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Principle of the head impulse (thrust) test or Halmagyi head thrust test (HHTT)

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The head impulse or head thrust test was first described by Halmagyi and Curthoys in 1988.[1] It has acquired an increasingly important place in the clinical examination of the vertigo patient. It detects severe unilateral loss of semicircular canal (SCC) function clinically; it is more sensitive and specific than the traditional Romberg and similar tests; and it is particularly important in the emergency unit, where it can distinguish between vestibular neuritis and cerebellar infarction, which can both generate similar symptoms suggesting an initial attack of severe acute vertigo. The result of the head thrust test is definitely normal in a patient with a cerebellar infarction but abnormal in a patient with vestibular neuritis.

General physiological background: the push-pull principle of the vestibulo-ocular reflex

The peripheral vestibular sensors transmit motion to the brain through frequency encoding. Like FM radios, our brains continuously receive 'frequency modulated' signals. A normal resting discharge rate of approximately 90 spikes per second is modulated such that any increase in this rate corresponds to excitation and a decrease to inhibition. The polarisation of the hair cells in the horizontal semi-circular canal is such that deflection of the stereocilia in the cupula towards the kinocilium (ampullo- or utriculopetal) results in hair cell depolarisation and the activity of the primary afferent neurons therefore increases. Deflection of the stereocilia away from the kinocilium (ampullo- or utriculofugal) results in hair cell hyperpolarisation and decreased primary afferent neuron activity.

The orientation of the left and right semi-circular canals in the head is such that any movement always induces an antagonistic response in both canals. Horizontal head movements in the yaw plane are an example. During rightward head rotation, the endolymph in the lateral semi-circular canals on both sides lags behind, bending the cupula of the right SCC towards the vestibulum (ampullo- or utriculopetal) and simultaneously deflecting the cupula of the left SCC away from the vestibulum (ampullo- or utriculofugal). A key difference is the polarisation of the hair cells. Indeed, since the the hair cells in the right and left canals are implanted in opposing directions (in a mirror image fashion), the deflection on the "leading" right side induces the movement of the stereocilia towards the kinocilium, whereas the movement of the stereocilia is away from the kinocilium in the opposing, "following" ear. As a result of this "push-pull principle", the activity of right lateral SCC primary afferent neurons increases, and, at the same time, the activity of left lateral SCC primary neurons decreases with respect to the normal resting discharge rate.

The activity of the lateral SCC primary afferent neurons is modulated by horizontal head rotation. The firing rate increases in the leading ear (the ear towards the movement is directed) and decreases in the following ear. This is the push-pull principle of the VOR.

The right medial vestibular nucleus in the brainstem receives an increased input from the right lateral SCC primary neurons (no crossing). This excites the activity of type I secondary vestibular neurons. These excitatory neurons drive the leftward compensatory eye movements of the VOR, to ensure gaze stabilisation. However, commissural disinhibition from the left lateral SCC primary neurons also contributes to the excitation of the type 1 neurons. Both excitation of the right SCC and disinhibition of the left SCC are therefore needed for an optimal VOR.

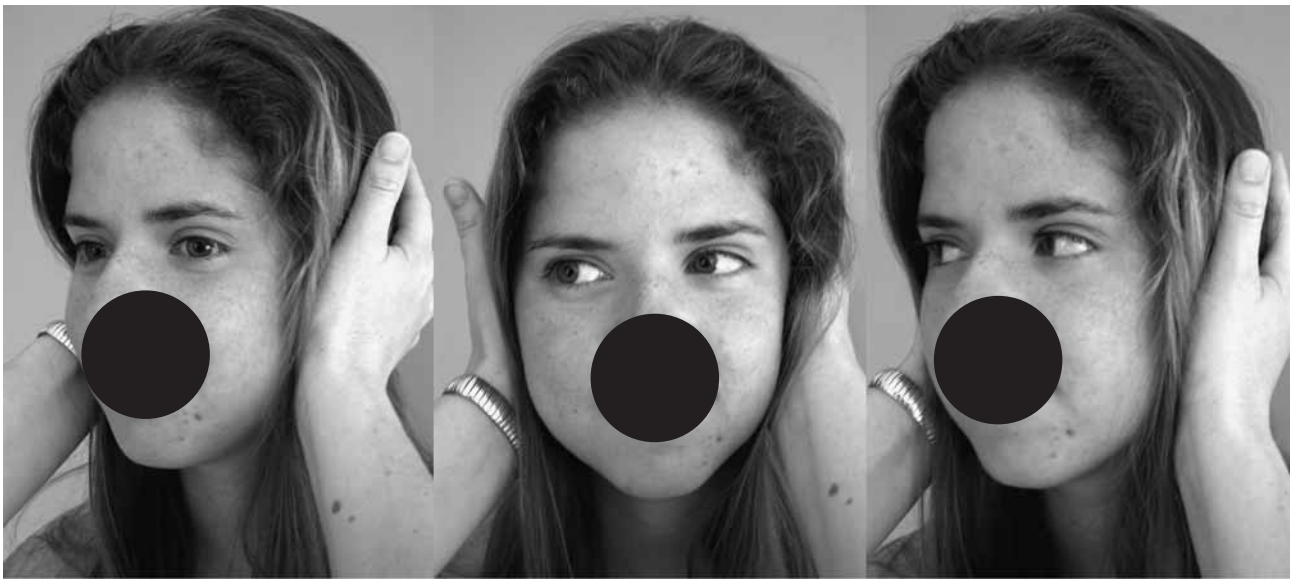

Figure 1
Left: the clinician holds the head of the subject firmly and turns it briskly to the left. Centre: After the rotation to the left, the subject maintains the gaze on the distant fixation point, *i.e.*, the eyes stay stable in space.
Right: After abrupt rotation to her right, the subject moves her eyes with her head and loses the target. A refixation is necessary to fixate the point again (not shown). The side towards the gaze fixation is lost is the deficit side, *i.e.*, the patient's right.

Head thrust test

The head thrust test is primarily based on the fact that inhibition of primary and secondary vestibular neurons cannot produce fewer than 0 spikes per second. Excitation can drive the discharge rate from 90 to 300 or more spikes per second. So when the healthy side is excited for a high acceleration head movement, the healthy side will generate the larger part of the VOR, since the disinhibition of the ipsilateral type-1 neurons by the contralateral SCC contributes relatively little to the VOR. Passive head impulses or thrusts should be typically rapid but with a small amplitude (± 20 degrees). Their velocity ranges up to 180 deg/s but high acceleration is particularly important (3000-4000 deg/s2). They have to be unpredictable since the patient very quickly learns to anticipate and this reduces the sensitivity of the test to a considerable extent. The examiner should therefore thrust the head of the patient firmly from left to right at random and from right to left a little later, *i.e.*, not immediately. The starting position should be such that the patient's head is turned slightly past the midline, and it should then be thrust just past the midline to the opposite side. Here, amplitude is low but acceleration can be considerable. This test demands some training, particularly with respect to the positioning of the hands on the side of the head and holding the head firmly. The instruction to the patient is to fix on a point in the distance behind the examiner.

When the subject's head is turned to the side of the lesion, the VOR is deficient and the eyes will move with the head so that they no longer fix on the point in the distance. The patient therefore needs a refixation saccade just after the thrust. When the head impulse is in the direction of the healthy side, the VOR will maintain the target on the fovea and no refixation saccade will be needed.

The head-thrust test is positive for the side that causes the refixation saccade upon thrust (Figure 1)

It is not only the lateral SCC that can be examined – this is, in a sense, a clinical approximation of the caloric test – but also the other SCC. Here, the patient's head must be thrust in the RALP or LARP planes (Right Anterior – Left Posterior or Left Anterior – Right Posterior SCC).[2]

References

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