Imaging of the facial nerve: A review of anatomy and pathology

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Authors: P. V. Patil, A. Johnson, D. Weinberg, R. Siripurapu, G. Potter; MANCHESTER/UK
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Learning objectives

The facial nerve (VII) is a mixed cranial nerve with very complex anatomy which is affected by a wide variety of primary pathologic processes and may also be secondarily involved in several disorders of the brain, temporal bone, and parotid gland. Imaging of patients with facial nerve dysfunction requires a thorough understanding of the anatomy of the facial nerve and a knowledge of common pathologies and their imaging appearances. A segmental anatomical approach and appropriate use of computed tomography (CT) and magnetic resonance imaging (MRI) imaging guided by clinical presentation is essential for the assessment of patients with facial nerve dysfunction.

The purpose of our educational exhibit is to:

1. To review the normal radiological anatomy of the facial nerve.
2. To illustrate various pathologies of the facial nerve that may present with facial nerve dysfunction.
3. To suggest a systematic imaging approach to the diagnosis of common pathologic processes affecting the facial nerve.
Background

The facial nerve is a complex nerve with motor, sensory, and parasympathetic fibers. The facial nerve provides motor innervation to the muscles of second branchial arch; it has a visceral motor function ( lacrimal, submandibular, sublingual glands and secretion of the nose), and conveys special visceral sensory taste fibers from the anterior two thirds of the tongue and general sensory fibres from the auricle and the wall of the external auditory meatus (Fig.1).

Imaging plays an important role in the evaluation of patients with VII weakness. To image VII adequately, it is important to understand its normal anatomy and relevant technical considerations regarding CT and MR to achieve high-resolution images of the nerve. A segmental anatomical approach and appropriate use of CT, MRI - or a combination of both - guided by clinical presentation, should enable the radiologist to assess facial nerve lesions.

Normal Radiologic Anatomy of the Facial Nerve

1. Supranuclear Control of VII: Upper Motor Neuron (Motor Cortex)

The supranuclear contribution originates from pyramidal neurons located in the lower third of the precentral gyrus of the frontal motor cortex. The cortical projection fibers for the upper face demonstrate incomplete decussation and project to the ipsilateral and the contralateral facial nuclei. However, the supranuclear fibers for the lower facial muscles completely decussate to the contralateral facial nucleus (Fig.2). Hence, unilateral supranuclear lesions often somewhat spare the functions of the upper face [1].

2. Intraaxial (Brainstem) Segment of VII:

- **Motor Nucleus**: The motor nucleus of the facial nerve lies in the lower third of the pons, behind the superior olivary nucleus, and medial to the spinal tract of the trigeminal nerve. From this origin, the fibers first pass backward and reach the posterior end of the nucleus of the abducens nerve. After looping around the abducens nucleus (internal genu), the fibers travel between the medial border of the trigeminal nucleus and the lateral edge of the facial motor nucleus (Fig.3.4). The motor fibers finally exit the caudolateral pons in the cerebellopontine angle (CPA) medial to cranial nerve VIII [1].
• **Nucleus Tractus Solitarius**: This nucleus receives taste sensation from the anterior two thirds of the tongue, and hard and soft palate, travelling within the chorda tympani (Fig.3) [1].

• **Sensory Nucleus**: The somatic sensations from the posterior aspect of the external auditory canal, pinna, and mastoid region travel through the nervus intermedius to the dorsal part of the primary sensory nucleus of trigeminal nerve within the pons (Fig.3) [1].

• **Parasympathetic Nucleus**: The parasympathetic component of the facial nerve originates from the superior salivatory nucleus in the dorsal pons. The facial nerve provides presynaptic parasympathetic fibers to the sphenopalatine and pterygopalatine ganglia for innervation of the lacrimal glands, and to the submandibular ganglion for innervation of the sublingual and submandibular salivary glands (Fig.1,3) [1].

3. **Peripheral (Intracranial-Intratemporal-Extracranial) Course of VII:**

• **Cisternal Segment in the Cerebellopontine Angle**: The motor root and the nervus intermedius emerge from the brain stem ventrolaterally and travel anterolaterally into the internal auditory canal (IAC), with the nervus intermedius between the motor root of the facial nerve and the vestibulocochlear nerve (hence its name) (Fig.5) [1,2].

• **Intratemporal Segment:**

  1. Intracanalicular segment: At the IAC, the nervus intermedius joins the motor root to form a common trunk, the nervi facialis, which lies within the anterosuperior segment of the meatus (Fig.6) [1,2].

  2. The first part of the facial nerve canal (labyrinthine segment): At the fundus of the IAC, VII enters the labyrinthine segment after passing through a small hole, the facial meatus. It passes between the ampulla of the superior semicircular canal and the cochlea to travel forward and downward (Fig.7,8A) [1,2].

  3. Genu of VII: At the geniculate ganglion, the nerve makes an abrupt sharp turn posteriorly, thus creating the external genu. This marks the beginning of the tympanic segment (Fig.8B). Exiting anteriorly from the geniculate ganglion is the first branch of VII, the greater superficial petrosal nerve (GSPN) [1,3].

  4. The second part of the facial nerve canal (tympanic segment): The tympanic segment continues along the medial wall of the tympanic cavity, medial to the incus. It courses along the upper edge of the oval window, and inferior to the lateral semicircular canal. At the origin of the stapedius tendon from the pyramidal process, the nerve turns inferiorly to become the mastoid segment (Fig.8C) [1,2].
5. The third part of the facial nerve canal (mastoid segment): VII travels along the posterior aspect of the external auditory canal to reach the stylomastoid foramen (Fig.8D). The mastoid segment has two branches: i) nerve to stapedius and ii) chorda tympani [1,2].

6. Stylomastoid foramen: VII leaves the petrous bone, seen as an enlargement of the mastoid segment of the bony canal (Fig.8D) [1,3].

• Extracranial Segment: VII immediately enters the parotid gland. Shortly afterwards, the nerve divides into its two terminal branches: the upper temporofacial nerve and a lower cervicofacial nerve at the posterior border of the ramus of the mandible [1,2].

Imaging Techniques for the Evaluation of the Facial Nerve

Imaging should be tailored to both the suspected pathology and clinical localisation of the lesion along the course of VII. Typically, if a facial palsy is localised to the cisternal or intracanalicular segments of the facial nerve or the pontine nuclei MRI is indicated. If the lesion can be localised to the mastoid, tympanic, or labyrinthine segments, high-resolution temporal bone CT is recommended. The proximal extracranial portion of the facial nerve in the parotid gland is best visualized with axial high-resolution T1-weighted images using a surface coil [4].

MRI:

MR scanning protocol for evaluation of VII should include a heavily T2-weighted sequence (e.g. FIESTA, CISS) as well as thin-section T1-weighted postcontrast sequences in axial and coronal planes. MRI allows evaluation of VII from the brainstem to the fundus of IAC and is particularly useful for determining the presence of perineural spread from parotid malignancies [2,4].

On high-resolution T2-weighted images, the normal facial nerve appears as a hypointense linear structure extending from the brainstem to the IAC, anterior to the vestibulocochlear nerve, surrounded by T2-hyperintense cerebrospinal uid (Fig.5). Enhancement of the postgeniculate (postganglionic) segments of the facial nerve can be a normal finding. However, asymmetric enhancement, nodularity, thickening, or extension to the cisternal/canalicular portions of the nerve are suggestive of disease [4,5].

CT:

High-resolution CT is the optimal modality for imaging the intratemporal course of VII, from the fundus of the IAC to the stylomastoid foramen, and allows assessment of the caliber and course of the IAC and bony facial nerve canal, as well as integrity of the bony
canal (Fig.8). In addition, CT has the advantage of demonstrating the relationship of the facial nerve canal to normal anatomic landmarks such as the ossicles, and otic capsule structures [4].

When the palsy cannot be definitively localised clinically, contrast-enhanced MRI should be performed first. Both MRI and CT are typically performed for the evaluation of tumors involving the facial nerve [4].
Fig. 1: Graphic image showing components of facial nerve nucleus and its peripheral connections.

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**Fig. 2:** Graphic image showing supranuclear control of facial nerve and effects of lower motor neuron lesion and upper motor neuron lesion.

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Fig. 3: Graphic image showing topography of facial nerve nuclei in the pons.

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Fig. 4: Course of the facial nerve within the brainstem. Axial T2-weighted image through the pons at the level of the facial colliculi (arrow) demonstrates the course of the facial nerve within the brainstem. Note the loop of the facial nerve (internal genu) around the abducens nerve nucleus (white circle). Motor nucleus of facial nerve (red circle); nucleus tractus solitarius (yellow circle); superior salivatory nucleus (blue circle).

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Fig. 5: Cisternal and canalicular course of the facial nerve. Axial heavily T2-weighted CISS image through the pons shows intracisternal and intracanalicular component of facial nerve (arrow) traversing the CPA cistern medial to vestibulocochlear (VIII) nerve.

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Fig. 6: Normal facial nerve inside the internal auditory canal. Sagittal CISS image shows the facial and the vestibulocochlear nerves (the latter formed by the cochlear, and the superior and inferior vestibular branches) in the IAC. The IAC is divided into four quadrants by the falciform crest (transverse crest) and the Bill bar (vertical bony structure), not visible on imaging. The facial nerve is located in the antero-superior quadrant, the cochlear nerve in the anteroinferior quadrant while the vestibular branches pass through the posterosuperior and posteroinferior quadrants respectively.

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Fig. 7: Graphic image showing intra-temporal segments of facial nerve.

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**Fig. 8:** Normal facial nerve canal on CT. Axial and coronal high resolution temporal bone CT images demonstrate the intracanalicular, labyrinthine (arrows in (A)), geniculate ganglion (arrow in (B)), tympanic (arrow in (C)), and mastoid (arrow in (D)) segments of the facial nerve.

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Findings and procedure details

Causes of the Facial Nerve Paralysis

The facial nerve can be affected by a number of different disorders resulting in weakness or paralysis of the facial musculature (Table 1). An important step in the clinical evaluation of facial paralysis is to try to discern whether a lesion is due to a central nervous system process (e.g. CVA, brain tumour, demyelination) or peripheral disease (e.g. Bell's palsy, middle ear infection/cholesteatoma, VII tumour) [4].

Initial clinical evaluation of patients with facial nerve dysfunction includes a detailed history and clinical examination. Clinically, it is usually possible to distinguish between upper and lower motor neuron causes of facial paralysis based on the observation that an upper motor neuron process tends to spare the upper facial musculature (because of the bilaterality of supranuclear control; see above), whereas a lower motor neuron process affects the upper and lower facial musculature. It may also be possible to localise, in some instances, the precise level of lower motor neuron facial palsy based on the affliction of the GSPN (lacrimation), nerve to stapedius (hyperacusis), and the chorda tympani (impaired taste and salivation). Lesions may, therefore, be classified as suprageniculate, suprastapedial, and suprachordal, respectively. In many instances, however, such localisation is not possible.

Central facial palsy is initially investigated using MR imaging. Peripheral facial nerve disorders may be evaluated using HRCT, MR, or both [1], as discussed above.

In this section, we demonstrate pathologies of the facial nerve beginning medially at the facial nerve nuclei and laterally to the peripheral divisions, in a segmental anatomical approach.

1. Central and Congenital Processes:

A variety of central pathologies can affect the intracranial portion of the facial nerve, including infarction, neoplasm (primary and secondary), and demyelination. The site of the intracranial lesion determines clinical presentation of the paresis or paralysis [4]. With central causes of VII palsy, involvement of other cranial nerves (particularly the abducens nerve) is much more common than isolated facial nerve palsy (Fig. 9) [2].

Congenital facial paralysis can occur as a result of facial nerve nucleus abnormalities in a variety of syndromes that include Moebius, Di George, Goldenhar, CHARGE, trisomy
13, and trisomy 18. Congenital hypoplasia or aplasia of VII can also occur as an isolated phenomenon and is best visualized by MRI (Fig.10) [4].

2. Facial Dystonias: Hemifacial spasm, or facial nerve vascular loop syndrome, is an isolated involuntary sudden contraction of the facial muscles caused by a vascular loop of the anterior cerebellar artery (AICA), posterior inferior cerebellar artery (PICA), or vertebral artery (VA) at the root exit zone. MR is the method of choice to demonstrate neurovascular conflict (Fig.11) [6].

3. Peripheral Causes of Facial Nerve Palsy:

A) Inflammatory Disorders:

Bell's palsy, the most common cause of facial paralysis, is characterised by an abrupt onset of facial weakness. MR imaging using gadolinium may demonstrate enhancement of the geniculate ganglion, labyrinthine segment, and proximal tympanic segment without significant enlargement of the nerve (Fig.12). Enlargement of the intratemporal segments of the facial nerve should raise suspicion for tumour. MR imaging in the setting of classic Bell's palsy is probably of limited value. However, any atypicality in presentation (e.g. insidious onset, progressive course, delayed recovery) may warrant imaging with MRI. A wide variety of inflammatory processes may cause facial palsy, including Lyme disease, Ramsay Hunt syndrome, sarcoidosis, Guillain-Barre syndrome, diabetes mellitus, connective tissue disorders, and leprosy (Fig.13) [1].

The facial nerve may also be secondarily involved in inflammatory disorders of the temporal bone. About 5% of patients who have acute otitis media and 1% of patients who have cholesteatoma may present with facial nerve paralysis (Fig.14). Malignant otitis externa may also be associated with facial palsy. The tympanic segment of VII is most vulnerable to such involvement. Subtle erosion of the facial nerve canal may be impossible to detect, especially because the canal wall is inherently thin. However, gross invasion of the facial nerve canal is usually demonstrable using HRCT [1]. Cholesteatoma is typically associated with high signal intensity on diffusion-weighted b1000 sequences and low signal intensity on ADC (a valuable key to the diagnosis, particularly in differentiating chronic otitis media from cholesteatoma) (Fig.15) with newer DWI techniques allowing better detection of small lesions due to decreased susceptibility artifact and providing a very useful imaging tool in the postoperative setting, reducing the need for second-look surgery and its associated morbidity [7].

B) Facial Nerve Trauma
Fractures of the temporal bone may be associated with facial paralysis in up to 50% of cases. Facial paralysis is usually delayed, incomplete, and transient. Although any fracture of the temporal bone can result in facial nerve injury, it is more likely to occur with transverse (38%-50% of cases) rather than with longitudinal (20%) fractures. Immediate facial paralysis usually indicates severe nerve injury (transection), whereas paralysis of delayed onset is usually due to an intramural hematoma. Fractures are best demonstrated on HRCT (Fig.16) [1].

C) Neoplasms:

Tumors of the facial nerve usually present with progressive facial paresis or paralysis. Facial paralysis that does not evolve in a pattern such as that typical of Bell's palsy (e.g. insidious onset, progression for more than 3 weeks, persistence for longer than 6 months) should be investigated for the possible tumour. The facial nerve may be affected by both primary neoplasms, or secondarily involved, either by tumours of the temporal bone or by perineural spread of extratemporal malignancy [1].

Metastasis commonly affects the petrous apex and the internal auditory meatus. CT typically shows erosion and destruction of cortical bone, with MRI demonstrating enhancement on contrast-enhanced T1-weighted images (Fig.17). Margins may be hazy and meninges may be thickened in cases of metastatic disease [2].

Lipomas can occur in the CPA or IAC, entrapping unmyelinated facial nerve fibers (Fig.18) [4]. Haemangiomas arise from the vascular plexus surrounding the facial nerve, most commonly in the perigeniculate region. Haemangiomas cause facial nerve dysfunction by direct neural compression or neural ischaemia due to vascular steal. Temporal bone HRCT can visualize mineralisation of ossifying haemangiomas, distinguishing these tumours from facial nerve schwannomas (Fig.19). MRI is useful for accurate identification of schwannomas, lipomas and haemangiomas (Fig. 20) [4].

Malignant tumors of the parotid can affect the extratemporal facial nerve. Primary malignant neoplasms include mucoepidermoid carcinoma, adenoid cystic carcinoma, adenocarcinoma, malignant mixed tumors, acinic cell carcinoma, lymphoma, and squamous cell carcinoma. In particular, adenoid cystic carcinoma frequently exhibits perineural spread (70-75%). MRI with gadolinium is useful for detecting perineural spread from parotid and minor salivary gland malignancies along the facial nerve (Fig.21,22) [4].

Facial schwannomas may occur along any segment of the facial nerve. CT and MR imaging play an important role in evaluation of site. On MR images, they may present as lobulated masses (when in the CPA cistern, IAC, tympanic segment, and parotid space) or segments of fusiform expansion (particularly labyrinthine and mastoid segments)
Tumours in the IAC are usually indistinguishable from vestibular schwannomas. CT best depicts the expansion of the bony facial canal produced by VII schwannomas [1].
<table>
<thead>
<tr>
<th>Type of Paralysis</th>
<th>Cause</th>
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<tbody>
<tr>
<td>Supranuclear</td>
<td>Cerebral infarction, haemorrhage, vascular lesions, demyelinating diseases, brain tumours, trauma</td>
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</table>
| Brainstem        | - Inflammation and infection: cerebritis, abscess, multiple sclerosis  
                    - Vascular: arteriovenous and cavernous malformations, stroke, Millard-Gubler syndrome  
                    - Tumour: lymphoma, brainstem glioma, metastasis  
                    - Congenital: absent facial nerve, Möbius syndrome  
                    - Trauma: brainstem injuries |
| Cisternal and intracanicular | - Inflammation and infection: viral infections, sarcoidosis, tubercular and bacterial infections, Ramsay Hunt Syndrome  
                                   - Vascular: vertebrobasilar dolichoectasia, vertebrobasilar and PICA or AICA aneurysms and loops, superficial siderosis  
                                   - Tumour: neurofibroma, schwannoma, meningioma, epidermoid, carcinomatous meningitis, leukaemia  
                                   - Trauma: basilar skull fractures |
| Intratemporal    | - Inflammation and infection: Bell's palsy, otitis media, mastoiditis, meningoencephalitis, Ramsay Hunt Syndrome, poliomyelitis, leprosy, Lyme disease, tuberculosis, syphilis, HIV  
                                   - Vascular: intratemporal ICA aneurysm, anomalous sigmoid sinus  
                                   - Tumour: glomus tympanicum or jugulare, haemangiomas, facial nerve schwannoma, choristoma, perineural tumour spread  
                                   - Congenital: abnormalities of facial nerve canal and course  
                                   - Trauma: surgery, fractures through facial nerve canal |
| Extratemporal    | - Inflammation and infection: malignant otitis externa, Ramsay Hunt syndrome  
                                   - Tumour: parotid tumours, metastatic tumours, perineural spread  
                                   - Trauma: forceps delivery; penetrating trauma; mandibular block anaesthesia; otologic, skull base, and parotid surgery |
| Miscellaneous     | Myasthenia gravis, diabetes, hypertension, hyperthyroidism, alcoholism, exposure to thalidomide or misoprostol, Möbius, Di George, Poland, Goldenhar, CHARGE, and trisomy 13 and 18 syndromes, Melkersson-Rosenthal syndrome, hereditary hypertrophic neuropathy, temporal arteritis, polyarteritis nodosa and other vasculitides |

**Table 1:** Causes of facial nerve paralysis.

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**Fig. 9:** Pontine cavernous haemangioma. Axial T2-weighted (A) image at the level of the pons in a patient with familial multiple cavernous haemangiomas presenting with sudden onset of left VI and VII palsy demonstrate a pontine cavernous haemangioma centred to the left of the midline with a fluid level in keeping with recent haemorrhage. Gradient echo image (B) demonstrates further cavernous haemangiomas in the left frontal lobe and both temporal lobes.

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**Fig. 10:** Hypoplasia of the right facial nerve. Axial (A) and sagittal reformatted (B) heavily T2-weighted image shows a narrowed right IAC (green arrow) with hypoplastic VII and VIII cranial nerves (blue arrow and yellow arrow respectively).

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**Fig. 11:** Neurovascular compression in a patient presenting with right hemifacial spasm. Sequential sagittal reformatted heavily T2-weighted images demonstrate a vascular loop (yellow arrow) abutting the right facial nerve (blue arrow) at the root exit zone. The vestibulocochlear nerve (red arrow) traverses the CPA cistern lateral to the facial nerve.

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**Fig. 12:** Bell's palsy. Axial and coronal contrast-enhanced T1-weighted images reveal marked, asymmetric enhancement of the geniculate (yellow arrows in (A and D)), tympanic (yellow arrow in (B)), and mastoid (yellow arrow in (C)) segments of the right facial nerve. Normal enhancement of the postgeniculate (postganglionic) segments of the left facial nerve (blue arrows) due to surrounding veins. There should be no significant enlargement or nodularity of the nerve to confidently make a diagnosis of Bell's palsy.

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Fig. 13: Ramsay Hunt syndrome. Axial fat-suppressed, contrast-enhanced, T1-weighted images through the temporal bones of a patient who had a painful external ear vesicular eruption, otalgia and acute right facial paralysis show intense enhancement of the right facial nerve in the geniculate ganglion, canicular, labyrinthine (blue arrow in (A)), tympanic (blue arrow in (B and C)) and mastoid segments (blue arrow in (D)). Subtle enhancement and oedema of the soft tissues around the right ear (yellow arrow) represents cutaneous vesicular eruption. Soft tissue involvement is not associated with Bell's palsy and is more suggestive of Ramsay Hunt syndrome.

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Fig. 14: Cholesteatoma with right facial palsy. Axial (A, C) and coronal (B, D) CT images in a patient presenting with recurrent, transient right facial paraparesis show a multiloculated, smoothly marginated lesion eroding into the tympanic and proximal mastoid segment of the right facial nerve (yellow arrow) and lateral semicircular canal. Left facial canal (blue arrow) and lateral semicircular canal (black arrow) for comparison.

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Fig. 15: Cholesteatoma with right facial palsy. Axial T2-weighted image (A) and periodically rotated overlapping parallel lines with enhanced reconstruction (PROPELLER) diffusion weighted image (DWI) (B) in the same patient shows lesion to
be of intermediate signal intensity on the T2-weighted image and demonstrating high PROPELLER DWI signal intensity, compatible with cholesteatoma.

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Fig. 16: Trauma of the temporal bone with peripheral right VII palsy. Axial CT image demonstrates a complex fracture with a transverse component through the tympanic segment of the bony facial nerve canal (arrow). Note blood products in the middle ear cavity.

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**Fig. 17:** Leptomeningeal carcinomatosis. Coronal and axial T1-weighted postcontrast images showing diffuse leptomeningeal metastases with left VII and right V nerve involvement in a patient with breast carcinoma.

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**Fig. 18:** CPA lipoma. Noncontrast axial T1-weighted (A) and T2-weighted image (B) demonstrates a nodular hyperintense lesion in the right CPA (arrow). Postcontrast fat-saturated T1-weighted MR image demonstrated signal dropout and no associated enhancement in this lesion indicating fat content (not shown).

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Fig. 19: Facial nerve haemangioma. Axial CT image in a patient with a 2-month history of progressive right VII palsy demonstrates a mass with fine trabecular bony matrix causing widening of the genu of the facial nerve, labyrinthine segment and fundus of the IAC.

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Fig. 20: Facial nerve haemangioma. Axial heavily T2-weighted CISS (A) and postcontrast T1-weighted (B) MR images reveal a T2-hypointense, contrast-enhancing mass in the region of the left geniculate ganglion (yellow arrow in (A), white arrow in (B)). The mass is extending medially into labyrinthine segment and fundus of the IAC (blue arrow in (A)). Histopathology confirmed imaging findings.

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**Fig. 21:** Perineural spread of adenoid cystic carcinoma of the parotid. Axial T1-weighted (A, B) and T2-weighted MR images (C, D) in a patient with a 1-year history of progressive right VII palsy demonstrate a thickened and oedematous right facial nerve in its parotid (white arrow) and mastoid segments (blue arrow). Adenoid cystic carcinoma of the parotid (yellow arrow) and incidental right CPA arachnoid cyst (asterisk).

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Fig. 22: Perineural spread of adenoid cystic carcinoma of the parotid. Axial and coronal postcontrast T1- weighted MR images in the same patient demonstrate an enlarged, enhancing right VII in the parotid segment (yellow arrow in (A)), mastoid (yellow arrow in (B and D)) and tympanic segments (blue arrow in (C and D)) of the VII canal, indicating perineural tumour spread of adenoid cystic carcinoma of the parotid gland (red arrow) to the facial nerve. The tumour has also spread to the right mandibular nerve (white arrow in (B)) at foramen ovale via the auriculotemporal nerve. Left foramen ovale (green arrow (B)) for comparison.

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Fig. 23: Schwannoma of the right facial nerve. Axial T1-weighted (A, B), coronal T2-weighted (C) and coronal postcontrast T1-weighted (D) images in a patient with right peripheral facial nerve palsy demonstrates a dumbbell-like lesion appearing hyperintense on T2-weighted MR images and enhancing avidly with contrast, involving the extracranial parotid segment of the right facial nerve. Note extension of the schwannoma to the mastoid segment of the facial canal (arrow in (B)).

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Conclusion

The facial nerve is complex in its anatomy and may be involved by a wide spectrum of disease. In patients with facial nerve dysfunction, appropriate use of CT and/or MRI imaging guided by clinical presentation should enable appropriate radiological assessment and diagnosis.
Personal information

Department of Neuroradiology,
Greater Manchester Neurosciences Centre,
Salford Royal NHS Foundation Trust,
Manchester, UK
References