

1 **The 180-Degree Clinical Chair Rotation Test in Normal Subjects and Patients with Various**
2 **Peripheral Vestibular Abnormalities.**

3
4 **Abstract**

5 **Background**

6 Rotary chair testing (ROT) is the gold standard assessment for horizontal semicircular canal (SCC)
7 function, particularly in cases of suspected bilateral vestibular hypofunction.[1,2] Despite sensitivity
8 (0.747) and specificity (0.634) for peripheral vestibulopathy, its clinical adoption remains limited by
9 cost and spatial requirements. A clinic-based 180-degree chair rotation test, coupled with video-
10 oculography (VOG), offers an accessible alternative for assessing vestibulo-ocular reflex (VOR)
11 function across the full spectrum of peripheral vestibular disorders.

12 **Objective**

13 To assess the utility of the 180-degree clinical chair rotation test in characterizing per-rotatory
14 nystagmus, post-rotatory nystagmus, and VOR suppression in healthy normal subjects and in
15 patients with diagnosed peripheral vestibular abnormalities, including vestibular migraine (VM),
16 benign paroxysmal positional vertigo (BPPV), and Meniere's disease.

17 **Methods**

18 A total of 100 healthy volunteers and 100 patients with peripheral vestibular disorders were enrolled
19 across all age groups at the Vertigo and Balance Disorders Clinic, Bengaluru, India. The chair was
20 manually rotated 180 degrees by a single right-handed examiner, with a 10-second inter-rotation

21 gap, under three conditions: vision allowed (fixation), vision denied (fixation eliminated), and VOR
22 suppression. Eye movements were recorded using the 'Balance Eye VOG' system.

23 **Results**

24 Normal subjects demonstrated absent post-rotatory nystagmus (PRN) and intact VOR suppression.
25 Among patients with vestibular migraine, PRN was significantly more prevalent (59.5%) compared
26 with normal subjects (14.3%; $p < 0.001$). Non-vestibular migraine patients also showed
27 significantly elevated PRN positivity (43.1%; $p = 0.008$). Distinct VOG patterns were identified for
28 right posterior semicircular canal BPPV, lateral semicircular canal BPPV and Meniere's disease,
29 enabling lesion lateralization.

30 **Conclusion**

31 The 180-degree clinical chair rotation test, combined with VOG recording of per-rotatory
32 nystagmus, post-rotatory nystagmus, and VOR suppression, constitutes a simple, intuitive, and
33 clinically informative tool for assessing peripheral vestibular disorders. Further prospective studies
34 with larger sample sizes are warranted to validate normative thresholds and disease-specific
35 diagnostic criteria.

36 **Keywords:** *rotary chair test; video-oculography; vestibulo-ocular reflex; post-rotatory nystagmus;*
37 *Bilateral vestibulopathy, LSCC*

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44 **1. Introduction**

45 Vestibular function testing plays a key role in diagnosing disorders of the inner ear balance system.
46 Rotary chair testing (ROT), also known as rotational chair testing, evaluates horizontal semicircular
47 canal function and vestibulo-ocular reflex (VOR) pathways. Unlike caloric testing, which assesses
48 each ear separately at low frequency, ROT evaluates bilateral vestibular responses across
49 physiologically relevant frequencies.[1,2,8].

50 ROT has demonstrated good diagnostic utility in peripheral vestibulopathy and is included in the
51 diagnostic criteria for bilateral vestibular dysfunction. [2,4]It also provides information regarding
52 central vestibular compensation. However, its routine clinical use remains limited because of the
53 high cost and space requirements of motorised rotary chair systems, particularly in resource-limited
54 settings.[1,7]

55 The 180-degree clinical chair rotation test is a low-cost alternative using rapid manual chair rotation
56 combined with video-oculography (VOG) to assess per-rotatory nystagmus, post-rotatory
57 nystagmus (PRN), and VOR suppression. These parameters may provide clinically useful
58 information regarding vestibular function and lesion localization.

59 The present study aimed to evaluate these VOG parameters in healthy controls and patients with
60 peripheral vestibular disorders including vestibular migraine, benign paroxysmal positional vertigo
61 (BPPV), and Ménière's disease. [1,2,8]

62 **2. Materials and Methods**

63 **2.1 Study Design and Setting**

64 This prospective, observational, single-center study was conducted at the Vertigo and Balance
65 Disorders Clinic, Bengaluru, India. Informed consent was obtained from all participants. Ethical
66 principles were followed in accordance with the Declaration of Helsinki.

67 **2.2 Participants**

68 Two groups were enrolled:

69 • **Group A (Normal Controls):** 100 healthy volunteers with no history of vertigo, hearing loss,
70 neurological disease, or medications affecting vestibular function.

71 • **Group B (Patient Cohort):** 100 patients with peripheral vestibular disorders including vestibular
72 migraine (VM), posterior SCC BPPV (right PSCC BPPV), lateral SCC BPPV (geotropic variety),
73 and Meniere's disease across all age groups.

74 **2.3 Equipment**

75 Eye movements were recorded using the 'Balance Eye VOG' system (Figure 1), a binocular video-
76 oculography device that captures horizontal and vertical slow-phase velocity (SPV), amplitude, and
77 frequency of nystagmus in real time. The device provides simultaneous display of right eye (red
78 trace) and left eye (blue trace) horizontal nystagmus waveforms.



79

80 *Figure 1. Clinical setup demonstrating the Balance Eye VOG device and chair rotation technique at*
81 *the Vertigo and Balance Disorders Clinic, Bengaluru, India.*

82

83 **2.4 Test Procedure**

84 A single right-handed examiner performed all tests to ensure methodological consistency. The
85 subject was seated upright on a standard rotating chair. The chair was manually rotated 180 degrees
86 in either direction, with a minimum 10-second inter-rotation gap to allow nystagmus to decay.

87 Each subject underwent testing under three sequential conditions:

88 • Vision Allowed (Fixation): Subjects fixated on a stationary visual target during and after rotation,
89 assessing per-rotatory nystagmus.

90 • Vision Denied (Fixation Eliminated): The VOG goggles were occluded to eliminate visual
91 fixation, maximizing vestibular contribution to nystagmus and unmasking post-rotatory nystagmus.

92 • VOR Suppression: Subjects were instructed to fixate on their thumb, testing the ability of the
93 smooth pursuit system to suppress the VOR.

94 **2.5 Parameters Assessed**

95 The following VOG parameters were systematically evaluated for each rotation direction and each
96 vision condition:

- 97 • Per-rotatory nystagmus: Direction, slow-phase velocity (SPV), amplitude, and duration of
98 nystagmus occurring during rotation.
- 99 • Post-rotatory nystagmus (PRN): Presence, direction, SPV, and duration of nystagmus
100 persisting after cessation of rotation.
- 101 • VOR suppression: The ability to suppress per-rotatory nystagmus by optic fixation,
102 expressed as the VOR suppression ratio.

103 **2.6 Statistical Analysis**

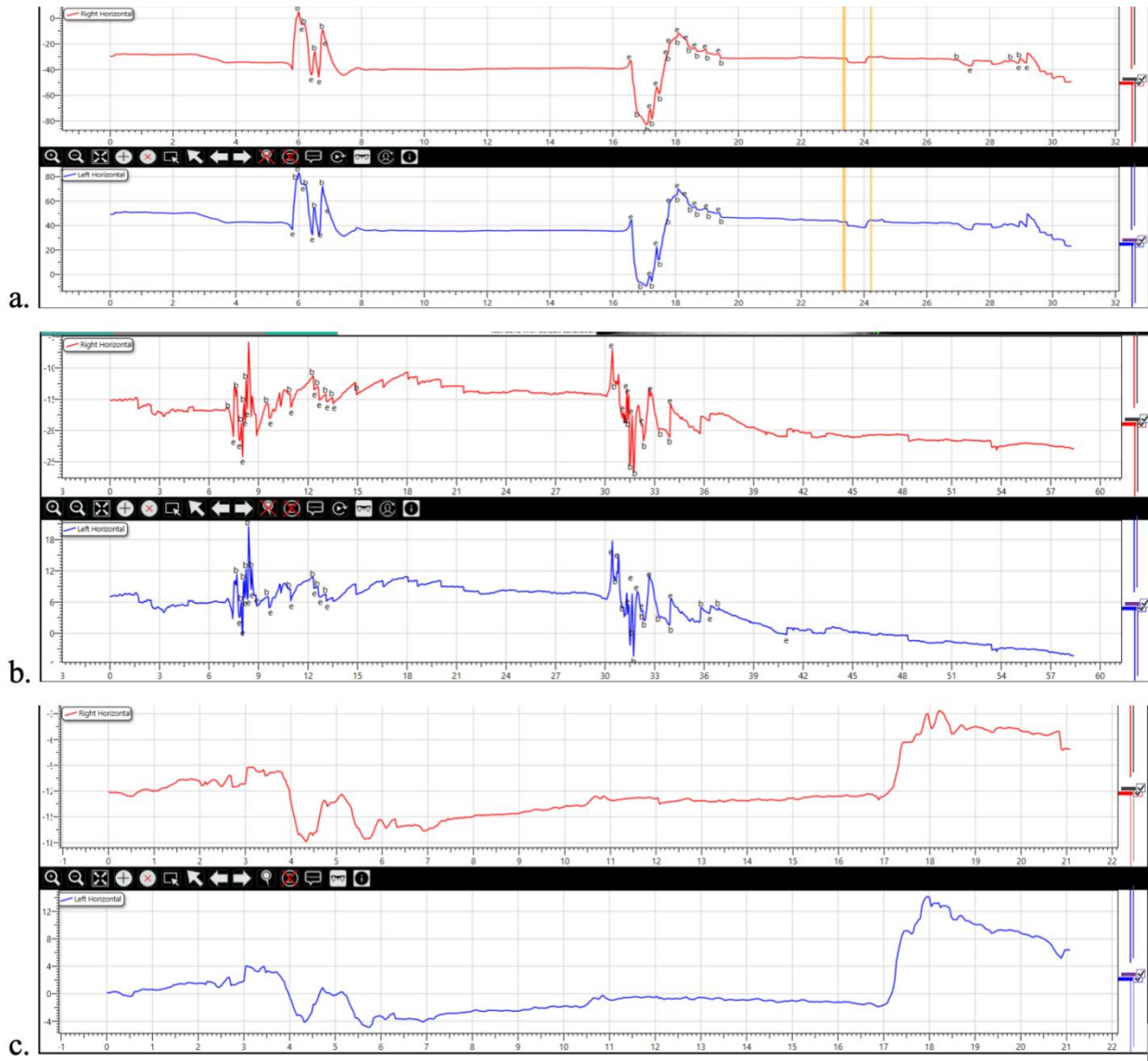
104 Categorical data were analyzed using the chi-square test or Fisher's exact test as appropriate.
105 Statistical significance was defined as $p < 0.05$. All analyses were performed using standard
106 statistical software.

107 **3. Results**

108 **3.1 Normal Subjects**

109 In 100 healthy volunteers, per-rotatory nystagmus was consistently elicited bilaterally following
110 chair rotation in both directions. The nystagmus beat toward the direction of rotation during rotation
111 and rapidly decayed with visual fixation. Post-rotatory nystagmus was absent or minimal in most
112 normal subjects. VOR suppression was intact, with marked attenuation of nystagmus SPV when
113 subjects fixated on a chair-fixed target.

114 Normative VOG waveforms under vision-allowed and vision-denied conditions are presented in
115 Figures 2a and 2b, respectively. Figure 2c illustrates intact VOR suppression in a normal subject.



116
117 *Figure 2a. Normative VOG waveform (vision allowed / fixation): Right eye (red) and left eye (blue)*
118 *horizontal nystagmus traces showing per-rotatory nystagmus with rapid decay and absent post-*
119 *rotatory nystagmus in a healthy normal subject.*
120 *Figure 2b. Normative VOG waveform (vision denied): Horizontal nystagmus traces with fixation*
121 *eliminated. The absence of post-rotatory nystagmus confirms intact vestibular decay in the normal*
122 *group.*

123 *Figure 2c. VOR suppression tracing in a normal subject: Near-complete suppression of per-*
124 *rotatory nystagmus by chair-fixed fixation demonstrates intact cerebello-ocular VOR suppression*
125 *pathways.*

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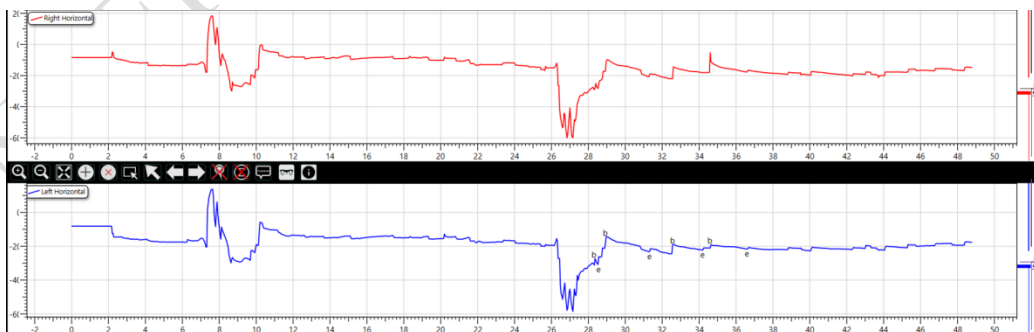
127 **3.2 Post-Rotatory Nystagmus: Vestibular Migraine vs. Normal**

128 Post-rotatory nystagmus was assessed as a binary outcome (positive/negative) and compared
129 between patient subgroups and normal controls. Table 1 summarizes findings for vestibular
130 migraine (VM) versus normal subjects.

131 **Table 1. Post-Rotatory Nystagmus in Vestibular Migraine vs. Normal Subjects**

| Group | PRN Positive n (%) | PRN Negative n (%) | Total | P-value |
|---------------------|-----------------------|-----------------------|-------|---------|
| Vestibular Migraine | 22 (59.5%) | 15 (40.5%) | 37 | < 0.001 |
| Normal | 4 (14.3%) | 24 (85.7%) | 28 | — |
| Total | 26 | 39 | 65 | |

132 Vestibular migraine patients showed a significantly higher rate of PRN positivity (59.5%, 22/37)
133 compared with normal subjects (14.3%, 4/28; $p < 0.001$). This highly significant difference
134 underscores the role of aberrant central vestibular processing in VM, manifesting as prolonged post-
135 rotatory nystagmus beyond the physiological decay observed in normals.



136

137 *Figure 3. Representative VOG waveform in a patient with vestibular migraine demonstrating post-*
138 *rotatory nystagmus (both directions) with prolonged slow-phase velocity decay, indicating*
139 *vestibular hyper-reactivity characteristic of this condition.*

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141 3.3 Post-Rotatory Nystagmus: Non-Vestibular Migraine vs. Normal

142 Table 2 presents the comparison of PRN prevalence in non-vestibular migraine patients against
143 normal subjects.

144 **Table 2. Post-Rotatory Nystagmus in Non-Vestibular Migraine vs. Normal Subjects**

| Group | PRN Positive n (%) | PRN Negative n (%) | Total | P-value |
|----------------------------|-----------------------|-----------------------|-------|---------|
| Non-Vestibular Migraine | 25 (43.1%) | 33 (56.9%) | 58 | 0.008 |
| Normal | 4 (14.3%) | 24 (85.7%) | 28 | – |
| Total | 29 | 57 | 86 | |

145

146 Non-vestibular migraine patients also demonstrated significantly elevated PRN positivity (43.1%,
147 25/58) compared with normal subjects (14.3%, 4/28; $p = 0.008$). This finding suggests a subclinical
148 vestibular activation component even in migraine patients not fulfilling formal vestibular migraine
149 diagnostic criteria, possibly reflecting shared central sensitization mechanisms.

150 3.4 BPPV – Right Posterior Semicircular Canal

151 In patients with right PSCC BPPV, the 180-degree rotation elicited characteristic per-rotatory
152 nystagmus with a paroxysmal burst of mixed vertical-torsional nystagmus consistent with posterior
153 canal activation. Post-rotatory nystagmus demonstrated an asymmetric pattern, with the direction
154 and amplitude consistent with an ipsilesional (right-sided) posterior canal origin. VOR suppression
155 was preserved, distinguishing this from central pathology.

156 3.5 BPPV – Lateral Semicircular Canal (Geotropic Variety)

157 In patients with LSCC BPPV (geotropic variety), chair rotation produced a characteristic direction-
158 changing horizontal post-rotatory nystagmus that beat toward the ground (geotropic) on both
159 clockwise and counterclockwise rotations, consistent with canalithiasis of the lateral SCC. The
160 pattern was distinctly different from normal subjects and from PSCC BPPV, reflecting free-floating
161 otolith debris within the lateral canal ampulla.

162 3.6 Meniere's Disease

163 In Meniere's disease patients, rotational testing revealed unidirectional post-rotatory nystagmus with
164 asymmetric SPV, reflecting reduced peripheral vestibular drive from the hydropic labyrinth.
165 Valsalva maneuvers performed during VOG recording unmasked Valsalva-induced nystagmus
166 (SPV 10.36°/s, right horizontal), consistent with the pressure-sensitive endolymphatic hydrops
167 mechanism. VOR suppression was impaired in proportion to vestibular paresis severity.

168 3.7 Summary of VOG Findings by Diagnosis

169 Table 3 provides a consolidated summary of VOG parameters across all diagnostic groups,
170 highlighting the distinctive pattern for each condition.

171 **Table 3. Summary of VOG Parameters by Diagnosis**

| Diagnosis | Per-Rotatory Nystagmus | Post-Rotatory Nystagmus | VOR Suppression | Key Feature |
|-------------------------|------------------------|-------------------------|-----------------|-----------------------------------|
| Normal Subjects | Present (bilateral) | Absent | Intact | No PRN; good VOR-S |
| Vestibular Migraine | Present | Positive (59.5%) | Mildly impaired | Post-rotatory nystagmus |
| Non-Vestibular Migraine | Present | Positive (43.1%) | Variable | Subclinical vestibular activation |
| Right PSCC BPPV | Present | Present | Intact | Paroxysmal |

| | | | | |
|-----------------------|------------|----------------------|----------|-----------------------------------|
| | | | | vertical/torsional burst |
| LSCC BPPV (Geotropic) | Present | Geotropic horizontal | Intact | Direction-changing horizontal PRN |
| Meniere's Disease | Asymmetric | Unidirectional | Impaired | SPV asymmetry; Valsalva-triggered |

172

173 4. Discussion

174 This study demonstrates that the 180-degree clinical chair rotation test, combined with portable
175 VOG recording, provides diagnostically informative vestibular parameters across a spectrum of
176 peripheral vestibular disorders. The three core parameters — per-rotatory nystagmus, post-rotatory
177 nystagmus, and VOR suppression — contribute complementary diagnostic information.[2,7].

178 The physiological basis of this test rests on cupuloendolymph mechanics of the semicircular canal
179 system. Rapid 180-degree rotation creates transient endolymph deflection of the cupula, generating
180 a VOR that drives compensatory eye movements. The magnitude and duration of evoked nystagmus
181 reflect the functional integrity of the peripheral vestibular end-organ and its central connections.

182 Unlike sinusoidal harmonic acceleration (SHA) testing performed in motorised chairs, the 180-
183 degree impulsive step rotation delivers a broadband stimulus that activates multiple frequency-tuned
184 VOR pathways.

185 The significantly higher rate of PRN in vestibular migraine patients (59.5% versus 14.3% in
186 controls; $p < 0.001$) aligns with evidence implicating central vestibular sensitization as a core
187 mechanism in VM. [8,10].The trigeminovascular system modulates brainstem vestibular nuclei

188 activity, resulting in prolonged neural discharge following rotational stimulation — reflected as
189 persistent post-rotatory nystagmus in VOG recordings. Even non-vestibular migraine patients
190 showed elevated PRN rates (43.1%; $p = 0.008$), suggesting a continuum of vestibular involvement
191 in migraine and raising the possibility that rotational testing may serve as an objective biomarker for
192 vestibular sensitization.[8,10].

193 The distinctive nystagmus patterns elicited in PSCC BPPV and LSCC BPPV mirror established
194 biomechanical models of canalith repositioning. In PSCC BPPV, the rotational stimulus produces
195 mixed torsional-vertical nystagmus congruent with ipsilateral posterior canal activation. In contrast,
196 LSCC BPPV demonstrates geotropic direction-changing horizontal nystagmus reflecting
197 ampullofugal deflection of the lateral cupula by free-floating debris. In Meniere's disease, rotational
198 VOG demonstrated SPV asymmetry, reflecting reduced vestibular gain of the hydropic labyrinth
199 [9,11]. An advantage of rotational testing in Meniere's disease lies in its ability to track central
200 compensation — progressive normalization of VOR asymmetry as the central vestibular system
201 adapts to unilateral peripheral hypofunction.

202 From a clinical utility perspective, the 180-degree test offers several advantages over conventional
203 motorized ROT. It requires no specialized capital equipment beyond a portable VOG device and
204 rotating chair, making it deployable in community clinics and resource-limited settings. The
205 manoeuvre can be performed by a single trained examiner in under five minutes, with results
206 immediately interpretable in office. The test complements clinical examination, head impulse test
207 (HIT), and caloric testing by providing additional information about central vestibular
208 compensation and bilateral function.

209 Limitations of the present study include the modest sample size in diagnostic subgroups, cross-
210 sectional design, and absence of inter-rater reliability data. Manual delivery of the rotational
211 stimulus introduces some variability in angular velocity compared with motorized systems. Future
212 studies should establish normative age-stratified thresholds, assess test-retest reliability, and explore
213 utility in central vestibular pathology and post-treatment monitoring.

214

215 **5. Conclusions**

216 The 180-degree clinical chair rotation test represents a simple, intuitive, and clinically practical tool
217 for the assessment of peripheral vestibular function. When coupled with portable video-
218 oculography, it enables reliable characterisation of per-rotatory nystagmus, post-rotatory
219 nystagmus, and VOR suppression - parameters that collectively facilitate lesion lateralization and
220 diagnosis across a broad spectrum of vestibular disorders. The significantly elevated post-rotatory
221 nystagmus in both vestibular and non-vestibular migraine supports its role as a sensitive marker of
222 central vestibular sensitization. Larger prospective multicenter studies are required to establish
223 robust normative data, disease-specific diagnostic thresholds, and longitudinal validity.

224 **Declarations**

225 **Conflicts of Interest:** The author declares no financial relationships or conflicts of interest relevant
226 to the subject of this article.

227 **Ethical Approval:** Informed consent was obtained from all participants. The study was conducted
228 in accordance with the ethical principles of the Declaration of Helsinki.

229 **Funding:** No external funding was received for this study.

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