REVIEW / Neuroradiology

Current and future imaging of the peripheral nervous system

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Abstract Peripheral nervous system (PNS) imaging is usually carried out by ultrasound and MRI. Thanks to its wide availability and excellent spatial resolution, ultrasound is a mature investigation with clearly established indications, particularly in entrapment syndromes and tumors. MRI is generally a second-line examination, which provides decisive additional information thanks to its excellent contrast resolution and its multiplanar abilities. This review describes the current methods for imaging the PNS, concentrating on acquisition techniques, normal results and basic pathological semiology. Ongoing and future developments are described in order to underline the forthcoming changes in this very dynamic field of musculoskeletal radiology.

The nervous system is divided into the central nervous system (CNS) which consists of the brain, spinal cord and retina, and the peripheral nervous system (PNS) which is made up of all of the other nervous structures [1].

The PNS is therefore made up of nerves, ganglions, various receptors and synaptic and motor nerve endings, which carry CNS commands to the effector organs and return internal and external sensory informations. Each structure is essential in transmitting the nerve impulse, but because of its length, specific histology function and exposure to diseases, the peripheral nerve remains the main focus of PNS imaging.

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A few pathophysiologic concepts

Histology

A peripheral nerve is a rope-shaped organ which has a specific concentric organization designed to guide, protect and nourish neuronal fibers within it. The main structure is the axon [1] which is a long extension of the neuronal cell body specialized in transporting the nerve impulse. It is constantly sheathed in a support cell, the Schwann cell, whose specialization differentiates two types of fibers:

- unmyelinated fibers, surrounded by a single small diameter layer of Schwann cytoplasm, which conducts slowly and mostly has sensory (pain) and vegetative functions;
- myelinated fibers, around which the Schwann cell wraps in multiple layers to form the myelin sheath. These myelinated fibers vary in diameter, are generally involved in rapid, responsive conduction and constitute all the somatic motor fibers and a large proportion of the sensory fibers.

These myelinated and unmyelinated peripheral nerve fibers lie in a loose connective tissue called the endoneurium, made up of fibroblasts, a collagen matrix and blood capillaries. They group in bundles, each bundle being demarcated by a solid concentric cell layer, the perineurium. Several fascicles (from a few units to a hundred) are grouped together to form the actual nerve trunk, which is separated from the surrounding environment by a dense fibrous connective tissue, the epineurium, within which many blood vessels, the *vasa nervorum*, circulate. This concentric histology [2] is shown schematically in Fig. 1.

Wallerian degeneration

After section or crush, axons forming the peripheral nerves are able to regenerate slowly [1].

In the first hours after an injury, the axon and the Schwann cells forming its myelin sheath begin to disintegrate. This starts immediately distal to the site of injury and follows a proximal to distal path. This phenomenon, due to the post-traumatic interruption of axonal flow, and therefore the shortage of the essential substances for the axon to survive, is known as Wallerian degeneration [3] and lasts between one and two weeks, depending on the length of the nerve involved.

Macrophages then clean up the cell debris and release growth factors which stimulate axonal development. Several buds therefore develop from the axon stump. One of these buds becomes predominant and keeps advancing in a proximal to distal direction, guided and stimulated by the remaining endoneurial environment. Nerve regrowth occurs at a rate of 1 to 2 mm per day and its effectiveness increases in positive correlation with the closeness of the section margins. Should the nerve margins be too far apart, the axonal buds will no longer have a guide and may grow anarchically, forming a mass, the amputation neuroma.

This clearly defined degeneration followed by axonal regrowth sequence only occurs completely and effectively in clear-cut nerve section or focal nerve compression. This can be seen in the experimental setting (by tying off the nerve in the nerve crush model [4]) or clinically in a wound from a sharp object such as a blade or a broken piece of glass.

Classification of post-traumatic peripheral nerve injuries

Traumatic nerve injuries, however, involve a far greater spectrum of damage, ranging from simple compression to total destruction with loss of tissue. The more disorganized the ultrastructure of the nerve has become, the less likely nerve regrowth is to occur.

Several nerve damage classifications have been described to reflect this relationship. The most widely used is the Sunderland classification (1978) inspired by the work of Seddon (1943) [5]. It consists of five stages [6]:

- Sunderland I (neuroparaxia according to Seddon): a local conduction block secondary to focal demyelination, therefore involving destruction of the myelin sheath but with no damage to the axon or rupture of the endoneurial support tissue. Recovery occurs fully within 12 weeks;
- Sunderland II (axonotomesis according to Seddon): this involves a loss of axonal continuity, with complete distal Wallerian degeneration. The supporting connective tissue is preserved and the endoneurial tubes guide the proximal to distal axonal regrowth ("Schwann staves"). Complete recovery usually occurs but requires several months, depending on the distance which has to be reinnervated;
- Sunderland III: the damage locally destroys the axon, the myelin sheath and the endoneurial tubes. The perineurium

![Figure 1. Schematic anatomy of the peripheral nerve.](image-url)
and epineurium remain intact. Regrowth is variable but generally incomplete because of incorrect orientation of the fibers and trapping in the endoneural scar;

- Sunderland IV: only the epineurium remains intact with endoneural scarring and loss of endoneural and perineural continuity. The scarring prevents regrowth of the fibers and results in the formation of a neuroma;
- Sunderland V (neurotmesis according to Seddon): the nerve is totally dislocated, affecting the endoneurium, epineurium and perineurium. As in stage IV, only surgery can offer a hope of nerve regrowth.

**Muscle denervation**

The section of a motor nerve has direct consequences on the innervated skeletal muscle, the first one being paralysis. Wallerian degeneration ultimately reaches the neuromuscular junction, which is thereby destroyed. The denervated muscle fiber will at first develop vasogenic edema [7]. These abnormalities are seen electromyographically from 48 hours [8], and a typical histological appearance is seen after 3 weeks [9]. These changes usually reverse when the endplate regenerates.

If the denervation persists, metabolic changes within the muscle will progress to muscle fiber atrophy and an increase in their fat content. Chronic denervation leads to diffuse fatty infiltration of the affected muscles after several months [10].

**Current PNS imaging methods**

PNS imaging relies mostly on ultrasound and MRI: computed tomography has a more limited input. Conventional radiography has only indirect applications, researching bony causes in entrapment syndromes (fracture, supernumerary bone, bone callus, etc.) [11,12].

**Computed tomography**

CT is useful in diagnosing intradural, intraforaminal and extraforaminal spinal nerve compression [13]. It is also useful in the etiological diagnosis of some entrapment syndromes, particularly if a bone-based, tumoural or vascular cause is suspected [14]: its good spatial resolution, excellent bone contrast and the ability to perform CT angiography are advantages in this situation.

CT can also be used to diagnose peripheral nerve tumors [15]: it has good spatial resolution and can easily detect a mass [16], although it may be difficult to confirm its neural origin. It is also a useful investigation in type 1 neurofibromatosis [17], where it provides a precise staging of the neurofibromas, investigating for gastro-intestinal, pulmonary or urinary compressions and related bone deformities.

Apart from these indications, and although the large nerve trunks are often clearly visible on CT [18] (Fig. 2), its contrast resolution is inadequate for detailed examination and identification of pathological changes in the neuronal microstructure.

The advantages and disadvantages of CT are summarized in Table 1.

**Ultrasound**

The arrival of high frequency probes and refinement in electronics have resulted in a clear improvement in the spatial resolution of ultrasound. Peripheral nerves, which are generally very superficial, have benefited from these new developments, as already demonstrated in 1987 [19]. Many subsequent studies [20–25] have confirmed the benefits of the method and its advantages (Table 2).

Using musculoskeletal presets and high frequency linear probes (10 to 17 MHz), the majority of peripheral nerve trunks, i.e. the median, radial, ulnar, sciatic, common fibular and tibial nerves, can be examined [22].

The nerve is identified and its perineuronal environment studied on axial sections in which the peripheral nerve appears as an oval structure, consisting of a network of hypoechoogenic fascicles separated by hyperechogetic septa. In longitudinal sections, it appears as a tube containing hypoechoogenic bands separated by hyperechogetic lines (Fig. 3). The hypoechogenic bands correspond to the nerve
Typical bundles of Doppler appear scarcely as degenerative changes, and their characteristic features distinguish them from other lesions.

In the peripheral nervous system (PNS), ultrasound is a reliable and operator-dependent imaging modality. It has excellent spatial resolution, no contraindications, and allows dynamic investigations. Interventional ultrasound with diagnostic and therapeutic procedures is readily available and low-cost.

<table>
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<tr>
<th>Advantages</th>
<th>Disadvantages</th>
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<tr>
<td>Excellent spatial resolution</td>
<td>Operator dependent</td>
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<tr>
<td>No contraindications</td>
<td>Relatively long learning curve</td>
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<td>Dynamic investigations possible</td>
<td>Poor contrast resolution</td>
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<td>Investigation of a large part of the nerve path</td>
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<td>Intervventional ultrasound with diagnostic and therapeutic procedures</td>
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<td>Readily available</td>
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Figure 3. Twenty-eight-year-old man. Ulnar nerve ultrasound at the wrist in axial and longitudinal sections. 12-MHz linear probe. Typical fascicular appearance.

bundles and the hyperechogenic lines are the epineural supporting connective tissue [26]. However, less than 30% of the true number of nerve fascicles are actually seen on ultrasound and this proportion declines with the frequency of the probe used [27].

This characteristic fascicular structure allows peripheral nerves to be distinguished from tendons, which have a fibrillar echostructure. Nerve mobility during movements is of low amplitude, unlike the neighboring muscles and tendons. Doppler studies are usually negative on a normal nerve [24].

In pathology, ultrasonography performs well in the diagnosis of neoplastic diseases. These are generally benign tumors (neurofibromas and schwannomas) and appear as an oval or fusiform tissue lesion with regular, clearly demarcated outlines. They are hypoechogenic and relatively homogeneous, with posterior acoustic enhancement [15,28]. The proximity of the mass to the nerve is an essential diagnostic feature [29]. Some signs can suggest a histological subtype such as a lesion centered relatively to the nerve course which is more common in schwannomas. The ultrasound distinction between schwannomas and neurofibromas remains however arbitrary [30] and in any event of limited use in practice.

Ultrasound has two roles in entrapment syndromes:

- to look for nerve morphology abnormalities as a result of compression, which may produce two major signs [22]:
  - a segmental change in diameter, usually focal thinning at the point of compression and downstream enlargement immediately after the compression; an increase in nerve diameter may also be seen proximal to the compressed area [24]. Some authors propose the measurement of nerve cross-sectional area or the flattening ratio [25], with cut-offs to distinguish patients from healthy controls,
  - a loss of the usual fascicular echostructure, the nerve becoming hypoechogenic in which nerve fascicles are difficult to visualize;
- to identify lesions in the perineuronal environment responsible for compression: soft tissue tumors, osteoarticular abnormalities such as synovial cysts, tenosynovitis or supernumerary tendons and vascular abnormalities.

In polyneuropathy, some studies [31] have shown a significant increase in nerve diameter (mostly the median nerve), particularly in hereditary Charcot-Marie-Tooth neuropathy [32,33].

Magnetic resonance imaging

At the beginning of the 1990s, Howe & Filler team proposed an examination entirely dedicated to the visualization of the PNS with suitable sequences optimizing the contrast between nerves and their environment, called neurography [34,35]. The use of phased-array coils and a high field imager significantly improve the signal to noise ratio and the spatial resolution, allowing the neuronal ultrastructure to be examined [36,37].

The protocols use [37–40]:

- T1-weighted spin-echo sequences, providing good morphological evaluation and excellent spatial resolution;
- T2-weighted spin-echo sequences, which have good contrast resolution;
- these “simple” T2 sequences have been replaced by “neurography” sequences [37,41] which are highly weighted T2 sequences using long echo times (100 ms) combined with fat signal suppression. Vascular saturation bands are usually added on both sides of the region being investigated in order to reduce the vascular inflow signal. The field of view is limited to what is strictly necessary, with a matrix as large as possible and fine sections (2 to 4 mm) in order to maximize the spatial resolution. Either spin-echo sequences (SE or TSE) with fat saturation, or Short Tau Inversion Recovery (STIR) sequences which provide more reliable homogeneous fat suppression are used;
- T1 weighted sequences after gadolinium injection: these are occasionally required in tumour or inflammatory diseases.

The section planes should be adjusted depending on the region being examined: an axial plane is essential [40] and occasionally sufficient but is often combined with a perpendicular plane.

The advantages and disadvantages of MRI are summarized in Table 3.

MRI findings on the peripheral nerve are consistent with its anatomy [36].

On T1 weighted sequences, the nerve appears isointense with muscles, with a peripheral hyperintense halo, representing the epineural fat [38,42]. The isointense nerve bundles can be distinguished within the hyperintense connective epi- and perineural tissue [39] in the major nerve trunks with high-resolution acquisitions.

On T2 weighted sequences, the nerve is moderately hyperintense. The fascicles within the large nerve trunks exhibit a more pronounced hyperintensity due to the endoneural fluid [39] (Fig. 4).

Non-pathological nerve structures demonstrate no significant enhancement after contrast injection (because of the presence of the blood-nerve barrier).

As with ultrasound, pathological features concern the morphology and signal of the nerve structure:
- flattening of the nerve, particularly in areas liable to be compressed, is abnormal. This sign is particularly valuable if it is segmental and associated with an increase in diameter of the upstream nerve segment;
- increase in diameter, particularly a segmental increase preceded and followed by a nerve of normal diameter, is also considered pathological;
- hyperintensity on "neurography" sequences: this is reported to be a result of a decrease in axonoplasmic flow due to neuronal degeneration and peri- and endoneural edema [37,41];
- a loss of the fascicular structure on the T2 and particularly T2FS weighted sequences also indicates disease and is due to the same causes;
- moderate contrast enhancement on T1 weighted sequences after gadolinium injection: this reflects a breach of the blood-nerve barrier.

![Figure 4](image-url)

**Figure 4.** Twenty-eight-year-old man. Sagittal T1 and T2FS MRI sections of the median nerve in the carpal tunnel on a 3T scanner. Note the peripheral hyperintensity on the T1 weighted sequence, representing the epineural fat, and the fascicular appearance with hyperintensity of the tubes on the T2FS weighted sequence.

| Table 3 Advantages and disadvantages of MRI to examine the peripheral nervous system (PNS). |
|-------------------------------|--------------------------|
| **Advantages**                | **Disadvantages**        |
| Excellent contrast resolution | Contraindications to MRI |
| Very good spatial resolution  | Availability and         |
| Three-dimensional planes      | appointments delays      |
| No limitation in examining    | High cost                |
| deep structures               | Static investigation,    |
| Good investigation of muscle mass (signs of denervation) | occasionally performed |
| Research and development      | in un-physiological      |
| potential                     | positions                |
| Investigation is less         | Limited field of view    |
| operator dependent            | Long acquisition time    |

MRI also allows a good appreciation of the overall path of the nerve: a non-physiological deviation or change in direction is evidence for compression or a pathological adhesion. It is therefore an excellent investigation to assess entrapment syndromes [21,43], particularly for recurrences or failures after surgery [12,44,45].

MRI is also extremely useful for examining the perineural environment: its excellent contrast resolution and its ability to carry out multiplanar acquisitions facilitates the diagnosis of intrinsic [15,28] or extrinsic compressive lesions.

MRI can also be useful for the positive diagnosis of inflammatory polyneuropathies by demonstrating contrast uptake and increased diameter of the cauda equina nerve roots [46,47]. An old study showed that in some cases the diagnosis was confirmed by MRI findings despite a negative electroneuromyogram [48].

Wallerian degeneration is visible experimentally on MRI as early as 24 hours after the initial injury [49]. It produces a clear hyperintense T2 signal [50], with loss of usual fascicular structure and an overall increase in nerve diameter. These abnormalities are due to an increase in water content as a result of intra- and perineural edema, secondary to rupture of the blood-neuronal barrier and influx of phagocyte cells.

It is therefore theoretically possible to distinguish neuropaxia (no abnormalities on imaging), axonotmesis (Wallerian degeneration without loss of nerve continuity) and neurotmesis (Wallerian degeneration with complete loss of continuity) [51] by MRI. In practice, the distinction is not that clear and is primarily based on clinical and electromyographic findings.

Finally, muscle denervation can be seen on MRI and is a valuable indirect evidence of damage to the nerve innervating the muscle area in question [52]. As a general rule, the nervous lesion is located more proximally than the muscle abnormality [39]. Muscle denervation is seen on MRI as two chronologically consecutive appearances [10,37,38]:

• initially the muscle remains normal in size and morphology, with clear global hyperintensity on T2 weighted sequences (Fig. 5). The edema is visible early, experimentally from 48 hours [53], and persists throughout the acute and subacute phase of denervation, usually for less than 10 weeks and very rarely for more than 6 months. The intensity of the increased T2 signal is reported to be proportional to the severity of the nerve damage [54]. Significant muscle enhancement is seen at this phase after gadolinium injection;

• secondarily, loss of muscle volume associated with a hyperintense T1 signal (Fig. 6) is seen: this is the fatty amytrophic stage characterizing the chronic phase of denervation, which occurs in the months following the initial lesion and persists if reinnervation does not occur [10].

Developments and future prospects

Volume neurography

3D volume acquisitions have been achievable on MRI for many years and several authors have reported application of this technique to the peripheral nerve system [55,56], with acquisition of a T1 weighted and T2 STIR weighted three-dimensional sequences.

They provide high quality isotropic neurography, with the possibility for curvilinear multiplanar reconstructions and post-treatment such as MIP or image fusion, which are particularly useful in examining complex anatomical structures like the brachial plexus. This sequence can already be used in everyday practice without changing equipment.

Figure 5. Forty-eight-year-old man, one month after an injury causing complete section of the collateral ulnar and radial nerves in his left thumb. T2FS weighted axial section. Hyperintensity on the T2 sequence in the thenar eminence due to acute muscle denervation.

Figure 6. Eighty-five-year-old woman, who had a whole body MRI to investigate camptocormia. T1 weighted axial slices. Stage IV chronic fatty atrophy in the anterior tibial and left soleus muscles, and stage II-III in the right soleus muscle, due to sciatic sequelae.
Microneurography

This technique [57] optimizes the spatial resolution of neurography images, using very high magnetic fields, extremely powerful gradients and specific coils. Using a 9.4 Teslas scanner and gradients of 400 mT/m, images of anatomical parts can be obtained with a spatial resolution of 30 μm. These acquisitions of stunning histological precision show us what could be done with neurography when the MRI method is pushed to its limits.

This can be compared to very high frequency ultrasound, which uses dedicated probes to increase the spatial resolution, at the expense of the investigation depth. 55 Mhz microprobes are used in ex vivo animal research and provide a theoretical spatial resolution of 30 μm [58].

Dedicated contrast media

Two molecules may be useful in PNS MRI investigation:

- Superparamagnetic MR contrast agents (SPIO - Endorem or Resovist) and Ultrasmall Superparamagnetic contrast agents (USPIO - Sinerem), containing iron oxide particles, which identify the inflammatory response thanks to their affinity for the macrophage system. Accumulation of these molecules produces a hypointense signal on gradient-echo acquisitions through a paramagnetic effect. Bendzus [59] was able to visualize macrophage influx from day 1 to day 8 in Wallerian degeneration and Stoll [60] demonstrated PNS inflammation with contrast uptake beginning before clinical symptoms and resolving at the peak of disease in an animal model of the Guillain-Barré syndrome. The problem with these applications is that the products are not available commercially;

- gadofluorine belongs to the family of micellar contrast media, which is still experimental, and binds to the areas of nervous demyelination, producing a hypointense T1 signal until remyelination is complete. It appears to be both highly sensitive and specific, and can detect focal demyelination lesions. This may be useful in post-treatment follow-up of autoimmune and inherited demyelinating diseases [61,62].

Contrast-enhanced ultrasound

An animal study has shown that contrast-enhanced ultrasound is feasible in peripheral nerve imaging [63], enabling quantitative measures of PNS perfusion. Pathology applications have been suggested, particularly in traumatology to monitor grafted nerve injuries.

Spinal ganglion morphometry

The spinal ganglion is formed by the cell bodies of the primary sensory neurons. Its morphology can therefore change in peripheral nervous diseases and indirectly reflect these.

In an experimental study on mice using a high field (7 Teslas) imager, West showed that 3D MRI measurement of spinal ganglion volume correlated well with histological measurements, which themselves were directly proportional to the number of neuronal cell bodies [64]. A reduction in spinal ganglion volume therefore reflects a loss of neurons and appears to be an objective and early means of measuring sensory neuronal damage following peripheral nerve lesion.

In contrast, an increase in the size of the spinal ganglion has been found in chronic inflammatory demyelinating polyneuropathy. This increase can also be observed in inherited neuropathies or those secondary to lymphoma, and is believed to be due to inflammatory and edematous changes with demyelination and remyelination cycles. Measurement of cervical and lumbar spinal ganglia with coronal STIR acquisitions can then be used to diagnose affected subjects [65]. The hypertrophy also correlates positively with the electrophysiological abnormalities.

MRI spectroscopy

This technique is used to identify and quantify several metabolites thanks to their resonance frequency differences. Its major limitation is its poor contrast resolution (routinely 7 to 10 mm) which is not really compatible with examination of peripheral nerves. In an old publication, Baldassarri [66] astutely circumvented the problem by examining muscle metabolism during nerve regeneration with 31P spectroscopy to quantify high-energy metabolites and estimate intramuscular pH. Muscle tissue alkalization was found during the denervation phase. These abnormalities disappeared following complete regeneration of the motor nerve, allowing it to be monitored indirectly.

A recent study [67] on animal models used 19F MRI spectroscopy and perfluorocarbon labeling. Perfluorocarbon has affinity for the macrophage system and its binding is therefore an indirect marker of neuronal inflammation. This technique does not yet, however, seem applicable to human beings.

Diffusion, diffusion tensor imaging and tractography

The description of peripheral nervous system appearance on diffusion-weighted sequences is relatively recent. Takahara [68,69] introduced the concept of diffusion-weighted MR neurography (DW-MRN), based on diffusion-weighted sequences with baseline signal suppression. These sequences optimally showed the path of nerve trunks, particularly on MIP reconstructions. The technique has been described in several anatomical sites [70], and even successfully in wholebody acquisition [71], which could enable the entire PNS to be examined in a single investigation. Its spatial and contrast resolution, however, are still inadequate.

Most authors who study diffusion actually concentrate on diffusion tensor imaging and tractography, an MRI technique used to produce an in vivo reconstruction of the path of neuronal fibers based on their anisotropism. The first tractography reconstruction of a peripheral nerve dates back to 2004, when Skoril [72] used a CNS tractography protocol on the sciatic nerve and demonstrated that the technique could be used on three healthy individuals. Many authors have since confirmed the validity of the technique and have looked for applications in pathology, particularly to diagnose carpal tunnel syndrome [73,74]. The tractography sequences can be optimized [75] in order to obtain high quality reconstructions for the large nerve trunks (Fig. 7).
The techniques used to examine the peripheral nervous system, either using ultrasound or MRI, are now robust and have the clear advantage of being able to examine the perineural environment, which is not easily assessed by clinical examination alone or by electromyography [76].

Whilst it is currently of limited use in the management of polyneuropathies, imaging is a useful diagnostic complement in focal mononeuropathies [42], particularly in case of atypical nerve entrapment syndromes which do not appear to be idiopathic [11,43].

Alongside these established uses, the many developments, particularly in MRI, are opening new perspectives such as monitoring axonal regeneration and improving our understanding of peripheral nervous pathophysiology.

**Disclosure of interest**

The authors declare that they have no conflicts of interest concerning this article.

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