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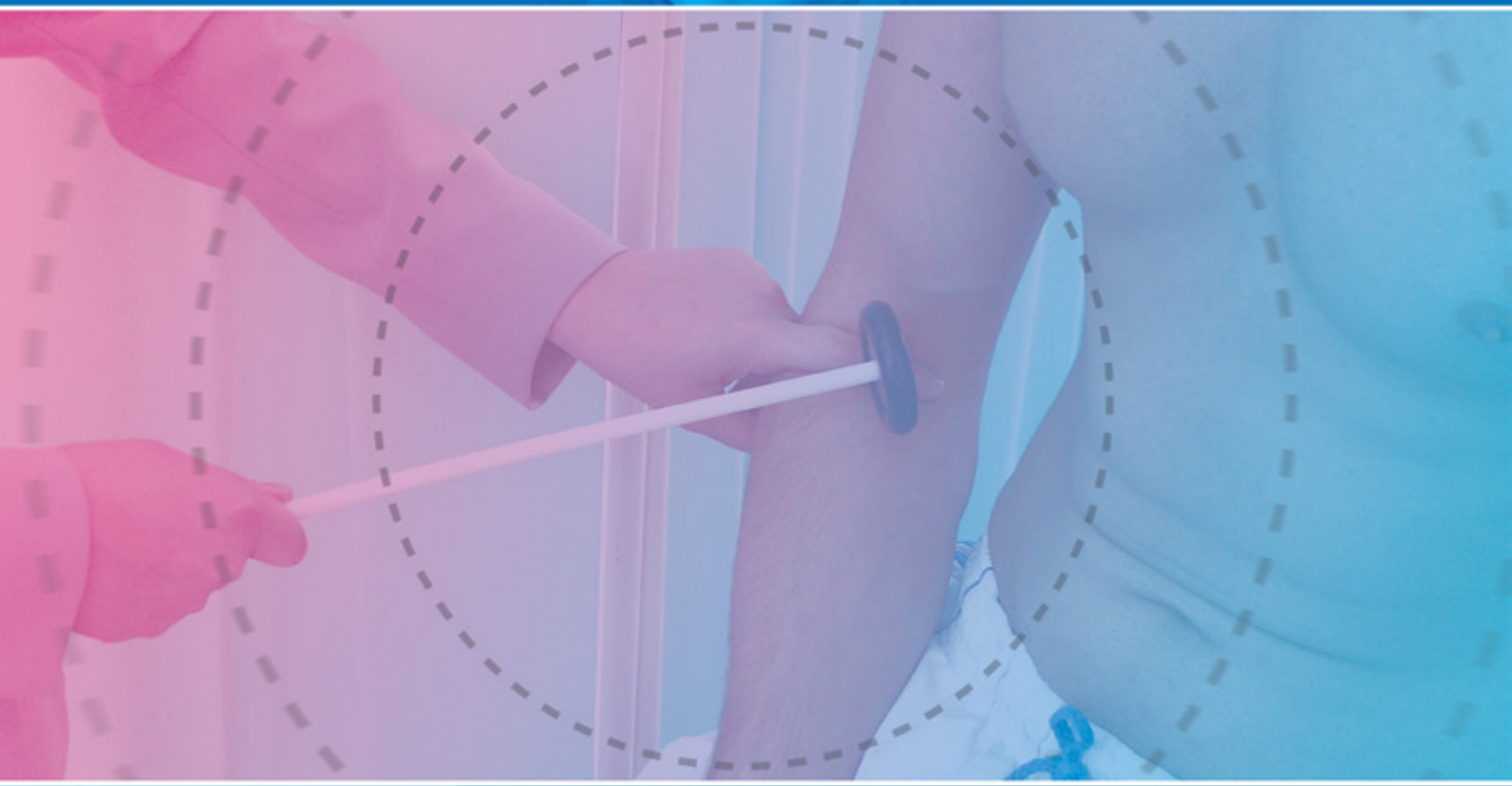

A Peer Reviewed International Journal for the Advancement of Clinical Skills

- 'docendo ac discendo' - 'by teaching and learning'

In this issue:

With proceedings from

The 8th International ePortfolio Conference

Clinical Training Associates & Pelvic Examinations
WHO 'Five Moments for Hand Hygiene'
Holistic approach to resuscitation
Cranial nerve examination

EIfEL
Building a Learning Europe

Executive Board

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Congratulations to Mr Ronak Ved of Cardiff Medical School (UK) on successfully winning The IJOCS Award 2010 - presented for creativity and excellence in the field of Clinical Skills.

The International Journal of Clinical Skills looks forward to contributing positively towards the training of all members of the healthcare profession.

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Foreword

We want raw ePortfolio data, and we want the data now

Patients trust that healthcare professionals will possess the clinical skills to provide safe and effective treatment. Serious failures of medical care, through the actions of individuals and the inaction of organisations, have shaken that trust and led to a re-examination of the process of registration. In many countries and disciplines, continued registration now depends on the documentation of continuing professional development. Some jurisdictions, such as the UK, have gone further and are planning more comprehensive evaluation of clinical performance for revalidation. In all cases, assessment is based on some form of ePortfolio.

"An e-portfolio is a purposeful aggregation of digital items – ideas, evidence, reflections, feedback etc, which 'presents' a selected audience with evidence of a person's learning and/or ability." Sutherland and Powell (2007)

Presenters in the healthcare ePortfolio track at the 8th International ePortfolio Conference, London (July 2010) described a wide range of ePortfolios being used or being developed for allied health, dental surgeons, surgeons, physicians, nurses, medical education, foundation medical graduates. ePortfolios are used by students to evidence acquisition of clinical skills for initial registration, by new graduates to collect evidence of competence for credentialing and by trained staff for evidence of consistent expert performance. As Stuart Cable from the Royal College of Nursing (UK) explained:

"[the ePortfolio] enables nurses to demonstrate their competence in different areas of nursing practice. They are able to capture 'just-in-time' reflections on their practice or a learning experience and then re-present this evidence for different purposes, for example, personal development planning, competence demonstration and educational accreditation of prior learning." (Stuart Cable, Proceedings of the ePortfolio Conference, Maastricht, 2007)

The need for repurposing the same set of collected data across time was confirmed by many of the International ePortfolio Conference presenters: as their careers develop, healthcare professionals will be required to transition across several ePortfolio systems, from those used during initial training, continuing professional development, quality assurance procedures and, at regular intervals, to support reaccreditation processes.

To support evidence of informed and reflective practice, healthcare professionals collect evidence from a variety of sources and data systems, such as patient personal health records, laboratory test analysis, clinical diaries, feedback from peers and patients. Unfortunately, all these different pieces of information are usually stored in independent information silos, making the work of ePortfolio construction and assessment more difficult, notwithstanding that silos make data errors more likely to occur and less likely to be corrected. As most individual ePortfolios also create their own data silos, it reduces the ability to share relevant and critical information across a profession to advance professional practice.

While the initial idea of repurposing ePortfolio data rests on the editing work of an individual compiling a new document, there is an alternative and more radical way of exploiting ePortfolio data: data freedom, i.e. allowing a wide range of online services to exploit raw ePortfolio data.

Imagine a world in which all data created by a healthcare professional when interacting with patients, teachers, colleagues and organisations is securely stored in a Personal Data Store (PDS), creating a 'life log'. Imagine that patients in the healthcare ecosystem have their own personal data stores and can share the contents, under their control, with the people and services they trust. Imagine a world where everyone would be able to choose any health ePortfolio services while being fully interoperable with those used by various institutions with which healthcare professionals interact.

Imagine a world where the performance of students at several medical schools could be confidentially mined to identify best practice for teaching clinical skills. Imagine a service collecting data from the personal data stores of all the staff of a hospital to conduct audit procedures. Imagine another service identifying the need for training and linking it to workshops on particular topics at a conference or a review in a journal. Imagine a service mining anonymous healthcare data collected in personal data stores by a patient's support group. What Amazon® and Google® can do with their global data stores to identify patterns and trends and target advertising, we can do, with personal data stores for the benefit of healthcare, professional education, patient safety and society in general.

Such a world is possible. It was presented by ElfEL at the launch of the Internet of Subjects (www.iosf.org) during the 8th International ePortfolio Conference. The Internet of Subjects supports the programme that Sir Tim Berners-Lee, the inventor of the Internet, called for: *"we want the data raw, and we want the data now!"* To achieve that goal, which is to facilitate reuse, repurposing and exchange of data, we need to achieve the separation of data from the applications and services producing and exploiting it; applications and online services must remain the servants, not the masters, of our personal data.

In the near future institutions will not have to select the ePortfolio platform for their students or professionals; it will be an individual choice. On the other hand, educational institutions, professional communities and public healthcare authorities will have the opportunity to develop a number of innovative services, based on the exploitation of the raw data contained in personal data stores. For example, with an Internet of Subjects, data collected by students and trainees for assessment of progress or by trained staff for revalidation could be used, with permission, for other useful purposes such as quality assurance, needs analysis and career planning.

By providing access to raw data in personal data stores (anonymised and under the full control of individuals) to the services of their choice, healthcare professionals and communities would have the foundations to support the development of lively learning communities, for the benefits of their members, patients and society at large. Data collected whilst compiling an ePortfolio is too rich to be limited to a unique usage. We want raw ePortfolio data, we want it now, to contribute amongst other things, to the improvement of the continuing education of healthcare professionals.

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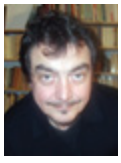

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Cranial nerve examination

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Pathology

Abstract

Cranial nerve examination is often perceived by some as a challenging examination which is time consuming, difficult to interpret and often involves sparsely distributed or unavailable equipment.

This paper summarises key points involved in cranial nerve examination and is intended to make junior doctors feel more confident at performing and interpreting the examination. A summary table of common cranial nerve lesions and their signs and symptoms is provided.

General inspection

A full neurological history prior to examination should allow the examiner to assess the patient's speech for abnormalities such as dysphonia, dysarthria or dysphasia and question the patient directly regarding olfactory, visual, taste, auditory, speech and swallowing disturbances. A general bedside inspection should specifically include assessment of the patient for the presence of facial asymmetry, ptosis, postural deformities and presence of involuntary movements, for example, tremor, tics or chorea.

For the sake of simplicity, throughout this paper the words 'cranial nerve(s)' are represented by the abbreviation 'CN'.

CN I (Olfactory nerve)

Function: The olfactory nerve consists of sensory fibres conducting impulses from the mucous membrane of the nose. CN I is responsible for sense of smell.

Test: Ask the patient if they have noticed any change in their sense of smell. Olfaction can be tested by occluding one nostril and asking the patient to identify and distinguish common strong smelling substances [1]. These can include coffee, orange or peppermint. A lesion involving the olfactory nerve will result in unilateral anosmia; however, bilateral anosmia can be due to a nasal problem such as the common cold; head injury leading to fracture of anterior cranial fossa or a large structural lesion, for example, an olfactory groove meningioma.

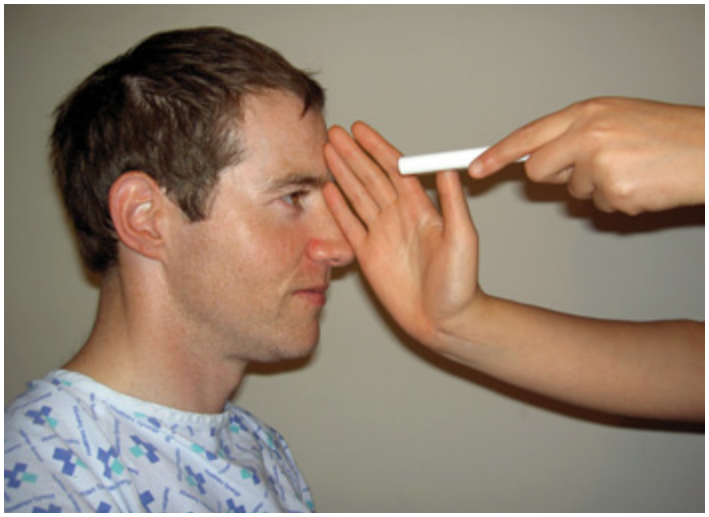
CN II (Optic nerve)

Functions: The optic nerve consists of sensory fibres conducting impulses from the retina. CN II is responsible for vision including visual acuity, visual fields, colour vision, light and accommodation reflexes.

Tests:
1. Visual acuity can be tested using a Snellen chart after correcting for any refraction errors [2].
2. Visual fields should be tested using a red pin that is held equidistant from yourself and the patient. The pin is gradually moved into the centre of vision until it is visible to both yourself and the patient.

3. Pupillary reflexes (direct and consensual reflexes) should be tested using a pen torch (Figure 1). Shining a light into the patient's eyes should make their pupils constrict. Both pupils should constrict at the same time, independent of which eye the light is actually focused on [3].
4. The accommodation reflex can be tested by asking the patient to focus on a distant object; then placing your finger near the tip of the patient's nose, ask them to focus on your finger, whilst you continue observing their eyes, which should converge and the pupils should constrict [1, 3].
5. Colour vision can be examined using standardised tests such as Ishihara plates.
6. Fundoscopy enables examination of the red reflex, optic disc and surrounding media. Look for any papilloedema, macular changes or retinal abnormalities such as diabetic retinopathy (haemorrhages, cotton wool spots and neovascularisation) and hypertensive retinopathy (silver wiring, A-V nipping, haemorrhages, cotton wool spots and rarely papilloedema) [4].

Figure 1: Assessment of pupillary light reflexes (direct and consensual) tested using a pen torch



CN III (Oculomotor nerve), CN IV (Trochlear nerve) and CN VI (Abducens nerve)

Functions: CN III, IV and VI are all involved in the control of eye movements and are therefore usually examined together.

The oculomotor nerve (CN III) consists chiefly of motor fibres supplying levator palpebrae superioris and all the extraocular muscles except for the lateral rectus muscle and the superior oblique muscle. CN III also controls constriction of the pupil and maintains an open eyelid.

The trochlear nerve (CN IV) consists of motor fibres supplying the superior oblique muscle.

The abducens nerve (CN VI) consists of motor fibres supplying the lateral rectus muscle [3].

Tests: Inspect the eyes for any signs of ptosis or a squint. Next examine eye movements by circumscribing a large H in front of the patient with your finger or a penlight. The patient should follow this pattern with their eyes only and their head should remain still. Check for paralysis and nystagmus. Ask the patient to report any diplopia.

In oculomotor nerve (CN III) palsy, the superior oblique and lateral rectus muscles are unopposed resulting in the eye being depressed and abducted (a divergent squint), sometimes referred to as "down and out" [1].

A trochlear nerve (CN IV) palsy results in the eye becoming elevated and outwardly rotated.

As CN III also controls constriction of the pupils, the accommodation reflex and pupillary reflex should be examined.

CN V (Trigeminal nerve)

Functions: CN V receives sensation from the face via three sensory divisions (ophthalmic, maxillary and mandibular) [5]. CN V also innervates the muscles of mastication (temporalis, pterygoids and masseter) [3].

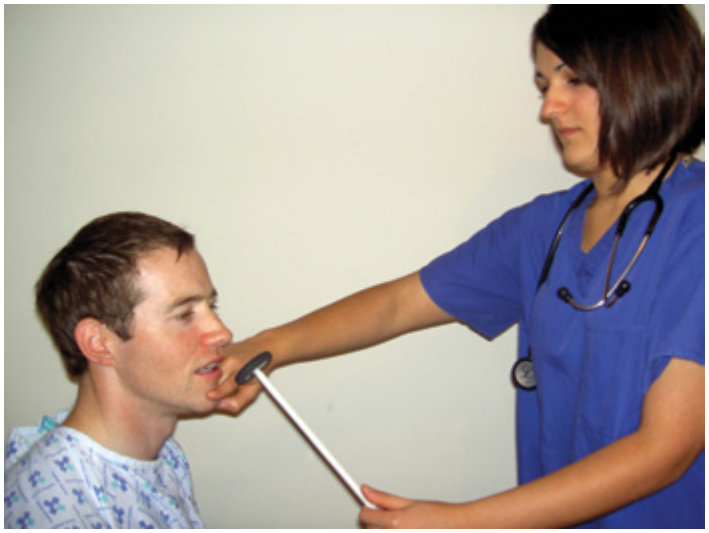
Tests: Test facial sensation in all three divisions. Ask the patient to close their eyes and using cotton wool and a 'neuro pin', test whether the patient can recognise sensation and differentiate between pain and light touch. Note, sensation should be tested on a normal part of anatomy (such as the chest) prior to testing in the three divisions.

The corneal reflex should be assessed by gently touching the cornea with cotton wool, resulting in stimulation of the afferent limb via the ophthalmic division of trigeminal nerve and subsequent bilateral blinking mediated by the facial nerve (efferent limb). Loss of the corneal reflex is a sensitive indicator of impaired sensation in the trigeminal nerve.

The motor function is assessed by checking for any signs of wasting of muscles of mastication: by palpating masseter and temporalis muscles on each side during clenching of the teeth and by asking the patient to open their mouth. A unilateral trigeminal nerve lesion will result in deviation of the jaw towards the damaged side. Subluxation of the temporomandibular joint can also result in this and should be considered as a differential.

Jaw jerk reflex should be tested by asking the patient to relax their jaw with their mouth open a little; placing a thumb on their chin and tapping with the tendon hammer (Figure 2). If there is bilateral upper motor neuron palsy, such as in bilateral cortical infarcts, the reflex will be brisk [3].

Figure 2: Assessment of jaw jerk reflex



CN VII (Facial nerve)

Functions: The motor part of CN VII supplies the facial muscles (muscles of 'facial expression', but not the muscles of mastication), scalp, platysma, stapedius and posterior belly of digastricus [2]. CN VII supplies taste sensation to the anterior 2/3 of the tongue via chorda tympani branch and also comprises the efferent limb of the corneal reflex [1]. It also supplies parasympathetic (secretory) fibres to the lacrimal and submandibular glands.

Tests: Carry out a general inspection for facial droop, asymmetry and abnormalities in eye closure. Examine the muscles of facial expression by asking the patient to demonstrate the following facial expressions [3]:

- Raise eyebrows (frontalis)
- Screw up eyes up tightly (orbicularis oculi)
- Whistling; also blowing out cheeks against a closed mouth (buccinator)
- Showing teeth (orbicularis oris)

Taste for anterior 2/3 of the tongue can be assessed by specifically asking the patient regarding any alteration in taste, as well by testing with salt and sweet solutions [2].

A complete ipsilateral palsy, hyperacusis and altered taste indicates a lower motor neuron CN VII lesion. Its causes can include Bell's palsy (75% of cases), meningitis, Lyme disease, brainstem lesions, cerebellopontine angle tumours (acoustic neuroma), systemic diseases such as diabetes and sarcoidosis, as well as other causes such as otitis media and base of skull fractures (Table 1) [2].

Bell's palsy is a condition of unknown aetiology and is a diagnosis of exclusion. The so-called Bell's phenomenon, commonly caused by Bell's palsy, is demonstrated by the patient attempting to close their eyes and unable to do so, the eyes will roll upwards. This can lead to dry eyes, and injury to cornea from trauma and abrasions should be avoided with the use of artificial tears and eye patching. Bilateral innervation to the upper face means that upper motor neuron palsy will result in contralateral lower 2/3 facial palsy with relative sparing of the frontalis (forehead muscle) and a weak, but usually well preserved, eye closure [1, 6, 7]. Its causes can include stroke and tumour involving the contralateral motor cortex or subcortical pathways [6].

CN VIII (Vestibulocochlear nerve)

Functions: Responsible for hearing and balance, CN VIII is comprised of two sensory components, the cochlear branch carrying the impulses for hearing, and the vestibular branch carrying impulses for rotation and gravity (essential for balance and movement). CN VIII is sometimes called the auditory-vestibular nerve or statoacoustic nerve.

Tests: You can grossly test hearing by whispering numbers in one ear whilst blocking the other ear and asking the patient to repeat them.

Rinne's test compares the patient's ability to hear a tone conducted via air and then bone (the mastoid process). Place the base of a vibrating 512Hz tuning fork on the mastoid process (Figure 3). Ask the patient to identify when the sound is no longer appreciated and then hold the vibrating top of the tuning fork one inch in front of the external auditory meatus (Figure 4). Ask the patient whether the sound is louder in front of the ear (air conduction) or when the tuning fork was placed on the mastoid process behind the ear (bone conduction).

Figure 3: Base of a vibrating 512Hz tuning fork is placed on the mastoid process during Rinne's test

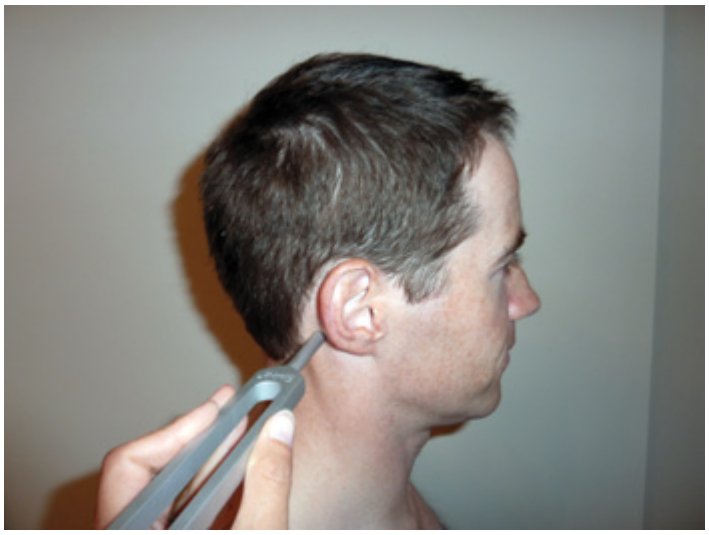
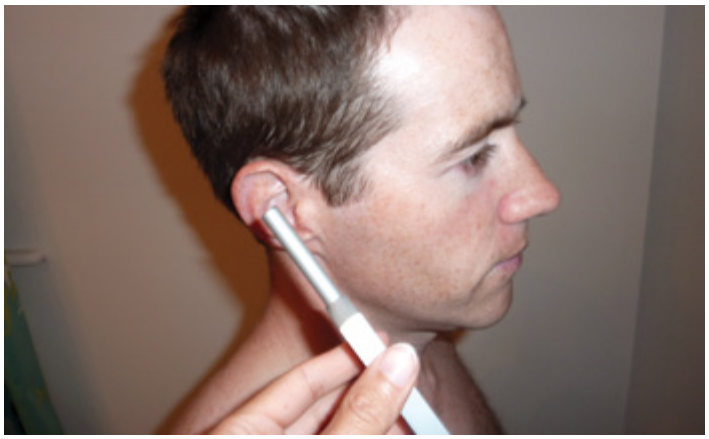


Figure 4: Vibrating tuning fork is held one inch in front of the external auditory meatus during Rinne's test



Normally the vibration is more audible at the external auditory meatus. If the vibration is not heard at the external auditory meatus (i.e. bone conduction is better than air conduction) then this suggests a conductive hearing loss and is termed 'Rinne negative'.

Rinne positive: when nerve deafness is present then the vibration is heard at the external auditory meatus, as air and bone conduction are reduced equally, so that the air conduction is better (as is normal) than bone conduction.

The Weber test compares bone conduction in both ears and can detect a unilateral hearing loss. It is valuable in distinguishing between a true and false 'Rinne negative'. The base of the vibrating 512Hz tuning fork is placed on the centre of the patient's forehead (Figure 5). The patient is then asked whether the sound is heard in the middle or to one side of the head. If the sound lateralises to one side this suggests:

- either a contralateral sensorineural hearing loss (sound will be heard louder in the healthy ear)
- or an ipsilateral conductive hearing loss (sound will be louder in the affected ear due to external noise reduction)

Figure 5: The base of a vibrating 512Hz tuning fork is placed on the centre of the patient's forehead during Weber's test

CN IX (Glossopharyngeal nerve) and CN X (Vagus nerve)

Function: CN IX consists of sensory fibres innervating the tonsils, pharynx, posterior 1/3 of the tongue and the middle ear. It receives general sensation, as well as special sensation (taste), from the posterior third of the tongue. It provides motor innervation to the stylopharyngeus and secretomotor parasympathetic fibres to the parotid gland [1, 6].

CN X:

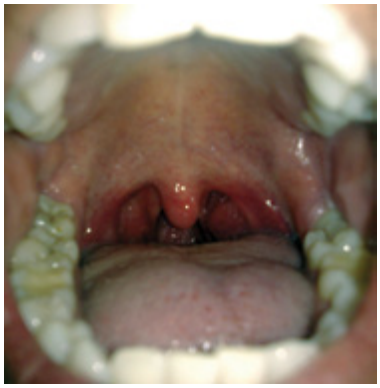
- provides motor fibres to soft palate, all pharyngeal muscles (with the exception of CN IX supplying the stylopharyngeus), intrinsic laryngeal muscles and the cricothyroid
- provides parasympathetic innervation to nearly all thoracic and abdominal viscera down to the splenic flexure
- constitutes the efferent limb of the gag reflex with CN IX being the afferent limb (stimulation of upper pharynx and tonsils)

This implies that the vagus nerve is responsible for varied tasks ranging from heart rate, swallowing and gastrointestinal peristalsis to speech, including the control of muscles for voice and resonance [1, 6, 8].

Tests: The patient should be specifically questioned regarding any speech or swallowing difficulties (dysphagia). This should be followed by a gross assessment of the patient's speech for dysphonia (usually hoarseness) or a 'nasal character'. Ask the patient to say 'aahh' and using a pen torch observe the elevation of soft palate and the uvula, which should be symmetrical with the uvula in the midline in normal cases (Figure 6).

The uvula is deviated to the normal side in cases of CN X palsy. Tactile sensation (CN IX) may be tested by gently touching the back of the palate with a wooden spatula and asking the patient to compare both sides. Gag reflex is not routinely tested due to its unpleasant nature and should be carried out if other tests demonstrate evidence of nerve dysfunction. Similarly taste in the posterior one-third of the tongue is not routinely tested. Clinical scenarios leading to dysfunction of these nerves are listed in Table 1.

Figure 6: Photograph demonstrating visualisation of uvula and soft palate



CN XI (Accessory nerve)

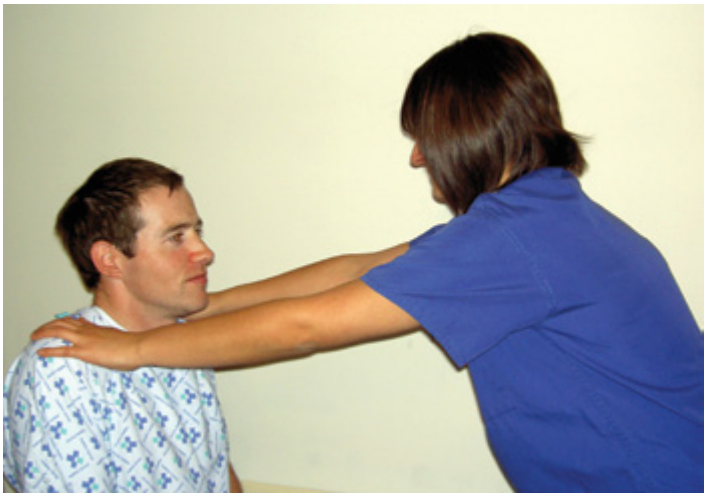
Function: CN XI provides motor innervation to the upper half of the trapezius and sternocleidomastoid muscles (SCM) [1, 3].

Tests: Both SCM and trapezius should be inspected and palpated for fasciculations, evidence of atrophy and bulk.

Assess the power of the SCM by asking the patient to turn their head against resistance (usually from the examiners hand palpating the SCM); for example, the left SCM is tested by asking the patient to move their head to the right with a hand placed on the right side of the chin stopping this movement.

Assess the power of the trapezius muscle by asking the patient to shrug their ipsilateral shoulder and maintain it in elevation during application of a downward force (Figure 7). Base of skull tumours may lead to paresis of CN XI along with other lower cranial nerves in the so-called 'jugular foramen syndromes'. Any surgery in the posterior triangle of the neck can lead to inadvertent injury to branches of CN XI with consequent paresis of trapezius.

Figure 7: Assessment of the power of each trapezius muscle by asking the patient to shrug the ipsilateral shoulder and maintain it in elevation whilst a downward force is applied



CN XII (Hypoglossal nerve)

Function: This nerve supplies motor innervation to all muscles of the tongue (except for palatoglossus which is innervated by CN X) [3].

Tests: The tongue should be observed whilst relaxed in the mouth for evidence of fasciculation. The patient should be asked to protrude the tongue and move it quickly from side to side allowing the examiner to assess for any evidence of wasting, as well as observe the speed of tongue movement. Its strength can be assessed by asking the patient to push their tongue into the side of their cheek whilst offering resistance with a fingertip on the cheek. Unilateral LMN lesions will lead to atrophy of the tongue and deviation of the tongue to ipsilateral side on protrusion. Bilateral LMN lesions causing bilateral wasting and fasciculations are usually part of bulbar palsy with involvement of CN IX, X, XI and XII leading to dysarthria, dysphagia and dysphonia and related to an underlying disorder like motor neurone disease. Bilateral UMN lesions usually lead to hypokinesia of the tongue with associated dysarthria and dysphagia – this so called 'pseudobulbar palsy' may be due to motor neurone disease, demyelination (e.g. in multiple sclerosis) or a vascular event [6]. The gag reflex is preserved in pseudobulbar palsy.

General Examination

Although not in the remit of this paper, depending on the clinical scenario it is of course important to perform general and specific examinations of other systems related to cranial nerve and neurological function. These will not be discussed in detail, but include:

- **the cardiovascular system;** including the heart rate, heart sounds and BP (may show signs of autonomic dysfunction)
- **the respiratory system;** diaphragmatic movement should be assessed as it is innervated by the vagus nerve [8]
- **the gastrointestinal system;** especially if any suspicion of hepatic encephalopathy; remember the importance of per rectum examination (PR) assessing for anal tone and sensation if cauda equina is part of the differential diagnosis [1]
- **the upper and lower limbs;** peripheral nerve examination including tone, power, co-ordination, reflexes and sensation [7]
- **the temporal arteries;** for pulsatility and tenderness to assess for temporal arteritis (important in elderly patients with headaches) [1, 6]
- **the carotid arteries;** auscultating for carotid bruits which may indicate carotid stenosis (carotid stenosis increases the risk of stroke and transient ischaemic attacks) [1, 2]
- **the Mini Mental State Examination (MMSE);** for assessment of cognitive function [9]

Table 1: Summary of the signs and symptoms caused by common cranial nerve lesions [1 – 4, 6, 7, 8, 10]

Cranial Nerve	Signs or symptoms of a lesion	Cause or Lesion
I (Olfactory)	Change in sense of smell	Nasal obstruction • Polyps or foreign bodies • Viral infections
	Unable to identify common substances	Neurological causes • Head injury • Nasofrontal tumours • Parkinson's disease • Alzheimer's disease
II (Optic)	Monocular blindness	Lesions of the eye • Cataracts • Intraocular haemorrhage • Retinal detachments
		Diseases of the optic nerve Multiple sclerosis (MS) Tumours
	Bitemporal hemianopia	Compression of optic chiasm • Pituitary tumour
	Homonymous hemianopia	Lesions of the optic tract • Vascular lesions • Neoplasm • Optic radiation
		Lesions of the occipital lobe
	Visual inattention	Parietal lobe lesions
	Reduced visual fields	Glaucoma Chronic papilloedema
	Marcus Gunn Pupil (relative afferent pupillary defect) observed during the swinging flashlight test [patient's pupils constrict less (therefore appearing to dilate) when the light swings from the pupil of the unaffected eye to the pupil of the affected eye]	Damaged optic nerve pathway – indicating a decreased pupillary response to light in the affected eye (this detects less light than the functioning pathway)
	Constricted pupil	• Horner's syndrome • Opiate overdose • Brainstem stroke
	Nystagmus	Physiological • Congenital • Visual impairment (difficulty fixing gaze) • Vestibular disease • Cerebellar disease
	Papilloedema	Increased intracranial pressure • Tumour • Abscess • Encephalitis
III (Oculomotor)	Divergent squint and diplopia	Paralysis of extraocular muscles (superior rectus, inferior rectus, medial rectus)
	Ptosis	Paralysis of levator palpebrae • Horner's syndrome • Myasthenia gravis
	Dilated pupil	Paralysis of sphincter papillae Tumour Aneurysm
		Brainstem stroke

IV (Trochlear)	Eye elevation and outward rotation and diplopia (on looking down)	Paralysis of superior oblique muscle • Tumour • Aneurysm
V (Trigeminal)	Localised pain and vesicular eruption	Herpes zoster infection
	Anaesthesia and dissociated sensory loss	Syringobulbia
	Brisk jaw jerk	Bilateral upper motor neuron (UMN) lesion
	Loss of corneal reflex, paralysed muscles of mastication and loss of facial sensation	CN V palsy • Neoplasm • Infection
VI (Abducens)	Convergent squint and diplopia (with all movements excluding adduction)	Paralysis of lateral rectus muscle • Tumour • Aneurysm
VII (Facial)	Unilateral complete facial paralysis and hyperacusis	Bell's palsy (inflammation of CN VII)
	Unilateral lower facial palsy	UMN lesion • Stroke • Tumour • MS
	Unilateral entire facial palsy	LMN lesion • Stroke • Tumour • MS
VIII (Auditory)	Conductive deafness	Ear disease • Otitis externa or Otitis media • Paget's disease • Perforated ear drum
	Sensorineural deafness	Congenital
		Acquired Presbycusis (ageing) Noise induced Ototoxicity (drugs)
	Attacks of dizziness and deafness	Acoustic neuroma (benign tumour). As it expands it may compress adjacent CN V – VII
IX (Glossopharyngeal)	Altered sensation to palate and pharynx	CN IX palsy • Base of skull tumour • Stroke or trauma
X (Vagus)	Weak cough or dysphonia	Lesion of the recurrent laryngeal branch
	Asymmetrical soft palate, loss of gag reflex	CN X palsy • Base of skull tumour • Stroke or trauma
XI (Accessory)	Loss of power to sternocleidomastoid (SCM) or trapezius muscle	CN XI palsy • Tumour • Stroke • Trauma
XII (Hypoglossal)	Tongue deviation or weakness	Lower motor neuron (LMN) lesion (towards side of lesion)
	Tongue fasciculation	Motor neurone disease

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