at both aspects or in both legs from the knees to all the toes.

The symptoms are provoked neither by movements of the limbs nor by stroking the skin. Neck flexion is the only way to bring on the pins and needles (L'Hermitte’s sign). Together with the extrasegmental and bilateral distribution of the painless paraesthesiae, positive neck flexion provides the clue to the diagnosis of incipient spinal cord compression.

Access the complete reference list online at www.orthopaedicmedicineonline.com

### Box 2.2

**Pressure on nerves: Summary**

<table>
<thead>
<tr>
<th>Structure</th>
<th>Symptoms Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Peripheral nerve</td>
<td>Numbness &gt; paraesthesia &gt; pain</td>
</tr>
<tr>
<td></td>
<td>Clearly delineated area</td>
</tr>
<tr>
<td>Nerve trunk</td>
<td>Paraesthesia &gt; numbness &gt; pain</td>
</tr>
<tr>
<td></td>
<td>Vaguely delineated area</td>
</tr>
<tr>
<td></td>
<td>Release phenomenon:</td>
</tr>
<tr>
<td></td>
<td>— The pins and needles appear after the compression has ceased</td>
</tr>
<tr>
<td></td>
<td>— There is a direct relation between the interval of onset and duration of compression</td>
</tr>
<tr>
<td>Nerve root</td>
<td>Pain &gt; paraesthesia &gt; numbness</td>
</tr>
<tr>
<td></td>
<td>Segmental distribution of symptoms</td>
</tr>
<tr>
<td></td>
<td>Chronological sequence of symptoms</td>
</tr>
<tr>
<td></td>
<td>Compression phenomenon:</td>
</tr>
<tr>
<td></td>
<td>— The pins and needles appear during the compression</td>
</tr>
<tr>
<td>Spinal cord</td>
<td>Completely painless (sometimes dural pain)</td>
</tr>
<tr>
<td></td>
<td>Extrasegmentally and bilaterally distributed paraesthesia</td>
</tr>
<tr>
<td></td>
<td>Positive neck flexion</td>
</tr>
</tbody>
</table>
References


