

SHAKING SPELLS, BLACKOUTS AND FACIAL NUMBNESS (slide CC10-1)

CASE 10: Answers:

1. Neurologic causes account for only about 10% of all cases of syncope. *By far the most common causes of syncope are cardiovascular or vasogenic in origin.* These include, from most common to least common, "vasovagal" episodes (the common "faint" in which intense emotional stress or physical pain produces increased vagal tone resulting in transient hypotension and bradycardia), postural hypotension, hypovolemia, cardiac arrhythmias, aortic stenosis, and various other less common causes. Hypoglycemia or drug intoxication should always be considered as well.

Neurologic causes of transient loss of consciousness include seizures, and transient ischemic attacks "TIAs." In seizures, involuntary movements may not always be a prominent feature, making them difficult to distinguish from other causes of transient loss of consciousness. TIAs are thought to be embolic events which rapidly reverse, leaving no lasting neurologic deficits. They are usually warning signs that precede strokes and should, therefore, be taken quite seriously. TIAs involving the cerebral hemispheres can lead to compromised consciousness, usually accompanied by focal neurologic deficits corresponding to the territory of the occluded artery involved. Rarely, TIAs occur in the vertebrobasilar circulation leading to transient ischemia of the brainstem reticular activating system and loss of consciousness. Finally, in the appropriate setting and in the absence of any other cause, "hysterical" fainting should be considered, in which the fainting behavior occurs in the absence of other physiologic changes.

The neurologic exam of our patient suggests the possibility of an intracranial lesion that could produce seizures and the other deficits described (see below). However, our patient gives a history of occasional palpitations in the past and chest tightness. Although her story is somewhat atypical, and in fact, much of the history is vague and poorly remembered, a cardiac cause must be ruled out despite her young age. Patients with unusual constellations of symptoms present one of the most difficult diagnostic dilemmas in clinical practice: Does the patient have a *psychiatric disorder* or an *incredibly rare disease*?

2. A. Episodes of **generalized shaking and loss of consciousness** suggests seizures. In a young person these could be either idiopathic, due to prior head trauma or an intracranial lesion. The history is inconsistent but the fact that in her most recent episode she recalls shaking and walking around before losing consciousness suggests the possibility of a more focal lesion producing focal seizures with secondary generalization.

The **facial sensory loss** corresponds to the third division of the left trigeminal nerve (CN V₃). This deficit is highly suggestive of a lesion near the foramen ovale where CN V₃ separates from the other divisions of the trigeminal nerve and exits the cranium.

Headache has been discussed previously (see Lab 3 Clinical Case), and, among other things, can be caused by muscle tension, visual disturbance, migraines, or intracranial mass lesion.

Double vision on left end gaze in the absence of any other findings suggests a mild dysfunction of the peripheral portion of CN VI. The course of this nerve is long as it ascends from the pontomedullary junction to exit at the inferior orbital fissure and it is thus highly susceptible to compression by intracranial mass lesions. Note that a central lesion of CN VI would produce additional deficits such as difficulty adducting the contralateral eye. A lesion involving the right CN III could also produce diplopia on left end gaze due to impaired adduction of the right eye. However, in this case the right eye should also have impaired vertical gaze and possibly a dilated pupil.

Unsteady gait has also been discussed previously (Lab 8 Clinical Case). In our patient the finding is subtle, elicited only by tandem and heel walking, but this *should not be present* in an otherwise healthy 24 year old. Among the many possible causes of unsteady gait, the most

pertinent in this case would be mild impingement on the corticospinal tract, spinocerebellar tract, pontine nuclei or vestibular nerves by an intracranial mass lesion.

B. Sensory loss in the CN V₃ distribution suggests a lesion near the foramen ovale on the left side. If the lesion is intracranial it could impinge on the cerebral cortex producing seizures, especially since the foramen ovale lies in the vicinity of the temporal lobe, a particularly epileptogenic area. A mass lesion at the base of the brain, in the middle cranial fossa near the foramen ovale could easily produce diplopia and unsteady gait as well, as described above. The two most likely candidates suggested for this mass lesion were meningioma or schwannoma of the trigeminal nerve (trigeminal neuroma). An large aneurysm was also possible.

MRI:

The above clinical findings were all "soft," i.e. could easily be fabricated in the absence of disease. However, due to the possibility of an intracranial mass lesion at the base of the brain, the intern on the cardiovascular service ordered an MRI scan. Recall that MRI is better than CT for a suspected lesion near the base of the brain where shadowing artifact can be a problem. The top two horizontal images (see slide) demonstrate the presence of a large enhancing mass in the left middle cranial fossa, indenting the medial aspect of the left temporal lobe. In coronal images (bottom right) the mass can be seen to extend through the floor of the cranial vault in the region of the foramen ovale. Note that it also lies adjacent to the cavernous sinus where CN VI runs. Subsequently, a CT scan with "bone windows" (see Introduction to Clinical Material p. 11) was done as well. The lower left image shows such a horizontal image through the base of the skull demonstrating a normal right foramen ovale, and a large ovoid erosion through the base of the skull in the region of the left foramen ovale.

Clinical Course:

The patient ruled out for any cardiac problems during her first day in the hospital and on the same day the above MRI was obtained and a neurosurgical consult was called. She was started on phenytoin to prevent further seizures, surgery was scheduled for a week later and she was sent home in the interim.

During a ten hour operation, the tumor was removed completely. It was found to arise from CN V₃ and was easily demarcated from adjacent structures. Pathological analysis revealed a trigeminal neuroma. This is a rare tumor, but is the second most common type of neuroma of the cranial nerves, after CN VIII (see Lab 4 Clinical Case-acoustic neuroma). She made an excellent recovery, with no deficits except for continued numbness of the left jaw.