The Active Head-Impulse Test in Unilateral Peripheral Vestibulopathy

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Background: The head-impulse test, which is sensitive and specific for detecting severe unilateral peripheral vestibulopathy, is an accepted part of the neurological examination, especially in patients with vertigo and balance disorders.

Objective: To discover if the head-impulse test is just as useful diagnostically when patients are asked to rotate their own heads, the active head-impulse test, rather than when the clinician does so as in the standard passive head-impulse test.

Methods: Clinical observation of compensatory saccades and search coil measurement of compensatory eye rotations, during active and passive horizontal head-impulses in 6 patients with total unilateral vestibular deafferentation.

Results: Clinical observation showed the expected compensatory saccades with rotations toward the side with the lesion with passive head-impulses but not with active head-impulses. Search coil recordings revealed 2 reasons for this. With active head-impulses not only was vestibulo-ocular reflex gain higher, but compensatory saccade latency was shorter resulting in an occult saccade that occurred during, rather than after, head rotation.

Conclusions: Passive head-impulses are necessary to detect a severe unilateral peripheral vestibulopathy; active head-impulses will produce a false-negative result.

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was measured using a search coil mounted in a mouth guard composed of dental impression material (President Putty; Colte Imports, Mahwah, NJ) in a disposable dental impression tray. Full details of the recording methods have been reported.6,7

We studied the VOR with active and passive head-impulses in 6 subjects with total unilateral vestibular deafferentation (5 men, 1 woman), aged 32 to 71 years, more than 2 years after vestibular schwannoma (acoustic neuroma) surgery. The head-impulse is a low-amplitude (15°-20°), high-acceleration (4000°/s²-6000°/s²) head rotation, in this case horizontal, with a peak velocity of 150°/s to 350°/s. Details of the head-impulse test have been previously published.6,7 A passive head-impulse is delivered by an investigator who stands behind the patient and firmly holds the patient’s head; at unpredictable times the investigator rapidly turns the patient’s head to the left or right by 15° to 20° while the patient tries to keep staring at the target. The target is an earth-fixed laser dot located 94 cm straight ahead of the left eye. Active head-impulses were made by the patients themselves: each was asked to make rapid horizontal head rotations with velocities that fell within a template envelope representing the upper and lower 95% confidence intervals of the velocity of all passive head-impulses. The template was shown on a screen throughout the experiment. As the patient made an active head-impulse its velocity profile was instantaneously displayed on the head-velocity template (Figure 1). With this feedback all patients learned to make active head-impulses that fell within the passive head-impulse velocity envelope. Only those head velocities that fell within the envelope were analyzed. The peak velocities and durations of active and passive head-impulses were therefore matched. Written informed consent was obtained from subjects prior to testing, according to the Declaration of Helsinki. The experimental protocol was approved by the human ethics committee of the Central Sydney Area Health Service, Sydney, Australia.

The VOR with active and passive head-impulses was analyzed from the onset of head rotation to final gaze fixation—about 500 milliseconds. The gain of the VOR was calculated by finding the gradient of a line fitted to the mean eye velocity plotted as a function of head velocity in a time frame window from 30 to 75 milliseconds of head rotation onset. Full details of the analysis methods have been previously published.8 Mean ±2-tailed 95% confidence intervals were computed for head and eye velocity. A 2-tailed t test (P < 0.05) was performed to determine whether a statistically significant difference existed between head velocities and accelerations and VOR gains with active vs passive head-impulses.

All 6 patients learned to make active head-impulses that matched the passive head-impulses within about 20 attempts. While it was easy to see the compensatory saccade with passive ipsilesional head-impulses—the clinical sign of canal paresis—we all had difficulty in doing so when the patient made active head-impulses because compensatory eye movements appeared smooth during most active head-impulses.

Using scleral search coil recordings, it was possible to make a valid comparison of the VOR in response to active vs passive head-impulses because the head-velocity profiles in the first 75 milliseconds were the same (Figure 2). The results were that while ipsilesional VOR gain was abnormally low with both active and passive head-impulses, with active head-impulses (mean ± SD, 0.38±0.1), it was almost twice that seen with passive head-impulses (0.18±0.02) (Figure 2). In contrast, contralateral VOR gain was only slightly higher with active head-impulses (0.78±0.08) than with passive head-impulses (0.68±0.06) (Figure 3).

Furthermore, the latency of the first saccade, which corrects the gaze position error accumulated during the ipsilesional head-impulse and forms the clinical sign of canal paresis,1 was shorter with active than with passive ipsilesional head-impulses. With passive ipsilesional head-impulses, the mean latency of the compensatory saccade was 219±102 milliseconds (mean ± SD) so that it invariably started only after the head had stopped rotating (Figure 2). In contrast with active ipsilesional head-impulses, the mean latency of the compensatory saccade was only 87±11 milliseconds, so that typically it would begin while the head was still rotating. Furthermore, since VOR gain was higher with active than with passive ipsilesional head-impulses, the accrued gaze error and therefore the mean amplitude of the compensatory saccade was lower with active (approximately 4º) than with passive head-impulses (approximately 12º). Consequently, with active ipsilesional head-impulses, the eye position response appeared to be almost perfectly compensatory for head rotation (Figure 2)—creating the misleading impression of a near-normal VOR.

The technique and interpretation of the passive head-impulse test is generally straightforward as long as one recalls that SCC afferent neurons have a tonic firing rate at rest—about 40 spikes per second in the squirrel monkey.9 Rotations away from an SCC (ie, in the off-direction) decrease the firing rate of the neuron whereas rotations toward an SCC (in the on-direction) increase its firing rate. With high-acceleration rotations, it is much
easier to saturate the firing rate of a vestibular neuron in the off-direction, that is, to silence it, than to saturate its firing rate in the on-direction. In a patient with only one functioning labyrinth, rapid head rotations toward the side with the lesion will silence SCC afferents on the intact side and saturate the gain (eye velocity divided by head velocity) of the VOR. Consider a patient with left vestibular neuritis: if the patient fixates a distant earth-fixed target while the clinician rapidly rotates the patient's head to the left, the passive head-impulse test, the patient will make 1 or 2 observable, compensatory, that is, rightward, saccades just after the head-impulse is over.

Figure 2. Passive (A) and active (B) head-impulse graphs. Typical active and passive, ipsilesional and contralesional head-impulses from a subject with unilateral vestibular deafferentation from just before the start of head rotation until just after the target is acquired. The start of head rotation is indicated by arrow 1, the start of the compensatory saccade by arrow 2, and the end of the compensatory saccade by arrow 3. With a passive ipsilesional head-impulse, a large gaze error is generated and it persists until after the end of head rotation, when it is corrected by a single saccade. With an active ipsilesional head-impulse, a compensatory saccade occurs during the head rotation so that the resulting gaze error is small. The higher initial vestibulo-ocular reflex gain with active head-impulses plus an early compensatory saccade result in an eye rotation response that is clinically indistinguishable from the head rotation stimulus. Contralesional head-impulses, both passive and active generate a vestibulo-ocular reflex within the normal range. All angles are shown in rotation vectors (RVs). Multiplying the RV value by 100 gives the approximate value in degrees. Hence, 0.1 RV is about 10°. H indicates the head position and velocity data; E, the eye position and velocity data.

Figure 3. The mean±2-tailed 95% confidence intervals of head and eye velocities in the initial 75 milliseconds of active and passive ipsilesional (A) and contralesional (B) head-impulses from all subjects with unilateral vestibular deafferentation. Active and passive head-impulse velocity ($H_{\text{Active and Passive}}$) is shown as dotted black lines, eye velocity ($E_{\text{Active}}$) in response to active head-impulses as solid black lines, and eye velocity ($E_{\text{Passive}}$) in response to passive head-impulses as solid gray lines. Eye velocity has been inverted to aid comparison with head velocity. No statistically significant difference exists between the active and passive head-impulse velocities in the initial 75 milliseconds of ipsilesional or contralesional head rotations. A, Significant differences exist between the eye velocity responses to passive vs active ipsilesional head-impulses: the eye velocity response is higher (initial gain) with active than with passive head-impulses. Consequently, with active head-impulses, compensatory eye velocity at first closely follows the head velocity profile and then near peak head acceleration it diverges from head velocity. In contrast with passive ipsilesional head-impulses the initial compensatory eye response has lower magnitude and increases monotonically after onset. B, With contralesional head-impulses the magnitude of the eye velocity response to both active and passive impulses is higher than with ipsilesional head rotations and increases monotonically after onset. With active head-impulses the eye and head velocities are nearly matched. C, The mean±2-tailed 95% confidence intervals of horizontal eye velocity as a function of head velocity with active and passive ipsilesional and contralesional head-impulses. The statistically significant differences between active and passive impulses, in both directions of head rotation, are apparent even at low head velocities. Active head-impulses, in both directions, are associated with higher initial gains than passive head-impulses. The gains with contralesional head-impulses are higher than with ipsilesional rotations.
This is the head-impulse sign and it indicates that the gain of the horizontal VOR, which is generated from inhibition of the sole functioning, right, lateral SCC, rather than from excitation of the nonfunctioning left lateral SCC, is severely defective. In other words, the VOR gain is much less than 1.0. In contrast, when the clinician rotates the patient's head to the right, there will be no compensatory saccades indicating that the gain of the VOR, generated by excitation from the right lateral SCC plus any inhibition from the left lateral SCC, is close to 1.0. Not only can the compensatory saccades be observed clinically, the VOR deficit can be measured ocularographically2-7,10-12 not only from lateral but also from individual vertical SCCs.5,11

The present results show that active head-impulses, unlike passive head-impulses, are not an effective clinical way to show severe unilateral loss of vestibular function. They confirm that in unilateral vestibular loss ipsilesional VOR gain is abnormally low with both active8,9 and passive head-impulses5,7,12; however, they also show that the VOR gain is significantly higher with active than with passive head-impulses. Although ipsilesional VOR gain is still low with active head-impulses resulting in a measurable, and potentially observable, gaze-error correcting compensatory saccade, the saccade latency is shorter with active head-impulses than with passive head-impulses so that the saccade is occult in that it occurs during, rather than after, the head-impulse. Because the velocity profile of the compensatory saccade resembles that of the head-impulse itself, it is easy clinically to mistake the compensatory saccade for a normal compensatory VOR and consider the findings of the head-impulse test to be normal. Consequently, for the diagnosis of a unilateral peripheral vestibulopathy, active impulses have 2 disadvantages compared with passive head-impulses: they not only produce a higher VOR gain, but they also generate an earlier compensatory saccade that can mimic a normal VOR. Why is VOR gain higher and compensatory saccade latency shorter with active than with passive head-impulses? There is no theoretical reason why the VOR gain should be higher and there are no animal data because it has not proven possible to train an animal to make active head-impulses. Single neuron data from vestibular nucleus neurons in alert behaving monkeys show reduced activation with active vs passive head rotations.15 However, in these experiments the animal's goal was reflexion rather than fixation, a task that would benefit from a lower VOR gain. Active head rotations are normally made to reflex rather than to fix gaze and that is presumably why for human subjects it feels unnatural to rotate the head rapidly while trying to look straight ahead. Saccadic latency might be shorter with active head-impulses than with passive head-impulses because of a learning effect. Our subjects were trained to match the velocity profile of their own passive head-impulses with an active head rotation. Therefore, our subjects may have learned to predict their own gaze error and to make compensatory express saccades16 almost in time with head rotation. It is clear, therefore, that passive head-impulses rather than active head-impulses are needed to demonstrate a unilateral vestibular deficit.