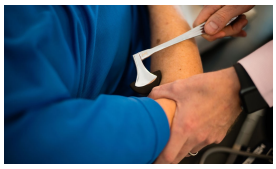


NEUROLOGICAL TESTING & PROCEDURES ON THE NORTHERN BEACHES

northern beaches neurology




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Neurological Consultants

Dr Neil Simon
Dr Peter Puhl
Dr Alexis Selby
Dr Christian Skulina
Dr Tejas Patel
Dr Jason Gu
Dr Natasha Gerbis

Locations

Dee Why Rooms

Suite 4101, Level 1
Dee Why Grand Commercial Tower
834 Pittwater Road (corner Sturdee Pde)
Dee Why NSW 2099

Northern Beaches Hospital Rooms

Suite 14a, Level 6
105 Frenchs Forest Rd W
Frenchs Forest NSW 2086

Contact details for both clinics:

T: (02) 9982 2270

E: reception@nbneuro.com.au W: www.nbneuro.com.au

F: (02) 9981 7880

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Contents

To Book a test or Consult:

Ring: (02) 9982 2270

Email: reception@nbneuro.com.au

Fax: (02) 9981 7880

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VESTIBULAR TESTING

When to order vestibular function tests:

Vestibular function tests can be useful to confirm vestibular dysfunction in patients presenting with the following complaints:

- Vertigo and dizziness
- Unsteady gait and recurrent falls

Limitations of vestibular function tests:

- Vestibular function tests are not useful to diagnose benign paroxysmal positional vertigo (BPPV)
- Occasional patients with Meniere's Disease will have normal vHIT and VEMP studies. In this case caloric studies may be required

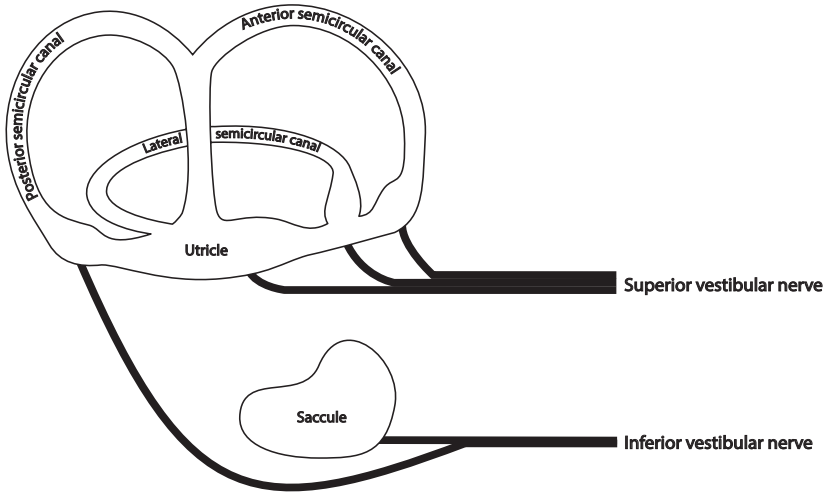


Figure 1: Diagrammatic representation of the vestibular apparatus and its nerve supply.

Vestibular function tests – What, how and why?

Northern Beaches Neurology has now acquired specialised laboratory equipment to assist in the assessment and diagnosis of dizzy patients.

A comprehensive battery of tests includes video head-impulse tests (vHIT), ocular and cervical vestibular-evoked myogenic potentials (oVEMPs and cVEMPs) and brainstem auditory-evoked responses (BAERs).

These tests assess all of the components of the vestibular apparatus as well as the peripheral and central components of cochlear nerve function.

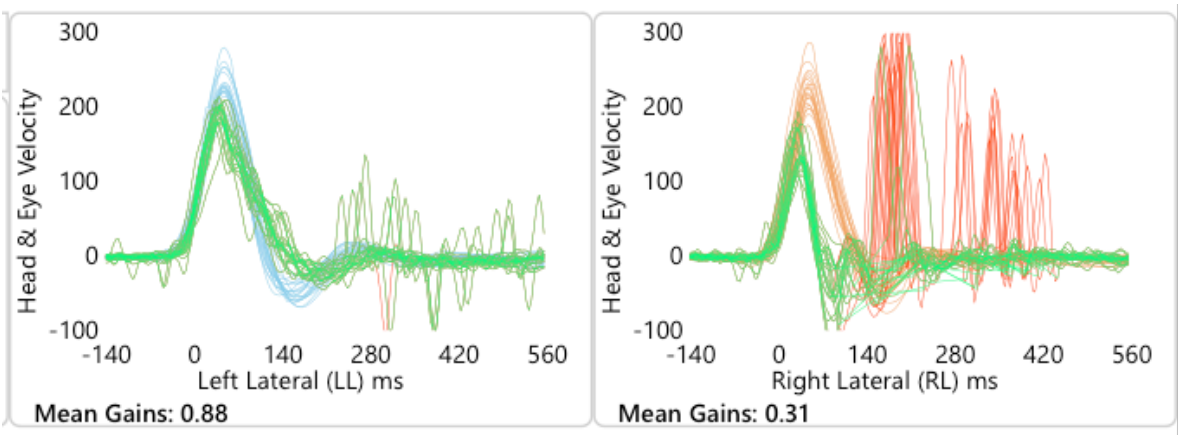
All of the tests are easy for the patient to tolerate and do not induce vertigo

Video head-impulse tests (vHITs)

vHITs measure the function of all six semicircular canals. This is a non-invasive test involving measurement of eye movement abnormalities in response to sudden brief head movements that is very easy for patients to tolerate.

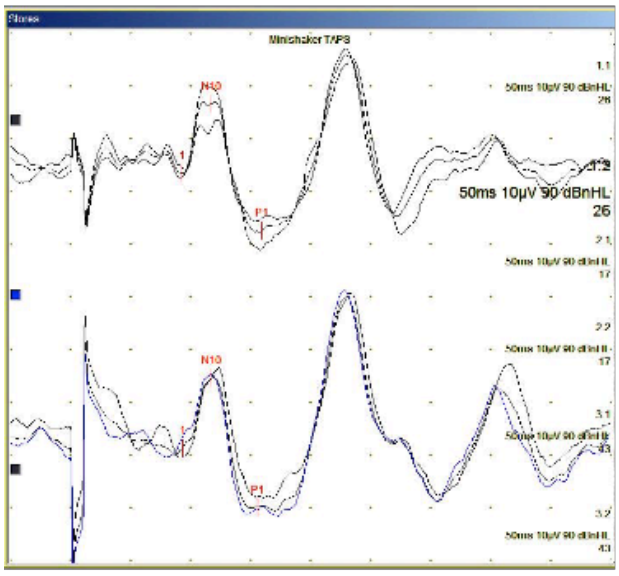
In brief, the patient is asked to focus on a point and their head is quickly rotated. In the normal situation the vestibulo-ocular reflex maintains fixation. In the case of a vestibulopathy, the vestibulo-ocular reflex does not function and the eye moves away from the target. There is then a voluntary saccade (rapid corrective eye movement) which brings gaze back to the target. This may be evident to the examiner when these saccades are 'overt' but they may be hidden in the eye movement 'covert' and not easy to see.

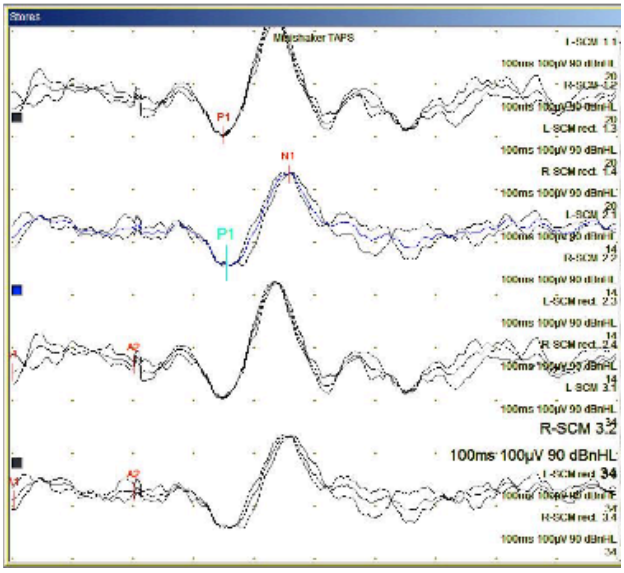
The vHIT goggles detect corrective saccades indicating semicircular canal dysfunction. The figure below shows a normal recording from the left lateral semicircular canal (green lines indicate normal eye movements) and both over and covert saccades (red lines).



Vestibular-evoked myogenic potentials (VEMPs)

Ocular VEMPs (oVEMPs) and cervical VEMPs (cVEMPs) assess the function of the utricle and saccule. This test is painless and uses a tapping stimulus to produce the response. Typical normal tracings are shown below.





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Interpreting the results

The table below demonstrates the testing abnormalities that will be evident in various vestibular disorders.

TEST	Healthy Subject	Superior Vestibular neuritis	Inferior vestibular neuritis	Unilateral vestibular loss
vHIT lateral semicircular canal (to the side of lesion)	✔	✖	✔	✖
vHIT anterior semicircular canal (to side of lesion)	✔	✖	✔	✖
vHIT posterior semicircular canal (to side of lesion)	✔	✔	✖	✖
Ocular VEMP (opposite side to lesion)	✔	✖	✔	✖
Cervical VEMP (to side of lesion)	✔	✔	✖	✖

NERVE AND MUSCLE ULTRASOUND

An Introduction to Nerve and Muscle Ultrasound

Neuromuscular ultrasound is a specialty diagnostic modality offered by Northern Beaches Neurology. Dr Neil Simon is an internationally recognised expert in this technique and he undertakes clinical and research ultrasound studies in patients with various nerve and muscle disorders.

Neuromuscular ultrasound has risen in prominence in parallel with improvements in ultrasound technology. Specifically, new machines are equipped with high-frequency probes that provide excellent resolution of even small nerves. Ultrasound complements nerve conduction studies and EMG in that it allows real-time assessment of anatomy while the neurophysiological studies provide functional data. Both studies together can provide a comprehensive assessment of a neuromuscular disorder, which may improve diagnostic accuracy and contribute to decision making about patient management. Ultrasound can also be performed at the time of the patient consultation or nerve conduction studies by the neurologist so can be tailored to the clinical scenario.

Most peripheral nerves can be seen on ultrasound, including smaller cutaneous nerves. A normal nerve has a typical honeycomb appearance on cross-section with dark nerve fascicles surrounded by bright connective tissue (see Figure 1). Nerve injury produces characteristic changes on ultrasound. An injured nerve usually becomes bigger and less echogenic or darker on the screen (see Figure 2). The fascicular pattern is often reduced. Doppler examinations of normal nerve are usually negative for blood flow but an injured nerve may show increased perineurial blood flow.

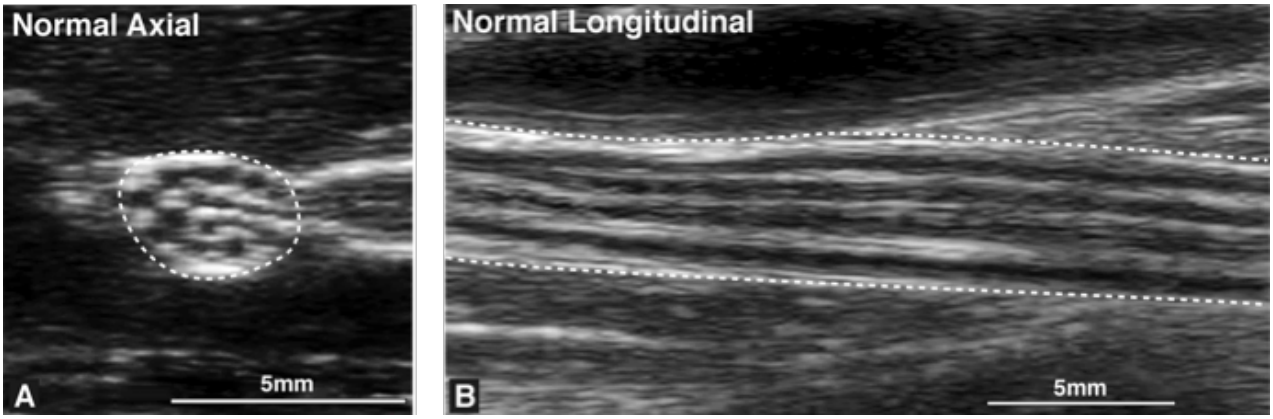

Figure 1: Ultrasound of normal nerve showing a honeycomb pattern on cross-sectional imaging (A) and the nerve fascicles running through the nerve on longitudinal imaging (B).

Indications for nerve and muscle ultrasound

Below is a list of some of the indications for nerve and muscle ultrasound:

Entrapment neuropathy

- Patients with unilateral *carpal tunnel syndrome* to exclude structural lesions contributing to nerve injury
- All patients with *ulnar neuropathy at the elbow* to determine if there is evidence of true nerve entrapment
- All patients with *peroneal neuropathy at the knee* to look for the uncommon case of nerve entrapment and to exclude intraneural ganglion cyst
- *Unusual neuropathies* such as radial tunnel syndrome / posterior interosseous neuropathy
- Where there is a focal neuropathy that has not been localised by nerve conduction studies
- Where there is a concern that a neuropathy is caused or contributed to by anatomic derangement e.g. fracture or mass

Peripheral polyneuropathy

- Suspected cases of inflammatory neuropathy such as CIDP
- Suspected cases of hereditary neuropathy such as CMT

(cont. over page)

Nerve ultrasound is particularly useful for identifying true nerve entrapment that may require surgical treatment. As a point of principle, carpal tunnel syndrome is almost always a nerve entrapment syndrome. Conversely, most other focal neuropathies are not caused by entrapment. In ulnar neuropathy at the elbow only about 25% of cases are related to entrapment, while the remaining cases are caused by extrinsic compression or traction of the nerve. The distinction is very important as surgical decompression may be the preferred treatment of symptomatic nerve entrapment that has not responded to conservative measures.

On the other hand, surgical decompression probably will not help a patient with focal neuropathy caused by extrinsic compression or traction.

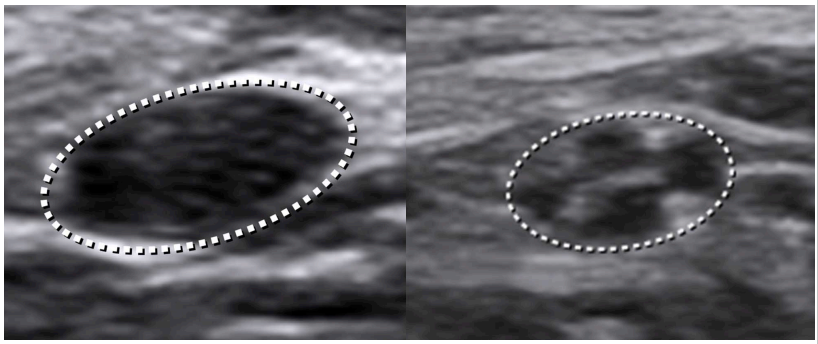

Figure 2: The left panel shows ultrasound of the ulnar nerve at the elbow of a patient with mild ulnar neuropathy at the elbow. The nerve is enlarged, less echogenic (darker) and has lost the normal fascicular architecture as compared with a normal ulnar nerve at the elbow shown on the right

Focal nerve injury may also be related to anatomic anomalies or abnormal anatomical structures such as accessory muscles, cysts or scar tissue. Identifying such abnormalities with nerve ultrasound may change the management offered to the patient.

Some patients are unable to tolerate nerve conduction studies and EMG. Nerve ultrasound may be an alternative diagnostic modality in this patient group, although generally having data from nerve conduction studies, EMG and ultrasound improves clinical decision-making.

Muscle ultrasound can also help in some instances. Muscle has a "starry night" pattern on ultrasound. That is, on ultrasound images most of the muscle is dark

Indications for nerve and muscle ultrasound (cont)

Muscle diseases

- Suspected diaphragmatic weakness
- Suspected *motor neuron disease* (assess for widespread fasciculations)
- Some cases of *progressive myopathy* to look for patterns of involvement (e.g. inclusion body myositis)
- Some cases of suspected *inflammatory myositis*

Ultrasound-guided nerve blocks and botulinum toxin

- Corticosteroid injection for *carpal tunnel syndrome*
- Corticosteroid injection for *meralgia paraesthetica*
- Some *botulinum toxin* injections e.g. deep neck muscles, piriformis syndrome, thoracic outlet syndrome

Ultrasound has some limitations, in particular assessing very deep nerve and muscle structures where resolution is decreased. Ultrasound can not examine structures lying under bone.

Dr Neil Simon can perform ultrasound studies for all of the above indications.

and there are scattered bright spots throughout corresponding to connective tissue and neurovascular elements (see Figure 3). In both myopathy and denervation muscle becomes more echogenic or brighter on ultrasound. In myopathy there is typically a homogenous 'ground-glass' pattern, while in chronic muscle denervation there is more of a patchy increase in brightness.

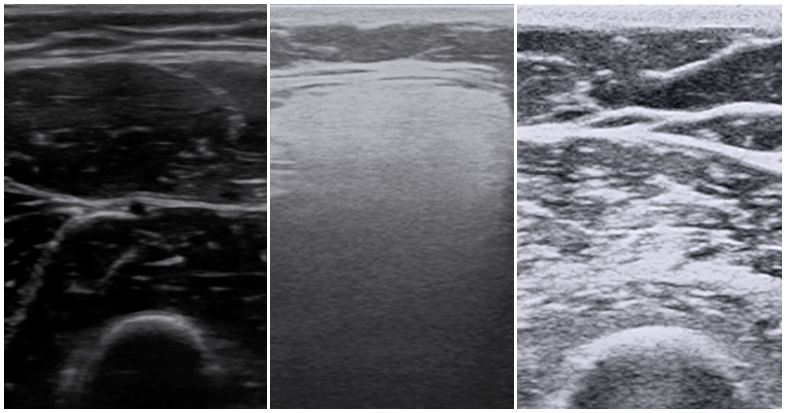

Figure 3: The left panel shows ultrasound of a normal quadriceps muscle with a typical starry night pattern. The middle panel is ultrasound of quadriceps in a patient with severe myopathy showing increased echogenicity with a ground-glass pattern. The right panel shows ultrasound of biceps in a patient with severe chronic denervation with evidence of patchy increase in echogenicity throughout the muscle.

Because ultrasound allows dynamic imaging muscle movements can also be measured. This can involve assessment of normal muscle movements, for example of the diaphragm. Ultrasound is the best diagnostic modality to assess the function of the diaphragm and should be considered a first-line investigation for patients with suspected diaphragm weakness. Ultrasound can also show abnormal or spontaneous muscle movements such as fasciculations, which can be helpful in the diagnosis of some neuromuscular disorders like motor neuron disease.

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NERVE CONDUCTION STUDIES: NCS

Nerve Recovery

In terms of the clinical relevance of the changes on nerve conduction studies, the nature of the nerve injury and the degree of injury to myelin and axons determine the time course of recovery and the likelihood of a complete recovery.

- Isolated myelin injury (or neuropraxia) generally recovers fully within 3 months if the cause of nerve injury has resolved.
- Isolated axonal injury (axonotmesis) takes longer to recover and recovery may be incomplete. Clinical improvements may be detected in a partial injury of nerve axons within a month or two, usually from residual uninjured axons forming sprouts and reconnecting with any denervated muscle. Injured axons will regenerate from the site of nerve injury and grow towards the target muscle at a rate of approximately 1mm per day. Measuring the distance between the

Nerve conduction studies and nerve injury: how to understand the numbers

Nerve conduction studies are one of the most important tests in neurology. They are used to diagnose peripheral nerve disorders such as focal or generalised neuropathy. While each nerve conduction study is interpreted by one of our neurologists, it is useful for a GP to understand the principals behind the study to help when reviewing the study report.

Essentially, during a nerve conduction study, electrical stimuli are passed through a nerve resulting in nerve activation. The resulting signal (nerve action potential) is transmitted along the nerve. In studies of sensory nerves, the nerve action potential is recorded by electrodes placed over the nerve some distance from the region of stimulation (usually 10cm or further). In studies of motor nerves, the nerve action potential travels along the nerve to a muscle resulting in activation (depolarization) of the muscle, which is recorded by electrodes placed over the muscle as a compound muscle action potential.

The nerve conduction study apparatus (electromyograph) measures the time taken for the

nerve injury and a weak muscle can help estimate the time when recovery is expected and this can range from several months up to two years. In general terms, significant recovery is usually not possible beyond two years from the injury.

In mixed myelin and axonal lesions there may be partial early improvement around the 3 month time point, corresponding to improved conduction from myelin regeneration and then a slower phase of recovery while axonal regeneration takes place.

It should be noted that some severe nerve injuries do not recover spontaneously. A neurotmetic injury involves injury to the axons and supporting connective tissue. In this case regenerating axons do not regenerate beyond the injury. Some severe axonotmetic injuries may also not recover if the distance between the injury and the target muscle is more than 60cm (e.g. a patient with a severe injury to the L5 nerve root will probably never great toe extension function).

signal to reach the recording electrodes following the stimulus. The amplitude of the resulting waveform is measured. In combination with the measurement of the distance between the recording and stimulation sites, the speed that the signal conducts is calculated and expressed as conduction velocity.

Nerve injuries can result in damage to the nerve myelin, axons or a combination of the two. Changes on nerve conduction studies can delineate the nature of the nerve injury.

Nerve axons are like the copper wires in a cable that conduct electrical charge. When there is injury to axons the amount of electrical signal travelling through the nerve diminishes. Nerve conduction studies show this as reduced amplitude of the resulting response. In a purely axonal nerve injury the amplitude will remain reduced irrespective of where the nerve is stimulated, as axons degenerate distal to the region of damage within a few days of injury.

Myelin is required for rapid salutatory conduction in nerves. Accordingly, if there is injury of nerve myelin, nerve conduction studies demonstrate reduced conduction velocity across the injured segment.

If myelin damage is severe, electrical signal transmission may not proceed through the injured segment resulting in conduction block. In this instance, stimulating the nerve proximal to the injured area results in a smaller response amplitude than if the nerve is stimulated distal to the nerve injury as injury to myelin alone does not result in axonal degeneration distal to the nerve injury.

Using the combination of these findings, the neurophysiologist can determine the site, nature and severity of nerve injury.

The figure below illustrates the various patterns of abnormalities on nerve conduction studies.

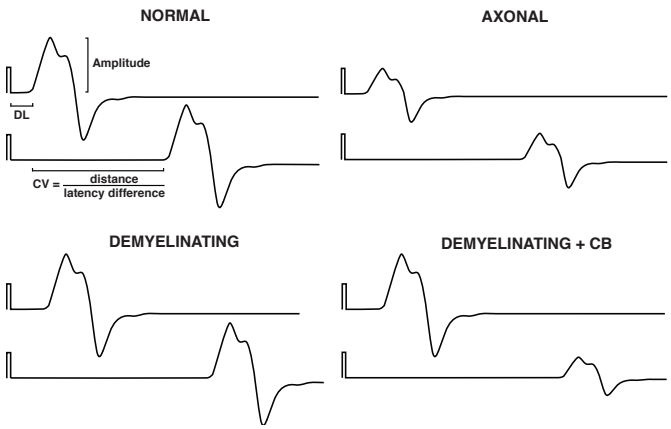

Figure 1: Key metrics are distal latency (DL), amplitude and conduction velocity (CV). With demyelinating abnormalities, DL is prolonged and CV is slow, but amplitude is relatively normal. Conduction block (CB) may also be associated with demyelinating abnormalities, with reduced amplitude with proximal stimulation. In axonal neuropathy, amplitude is reduced, without conduction block, with relatively preserved DL and CV

Figure from Simon NG, Kiernan MC. Neuropathies and motor neuron disease. In: Salardini A, Biller J (Eds). The Hospital

ELECTROENCEPHALOGRAPHY: EEG

When to order an EEG?

Indications for a routine outpatient EEG:

- Seizures
- Episodic neurological symptoms with a clinical suspicion of focal seizure
- Recurrent absence events in children
- Blackout where no other cause is identified
- Rapidly progressive dementia
- Inflammatory or infective brain disorders
- Altered mental status

EEG for the following indications are generally considered low yield:

- Headache and classic migraine
- Typical vasovagal syncope (note that in patients with recurrent syncope without a clear cardiac explanation EEG can be considered to look for epileptic syncope)
- Mood disorders
- Tic disorders
- Non-specific fatigue
- Postural dizziness

As part of a sweeping review of the Medicare Benefits Schedule, the Government has recently reviewed the ordering of EEGs and is considering introducing a national standardised request form for EEG. In general, patients referred by GPs for EEG at Northern Beaches Neurology have a clinically relevant indication for EEG. However, we take this opportunity to review the indications for EEG and also those indications in which a routine EEG may be considered low yield. There are also scenarios in which a prolonged EEG (>3 hours) is indicated.

Note that Northern Beaches Neurology now has the capacity to record prolonged EEGs in our Northern Beaches Hospital rooms.

A routine EEG involves applying multiple leads to a patient's head and then recording the underlying electrical activity for around 20-40 minutes. The EEG will initially be recorded with the patient awake and the patient will be asked to open and close their eyes (and may be asked to perform other actions).

The patient will usually be asked to hyperventilate.

Prolonged EEGs

Prolonged EEG is indicated in patients having frequent neurological events where focal seizure is being considered or in patients in whom non-epileptic seizures are suspected.

Please contact Northern Beaches Neurology to speak with a neurologist if there is a question regarding the usefulness of an EEG in a specific clinical situation.

Hyperventilation can provoke abnormalities on the EEG that may not be seen during quiet breathing.

Photic stimulation will usually be performed which involves flashing a light into the face of the patient at differing frequencies and watching for any brain response. In the normal situation there is often a photic driving response, such that a signal is recorded at the back of the brain corresponding to the frequency of photic stimulation.

Asymmetry of photic driving response may be an important indicator of brain dysfunction and photic stimulation provokes epileptic activity in some patients.

Sleep-deprived recordings are indicated at times. In a sleep-deprived recording the patient is asked to sleep less than 50% of the usual night's sleep. Our office will provide specific instructions if this is required. Sleep-deprivation stresses the brain and can increase the likelihood of showing epileptic activity. It also increases the likelihood of a patient sleeping during the test which can also show abnormalities that are only present during sleep.

At times, where a routine EEG has shown equivocal changes, the report may indicate that a sleep-deprived EEG is recommended. In this situation, the sleep deprived EEG may help confirm the diagnosis of epilepsy.

TREATMENT WITH BOTULINUM TOXIN

PBS Subsidy Criteria for Botox

Presently, Botox is subsidised by the Pharmaceutical Benefits Scheme for the treatment of chronic migraine.

For the subsidy to be available the patient needs to fulfil the following criteria:

- ≥6 months of ≥15 headaches per month with at least 8 migraine headache days
- Patient has tried and failed 3 recognised migraine preventative medications due to side effects or poor efficacy. Examples; amitriptyline, propranolol, verapamil, pizotifen and topiramate.

Botox may still be helpful in patients who do not fulfill these criteria but the patient will incur extra costs, as the Botox will need to be purchased privately.

The first clinical use of botulinum toxin was for the treatment of strabismus in the late 1970s. Since that time the medical indications for botulinum toxin (Botox ®, Dysport ®, Xeomin ®) continue to expand and many of the current uses are in patients with neurological disorders.

Until recent year most of our patients being treated with botulinum toxin were suffering with spasticity, cervical dystonia, hemifacial spasm and other forms of dystonia. In our clinic many of these injections are performed with EMG or ultrasound-guidance to improve accuracy of muscle targeting.

However, in 2015 a pivotal phase III trial (PREEMPT) of Botox in migraine was published indicating that patients with chronic migraine often dramatically responded to Botox. In the study, chronic migraine was defined as 15 or more headache days per month with at least 8 migraine headache days per month for at least 6 months.

Response (defined as ≥ 50% improvement in various indices) was seen in 70% of patients (see figure below from the PREEMPT trial).

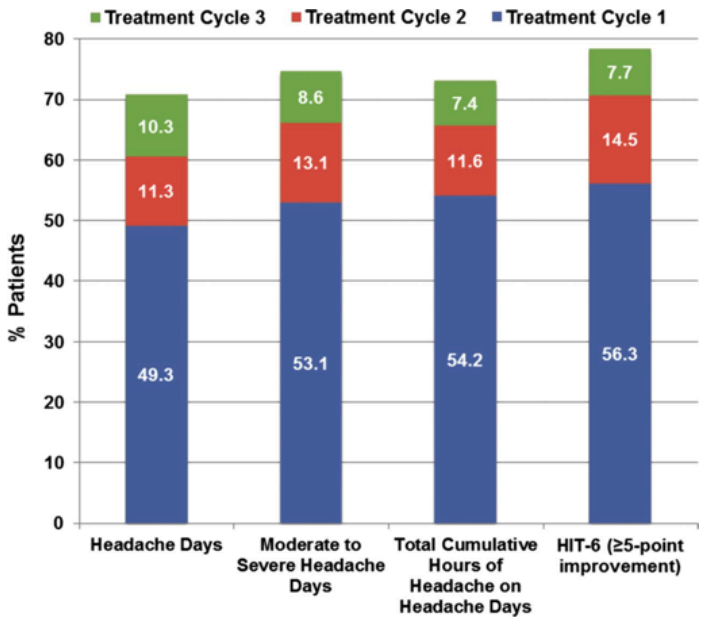

Figure 1. Number of patients responding to Botox injections for chronic migraine.

The neurologists at Northern Beaches Neurology are trained in performing Botulinum toxin procedures for various neurological disorders including Botox for chronic migraine as well as the treatment of severe hyperhidrosis (excessive sweating).

Side Effects

There are generally few side effects.

- Headaches can get worse for approximately a week before they then improve. Some patients complain of uneven eyebrows (brow ptosis) related to injections of the forehead.
- Occasional patients will experience mild neck weakness.
- There are other less common side effects which will be discussed in detail with the patient prior to the injections. It should be noted that all side effects are

This compares very favourably with oral migraine prophylactic mediation where response rates are typically less than 40%, often lower in patients who have failed multiple preventative medications.

As such, Botox is considered the most effective current treatment available for chronic migraine.

The Procedure

In terms of the procedure, Botox for chronic migraine is quite straightforward and generally tolerated by patients.

The procedure involves a series of **31 injections** around the face, head and neck following a standard pattern (as per image above).

The injections take about 5-10 minutes to complete. There is no special preparation before the procedure and there are no significant precautions required after the procedure.

After the first procedure, the patient is required to complete a daily headache diary.

A second procedure will be arranged for **3 months later** and this is suggested even if there has been a suboptimal response from the initial round of injections, a some patients respond well to second and subsequent rounds of injections where they did not respond well to the first. If there is a positive response, repeat injections are often scheduled approximately 3 monthly (and can not be more frequent than 12 weekly) as the biological action of Botox typically wears off at around 3 months. In some instances where there is a very good response, injections can be stretched out to longer intervals.

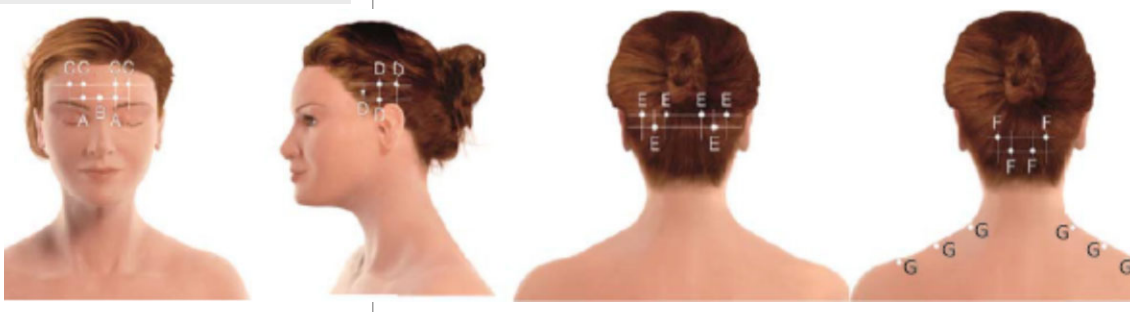

Figure 2 – Botox injection sites for chronic migraine (image copyright American Headache Society).

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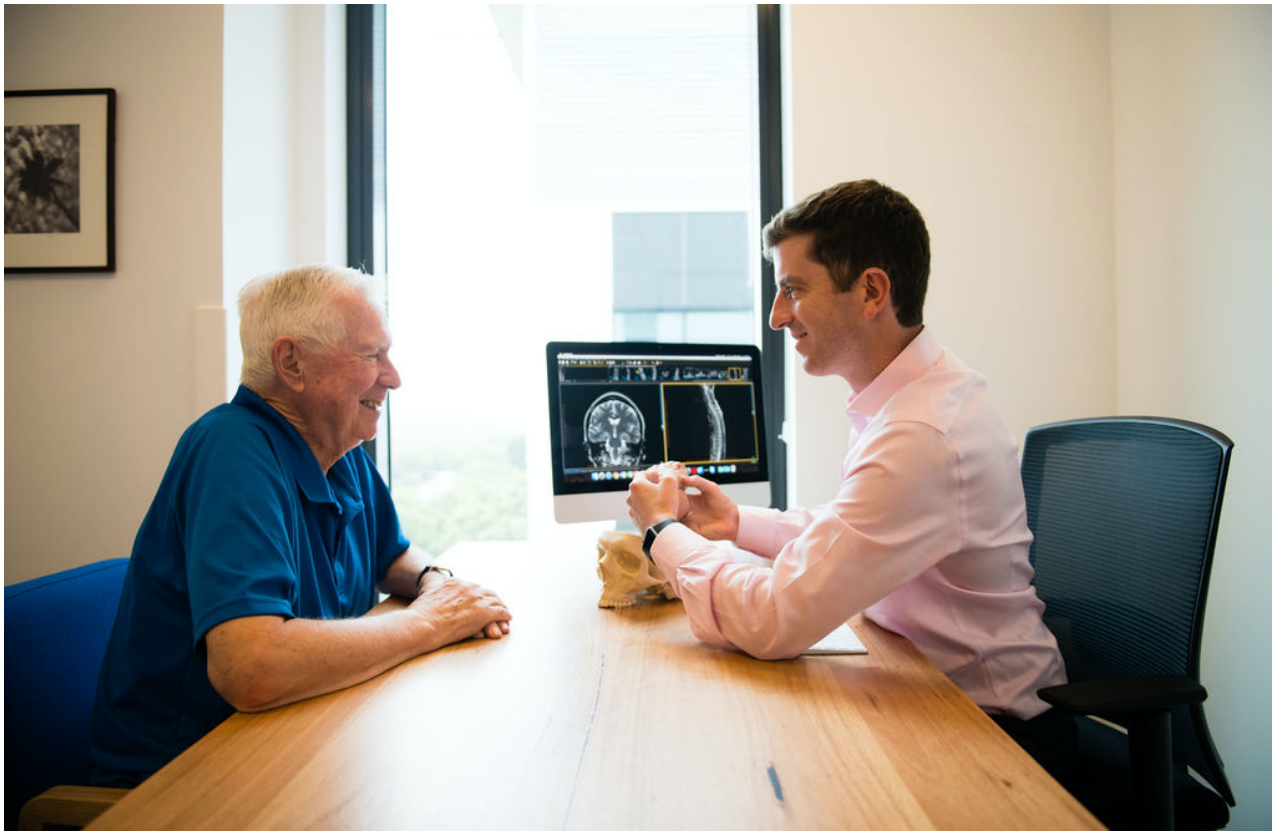

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