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# Comparison of the video head impulse test with the caloric test in patients with sub-acute and chronic vestibular disorders

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ABSTRACT

The aim of this prospective register-based study was to compare video Head Impulse Tests (vHIT) with caloric tests on 173 patients assessed by a tertiary Neurology referral centre who had been referred for investigation of dizziness or vertigo and whose symptom duration was one month or longer. Abnormal vHIT was defined as angular velocity gain (peak eye velocity/peak head velocity) less than 0.79 at 80 ms and 0.75 at 60 ms, which was two standard deviations below our institutions' lower limit of normal; together with refixation saccades. Abnormal bi-thermal caloric testing defined unilateral hypofunction as a 25% difference using Jongkee's formula and bilateral hypofunction was defined by the sum of the peak slow phase velocities over the four irrigations being <20°/s. Sixty patients had abnormal results on one or both tests, of whom 51 had unilateral and nine bilateral hypofunction. With caloric testing considered as the gold standard, the sensitivity (95% CI) of the vHIT was 18/52, 34.6% (22.0–49.1), and the specificity (95% CI) was 113/121, 93.4% (87.4–97.1). However vHIT was more sensitive in the nine patients with bilateral hypofunction with 100% abnormal vHIT results while only 4/9, 44% had abnormal caloric results. In conclusion these results support the continued use of both vHIT and caloric tests in patients with sub-acute and chronic vestibular symptoms, especially if the vHIT is normal.



## 1. Introduction

Portable video-oculography systems which measure the high frequency gain of the horizontal vestibulo-ocular reflex (HVOR) with the head impulse test have improved the diagnostic accuracy of suspected vestibular neuritis [1]. Although the video head impulse test (vHIT) is quick to perform, tightly fitting goggles, required to avoid goggle slippage, can be uncomfortable. Their use also requires care to avoid artefacts and good concentration by patients [2,3].

The bi-thermal caloric test, in use since the 1940s, has been the most common diagnostic test used to assess low frequency horizontal semicircular canal function. Disadvantages of caloric testing are that it is less physiological than high frequency testing [4], and responses to the caloric test are variable; being influenced by technique, patient alertness, the size of the external ear canal, and patient tolerance.

Although the caloric and the vHIT test different frequencies of the vestibular system the vHIT has lower sensitivity in detecting vestibular hypofunction than caloric testing in the following test situations: five days after recovery from an acute vestibular presentation [5], one month after vestibular neuritis [6], and in patients with chronic lesions, such as sporadic vestibular schwannoma [7].

The Vestibular Function Testing Unit of the Neurology Department at Wellington Hospital is a tertiary referral centre for the lower half of the North Island of New Zealand and has a catchment patient population of around 500,000. In this unit patients referred with dizziness routinely have both the caloric test and vHIT to measure HVOR gain.

### 1.1. Aim

To compare the sensitivity of the caloric test results with the vHIT results in patients with vestibular symptoms for one month or more, and to define the slow phase velocity on caloric testing at which a vHIT would be abnormal.

### 1.2. Hypothesis

That an impaired HVOR gain with an abnormal vHIT, with ipsilateral hypofunction, would only be seen with an ipsilateral caloric

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average peak slow phase velocity (aPSPV) $\leq 10°$/sec as suggested on previous clinical observation.

## 2. Methods

A prospective register of all patients assessed by the Vestibular Function Testing Unit was reviewed for October 2011 to December 2015. Patients were included for analysis if they had been referred for investigation of dizziness or vertigo with symptom durations of one month or more. All patients had vHIT followed by caloric testing performed on the same day.

vHIT was performed using the EyeSeeCam VOG system (Manufacturer EyeSeeTec, Munich, Germany) to measure the HVOR gain of the horizontal semicircular canals. The clinical testing protocol included a minimum of ten unpredictable (timing and direction) head impulses performed in the horizontal plane to each side with the patient seated and fixating on a small target on the wall at eye level and 1.5 metres distant. An adequate head acceleration of 2300–5900°/s$^2$ was achieved in each HIT to ensure the detection of a high frequency vestibular deficit [8,9]. An adequate acceleration was monitored in real time by ensuring a peak head velocity of between 150°/s and 300°/s with low amplitude head movements.

Instantaneous HVOR velocity gains were calculated by the EyeSeeCam VOG software at 80 ms and 60 ms. An abnormal response was defined using normative data from our previous study with an abnormal vHIT angular velocity gain (peak eye velocity/peak head velocity) below the 2 standard deviation lower limit of normal at 0.79 at 80 ms and 0.75 at 60 ms as defined in 60 historical controls aged between 20 and 80 years of age [2] and consistent with the literature [1]. Covert and/or overt re-fixation saccades were also required when defining an abnormal vHIT result.

Although not used to define an abnormal vHIT result, gain asymmetry ratios were also recorded [2,7] and were used in support of abnormal VOR velocity gains to alert us to any possible questionable results. The gain asymmetry ratio is defined as:

$$\textit{Gain asymmetry ratio}\ \% = \left[\frac{(\textit{Gain unaffected side} - \textit{Gain affected side})}{(\textit{Gain unaffected side} + \textit{Gain affected side})}\right] \times 100$$

Caloric testing was performed using the Disoft infra-red video-oculography system (Instrumentation DIFRA, 84, Rue de l'Eglise, B-4840 Welkenraedt, Belgium) with cold and warm water irrigations to both ears. Unilateral hypofunction (UVH) was defined by bi-thermal caloric testing when there was a canal paresis (CP) greater than 25% difference between the two ear peak slow phase velocities (PSPV) using the Jongkees' formula [10]. The average of the peak slow phase velocities (aPSPV) of nystagmus from both the cold and warm water irrigations on the affected side was then calculated. Bilateral vestibular hypofunction (BVH) was defined by the sum of the peak slow phase velocities over the four irrigations being <20°/s [11].

When there were abnormal test results with either investigation the ipsilateral vHIT VOR gain was compared with the ipsilateral caloric aPSPV in patients with both UVH and BVH.

### 2.1. Statistical analysis

Comparison of paired proportions was by McNemar's test. Sensitivity and specificity were estimated by the Clopper-Pearson exact binomial method. A rank correlation coefficient estimates the strength of association between different measurements of vestibular function. Logistic regression was also used to estimate the diagnostic performance of caloric testing in those with UVH with vHIT treated as a gold standard and as summarised by the Area Under Curve for the Receiver Operator Characteristic curve and an illustrative sensitivity and specificity at particular cut-off values for caloric testing. In this analysis caloric testing for those with UVH was defined as greater than 25% on the Jongkees formula.

SAS version 9.4 was used.

## 3. Results

A total of 185 patients were investigated with both tests over 26 months with 12 patients excluded from analysis due to technically inadequate measurements with vHIT testing. No caloric test results were rejected. The mean (range) age of the remaining 173 patients was 52 years (19–87), 97 were female, and symptom duration ranged from 1 month to 50 years.

Of these 173 patients, 60 had abnormal results on at least one testing regime. The mean age (range) of these 60 patients was 57 (24–79) years, and 27 were female.

The 60 patients were referred for vestibular testing by Neurologists (N = 39), Physiotherapists (N = 14), General Practitioners (N = 5), Ear Nose and Throat specialists (N = 1) and General Medicine (N = 1). The referring clinicians suspected that all patients had a peripheral vestibular origin for all or some of their symptoms and included diagnoses of possible vestibular neuronitis (N = 29), Ménière's Disease (N = 3), Benign Paroxysmal Positional Vertigo (N = 3), gentamicin ablation (N = 1), acoustic neuroma excision (N = 1), gentamicin ototoxicity (N = 2) and bilateral vestibular hypofunction (N = 2). A normal central nervous system (CNS) examination was present in 37 patients at the time of referral from within the Neurology Department four of which had suspected vestibular migraine. Another 12 patients had both CNS as well as suspected peripheral vestibular conditions (including cerebellar degeneration (N = 6), stroke (N = 2), and one each of dementia, head injury, multiple sclerosis, and another CNS inflammatory condition). Symptoms consistent with Persistent Postural and Perceptual Dizziness (PPPD) were present in three patients.

Table 1 shows the contingency table of abnormal results by testing regime. Of the 60 patients with abnormal results on either or both of the vHIT and caloric test, 51 had partial or total UVH. Of

**Table 1**
Contingency table of vestibular function test outcomes.

| vHIT[1] | Caloric test | | Total |
|---|---|---|---|
| | Abnormal | Normal | |
| Abnormal | **18** (**14** UVH[2]/**4** BVH[3]) | **8** (**3** UVH[2]/**5** BVH[3]) | **26** (**17** UVH[2]/**9** BVH[3]) |
| Normal | **34** (UVH[2]) | **113** | **147** (**34** UVH[2]) |
| Total | **52** (**48** UVH[2]/**4** BVH[3]) | **121** (**3** UVH[2]/**5** BVH[3]) | **173** (**51** UVH[2]/**9** BVH[3]) |

[1] Video Head Impulse Test.
[2] Unilateral Vestibular Hypofunction (UVH).
[3] Bilateral Vestibular Hypofunction (BVH).

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these, 14 patients had both abnormal vHIT and caloric tests, 34 had abnormal caloric tests only and three abnormal vHIT only. Nine patients had BVH and all nine (100%) had bilaterally abnormal VOR gains at both 80 ms and 60 ms with vHIT while only 4/9 (44.4%) had abnormal caloric test results. The full testing results are given in the Appendices.

A higher proportion of patients had an abnormal caloric test, 52/173 (30.1%), compared with the vHIT, 26/173 (15.0%). The paired difference (95%CI) was 15.0% (8.0 to 22.0), P < .001. If the caloric test is defined as the gold standard then the sensitivity (95% CI) of the vHIT was 18/52, 34.6% (22.0–49.1) and the specificity (95% CI) was 113/121, 93.4% (87.4–97.1).

Analysis of vHIT results uses the mean of VOR gains at 80 ms and 60 ms as there was no clinically important difference between these two measurements of VOR gain on analysis (See Appendix 1). This was also the case in our previous control data [2].

The Figure 1 shows a plot of CP, with PSPVs, using Jongkees' formula, expressed as a percentage, against the mean of the two measurement frequency vHIT VOR velocity gains for the abnormal side, for those patients with UVH. Note that a negative correlation means that as the mean of the VOR gains on vHIT decreases, the percentage CP increases. The rank correlation coefficient for the strength of this association was −0.43, P = .002 (See Appendix 2).

The Figure 2 shows a plot of aPSPV against the mean of the two measurement frequency vHIT VOR velocity gains for the abnormal side, for those patients with UVH. Note that a positive correlation coefficient means that as the mean of the VOR gains on vHIT increases, the aPSPVs also increase on caloric testing. The rank correlation coefficient for the strength of this association was 0.44, P = .001.

There were 14 patients in whom both tests were abnormal with 10/14 (71%) having aPSPVs on the affected side of <10°/sec. However 4/14 had aPSPVs > 10°/sec ranging from 10.2 to 23.8°/sec. There were an additional 9 patients with normal vHIT results and abnormal caloric results with aPSPV of <10°/s. Therefore there wasn't a clear relationship between aPSPV and an abnormal vHIT.

Four patients, who had moderate to severe UVH and positive results on vHIT and caloric testing, also had decreased VOR gains on the contralateral side (0.64–0.74 at 80 ms and 0.59–0.75 at 60 ms). All these patients had high degrees of canal paresis, ranging from 63 to 94%, and all had VOR gains of 0.31 or less on the affected side. Two were three months after symptom onset, one eight months and one 18 months after symptom onset.

The diagnostic performance of CP defined as the CP of PSPVs, using Jongkees' formula, compared with an abnormal vHIT is summarised in the Receiver Operating Characteristic (ROC) curve shown in Figure 3. Overall the diagnostic performance of CP was only moderate with the Area Under the Curve for the ROC only 0.66. At a cut-off value of 25% the sensitivity for vHIT abnormality

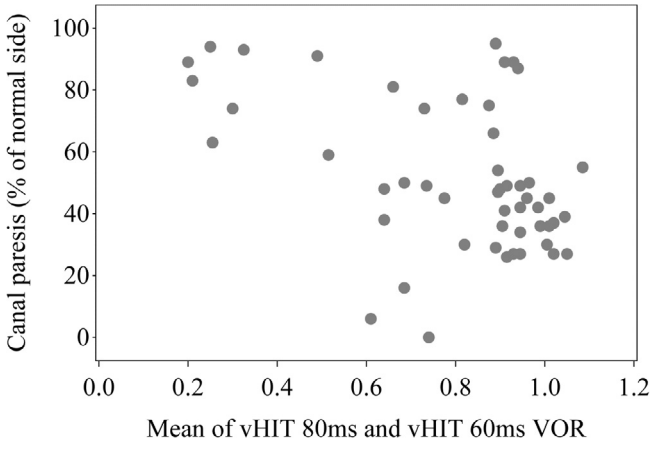


**Figure 1.** CP with PSPVs using Jongkees' formula against mean of two vHIT measurement frequencies for the VOR.

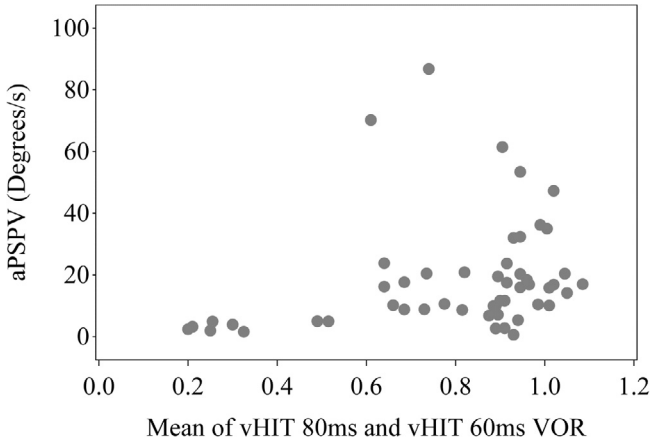


**Figure 2.** aPSPV of nystagmus against mean of two vHIT measurement frequencies for the VOR.

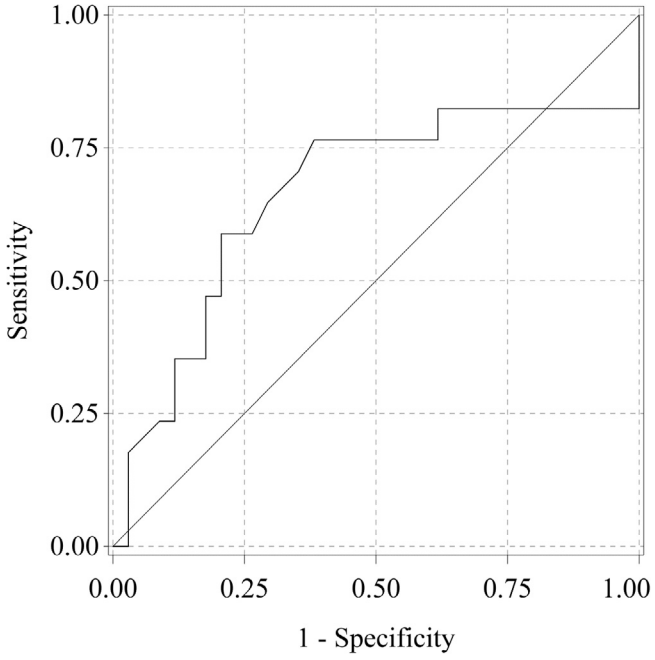


**Figure 3.** Receiver Operator Characteristic curve for CP against a vestibular abnormality defined as an abnormal vHIT.

**Table 2**
Summary of gain asymmetry measurements for vHIT.

| | Gain asymmetry ratio (%) | | |
|---|---|---|---|
| | Mean (SD) | Median | Range (minimum to maximum) |
| vHIT and Caloric tests both abnormal N = 14 | 29 (16.7) | 24.5 | 7–59 |
| Caloric abnormal and vHIT normal N = 34 | 3.6 (2.8) | 3.0 | 0–13 |
| vHIT abnormal and Caloric normal N = 3 | 11.7 (4.2) | 13 | 7–15 |

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was only 14/48 (29.2%) and because of so few events at this cut-off the specificity was 0/3 (0%). The optimal diagnostic cut-off by inspection was a CP of about 42% with a sensitivity of 13/30 (43%) and a specificity of 17/21 (81%).

Although in this study we didn't use abnormal gain asymmetry ratios (GA) to define a positive vHIT these were recorded as a consistency check with the two main measurements. Table 2 summarises these.

Past research reports GAs of greater than 8.5% as abnormal [7] consistent with our own results [2] with 95th percentile for normalised GA of 9.2% at 80 ms and 8.8% at 60 ms. In this study 3/51 patients had inconsistent GAs. Where both tests were abnormal 1/14 had a normal GA of 7% (0.69 at 80 ms/0.68 at 60 ms/50% left canal paresis). In the group with caloric abnormal/vHIT normal 1/34 had an abnormal GA of 13% (0.90 at 80 ms/0.99 at 60 ms/34% left canal paresis). In the group with vHIT abnormal/caloric normal 1/3 had a normal GA of 7%.The vHIT abnormality determined by low VOR gains (0.73-right and 0.78-left at 80 ms and 0.67-right and 0.77-left at 60 ms.) may be a false positive as the GA was normal with 0% canal paresis on caloric testing with aPSPV of 86.7 on the right.

## 4. Discussion

Of the 173 patients assessed for vestibular function in this study unit, 60(35%) patients with vestibular symptoms had abnormalities on one or both of the two vestibular investigations. In general the caloric test was more sensitive in detection of vestibular hypofunction than the vHIT. These findings are similar to other studies reporting vestibular function testing in patients with chronic vestibular symptoms with very similar sensitivity for vHIT compared with caloric testing. Mahringer and colleagues reported that 33% of patients with symptom onset greater than five days with a unilateral weakness on caloric testing also had a pathological vHIT test [5]. In our study 17/51 (33%) with a UVH had an abnormal vHIT. Three of our abnormal vHIT cases had a normal caloric test. In a study investigating vestibular schwannoma, 3–6% of cases with UVH had an abnormal vHIT with normal caloric test [7].

Our finding, that for UVH a greater CP is associated with a greater probability of a positive vHIT, is also consistent with past research [6]. We found sensitivities quite poor below a CP of 42%, and at this level sensitivity compared with vHIT was only 43%. Clinically this means that a positive vHIT indicates a more severe level of hypofunction and for diagnostic purposes suggests a caloric test is not necessary. However where the vHIT result is negative, caloric testing is still necessary to determine a milder hypofunction in the UVH group.

We found that those patients with partial CP with aPSPV > 10°/s were less likely to have an abnormal vHIT result but this was not consistent. Our hypothesis that a positive vHIT would only be seen with an ipsilateral caloric aPSPV $\leq$ 10°/sec was therefore rejected.

Our results suggest that as previously recognised the vHIT and the caloric test assess horizontal canal function at different frequencies [12] and that the higher frequencies are more likely to recover over time. This was shown by Bartolomeo et al. [6] when reporting results in 29 patients with acute unilateral vestibular neuritis who all had both abnormal vHIT and caloric tests on initial testing. When retested at one month post-acute vestibular neuritis 13/29 had mild caloric deficits of <40% but normal vHITs. In 4/29 patients with moderate caloric deficits (between 40% and 62.5%), 2/4(50%) had an abnormal vHIT. In 12/29 patients with severe caloric deficits of greater than 62.5% the vHIT was abnormal in all 12/12(100%). Therefore the vHIT had normalised in 15/29 (51.8%) patients and the caloric results had normalised in 8/29 (27.6%) at one month post onset. Comparing our results of chronic patients by using the same criteria to group caloric results, an abnormal vHIT was seen in 1/15 (6.6%) with a mild caloric deficit, in 4/17 (23.5%) with a moderate caloric deficit and 9/16 (56.3%) with a severe caloric deficit. Our lower proportion of patients with abnormal vHITs in the moderate and severe groups are probably indicative of further recovery of high frequency vestibular function with longer periods of time from initial onset.

We considered BVH separately from UVH. Whereas caloric testing was more sensitive than vHIT in detecting UVH, the opposite was true in the nine patients with BVH. The literature is unclear on the level at which bilateral vestibular failure is diagnosed from caloric results. We modified the criteria used in past studies [11] which determined caloric responses <5°/sec for each of the four separate irrigations (hot and cold irrigations in both ears) indicated BVH. We used the $\sum$PSPV of the four irrigations being $\leq$20° resulting in 4/9 (44%) cases meeting this criteria on caloric testing. However, this is probably an indication of near total BVH. Whereas UVH with partial loss of function is reasonably defined there may be patients with a partial BVH on caloric testing whose results are still within the normal range. We found that in three patients with decreased HVOR bilaterally, indicating BVH, the caloric results had $\sum$PSPVs between 38.5 and 98.4. Most other studies have only looked at unilateral cases.

It remains important with vestibular symptoms in the absence of a deficit on one test to determine function with both higher and lower frequency testing. These test different aspects of the canal function possibly in relation to different hair receptor cell structure and function. The findings in our study of lower sensitivity of vHIT compared with caloric testing in UVH and higher sensitivity in BVH, may be explained in time by emerging research looking at the different functions of the two vestibular hair cell receptor types and possible selective loss in different conditions and different recovery rates or as suggested by Nguyen et al. [13] due to a disorder affecting the vestibular nerve afferent firing rates. However in Ménières's disease McGarvie et al. [14] found that the model of different frequency responses in the hair cell receptors and afferent complex was unlikely to explain the different results with vHIT and caloric testing. They hypothesised that a more likely model is that caloric responses are reduced due to a physical enlargement of the membranous duct due to hydrops thus reducing any thermally induced pressure changes across the cupula for this group of patients. However this doesn't explain the differences in results between the two tests in other conditions.

There is a risk of false negatives if only one of the tests is performed. In our study of chronic unilateral cases, vHIT testing alone would have missed 34/60(56%) cases that showed a deficit on caloric testing, while caloric testing alone would have missed about 8/60(13%) that had an abnormal vHIT. Caloric testing alone would have missed 55% of our BVH cases.

## 5. Conclusion

vHIT is a very useful diagnostic tool both at the bedside and in clinic. However in this study, we found evidence that supports the continued use of both vHIT and caloric tests in patients with subacute and chronic symptoms, particularly if the vHIT is normal. The vestibular system can have differential deficits with both high and low frequency testing.

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## Appendix 1. Individual patient testing results for patients with UVH

| % Canal paresis as per the Jongkee formula | Video head impulse test (units) | | Average peak slow phase velocity degrees/s |
|---|---|---|---|
| | 80 ms | 60 ms | |
| 0 | 0.76 | 0.72 | 86.75 |
| 6 | 0.61 | 0.61 | 70.2 |
| 16 | 0.71 | 0.66 | 17.65 |
| 26 | 0.9 | 0.93 | 17.5 |
| 27 | 0.96 | 1.08 | 47.25 |
| 27 | 0.96 | 0.93 | 53.4 |
| 27 | 0.92 | 0.94 | 32 |
| 27 | 1.01 | 1.09 | 14.14 |
| 29 | 0.91 | 0.87 | 9.95 |
| 30 | 1.00 | 1.01 | 35 |
| 30 | 0.82 | 0.82 | 20.85 |
| 34 | 0.9 | 0.99 | 32.35 |
| 36 | 0.89 | 0.92 | 61.45 |
| 36 | 1.01 | 1.01 | 15.8 |
| 36 | 0.96 | 1.02 | 36.2 |
| 37 | 0.96 | 1.08 | 16.85 |
| 38 | 0.67 | 0.61 | 23.8 |
| 39 | 1.02 | 1.07 | 20.4 |
| 41 | 0.89 | 0.93 | 11.65 |
| 42 | 0.95 | 0.94 | 15.95 |
| 42 | 1.01 | 0.96 | 10.45 |
| 45 | 0.79 | 0.76 | 10.6 |
| 45 | 1.00 | 0.92 | 18.4 |
| 45 | 1.00 | 1.02 | 10.1 |
| 47 | 0.89 | 0.90 | 19.5 |
| 48 | 0.93 | 0.87 | 11.65 |
| 48 | 0.65 | 0.63 | 16.2 |
| 49 | 0.93 | 0.54 | 20.45 |
| 49 | 0.93 | 0.9 | 23.7 |
| 49 | 0.93 | 0.96 | 20.3 |
| 50 | 0.69 | 0.68 | 8.8 |
| 50 | 0.99 | 0.94 | 16.9 |
| 54 | 0.97 | 0.82 | 7.1 |
| 55 | 1.03 | 1.14 | 17 |
| 59 | 0.52 | 0.51 | 4.95 |
| 63 | 0.27 | 0.24 | 4.9 |
| 66 | 0.92 | 0.85 | 9.95 |
| 74 | 0.29 | 0.31 | 3.9 |
| 74 | 0.73 | 0.73 | 8.85 |
| 75 | 0.88 | 0.87 | 6.8 |
| 77 | 0.86 | 0.77 | 8.65 |
| 81 | 0.69 | 0.63 | 10.2 |
| 83 | 0.21 | 0.21 | 3.2 |
| 87 | 0.95 | 0.93 | 5.35 |
| 89 | 0.16 | 0.24 | 2.4 |
| 89 | 0.93 | 0.93 | 0.6 |
| 89 | 0.91 | 0.91 | 2.75 |
| 91 | 0.53 | 0.45 | 5 |
| 93 | 0.34 | 0.31 | 1.6 |
| 94 | 0.27 | 0.23 | 1.95 |
| 95 | 0.92 | 0.86 | 2.65 |

## Appendix 2. Individual patient testing results for patients with BVH

| Caloric ∑PSPV | vHIT 80 msR | vHIT 80 msL | vHIT 60 msR | vHIT 60 msL | Gain asymmetry |
|---|---|---|---|---|---|
| 6.5 | 0.22 | 0.05 | 0.21 | 0.04 | 53%L |
| 6.9 | 0.12 | 0.33 | 0.11 | 0.37 | 50%R |
| 6.9 | 0.06 | 0.05 | 0.04 | 0.01 | 0% |
| 14.3 | 0.51 | 0.57 | 0.33 | 0.44 | 9%R |
| 25.8 | 0.33 | 0.15 | 0.31 | 0.18 | 24%L |
| 30.9 | 0.31 | 0.41 | 0.32 | 0.31 | 21%R |
| 38.5 | 0.19 | 0.22 | 0.29 | 0.19 | 0% |
| 44.3 | 0.50 | 0.62 | 0.50 | 0.63 | 8%R |
| 98.4 | 0.44 | 0.28 | 0.37 | 0.24 | 20%L |

R = right, L = left.

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