Abstract

Rotatory chair testing of the horizontal semicircular canals is an important but specialized tool in vestibular examinations. This review summarizes stimulus protocols, basic physiology and the current knowledge. It will be shown that the value of rotatory testing in clinical practice is part of a vestibular test battery in high end vestibular and balance centers but not in a general clinical practice.

Introduction

Different vestibular tests are routinely used to identify vestibular failure of the horizontal vestibulo-ocular-reflex (VOR): the bithermal caloric irrigation (CI) [1] and the new video-head-impulse test (vHIT) [2]. Both methods test the dynamic responses of the horizontal angular VOR and could identify a unilateral vestibular failure (UVF). However, the vHIT does not replace CI as both tests are different in identifying a unilateral vestibular failure [3-6]. A third technique was used to test the horizontal VOR for years and is still used in specialized vertigo clinics, but the sensitivity to identify unilateral vestibular failure is low and depends on the test profile, the disease itself and the stage of the disease [7-10]. This paper reviews current knowledge of rotatory testing of the horizontal VOR and the application in a clinical work up. Passive rotatory testing does not replace the bithermal caloric irrigation or the vHIT, but adds further information for diagnostic purpose. This review does not include rotatory testing around other axis as sagittal or inter-aural, as these are generally only used in scientific setups.

Equipment

Today motor driven rotatory chairs (Figure 1A) are widely commercially available and able to apply defined and reproducible rotational vestibular stimuli (defined parameters: e.g. velocity, acceleration, frequency) around an earth vertical axis located in the center in between the two labyrinths. During the test, the patient’s head is stabilized by a head holding device, while tilted by 30 degrees forward to optimal align the horizontal semicircular canals (SCC) with the horizontal space plane (Figure 1B). To measure the VOR, the eye movements are recorded by means of video-oculography (VOG) or electronystagmography (ENG). It is important to enable fixation by a special VOG-mask, or eye closure with ENG, to measure the VOR in total darkness. Other factors effecting rotatory testing are stress, fatigue, state of mental alertness and habituation [11].

Physiological basics of the VOR

The VOR drives the eyes in the opposite direction of the head movement but in the same plane. If the eye and head are exactly moving in opposite direction of equal velocity the VOR-gain (ratio of eye to head velocity) is one. The head acceleration is detected by SCCs in the cupula. The information is conveyed to the vestibular nuclei by the vestibular nerve, which has a steady firing rate of about 100 spikes/s if no motion is applied [12]. Remember the SCCs are arranged in push-pull pairs, e.g. rotation to the right increases the firing rate of the right while it decreases the of the left SCC. If the stimulus is fast enough one SCC goes into an inhibitory cut off, while the other SCC further increases firing. This principal is very important to understand high frequency step stimuli, e.g. the vHIT, which test only one SCC at a time. In general, stimuli used in rotatory testing are too slow to cause this inhibitory cut off as a stimulus of more than 200°/s and an acceleration of 2000-5000°/s² are required.
Pendulum model

The physiological properties of the SCC are best described by the pendulum model, which describes the cupula displacement during stimulation. The stimulus is head acceleration (a force) which is opposed by three restraining forces: an elastic force, a force due to the viscosity of the endolymph-cupula system and an inertial force. For most natural movements, the elastic and inertial forces are negligible. Accordingly, the viscous force is counteracting the force applied to the head [11, 13].

Based on this model the results of cupula deviation caused by rotatory chair stimuli with slow stimulus velocities could be predicted as shown in a simulation (Figure 2). A constant acceleration stimulus causes an increasing and then saturating response (Figure 2A), a step-response an initial upraise followed by an exponential decay (Figure 2B) and a sinusoidal stimulus a response in phase with the stimulus velocity in the frequency range from 0.01 to 4Hz [11] (Figure 2C). During eye movement recordings of such rotatory stimuli very similar responses of the slow phase velocities are observed as predicted in the pendulum model.

Short-term adaptation

There is one phenomenon which could not be explained by the pendulum model that is the short-term adaptation. This adaptation should be distinguished from long-term adaptation to visual stimuli, which e.g. changes the gain of the VOR to adjust to the magnification of new goggles. Short-term adaptation occurs during long lasting constant velocity stimulations and can be measured in the step paradigms and has a time constant of about 80s. During a stimulus stop, after a constant rotation lasting minutes, an adaptive process decrease the output response of the cupula and causes a drift of the eyes in the opposite direction before it comes back to zero velocity (Figure 3). The adaptation is already observed in the vestibular afferents, especially in the irregular ones [14, 15]. The source of this process is located in the periphery, brainstem and cerebellum [16]. It is hypothesized that it is useful to maintain the D.C. balance of the right and left SCCs [13]. The short term adaptive mechanism is especially prominent in infants and in cerebellar lesion and also responsible for change in quick phase direction in period alternating nystagmus [16].

Velocity storage

The signals of the cupula are further processed in the brainstem network between right and left vestibular nuclei. There is dissociation in a direct and an indirect pathway. The direct pathway is the three-neuron arc, known as the VOR, and the indirect pathway is known as the ‘velocity storage’. Latter causes the postrotatory vestibular time constant to increase from the cupula time constant of about 4s up to 20s (Figure 4). Functions of this ‘velocity storage’ are to better transduce the low frequency components of the VOR (<0.03Hz) [17], to reorient the eye velocity in direction of the gravito-inertial acceleration and to differentiate linear acceleration from gravity [16]. This function is implemented in the cross commissural pathways between the right and left sided vestibular nuclei and under control of the cerebellum, e.g. the nodulus and uvula.

Rotatory chair: step test

Step-stimuli have a high acceleration and a broad spectrum of different frequencies and are known to be a suprathreshold stimulus for the horizontal SCC. To apply step stimuli there are technical limitations by the chair. It is technical easier to stop the chair with a break to obtain high negative acceleration, than to use the motor to accelerate the
Quantification of the rotatory step-test

Quantification of the responses could be done by an exponential fit of the desaccaded slow phase velocity (SPV). K: SPV = offset SPV + K × ΔV × e^{-t/TC} sensitivity coefficient, ΔV: change in stimulus velocity, TC: time constant, t: time). This fit provides the offset SPV as a measurement of the short-term adaptation, the TC as an estimate of the pendulum model and a maximal SPV value. The long time constant of the pendulum model and short-term adaptation TC could be estimated from the maximal responses and the time of response reversal [20]. Alternatively, the duration of the decaying SPV could be directly read from the curves: the gain, the duration until zero is reached and the offset. To calculate side differences (SD) the following formula could be used: SD = right-left/right+left (right: rightward; left: leftward stimulation) for e.g. gain, TC and offset. Each laboratory should use its own normal values (e.g., mean ±SD; TC: 19±6s; cut-off SD of TC: 25%). The best parameter to find a side difference is maximal slow phase velocity in sinusoidal and step stimulation. Other parameters as the duration of the response to a step, the primary time constant of the exponential decay and the adaptation time constant identified only 1/3 of unilateral vestibular failure [8]. The faster the stimulus the higher the proportion of identified vestibular failure was, in a velocity range of 16 to 256 °/s.

Rotational intensity damping test (RIDT)

The step test is often performed together with a subliminal stimulus (Figure 2A) and then called the rotational intensity damping test (RIDT). This test uses a slow ramp of constant acceleration (e.g. 3°/s², for 30s) until 90°/s is reached. After e.g. 180s at constant velocity the chair is abruptly stopped in 0.3s, reaching a deceleration force of 270°/s² [18, 21]. The expected responses are shown in Figure 2A and 2B.

The advantage of this stimulus is fourfold: 1. slow acceleration is more comfortable for the patient and lead less to vertigo and vomiting to reach a steady state velocity; 2. a subliminal stimulus brings latent pathology to light; 3. this stimulus is less affected by any startle reaction compared to the stop as in the end of the RIDT; 4. the TC of the velocity storage could be determined without major short term adaptation. Normative values depend on the setting and have to be established in each lab.

VOR-tilt-suppression

There is another important test which is based on the step-test. Due to the high variability of the vestibular time constant even in healthy controls, this test uses an intra-individual comparison. The postrotatory response is used as described before. Additionally, the test is repeated and immediately at the time the chair stops, the patient’s head is tilted forward, which causes a decrease of the vestibular response, by addition of an otolith stimulus. The results of this test are compared to the normal postrotatory response [22-24]. This response is processed in the cerebellum, the uvula and nodulus. In case of a unilateral lesion of the uvula and nodulus the vestibular response is not affected by the head tilt (Figure 5B compare to A).

Rotatory chair test: pendular (sinusoidal) tests

One other important testing method is sinusoidal stimulation with the rotatory chair (Figure 2C). In general, the frequencies range from about 0.01 to 1.28 Hz with the peak velocity kept at a constant level of about 50-60°/s. The values at the high end are limited by capability of the rotatory chair. In comparison to vHIT and CI, the
sinusoidal stimulation tests the middle range of frequencies of the VOR. Pendular testing could be applied in different forms: e.g., at an isolated frequency or as mixed frequencies, known to “sum of sines” which test defined frequencies at the same time. Another well-known test is the sinusoidal harmonic acceleration test, which increases frequency step wise.

**Quantification of the rotatory pendular-tests**

The responses to a sinusoidal stimulus could be quantified with a simple sinus fit of the slow-phase velocity (SPV). \( SPV = \text{Maximal SPV} \times \sin(2 \pi f \times \text{Time} + \text{Phase}) \). Side differences can be calculated by the maximal SPV values \( SD = \text{right-left/ right+left} \) (right: rightward; left: leftward stimulation).

In our lab we find the following values in controls using a constant maximum speed of 62.8°/s as mean ± standard deviation: gain values in percentage, 0.025 Hz: 22±7; 0.05 Hz: 25±9; 0.1 Hz: 27±11; 0.2 Hz 27±11; phase lead values in degree 0.025 Hz: 14±10; 0.05 Hz: 11±8; 0.1 Hz: 7±6; 0.2 Hz 4±5.

From the phase lead, e.g. at 0.01 Hz stimulus frequency, one can calculate the TC of the velocity storage. Remember it is always useful to test several frequencies to assess the vestibular function.

**VOR-Cancellation**

In the same setting and with similar parameters used in rotatory testing, the VOR-cancellation is tested. The only difference is that the patient is fixating a target which is head stationary, moving at the same velocity in space as the chair. Under this circumstance the VOR which is always elicited, has to be canceled by a central signal of opposite direction. The VOR-response is reduced and the eyes do not move in the head anymore. The necessary signal is generated by the smooth pursuit system in the cortex, brainstem and cerebellum. Neurons important in VOR-suppression are located in the flocculus and paraflocculus of the cerebellum, where smooth pursuit signals and VOR-signals are available [25]. These signals are projected on the eye-head cells vestibular nuclei, which directly project to the motoneurons. Again, there might be direction specific deficits as the pursuit is processed direction specific in the right and left flocculus.

**Quantification of VOR-cancellation**

The VOR-cancellation is quantified similar to the pendular VOR and a ratio of the SPV during VOR-cancellation to SPV during the VOR is calculated. This ratio should be less than 50% in our lab in controls.

**Clinical Application**

**Findings in unilateral vestibular failure**

A typical example of a subacute unilateral vestibular failure is shown in Figures 6 and 7, which has a pathological vHIT after rightward rotation and a side difference in CI. The step-responses in our example (Figure 7A) show a decreased gain and a decreased TC on the side of the lesion (Figure 7B) and additional an offset shift caused by a spontaneous nystagmus in unilateral vestibular failure. These findings might be caused by three processes: spontaneous nystagmus which is superimposed, saturation of inhibition of the intact labyrinth which increase the gain asymmetry and an asymmetry loss of the velocity storage which decreases the time constant of the lesioned side [26].

In sinusoidal stimulation two observations are important and could be found in unilateral vestibular failure, a phase lead and a right-left asymmetry (Figure 7C). Greater than normal phase leads are observed in sinusoidal stimulation in the low frequency range (less than 0.1Hz) and decrease with increasing stimulus frequency. They are caused by the dysfunctional ‘velocity storage’. In contrast, right-left asymmetries increase with increasing stimulus velocities, and are caused by the spontaneous nystagmus and saturation of inhibition of the healthy side at higher frequencies [26].
Remember, due to central compensation the pathological phase lead and asymmetries disappear over time. However, a persistence of a velocity storage deficit could persist.

**Usefulness of the rotatory tests to identify a unilateral vestibular failure**

It is known that CI, rotatory chair tests and vHIT do not correlate well in unilateral vestibular failure [3,4,9,27]. And especially CI and vHIT could not substitute one another. Rotatory testing does not identify a unilateral vestibular failure with high sensitivity in chronic stage [8,28,29] and is not very useful in acute disease. In acute unilateral vestibular failure there is majorly a DC bias caused by the spontaneous nystagmus and an asymmetry between ampullofugal and ampullopetal stimulation of the contralateral healthy labyrinth [28]. In chronic unilateral vestibular failure of less than 50% side difference CI, rotational testing could identify only a third of the cases [8]. In other studies unilateral vestibular failure could be best predicted by CI in combination with rotatory testing [7,30] and better than with CI alone. Different pathophysiologic changes (vestibular neuritis, Menière’s disease, and viral labyrinthitis) could to some extend be dissociated using a logistic model and CI and rotatory testing [7]. In Menière’s disease heterogeneous pattern in CI and rotatory test were observed [31]. For unilateral vestibular failure in the sinusoidal test the best frequencies are 0.01, 0.1 and 0.05 Hz (in the order of diagnostic capacity in a sinusoidal harmonic acceleration test) [9].

It is important to note, that in some patients with unilateral vestibular failure an isolated high frequency deficit with normal CI and rotatory testing, but with a pathological vHIT could be observed and in others only a deficits in the low frequency range with a pathological SD in CI and normal vHIT and normal rotational testing [3,32-35].

**Examining compensation after unilateral vestibular failure**

Sinusoidal stimulation is used in general to test for compensation in vestibular failure [10,36]. The asymmetry between ampullofugal and ampullopetal stimulations decrease during vestibular compensation over time but does not disappear [37,38]. The asymmetries are more prominent during high intensity stimuli, as high acceleration small pulses of high frequency high-acceleration sinusoidal stimuli [38-40]. It was observed that compensated unilateral vestibular failure show increased phase lead and reduced gain at low frequencies below 0.1 Hz [41]. Those findings could last at least 6 month [42] up to 10 years [38]. In contrast, it was shown that gain asymmetry of rotatory testing contributed to the diagnosis of unilateral vestibular failure only in the first 50 days after symptom onset [43].

The visual consequences of the very low frequency VOR are minimal as these deficits could be compensated for by the visuomotor system and eye movements. The VOR changes high frequencies tested with the vHIT and medium frequencies, tested with the rotatory chair might be important to explain the symptoms of the patients with not compensated vestibulopathy.

**Findings in bilateral vestibular failure**

Figure 8 shows an example of bilateral vestibulopathy with a bilateral caloric unresponsiveness, a bilateral pathological vHIT and the results of rotatory testing. Rotatory step-testing shows no (Figure 8) or decreased postrotatory responses on both sides. This is caused by the bilateral missing input in the system which causes the ‘velocity storage’ to fail. Sinusoidal stimulation causes in this case nearly no response (Figure 8C). In general, a phase lead and gain decrease (Figure 9) is observed in sinusoidal rotatory test compared to controls.

**Usefulness of the rotatory tests to identify a bilateral vestibular failure**

Rotatory testing is ideal for examining patients with a bilateral vestibular failure, as the variance of the slow phase velocities is less compare to caloric irrigation. Therefore, the...
disease is identified earlier [11]. Furthermore, rotatory testing is not dependent on the anatomy of the external auditory canal, middle ear and temporal bone, which are important factor in CI. There could be nearly no response to CI but good responses in sinusoidal testing. To diagnose a bilateral vestibulopathy frequency dependent gain and phase cut-off values are necessary. In comparison to the vHIT, the rotational response in midrange frequency are often much better, especially if actively performed, as probably central compensating mechanism kick in to enhance the response [11].

Bilateral vestibulopathy could have deficits not only in the low, middle or high frequency range and show very heterogeneous test results of CI, rotatory tests and vHIT [44]. The definition of this disease was very unclear and different definition have been applied including CI and rotatory testing, e.g. [11,45]. A current problem is the variety of the lesion patterns observed in this disease. From recent and past literature, it is clear, that a CI alone could not diagnose a bilateral vestibular failure but additional tests are necessary (Class I to VI evidence [10]). In line is the most current definition of the bilateral vestibulopathy of the Barany Society which is not published yet. Rotatory testing is not included but vHIT and CI, which might cause missing of certain disease. To my opinion, rotatory test should be further included in the diagnostic criteria, because VOR in middle frequency range is still important for keeping balance and stabilizing the eyes. This is illustrated in Figure 9. Patients with bilateral vestibulopathy were tested with the rotatory chair with sinusoidal stimuli, step stimuli and with the vHIT and CI. Normal values of controls are indicated by red lines. In the different test patients are not in all tests pathologic, but could also have normal values.

Findings in central vestibular deficits

In central vestibular lesion the results are very heterogeneous, especially to due diverse patterns of nystagmus activation. A direct differentiation of peripheral and central lesion is often not possible by rotatory stimuli. Hence, CI and vHIT are important to dissociate both. There are some tests and signs which help to diagnose a central vestibular lesion. The increase in VOR-gain is such a sign and was observed in cerebellar disease [46], and in other central disorders as microangiopathy. Another parameter is the vestibular TC which could be enhanced in lesion of the cerebellum [24] and the VOR-tilt-suppression to identify uvula and nodulus lesions [16, 22,23].

VOR-cancellation is a useful test to detect central lesion. In general, lesions of the smooth pursuit eye movement system lead to deficits in VOR-cancellation. But, in cerebellar and brainstem disease VOR-cancellation and smooth pursuit might be affected differentially [47,48]. One drug especially effect VOR-cancellation more than smooth pursuit, barbiturates [49].

Remember, to diagnose central vestibular disorders, all eye movements systems as fixation, saccades, smooth pursuit, optokinetic response, VOR and vergence have to be examined routine neurological examinations has to be performed.

Conclusion

The three tests CI, vHIT and rotatory chair testing do not test the same aspect of the horizontal VOR and therefore could not be replaced by each other. They should be part of a vestibular test battery. A direct comparison of rotatory testing of vHIT, CI and rotatory testing on a larger scale is missing so far. CI and VHIT are very powerful in identifying unilateral vestibular failure, but still the two tests must be used in sequence if necessary. Furthermore, the pathology of the vHIT and CI depends on the etiology of the disease. vHIT has the advantage of testing also the vertical SCCs, which the other tests could not. Rotatory testing is a specialized test to identify vestibular dysfunction of the horizontal VOR. The value of this test is especially important in bilateral vestibular failure, in central vestibular dysfunction, e.g. testing the central ‘velocity storage’, short-term adaptation, VOR-cancellation or VOR-tilt-suppression, and in testing the performance of the VOR-system using both, right and left SCCs. From this review it is clear that not each outpatient clinic needs a costly rotatory chair, but at high-end vertigo and balance centers they should be available. Instead, I recommend using both, vHIT and CI in sequence all in- and outpatient settings concerned with vertigo and dizziness.

References


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