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MAGNETIC RESONANCE

Magnetic resonance neurography: current perspectives and literature review

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Abstract

Magnetic resonance neurography (also called MRN or MR neurography) refers to MR imaging dedicated to the peripheral nerves. It is a technique that enhances selective multiplanar visualisation of the peripheral nerve and pathology by encompassing a combination of two-dimensional, three-dimensional and diffusion imaging pulse sequences. Referring physicians who seek imaging techniques that can depict and diagnose peripheral nerve pathologies superior to conventional MR imaging are driving the demand for MRN. This article reviews the pathophysiology of peripheral nerves in common practice scenarios, technical considerations of MRN, current indications of MRN, normal and abnormal neuromuscular appearances, and imaging pitfalls. Finally, the emerging utility of diffusion-weighted and diffusion tensor imaging is discussed and future directions are highlighted.

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Key points

- *Lesion relationship to neural architecture is more conspicuous on MRN than MRI.*
- *3D multiplanar imaging technique is essential for pre-surgical planning.*
- *Nerve injuries can be classified on MRN using Sunderland's classification.*
- *DTI provides quantitative information and insight into intraneural integrity and pathophysiology.*

Keywords MRN · MR neurography · DTI · Peripheral nerve · MRI

Introduction

Magnetic resonance neurography (also called MRN or MR neurography) refers to MR imaging dedicated to the peripheral nerves. It is a technique that enhances selective multiplanar visualisation of the peripheral nerve and pathology by encompassing a combination of two-dimensional (2D), three-dimensional (3D) and diffusion imaging pulse sequences [1]. While large perineural lesions and denervation muscle changes are quite apparent on conventional MRI, the relationship of lesions to the nerve architecture is more conspicuous on MRN [2, 3] (Fig. 1). Direct depiction of neuromuscular imaging alterations is highly valuable because it is helpful for both referring physicians and patients to objectively visualise the consequences of neuromuscular pathology (Fig. 2) [4, 5]. This article reviews the pathophysiology of common peripheral nerve abnormalities, describes technical considerations of MRN, discusses current indications for MRN, and highlights normal and abnormal neuromuscular appearances and imaging pitfalls on MRN examinations.

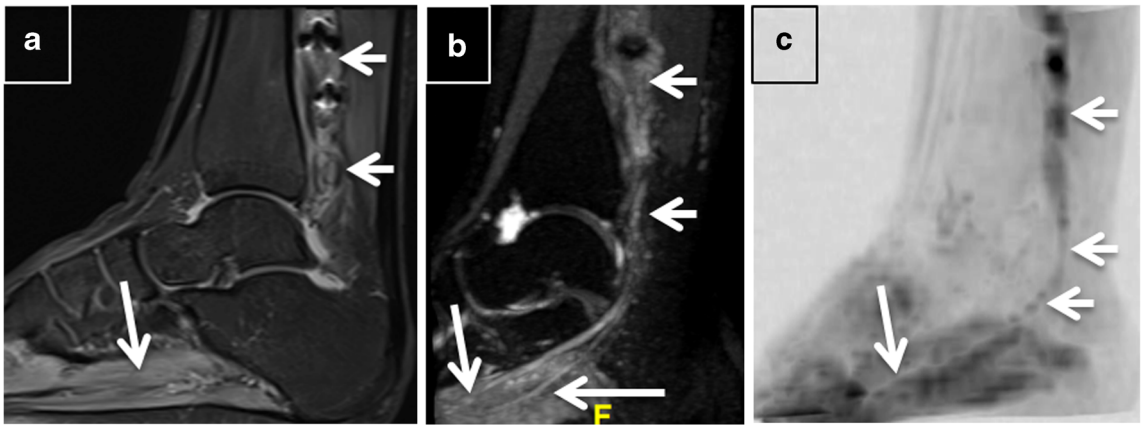

Fig. 1 A 23-year-old woman with prior right leg and ankle injury and tendon surgery presenting with ipsilateral foot weakness and pain. (**A**) Sagittal proton density-weighted (PDW) image shows heterogeneous soft tissue thickening along the course of the tibial nerve (*small arrows*) with adjacent susceptibility artefacts. Denervation changes are seen as hyperintense signals in the plantar muscles (*large arrow*). (**B**) Three-dimensional magnetic resonance neurography (MRN) image shows high-grade tibial nerve injury and large neuroma in continuity (Sunderland class IV) demonstrated by a heterogeneously thickened nerve segment (*small arrows*). Notice the hyperintense but otherwise normal-calibre medial and lateral plantar nerves (*large arrows*). (**C**) Inverted scale trace maximum intensity projection (MIP) image from diffusion tensor imaging (DTI) confirms the neuroma in continuity with selective depiction of abnormally hypointense nerve (*small arrows*) and muscle denervation changes (*large arrows*)

Finally, the emerging utility of diffusion-weighted imaging (DWI) is discussed and future directions are highlighted.

Peripheral neuromuscular anatomy and variations

The peripheral nerves have a uniform but complex architecture (Fig. 3). The axon, a basic building block, is covered by connective tissue layers known as the endoneurium, perineurium and epineurium [6]. In young adults, although there is tight compact appearance of the nerve with uniformly thick fascicles, more intraneural fat accumulates and prominent intraneural vessels appear with increasing age [7]. Asymptomatic streaky fatty infiltration of the muscles can be seen with aging, e.g. in gluteus maximus, pronator quadratus, semimembranosus, plantar foot and supinator muscles [8, 9].

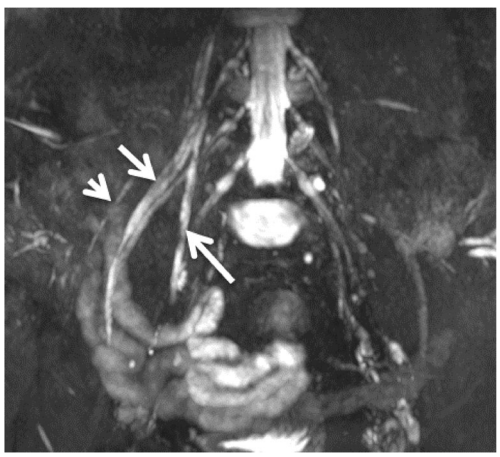

Fig. 2 A 63-year-old man with foot drop and clinically suspected right-sided sciatic neuropathy or radiculopathy. Electrophysiology revealed right tibial and sural neuropathy. Magnetic resonance neurography (MRN) of the right thigh, knee and lumbosacral (LS) plexus was performed to better localise and characterise the lesion. The nerves appeared normal in the thigh and knee (not shown). The LS plexus MRN image reveals an abnormally thickened and hyperintense right lateral femoral cutaneous nerve (small arrow), femoral nerve (medium arrow) and obturator nerve (large arrow). The left-sided plexus shows a normal appearance. The final diagnosis was multifocal inflammatory motor-sensory neuropathy

Pathophysiology of neuropathy as it pertains to imaging

The most common causes of peripheral neuropathy include trauma and entrapment. *Traumatic nerve injury* can occur

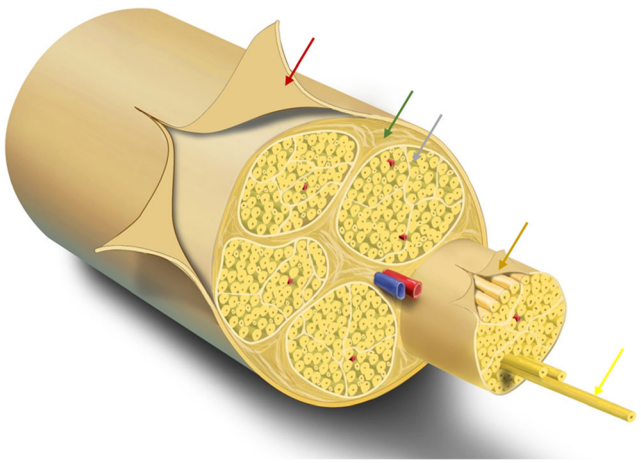

Fig. 3 Illustration showing the 3-dimensional architectural anatomy of the peripheral nerve. The axon (yellow arrow) and various covering layers are shown, i.e. endoneurium (*blue arrow*), perineurium (*mustard arrow*), internal epineurium (*green arrow*) and outer epineurium (*red arrow*)

because of stretch, crush or penetration, traditionally graded based on Seddon's classification. The degree of injury is further stratified based on Sunderland's I–V classification system, which aids in management planning (Table 1) [10–12]. Classes IV and V lead to significant nerve dysfunction and usually require surgical treatment. *Nerve compression* due to entrapment results in blockage of endoneurial fluid flow and vascular congestion. Over time, the unrelieved compression leads to myelin and axonal damage, nerve infarction and fibrosis. If the insult is relieved, the nerve heals by axonal regeneration and immature myelin formation [13, 14]. Body and tissue response may also include intraneural fat proliferation, which is particularly observed in diabetes mellitus, where the overall nerve calibre increases despite fascicular atrophy [15]. During healing, regeneration proceeds slowly from the spinal anterior horn cells or site of injury to the muscle end plate up to a rate of 1 mm/day for smaller nerves and 5 mm/day for the larger nerves [6, 16, 17]. Typical examples of repeated degeneration and regeneration of peripheral nerves are seen in the histology models of Charcot-Marie-Tooth disease (CMT) [18].

Electrophysiology versus imaging

Electrophysiology (EP) has long been considered the reference standard for neuropathy diagnosis and nerve regeneration. However, its limitations must be recognized. EP can be falsely negative in neurapraxia and, additionally, despite complete nerve transection in higher class injuries, it may take up to 7 days to detect a significant decrease or absence of sensory and/or motor nerve responses [19, 20]. Further limitations of EP procedures include difficulty in lesion localisation and physical discomfort to the patients.

Ultrasound (US) is excellent for evaluating superficial nerves and in interrogating large areas of extremities rapidly [21]. Increasingly, US is being employed for the diagnosis and follow-up of various neuropathies using the diagnostic criteria of nerve and/or fascicular enlargement [22]. MRN provides excellent spatial resolution (0.3–0.5 mm in-plane on 2D imaging and 0.9–1.5 mm isotropic on 3D imaging) [23]. It allows easy identification and characterisation of obliquely coursing peripheral nerves [24]. Due to its superior contrast resolution compared with US, signal alterations in nerves and muscles are much more conspicuous and the diagnosis of neuropathy on MRN can be accomplished using multiple different criteria even if the nerve is not enlarged (Table 2). Multiple MRN studies have shown moderate to excellent interobserver performance in determination of T2 nerve signal, nerve-to-vessel signal intensity ratio and nerve calibre with the intraclass coefficient ranging from 0.84 to 0.94 for larger nerves, such as sciatic and ulnar, and 0.56 to 0.72 for smaller ones

Table 1 Nerve injury classification (grading) based on Seddon's and Sunderland's classifications, electrophysiology and magnetic resonance neurography (MRN) findings

Sunderland class of nerve injury	Seddon class of nerve iInjury	Myelin	Axon	Endoneurium	Perineurium	Epineurium	Electrophysiology			MRN findings
							SNAP	CMAP	EMG	
I	*Neurapraxia*	Abnormal	Normal	Normal	Normal	Normal	Normal	Normal or CB	Normal but IP Decreased	Hyperintense nerve
II	*Axonotmesis*	Abnormal	Abnormal	Normal	Normal	Normal	Ampl Decreased	Ampl Decreased	SA and IP Decreased	Hyperintense and thickened nerve with/without prominent fascicles
III		Abnormal	Abnormal	Abnormal	Normal	Normal				
IV		Abnormal	Abnormal	Abnormal	Abnormal	Normal				Heterogeneous nerve signal with lateral or fusiform neuroma in continuity
V	*Neurotmesis*	Abnormal	Abnormal	Abnormal	Abnormal	Abnormal	Absent	Absent	No MUPS	Complete nerve gap

Please note muscle denervation change is typically absent in class I injury and full recovery is expected in class I/II injuries. In class III–V injuries, prognosis is guarded.

SNAP sensory nerve action potential, *Ampl* amplitude, *CMAP* compound motor action potential, *EMG* electromyography, *CB* conduction block, *IP* interference pattern, *MUPS* motor unit potentials, *SA* spontaneous activity

Table 2 Magnetic resonance neurography (MRN) findings of pathological nerves

Nerve characteristic	Abnormal MRN findings	Pulse sequence (s) for best observation
Calibre	Abrupt changes in calibre along the course or persistent diffuse enlargement without distal tapering	3-D T2W
Contour	Irregular contour	3-D T2W
Fascicle	Non-uniform, enlarged, atrophied or disrupted	2-D T2W
Signal	Abrupt signal alterations along the course, too bright or too dark signals, asymmetrical from the contralateral side or adjacent similar size nerves	3-D T2W and 2-D T2W
Continuity	Discontinuity or neuroma in continuity	3-D T2W and 2-D T2W
Tumour	Intra- or extraneural tumour	2-D T1W and 2-D T2W
Perineural fat planes	Fibrosis or space-occupying lesion	2-D T1W and 2-D T2W
Enhancement	Distal to dorsal nerve root ganglion	3-D T1W

[25–27]. Apart from lesion localisation and intraneural tissue characterisation (Table 3), MRN allows grading of the acuity or chronicity of muscle denervation, classifies nerve injury, detects polyneuropathies and aids in planning of subsequent image-guided perineural or intramuscular injections. *In vivo* neural regeneration studies on humans using MRI are being published, and decreasing muscle and nerve hyperintensity has been shown to correlate with nerve regeneration and functional improvement [28, 29]. Current indications of MRN are listed in Supplementary Table 4.

Technical considerations of magnetic resonance neurography (MRN)

Pulse sequences

The basic goal for all imaging sequences employed for MRN is to produce high-resolution scans with superior contrast and/or uniform fat saturation while simultaneously suppressing pulsation and breathing artefacts. Since nerves have longer T2 signals (e.g. 80– 100 ms) [30], and are often surrounded by bright fat, the preferred imaging sequence includes fast or

Table 3 Magnetic resonance neurography (MRN) imaging features of neuropathy and common lesions

Lesion site	Type	T1W Signal	fsT2W Signal	Aetiologies
Intraneural	Oedema	Hypointense	Hyperintense	Compression, inflammation or injury related neuropathy
Intraneural	Fat	Hyperintense	Hypointense/mixed signal	Chronic atrophic neuropathy, diabetes mellitus, lipoma, fibrolipoma
Intraneural	Fluid	Hypointense	Hyperintense	Ganglion cyst, leprosy abscess
Intraneural	Haemorrhage	Hyperintense	Hyperintense/mixed signal	Endometriosis, injury, intraneural malignancy
Intraneural	Fibrosis	Hypointense	Hypointense	Injury, chronic compression neuropathy
Intraneural	Benign mass	Isointense	Hyperintense	Perineurioma (motor loss, young age, honeycomb appearance of fascicles, diffuse enhancement), benign PNST (target sign – outer myxoid and inner collagenous tissue; fascicular sign – thickened nerve fascicles; tail sign – linear nerve extensions on either end of tumour; and bag of worms sign – plexiform clustered lesion), granular cell tumour, paccinian corpuscle granuloma (superficial lobulated lesion under the skin)
Intra- or perineural	Locally aggressive	Isointense	Hypointense/mixed signal	Desmoid, fibromatosis (T2 dark infiltrative lesions along the nerve or fascia)
Intra- or perineural	Malignant	Isointense/mixed signal	Hyperintense/ mixed signal	Malignant PNST, synovial sarcoma, fibrosarcoma, lymphoma (heterogeneous signal and enhancement with necrotic areas, large, peritumoral and /or perineural oedema, local infiltration, low ADC (<1.1x10-3 mm^2/s), distant metastases.
Intraneural	Mass mimicker	Isointense	Mixed or hyperintense signal	Amyloidosis (h/o multiple myeloma), idiopathic hypertrophic mono-neuropathy, post-traumatic neuroma in continuity

PNST peripheral nerve sheath tumour, *ADC* apparent diffusion coefficient

turbo spin echo (FSE/TSE) with spectral saturation using the adiabatic inversion pulse and Dixon technique [31]. The echo time should be kept above 60 ms, specifically to enhance the endoneurial fluid signal while minimizing signals from surrounding tissues with shorter T2 signals, such as muscle [32]. Furthermore, when used with diffusion tensor imaging (DTI) acquisitions, tighter echo spacing (e.g. <0.7 ms) and higher bandwidth reduce ghosting artefacts [33]. Recent advancements including modulated refocusing flip angles [34] combined with extended echo train lengths and parallel imaging have enabled 3D T2-weighted acquisitions using FSE/TSE for volumetric visualisation of the nerves in clinically feasible scan times [23, 35]. In addition, signal suppression of adjacent vessels can be accomplished by addition of motion-sensitizing driven equilibrium or diffusion moment [36–38] enabling visualisation of small nerves in the extremities and skull base. Contrast imaging is used in cases suspected of tumours or infections and for characterisation of atypical polyneuropathies. The suggested MRN protocols are highlighted in Supplementary Tables 5 and 6.

Scanners and coils

MRN can be performed on both 1.5 T and 3 T scanners, but when available, 3 T is preferred due to the increased SNR and faster 3D [39]. In addition, fascicular detail is much more conspicuous on 3 T, especially on the axial images, which are important to characterise and localise the neuropathies [40]. An exception to this is the presence of metallic implants, where 1.5 T imaging is preferred to minimize artefacts. For superior spatial resolution, a joint specific receiver coil should be used in a periarticular location, e.g. during carpal tunnel or cubital tunnel imaging [7]. The dedicated joint coil can be combined with a surface (flex) coil to evaluate the nerve over a longer distance. For plexus imaging, the anterior torso coil is combined with the spine coil to achieve a homogeneous signal.

Imaging pitfalls

Significant pulsation artefacts from vessels can obscure the nerve or cause it to be too bright due to overlap. Shorter echo spacing and re-orientation of the phase encoding gradient can help mitigate this artefact. Suboptimal fat suppression or magic angle artefact can lead to spurious differences in the signal intensity of the peripheral nerves. The presence of metal artefacts can obscure the local anatomy. Suboptimal fat suppression and low bandwidth can result in significant ghosting artefacts on diffusion imaging [41]. Finally, patient movement and off-centre imaging can potentially degrade imaging.

MRN imaging findings

Normal neuromuscular appearances

Normal peripheral nerves show uniform contour, a similar calibre to adjacent arteries and accompanying nerves, side-to-side symmetry and minimal visible branching [42]. The fascicular detail is conspicuous in axial images in extremity peripheral nerves measuring more than 3 mm. The outer epineurium and perineurium exhibit thin uniform hypointense linings. On contrast imaging, only the dorsal nerve root ganglion exhibits any appreciable enhancement [6].

Abnormal neuromuscular appearances

Pathological nerves show one or a combination of findings (Table 2), including nerve and/or fascicular signal alterations or discontinuity, altered calibre, irregular contour, intra- or perineural tumour or scarring, and abnormal enhancement (Fig. 2) [2, 5]. MRN can indirectly demonstrate intraneural pathophysiology as illustrated in Table 3 [42–44]. DTI further supplements anatomical MRN diagnostic accuracy. Breckwoldt et al. showed that in upper extremity neuropathy, T2W images and DTI perform similarly in isolation while a combination of MRN plus DTI results in increased diagnostic accuracy (area under the curve = 0.97) [44]. Muscle denervation oedema and/or atrophy are important findings in the diagnosis of neuropathy [2, 27]. As a rule, denervation changes occur distal to the site of insult and muscle signal alterations are diffuse without haemorrhage or fascial oedema. The injured nerves exhibit hyperintense signal on T2W images within 24 h of the insult [28, 45]. Sunderland Class I–III injuries demonstrate a uniform hyperintense signal and class I injury is short-lived with absent muscle signal alterations. Class IV injury shows a heterogeneous nerve signal with a focal fascicular abnormality and neuroma in continuity (Figs. 1 and 4). Class V injury shows a discontinuous nerve with end-bulb neuroma. Studies are needed to show if DTI can further supplement the differentiation of class I–IV injuries. MRN aids in characterisation of the lesions causing nerve entrapment, such as haematoma, ganglion, bony callous, etc. and demonstrates their relationship to the nerve in terms of its axial location, longitudinal extent or fascicular involvement [2, 6, 7]. Nerve and/or fascicular hyperintensity is maximum adjacent to the site of entrapment, which aids in lesion localisation. For example, sciatic nerve hyperintensity is most pronounced at the sciatic notch in the setting of piriformis syndrome. The increasing nerve calibre distinguishes severe from mild cases of neuropathy and DTI can also aid in the severity assessment [26, 46]. Commonly, the nerve enlarges proximal to the site of entrapment. If one also identifies distal nerve enlargement or ‘triple B sign’ (progressive nerve signal change from ‘bright to black to bright’ across the entrapment site), there is likely

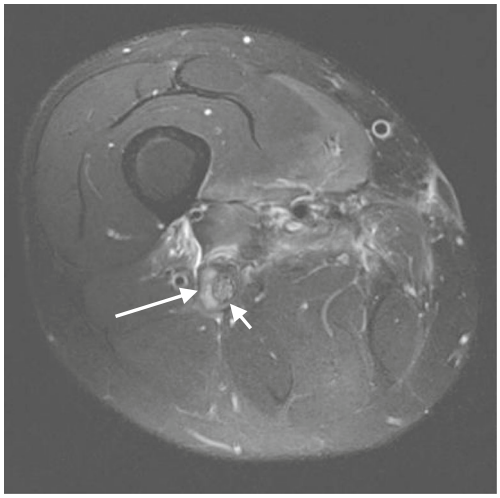

Fig. 4 Prior penetrating injury with Sunderland class IV and III injuries. Axial T2 SPAIR image shows a neuroma in continuity of the common peroneal nerve with loss of fascicular detail (*large arrow*) in keeping with a class IV injury. Notice the enlarged tibial nerve with hyperintense, albeit preserved fascicular detail (*small arrow*) in keeping with a class III injury. The patient underwent surgical reconstruction of the common peroneal nerve

severe nerve impingement or constriction that would typically require surgery (Fig. 5) [47, 48]. Peripheral nerves can be affected by a variety of tumours and tumour-like conditions (Fig. 6) [49, 50]. MRN and DTI can characterise the relationship of various lesions with nerve fascicles (Table 3) [51, 52].

Interpretation pitfalls

For accurate reporting, it is essential to be familiar with the expected regions of T2 hyperintensity related to magic-angle

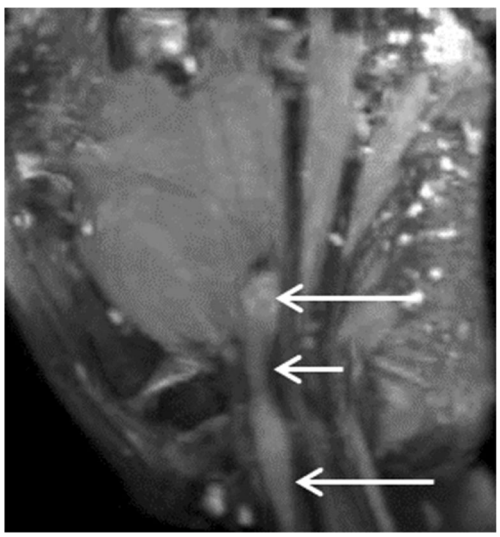

Fig. 5 Carpal tunnel syndrome and triple B sign. Coronal magnetic resonance neurography (MRN) image shows proximal and distal enlargement and abnormal hyperintensity (*large arrows*) of the median nerve across the transverse carpal ligament with flattening and decreased signal at the impingement site (*small arrow*)

artefacts. These include the greater sciatic notch for the sciatic nerve, genu of the femoral nerve and at turning points (e.g. lateral femoral cutaneous nerve at anterior superior iliac spine, C8 nerve at thoracic outlet, deep branch of ulnar nerve dorsal to the carpal tunnel and medial plantar nerve across the tarsal tunnel) [7]. It is important to evaluate the spatial extension of T2 signal alterations to be confident of neuropathy [53]. Mild hyperintensity of the nerves on T2W images can be seen in asymptomatic subjects [54]. Anatomical nerve branching variations are common. For example, sciatic variations are seen in about 6–25% of the population but only a minority develop piriformis syndrome.

Diffuse polyneuropathies

Diffuse or systemic neuropathies can be hereditary (CMT disease) or acquired due to a multitude of causes, and idiopathic (Fig. 2). While their diagnosis is primarily based on clinical findings and laboratory testing, electrophysiology and MRN supplement the diagnosis [53]. CMT manifests with symmetrical early-age neuropathy problems, high plantar arches, positive family history and symmetrically reduced nerve conduction velocities. MRI may incidentally exhibit substantial symmetrical thickening of nerves (CMT type I-demyelinating variety) or milder thickening (type II-axonal type). MRN and DTI increase conspicuity of the nerve lesions and can detect superimposed entrapment(s) [55–57].

Most acquired neuropathies, however, produce asymmetrical findings. One such common condition, chronic inflammatory demyelinating neuropathy (CIDP), usually shows a downhill course and typical albuminocytological dissociation. MRN shows significant thickening and hyperintensity of nerves in the lower limbs > upper limbs and no substantial enhancement [58]. Multifocal motor neuropathy presents with pure motor neuron weakness. Typically, ganglioside M1 antibodies are identified on serology and MRN shows mild to moderate thickening and hyperintensity of the upper limb or brachial plexus nerves [59]. In radiation neuropathy, MRN shows diffuse nerve thickening and T2 hyperintensity localised to the radiation field with regional oedematous muscle changes in acute-to-subacute stages. Nerve kinking from fibrosis is seen in chronic stages [7]. Diabetes mellitus is a common cause of peripheral polyneuropathy, and diabetic amyotrophy is a common differential finding of radiculopathy in adult patients. On MRN, a diffuse increased signal in bilateral sciatic and tibial nerves can be seen in the lower limbs, with nerve thickening and DTI parameter alterations [60–62]. In diabetic amyotrophy, MRN shows bilateral prominent and hyperintense lumbosacral (L4-S2) nerve roots and paraspinal branches and femoral and/or sciatic nerves. While local infections can affect the nerves, *Mycobacterium leprae* is a known neurotropic pathogen. MRN can show moderate to marked

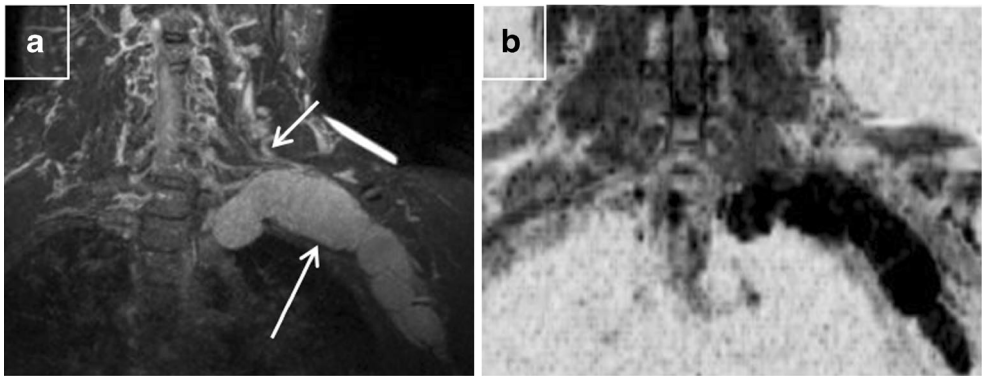

Fig. 6 Benign peripheral nerve sheath tumour. A 45-year-old man with left shoulder swelling. (**A**) Coronal magnetic resonance neurography (MRN) image shows a large peripheral nerve sheath tumour (*large arrow*) displacing the nerves anteriorly and superiorly (*small arrow*). (**B**) Corresponding ADC map shows a high ADC value of 2.5–2.7 × 10–3 mm^2/s in keeping with a benign peripheral nerve sheath tumour. The tumour was stable for more than 2 years

thickening of the nerves with perineural oedema and/or intraneural abscess. Finally, polyneuropathy can occur due to amyloidosis, especially in the setting of underlying paraproteinemia. It manifests as diffuse thickening of the nerves and fascicles with nodularity and mixed or predominantly hyperintense signal foci [63].

Role of diffusion imaging

DWI and DTI application in the peripheral nerve system is challenging because of the need for high spatial resolution over the long nerve course, low water proton content of the nerves and relatively limited diffusion within them. On qualitative evaluation, the signal in the neuropathic nerve is often more conspicuous on DWI trace images compared with T2W images. The quantitative evaluation generates fractional anisotropy (FA) and apparent diffusion coefficient (ADC) [64]. Research has shown an age- and location-dependency of FA values. Neuropathy leads to increased ADC and decreased FA. Quantitative data from the initial animal studies has shown FA increases towards normalcy with nerve regeneration [65]. DTI generates tractography (Fig. 7), and it is important to understand that the fibres seen on tractography are not anatomically identical to the individual fascicles. However, if fascicles are compressed, oedematous or interrupted, correspondingly, the fibres on tractography are thinned, distorted or interrupted [66].

Impact of MRN and future directions

Dedicated nerve imaging has been shown to impact both diagnostic thinking and therapeutic management of patients

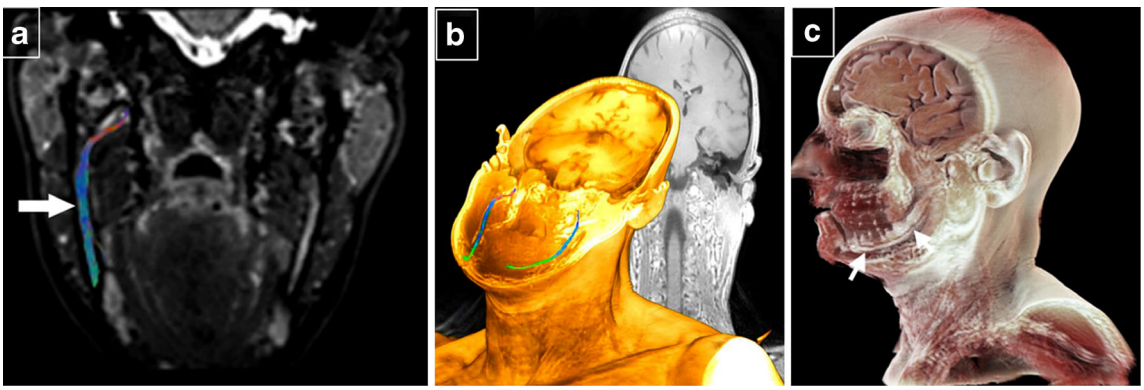

Fig. 7 Representative images illustrating the current status of diffusion tensor imaging (DTI) and maximum possible image quality employing diffusion weighted and diffusion tensor imaging in the corresponding anatomical regions. (**A**) Oblique coronal 3D diffusion-weighted PSIF image of the facial region with an overlaid tractograpy image in a healthy volunteer shows the right inferior alveolar nerve within its intra-osseous course through the mandible (image generated on Syngovia, Siemens, Erlangen, Germany). (**B**) The tractography fibres can also be overlaid onto a 3-dimensional (3D) model that was created by acquiring a 3D MRI data set using ultra-short echo time sequence. It can help in planning complex orthodontic surgery with visualisation of the important neurogenic structures. (**C**) Virtual anatomical images from a fusion of diffusion-weighted and high-resolution morphological magnetic resonance neurography (MRN) images allow photorealistic representations of the true *in vivo* anatomy in an individual

suspected of having peripheral neuropathy [67–69]. In a recent study, MRN was shown to reduce unnecessary surgeries by 17% and MRN increased the confidence of the surgeon in the diagnosis and management of neuropathy [67]. MRN accuracy is well reported [70]; however, technical developments continue and outcome studies using homogeneous and larger patient populations are still needed. Cost analysis would be useful to determine the overall burden of this emerging imaging modality.

Conclusion

MRN is a useful modality for direct demonstration of neuromuscular pathology and characterisation and is playing an increasingly important role in the management of various peripheral nerve disorders.

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Compliance with ethical standards

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Conflict of interest The authors of this manuscript declare relationships with the following companies

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