The Weekly Probe

8th July 2016 **Volume 13 Issue 21**

Welcome back- Welcome back to Carmel who comes back to work this week. I think there is a nice message to give to your patients – there is always hope when you are placed in Garrawarra - is not the "final destination" – there is always a chance of release if you take your tablets and don't touch the staff. Welcome back!

Up-to-date + CIAP - UpToDate is now available via CIAP on any networked hospital PC (without a login or password). There is a link to CIAP on the SWSLHD Intranet webpage.
To access CIAP at home/offsite, register for a personal CIAP login and password on any networked hospital PC. Once activated you can access CIAP, including UpToDate, offsite with this login and password. Select the Quick link to UpToDate on the CIAP home page.
After opening UpToDate via CIAP, you can also register for a personal UpToDate login and password by selecting the Log In/Register tab in the upper right hand corner. You can also update your existing UpToDate login and password
If you skip the UpToDate login, you can still access UpToDate content via CIAP. However you will not maintain your UpToDate account details, including CPD points unless you login to UpToDate.

Opioid App – Does your phone have any more room for a new app? The faculty of pain Medicine have developed a new FREE app (accessible through the Google play or the App store) which aims to help calculate dose equivalence of opioid analgesic medications. Worth a look!

THIS WEEK

Last week we discussed nystagmus and extraocular movement disorders with a focus on the clinical assessment on the dizzy patient. This week we'll talk about the specific causes of dizziness / vertigo and look at those conditions which cause these symptoms.

DIZZINESS and VERTIGO

TERMINOLOGY

We often "drill down" when a patient presents with dizziness- " is it vertigo or is it lightheadedness or disequilibrium". Vertigo is an illusion of rotation due to a disorder of the vestibular system.

However there are limitations with this approach for a number of reasons.

- Descriptions are imprecise – one study of 300 pts found > ½ were unable to report which symptom type accurately described their experience.
- The type of dizziness does not appear to be a trustworthy predictor of the underlying cause.
  - one study found that the presence of vertigo c/w other types of dizziness was found to predict stroke with equal likelihood
- The type of dizziness was inconsistently described by patients known to cause acute vestibular syndrome such as post fossa CVA, cerebellar haemorrhage, and vestibular neuronitis

The "take home message" is that if the patient does not describe true" vertigo" this does not rule out a central cause. Use the "whole package" of history, associated features, risks, exam, progress and "gestalt" to work out a provisional and differential diagnosis.

In view of this, our initial screen is to look for systemic issues which may have caused the dizziness- hypoglycaemia, cerebral hypoperfusion (due to arrhythmias, postural vasomotor changes etc), intoxication / toxicity, electrolyte abN , endocrine or metabolic issues etc etc .

The next process is to delineate those with **acute vestibular syndrome** (AVS) which is defined by some as acute dizziness accompanied by nausea or vomiting, unsteady gait , nystagmus and intolerance to head movements.

Our next consideration is to try to differentiate between *central* (lesions of the CNS) or *peripheral* (lesions of the vestibular labyrinth or nerve or both).

Central causes of acute vestibular syndrome

Post fossa CVA	83%
- ischaemia 79% / haemorrhage 4%	
MS	11%
Uncertain / misc	6%

Why worry?

- Not uncommon - ~ 18% of strokes are located in the posterior fossa
  ~ 50-70% are associated with dizziness

- Subtle – Posterior circulation strokes are not uncommonly misdiagnosed with symptoms may be subtle. This may be related to the fact that a significant % of patients are younger (1 in 5 < 50yo in the Kattah article below) , and do not have traditional risks for strokes – vertebral artery dissection is a common cause for strokes in this age group.

- The consequences of such misdiagnoses can be profound with one study of missed cerebellar infarcts having ~ 40% mortality (other studies 2-57%depending on the LOC at time of presentation). Deterioration often occurs in the days after the event peaking at ~ 3 days.

Last week we discussed the assessment of oculomotor movements with a focus on the assessment of the dizzy / vertiginous patient. Have a look at this and the links to a number of videos and references as a background for the following info.

One additional test not mentioned that may be considered:

- Head shaking nystagmus — Head shaking nystagmus is elicited by the patient shaking the head from side to side for 15 to 40 seconds with eyes closed. The shaking causes vigorous stimulation of the horizontal semicircular canals of both sides. When the shaking stops, the patient then opens his or her eyes and attempts to look straight ahead. If both sides were equally activated, the eyes will be still. If there is unilateral labyrinthine damage, the asymmetrical neural output will generate a nystagmus that beats away from the damaged side. This test will be normal in patients with bilateral symmetric disease, unlike rapid head thrusts or caloric responses. The test is abnormal in patients with unilateral **central** or peripheral vestibular lesions (nystagmus is contralateral to the lesion in peripheral vestibulopathy, nystagmus produced by head shaking in patients with medullary lesions is ipsilateral to the lesion)

Peripheral -benign positional paroxysmal vertigo, vestibular neuronitis, Meniere's disease, head trauma, acoustic neuroma, otosclerosis, herpes zoster, cholesteatoma, perilymph fistula (post trauma), aminoglycosides;

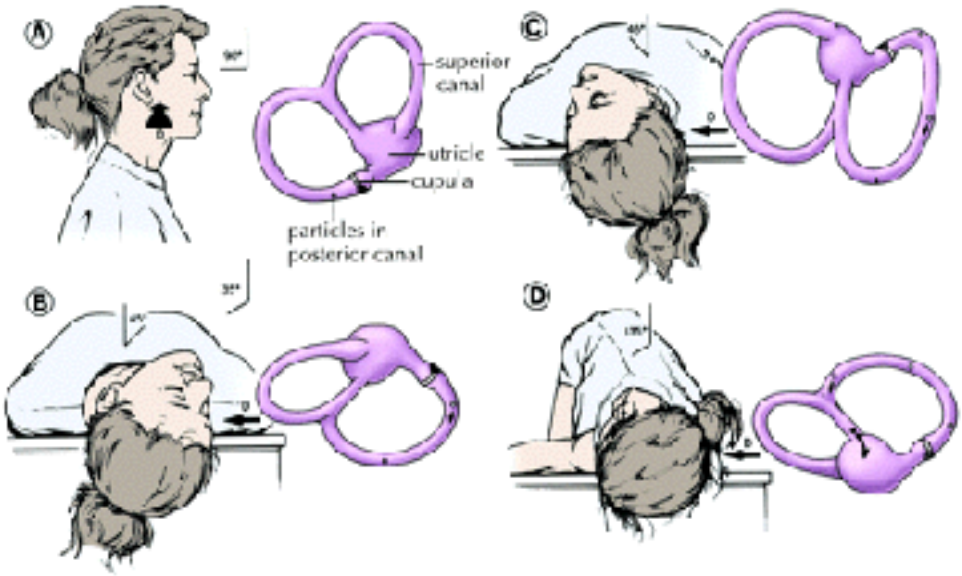
BPPV is the commonest cause of vertigo.

- Diagnostic criteria for BPPV were at least five attacks of vertigo lasting less than 1 min without concomitant neurological symptoms and invariably provoked by self-reported typical changes of head position especially vertical changes (eg. lying down, turning over in the supine position, reclining the head, rising up from supine position and bending forward).
- 80% of patients experience a rotatory vertigo and 47% experience a floating sensation.
- The attacks of vertigo typically last fewer than 30 seconds, however, some patients overestimate the duration by several minutes. BRIEF, INTENSE Patients with BPV also have normal hearing.
- Note there is no role of the Hallpike's manoeuvre in a patient with ongoing nystagmus, a scenario not expected as the patient with BPPV symptoms resolve with rest and time.
- The vertigo begins after a delay & is fatigable- vertical with rotational component .

Another take home message is that any head movement amplifies the disparity in vestibular tone and dizziness from any cause can be exacerbated by head movements.

Editor: Peter Wyllie

- Treat BBPV with Epley's manoeuvre, which is thought to successfully treat BPPV in up to 80% of patients- see below – find in Intranet under Clinical – medical – Neurology .



- Most recommend ~ 30seconds or till the vertigo eases between each stage.
- Overall, the PRM should take less than 5 minutes to complete. Patients are then typically asked to remain upright for the next 24–48 hours in order to allow the otoliths to settle, so as to prevent a recurrence of the BPPV.
- ETG has a collection of exercises patients may try at home – with pictures!!

- Lateral semicircular canal BPPV is a variant in which the nystagmus is horizontal and beats towards the lowermost ear- rotate the patient from their bad side to their good side and encourage the patient to only sleep on their good side

***Meniere's disease*:** consists of a clinical triad of vertigo, tinnitus & deafness.

- Typically, attacks commence with an aura of fullness of the ear, leading to progressive tinnitus, ipsilateral fluctuating hearing loss and vertigo.
- Nystagmus is present only during the attack.
- The commonest age of onset is 30-60 years. The attacks are paroxysmal. Episodes lasts hours typically yet can be 20min to days- tends to be recurrent .

(Note that patients may rarely present with acute , unilateral complete hearing loss via ischaemia of the inner ear , via occlusion of the anterior inferior cerebellar artery.

***Vestibular neuronitis*:**

- Onset- produces subacute onset (see comment on cerebellar and brainstem strokes below) of vertigo & nausea with no auditory signs or symptoms.
- Symptoms peak in the first day and begin the improve within a few days yet take weeks to months for full recovery.
- The sense of self-movement is present with the eyes closed and is made worse by head movement and/or lying down, and is reduced by keeping the head still and sitting upright. Associated autonomic symptoms of nausea and vomiting are prominent.
- Test for hypofunction of the affected side- +ve head impulse test with visual suppression of nystagmus.
- A recent trial suggested that early steroids may be beneficial two encouraging therapeutic trials of steroid therapy in patients presenting with acute vestibular neuritis. The latter study found that corticosteroids, but not antiviral therapy , significantly reduced the degree of vestibular hypofunction, as measured by the VOR response to caloric irrigation, at 12 months. The effect of treatment on symptoms, however, was not assessed. Thus current knowledge is that initiating steroids within 3 days of vertigo onset (100 mg methylprednisolone reducing course over 3 weeks) appears to improve the long term outcome regarding peripheral vestibular function. As this is not available in Australia Therapeutic guidelines recommends prednis(ol)one 1 mg/kg (up to 100 mg) orally, daily in the morning for 5 days, then taper dose over 15 days and cease. (Big doses!) .:

Acute labyrinthitis = vestibular neuronitis with additional auditory involvement.

***Migrainous vertigo*-** apparently this is the second most common cause of vertigo yet under recognised (agree!!) A diagnosis of exclusion with a spectrum of symptoms – +/- headaches or aura.

Editor: Peter Wyllie

Like migraines this may be recurrent with identifiable triggers. Normal neuro exam. Remember bleeds and vertebral artery dissection may cause headaches +/- neck pain.

Labyrinthine haemorrhage is a rare but increasingly documented cause of acute vertigo and deafness. This is particularly relevant in patients on NOACs, warfarin etc.

Central -cerebellar or brainstem strokes / TIAs, brain tumours, MS, vertebrobasilar migraine.

Cerebellar / Brainstem Infarction – Larger strokes are associated with brainstem symptoms such as diplopia, dysarthria, limb ataxia, dysphagia, weakness or numbness. However ~ 10% of pts present with isolated vertigo (medial PICA occlusion).

- Onset - Cerebellar stroke onset usually has a sudden onset (i.e. within seconds).
- Risks- embolic source in 25-40%. Note that with traditional risk factors may not help
    - ABCD2 score – far from perfect – one study of patients with dizziness found stroke frequency of 1% (5/512) with ABCD2 scores < 4 c/w if score 8.1% (32/395) had stroke
    - Preceding craniocervical trauma may be warning of potential vertebral artery dissection – yet note that ~1/2 have no identifiable history of trauma
- Gait changes – 70% unable to walk unaided yet 29% have min or moderate imbalance with ambulation
- Other associated symptoms
    - May have proceeding symptoms (vertigo) may be seen in ~ 60% of patients with brainstem CVAs
    - occipital headache (+/- neck pain) in patients with ischaemic cerebellar strokes in addition to those strokes associated with vertebral artery dissection- normally sudden, severe and sustained. Headaches also seen with vestibular migraines.
    - hearing loss – sudden complete hearing loss is almost always peripheral in aetiology and is caused by occlusion of the internal auditory artery (IAA) a branch of the ant inf cerebellar artery.
    - Disproportionate vomiting or gait unsteadiness is thought to be suggestive of a central aetiology esp infarcts of the superior cerebellar artery.
- Other associated signs:
    - As discussed last week vertigo / dizziness with other cranial nerve, long tract, brain stem signs are suggestive of a brainstem / cerebellar infarction.
    - As also discussed, other confirmatory test for central causes from the HINTs exam include a positive test of skew, a negative head impulse test and direction changing or gaze evoked nystagmus. No visual suppression of nystagmus and pursuit / saccadic pursuit problems are also suggestive of a central aetiology
    - Purely horizontal, rotational, vertical nystagmus is suggestive of a central cause
    - Gait unsteadiness or truncal ataxia are both significant indicators of a central aetiology
    - However studies have found that although the presence of neurological symptoms or signs is predictive of a central cause, the absence is a relatively poor predictor of a peripheral cause.
    - Not counting oculomotor findings or severe gait or truncal ataxia, the absence of neuro signs excludes only 1 of 5 strokes in AVS.

- TIAs – may be suggested by frequent brief vertigo attacks lasting minutes rather than hours

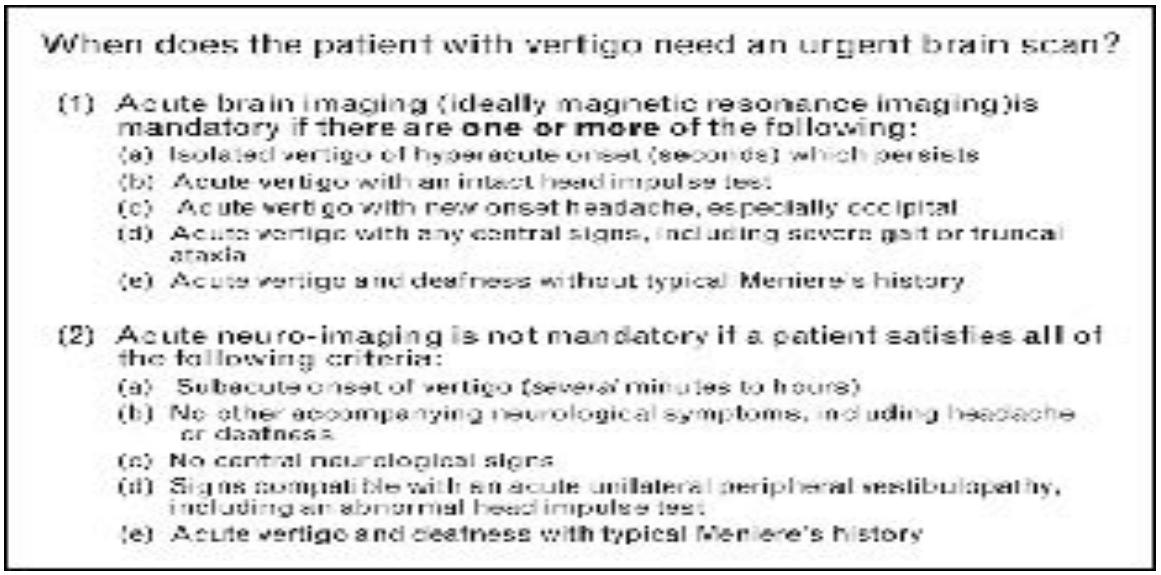
Other causes to consider:

- **Posterior Fossa lesions**- may have only vague imbalance and some positional vertigo with no other clinical abnormalities.
- **Hydrocephalus** – can present with imbalance rather than incontinence and memory loss.
- **Bilateral Vestibulopathy** – most commonly seen as consequence of aminoglycoside (gentamicin) toxicity. – this causes ataxia and oscillopsia (perception that the visual fields jump or oscillate) - +ve head impulse test in all directions. Gait abnormalities may only be exposed on soft surfaces esp when visual prompts are lost ie eyes closed . Note that gentamicin does not cause deafness or tinnitus.
- **Bacterial labyrinthitis**
- **Wernicke syndrome**
- **Brainstem encephalitis**
- **Hypertensive encephalopathy**
- **Drug intoxication (anticonvulsants , ketamine, alcohol, carbon monoxide)**
- **CNS inflammation / meningitis / Ramsay Hunt**

PUTTING IT TOGETHER

Editor: Peter Wyllie

Patients suspected of having a central cause for vertigo should be investigated & admitted to hospital with the direction of investigation determined by the provisional and differential diagnoses.
Below is one author's suggestions for initial imaging


When does the patient with vertigo need an urgent brain scan?

(1) Acute brain imaging (ideally magnetic resonance imaging)is mandatory if there are **one or more** of the following:
- (a) Isolated vertigo of hyperacute onset (seconds) which persists
- (b) Acute vertigo with an intact head impulse test
- (c) Acute vertigo with new onset headache, especially occipital
- (d) Acute vertigo with any central signs, including severe gait or truncal ataxia
- (e) Acute vertigo and deafness without typical Meniere's history

(2) Acute neuro-imaging is not mandatory if a patient satisfies **all** of the following criteria:
- (a) Subacute onset of vertigo (several minutes to hours)
- (b) No other accompanying neurological symptoms, including headache or deafness
- (c) No central neurological signs
- (d) Signs compatible with an acute unilateral peripheral vestibulopathy, including an abnormal head impulse test
- (e) Acute vertigo and deafness with typical Meniere's history

Couple of points on the imaging modality:

- Note that CT is a insensitive for the for acute stroke (~ 16%) – especially in the posterior fossa. Consider if bleeding or tumour is a significant possibility – if negative with these patients + ongoing concern or if acute ischaemia is a potential cause then MRI if the optimal choice for imaging .
- The gold standard is Diffusion- weighted Imaging (DWI) MRI
- However even early MRIs have their limitation – one study found a false negative rate of ~ 12% for brainstem / cerebellar pathology when performed < 48 hrs post event
    - This was one finding of the Kattah HINTS article referenced below and discussed last week, where they found that when used in patients with acute vestibular syndrome with at least one stroke risk factor, the 3 stage HINTs test was more sensitive than early MRI

Refs

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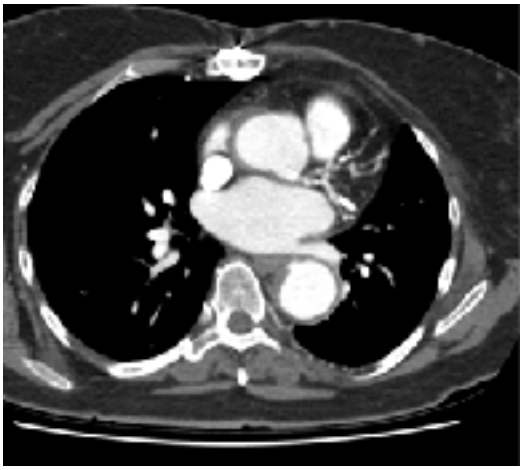
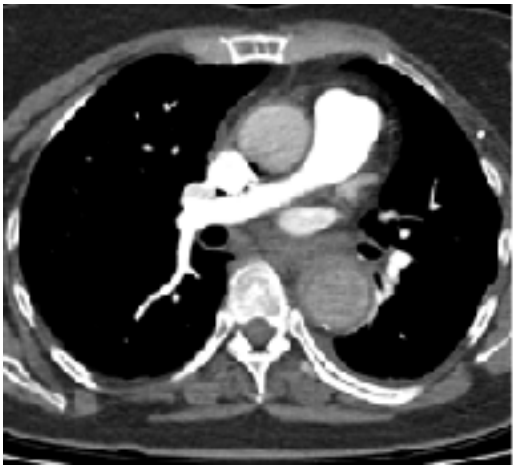
NEXT WEEK'S CASE

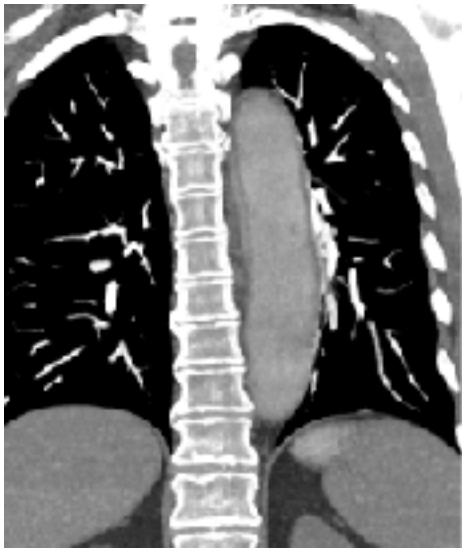
A break from the neuro stuff for a week

Editor: Peter Wyllie

A 66yo lady with a Hx of HT'n and previous type A aortic dissection represents with left sided posterior chest pain. No BP differences – no regurg – normal exam. The aortogram (A) is reported as normal and CTPA (B)+ (C) is performed

A 

B 

C 

What is going on?

JOKE / QUOTE OF THE WEEK

Please forward any funny and litigious quotes you may hear on the floor (happy to publish names if you want)

THE WEEK AHEAD

Tuesdays - 12:00 – 13:45 Intern teaching -Thomas & Rachel Moore
Wednesday 0800-0900 Critical Care Journal Club. ICU Conf Room / 12.00-1.15 Resident MO in Thomas & Rachel Moore
Thursday 0730-0800 Trauma Audit. Education Centre / 0800-0830 MET Review Education centre / 1300-1400 Medical Grand Rounds. Auditorium.

Editor: Peter Wyllie