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Original Article

Assessing the Vestibulo-ocular Reflex of Contralesional Sides According to Head Impulse Velocity Utilizing the Video Head Impulse Test in Patients with Vestibular Neuritis

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BACKGROUND: There is a lack of comparative studies examining changes in vestibulo-ocular reflex (VOR) gain with head velocity in the video head impulse test (vHIT) of patients with vestibular neuritis (VN). Thus, the purpose of present study was to identify the effect of head impulse velocity on the gain of the VOR during the vHIT in patients with VN.

METHODS: Head impulse velocities ranging from 100%-200°/s [158.08 ± 23.00°/s in the horizontal canal (HC), 124.88 ± 14.80°/s in the anterior canal (AC), and 122.92 ± 14.26°/s in the posterior canal (PC) were used during vHIT trials of 32 patients with VN. Differences in VOR gain on the ipsilesional and contralesional sides according to head velocity were analyzed.

RESULTS: The mean VOR gains in ipsilesional side were decreased to 0.47 in the HC and 0.56 in the AC, leading to marked asymmetry compared to the contralesional side; PC gain was relatively preserved at 0.82 in the ipsilesional side. The mean head impulse velocity applied during vHIT trials in each semicircular canal plane did not differ bilaterally. On the contralesional side, VOR gain was negatively correlated with head impulse velocity ($R^2 = 0.25$, $P = .004$ in HC; $R^2 = 0.17$, $P = .021$ in AC; $R^2 = 0.24$, $P = .005$ in PC), while VOR gain on the ipsilesional sides of the HC and AC was not.

CONCLUSION: Head impulse velocity may have a differential impact on VOR gain, depending on the degree of deficit. Increasing head velocity in vHIT may be considered to identify subtle deficits on the contralesional side of patients with VN.

KEYWORDS: Vestibulo-ocular reflex, video head impulse test, vestibular neuritis

INTRODUCTION

The vestibulo-ocular reflex (VOR), which consists of a 3-neuron arc involving the afferent vestibular nerve, vestibular nuclei in the brainstem, and oculomotor nuclei, is a crucial neural mechanism that enables stable vision under dynamic conditions.[1] The video head impulse test (vHIT) is a relatively recent clinical vestibular test that quantitatively assesses the function of the semicircular canals, which are angular acceleration detectors responsible for initiating the VOR.[2] The main pathological findings of vHIT are reduced VOR gain, which is defined as the ratio of eye movement velocity to head velocity,[3] and the presence of corrective saccades, which are compensatory mechanisms of visual fixation that occur in response to fast head movement,[4,5] and are typically observed in patients with vestibular neuritis (VN), which is characterized by the sudden onset of acute continuous vertigo, motion sickness, gait instability, and spontaneous nystagmus beating toward the contralesional side lasting longer than 24 hours.[6] The superior or total vestibular nerve is most often affected by VN, while the inferior division only is rarely affected.[7] This pattern also can be found using vHIT results.[8]

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In clinical practice, VOR gain values below 0.8 in the horizontal canal (HC) and below 0.7 in the anterior canal (AC) and posterior canal (PC) with or without corrective saccades are typically considered abnormal.[9] The VOR gain during vHIT is primarily influenced by the integrity of the canal and its neural connections. In vestibular disorders, VOR gain can be significantly reduced or altered depending on the extent of damage to the semicircular canal and its neural connections to the superior/inferior vestibular nerve.[4,10] In addition, various technical factors such as target distance, equipment calibration, eye-tracking accuracy, goggle slippage, and examiner expertise can also have a significant impact on VOR gain.[11,12] The secondary factors affecting VOR gain include canal stimulated, head impulse velocity, gender, handedness of the examiner, room lighting, and monocular vs. binocular recording. Of the various secondary factors that affect VOR gain, head velocity and canal stimulated are known to have a significant effect.[11] In addition, multiple studies revealed a negative correlation between head impulse velocity and VOR gain in vHIT trials.[13-15] Previous studies conducted in normal populations have shown that all canal gains decrease with increasing head impulse velocity.[13,14] Therefore, it is crucial to control and standardize head impulse velocity during vHIT to ensure consistent and comparable results. However, despite the potential influence of head velocity on VOR gain, there is a lack of comparative studies examining changes in VOR gain with head velocity in the vHIT testing of patients with VN. In this context, investigating the influence of head velocity on VOR gains has significant importance, particularly in VN, providing insights into the degree of vestibular damage and compensatory mechanisms, contributing to enhanced diagnostic accuracy and refined management strategies. Therefore, the aim of this study was to investigate the relationship between head impulse velocity and VOR properties, including gain and the presence of corrective saccades, in patients with VN using vHIT.

MATERIAL AND METHODS

Subjects

Thirty-two patients with VN (mean age, 54.48 ± 15.71 years; 14 men, 18 women) were retrospectively reviewed at Chosun University Hospital from March 2021 to March 2023. Inclusion criteria for patients with VN consisted of the existence of acute vestibular symptoms characterized by acute and persistent vertigo, spontaneous horizontal-torsional nystagmus directed towards the opposite side, impaired gait, and symptoms such as nausea or vomiting lasting for less than 3 days following the onset of symptoms.[6] Patients who had previously experienced various audiovestibular diseases, such as Meniere's disease, vestibular migraine, sensorineural hearing loss, chronic otitis media, and past VN, were not included in the subsequent analysis. All patients completed vestibular function tests, including the bithermal caloric test and vHIT, within 5 days of symptom onset. The time from symptom onset to test was 2.53 ± 1.54 days. Brain magnetic resonance imaging verified the absence of stroke, bleeding, or cerebellopontine angle malignancies in all patients.

The Institutional Review Board of the Chosun University Hospital approved this study (Approval No: CHOSUN 2023-07-020; Date: July 20, 2023), and written informed consent has been obtained from all patients.

Biothermal Caloric Test

Bithermal caloric tests were conducted using SLVNG; SLMED equipment from Seoul, South Korea. The patient was placed in the supine position on a chair reclined at a 30° angle to align the horizontal semicircular canals in a vertical position. The external auditory canal was flushed sequentially with a continuous stream of water at temperatures of 30°C and 44°C, each for a duration of 30 seconds. Electronystagmography was used to measure induced nystagmus in a dark room while the patient's eyes were open. Analyzed in this study was the maximal slow-phase eye velocity of nystagmus induced by each ear. This analysis aimed to identify unilateral canal weakness and directional preponderance, as determined by applying the Jongkees formula. Prior to analysis, the degree of spontaneous nystagmus was automatically corrected. Abnormal canal weakness was characterized as having an asymmetry of more than 25% according to the Jongkees formula.[16]

Video Head Impulse Test

The 3 paired semicircular canals were assessed utilizing 3-dimensional vHIT (ICS Impulse; Otometrics, Taastrup, Denmark) together with data acquisition software. The patients were placed at 1 m from the target, with their eyes fixed at eye level. In order to guarantee the dependability of the test procedure, goggles were securely attached to the patient's head using an elastic band to reduce the likelihood of slipping, which may have led to inaccurate results. Following the calibration of eye position, the technician proceeded to manually execute more than 20 rotations. These rotations involved rotating the head by 15°-20° and lasted for 150-200 milliseconds. The rotations were conducted on both sides of each plane in a random and unpredictable sequence. The manufacturer's instructions specified goal head velocities of 150~200°/s for HC and 100~150°/s for vertical canals (AC and PC). Due to the challenges in obtaining precise and consistent angles for vertical semicircular canal head rotations in vHIT, a highly skilled technician physically conducted all procedures on both sides of each plane. The analysis utilized vHIT factors such as VOR gain and the properties of corrective saccades, including their incidence and amplitude. The VOR gain in the 3 semicircular canals was estimated by calculating the ratio of the area under the curve for the eye velocity to the area under the curve for the head velocity. The device automatically determined these areas. Abnormal VOR gain was determined when the HC was less than 0.8, and the AC and PC were both 0.7[10] or when covert and/or overt saccades were present. Pathologic corrective saccades were defined as overt and/or covert saccades with similar amplitude and latency for more than 20% of the total vHIT trials.[17]

MAIN POINTS

- The study reveals that head impulse velocity significantly affects the gain of the VOR on the contralesional side in patients with VN, indicating a negative correlation.
- This study found that while ipsilesional VOR gain in VN patients was not significantly influenced by head impulse velocity, contralesional VOR gain, which remained relatively normal, was affected, indicating that current vHIT velocity guidelines may not adequately detect subtle vestibular deficits.
- These findings suggest the need for further research to standardize the vHIT protocols, potentially improving the assessment and management of vestibular disorders.

Statistical Analysis

The statistical analysis was conducted using Statistical Package for the Social Sciences (SPSS) software version 27.0 (IBM SPSS Corp.; Armonk, NY, USA). The normality of continuous data was assessed using the Shapiro–Wilk test. The threshold for statistical significance was established at $P < .05$. Quantitative parameters are expressed as the average value ± the standard deviation. In this study, the selection of statistical tests such as the independent *t*-test, Mann–Whitney *U*-test, and Spearman correlation analysis was based on the normality of the sample.

RESULTS

Clinical Vestibulo-ocular Reflex Properties for Patients with Vestibular Neuritis

The differences in VOR gain and head impulse velocity between the ipsilesional and contralesional sides evaluated using vHIT are detailed in Table 1 and Figure 1. The VOR gains were smaller, predominantly in the ipsilesional HC (iHC) (0.42 ± 0.20) and 0.92 ± 0.16, $P < .001$ in contralateral HC (cHC), independent *t*-test) and AC (0.56 ± 0.20 in iAC and 0.89 ± 0.12 in cAC, $P < .001$, Mann–Whitney *U*-test), and were reduced by 48%-51% contralesionally, leading to marked asymmetry. However, the PC gain was not significantly different from the contralesional side (0.82 ± 0.14 in iPC and 0.88 ± 0.17 in cPC, $P = .19$, independent *t*-test). The head impulse velocity during the vHIT was not significantly different in each semicircular canal plane (Table 1). The mean head impulse velocities and confidence intervals are listed in Table 1. In the bithermal caloric test, canal weakness was measured to be 55.07 ± 24.64%. Among the patients, 29 (90.6%) showed abnormal canal weakness, defined as being greater than 25%.

Correlation Between Vestibulo-ocular Reflex Gain and Head Velocity

In the correlation analysis, the vHIT gains of the iHC ($R^2 = 0.06$, $P = .170$, Spearman's rank correlation) and iAC ($R^2 = 0.03$, $P = .372$, Spearman's rank correlation) groups were not significantly associated with head velocity (Figure 2). However, the relatively preserved VOR gain of the iPC group compared with that of the iHC and iAC groups was negatively correlated with head velocity ($R^2 = 0.20$, $P = .011$, Spearman rank

Table 1. Vestibulo-ocular Reflex Gain and Head Velocity of Each Semicircular Canal in Patients with Vestibular Neuritis

	Gain		*P*	Head Impulse Velocity (°/s)		*P*
	I	*c*		*i*	*c*	
Horizontal Canal	0.47 ± 0.20 (0.40-0.55)	0.92 ± 0.16 (0.86-0.97)	*<.001*[a]	162.81 ± 23.28 (154.42-171.21)	153.34 ± 22.08 (145.38-161.30)	*.09*[b]
Anterior Canal	0.56 ± 0.20 (0.48-0.63)	0.89 ± 0.12 (0.85-0.94)	*<.001*[b]	126.74 ± 16.02 (120.96-132.51)	123.03 ± 13.48 (118.17-127.89)	*.42*[b]
Posterior Canal	0.82 ± 0.14 (0.77-0.87)	0.88 ± 0.17 (0.82-0.94)	*.19*[a]	122.13 ± 12.35 (117.67-126.58)	123.72 ± 16.10 (117.91-129.53)	*.89*[b]

The value in parentheses below the mean ± SD represents the 95% CI.
[a]Independent *t*-test.
[b]Mann–Whitney *U*-test.

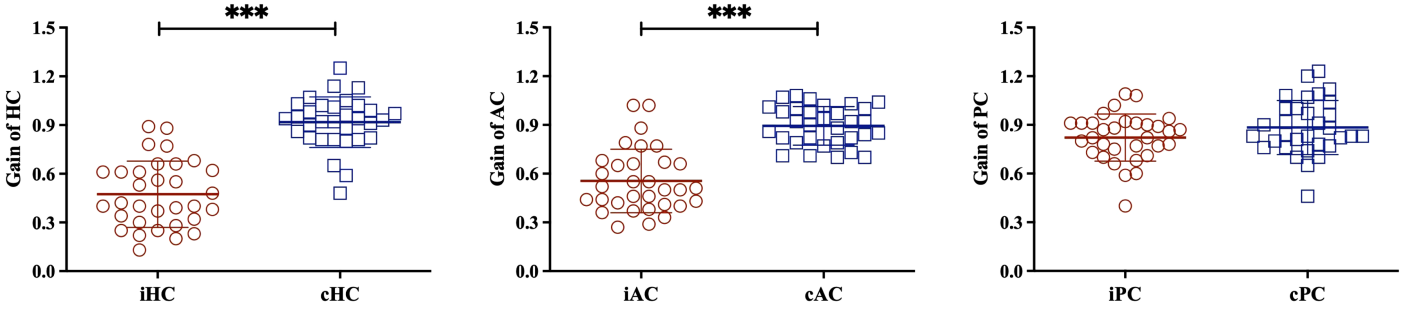

Figure 1. The VOR gain of the ipsilesional (i) and contralesional (c) sides of 3 semicircular canals in patients with vestibular neuritis. Compared with contralesional sides, the VOR gain of the ipsilesional sides was reduced significantly in the HC and AC. ***$P < .001$. AC, anterior canal; c, contralesional; HC, horizontal canal; i, ipsilesional; PC, posterior canal; VOR, vestibulo-ocular reflex.

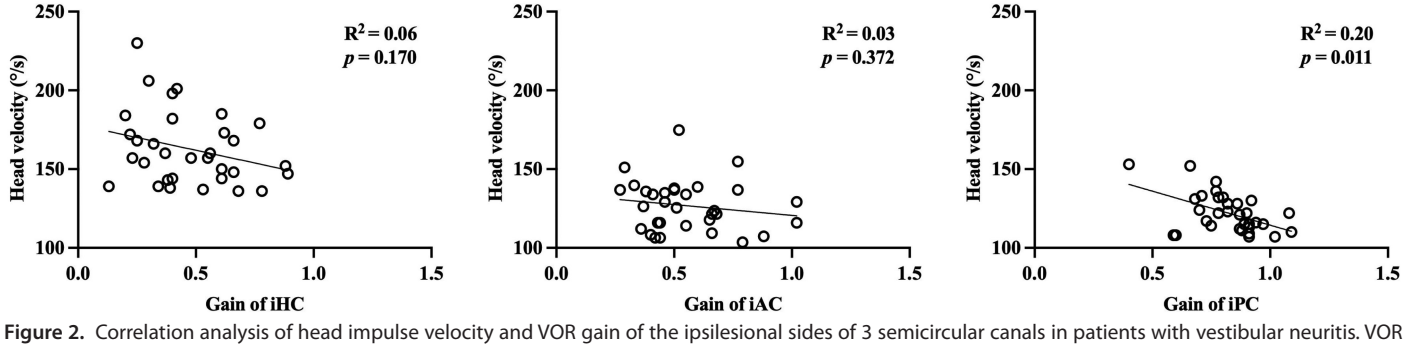

Figure 2. Correlation analysis of head impulse velocity and VOR gain of the ipsilesional sides of 3 semicircular canals in patients with vestibular neuritis. VOR gain tended to decrease with increasing head impulse velocity in all semicircular canals, but statistical significance was found only in the posterior canal, which had relatively preserved gain. AC, anterior canal; HC, horizontal canal; i, ipsilesional; PC, posterior canal; VOR, vestibulo-ocular reflex.

correlation) (Figure 2). In contrast to the ipsilesional sides, the vHIT gain on the contralesional sides of all 3 semicircular canals showed a negative correlation with head velocity ($R^2 = 0.24$, $P = .004$ in cHC; $R^2 = 0.17$, $P = .021$ in cAC; and $R^2 = 0.24$, $P = .005$ in cPC) (Figure 3). We further analyzed the correlation between head velocity and the incidence of corrective saccades, including covert or overt saccades, during all vHIT trials. Corrective saccades were observed in 87%, 30%, and 19% of HCs, ACs, and PCs, respectively. On the contralesional sides, corrective saccades were observed in 8%-13% of cases for each semicircular canal, which is consistent with other reports.[18] However, no significant differences were observed between the incidence of corrective saccades and head velocity.

DISCUSSION

This retrospective study analyzed the effect of head impulse velocity on bilateral VOR properties, including gain and the presence of corrective saccades, in patients with VN using vHIT. The results of this study revealed that the VOR gain in the iHC and iAC groups was not significantly associated with head impulse velocity. However, the relatively preserved VOR gain in the iPC was negatively correlated with head impulse velocity ($R^2 = 0.20$, $P = .011$, Spearman rank correlation). In contrast, on the contralesional sides, the VOR gain in all 3 semicircular canals was negatively correlated with head velocity ($R^2 = 0.24$, $P = .004$ in cHC; $R^2 = 0.17$, $P = .021$ in cAC; and $R^2 = 0.24$, $P = .005$ in cPC) (Figure 3). These results indicate that the influence of head impulse velocity on VOR gain differs between lesioned vestibular systems, suggesting a complex interaction and compensatory mechanism that depend on the degree of residual canal function.

Although there is some variation in reports, the VOR gain in normal individuals is typically around 1.0, with no significant effect of age.[13,18,19] In normal vestibular function, VOR gain is known to remain around 1.0 on average, due to a linear increase in the VOR-induced eye movements with increasing head impulse velocity, up to 350°/s.[19] However, several studies have investigated the effect of head impulse velocity on VOR gain in normal populations. Marion-Soler et al.[13] reported the effects of different ranges of head impulse velocities from 70°/s to 200°/s on VOR gain and found that VOR gain reduced with increasing head impulse velocity. Pogson et al[14] also found a negative correlation between the head impulse velocity and VOR gain. In fact, these reductions in VOR gain should be viewed as a process whereby the relatively high gain at low velocity normalizes to 1.0, as velocity increases; hence, the authors emphasize the importance of sufficient head impulse velocity to avoid missing unilateral vestibular loss.[20] In general, the oculomotor control system (smooth pursuit response, optokinetics, cervico-ocular reflex) is involved in vision stabilization in head velocity of under 100°/s in conjunction with the VOR system, referred to as vision-vestibular interaction; therefore, head impulse velocities of 200-400°/s are known to be appropriate for evaluating VOR using pure vestibular stimulation.[21,22] Recently, the vHIT manufacturer's recommended head impulse velocity is 150-200°/s for the HC and 100-150°/s for the vertical canals, which means that the vHIT can sensitively measure VOR at a lower velocity[23] and compensate for technical limitations such as goggle slippage at excessive head velocity.[12] Vestibular neuritis is the most common peripheral cause of acute vestibular syndrome (AVS) and is characterized by the presence of acute vertigo, spontaneous nystagmus beating toward the contralesional side, and gait instability lasting for more than 24 hours.[6] On vHIT, ipsilesional VOR gain reduction with large corrective saccades is a characteristic finding in patients with VN.[3,19] However, several studies also observed a subtle reduction in VOR gain on the contralesional side.[2,24] Subtle contralesional gain reductions possibly reflect contributions to the VOR of vestibular commissural inhibition by the contralesional semicircular canals. Thus, the contralesional gain of the VOR in patients with VN can be considered a potential deficit in the VOR.[19] In normal vestibular function, eye movements are thought to increase linearly beyond normal impulse velocities, with the VOR gain remaining around 1.0 on average. In contrast, compensatory eye movements induced by the VOR do not increase proportionally as the head impulse velocity increases in the case of vestibular deficits; therefore, it is believed that the faster the velocity, the lower the VOR gain. A VOR with a highly accelerated head impulse is simultaneously saturated by ipsilateral excitatory and contralateral inhibitory signals which can be explained by the push–pull arrangement. The contralesional afferents are progressively suppressed as head velocity increases, until mainly the excitatory afferents from the ipsilesional side contribute to the VOR.[25] In the deficit of excitatory input from the ipsilesional side, the VOR gain decreases rapidly as the head velocity increases. When the disfacilitation of afferents from the contralesional side is absent, the VOR gain falls less as velocity increases because its contribution is limited at higher velocity. Despite this theoretical background, in this study, the ipsilesional gain of the VOR was not significantly affected by the applied head impulse velocity, except for the PC, but the contralesional VOR, which has a relatively normal gain, was found to be affected despite adherence to the recommended velocity by the vHIT manufacturer. This means that the current manufacturer's vHIT head impulse velocity guidelines (HC < 200°/s, AC and PC < 150°/s) can yield relatively sensitive gain values in the presence of vestibular deficits but are insufficient to detect subtle deficits.

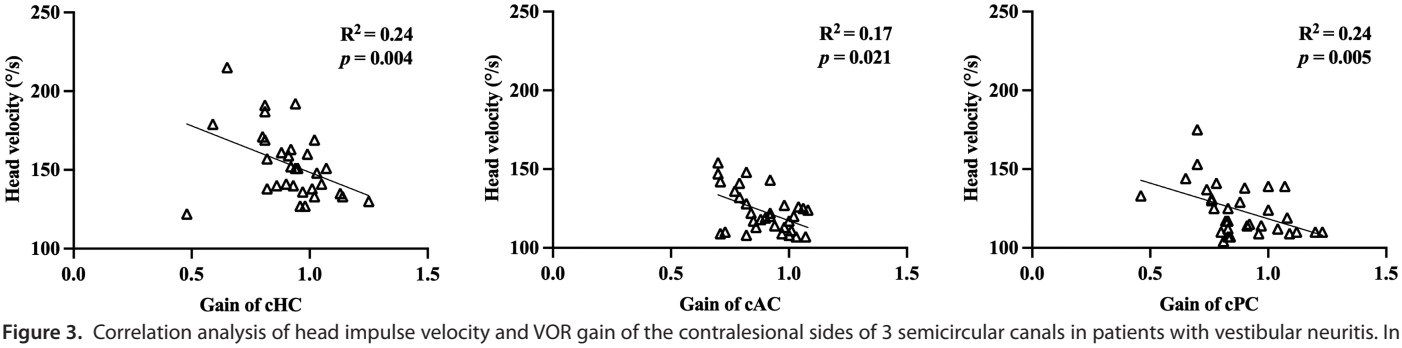

Figure 3. Correlation analysis of head impulse velocity and VOR gain of the contralesional sides of 3 semicircular canals in patients with vestibular neuritis. In contrast with the ipsilesional sides, the VOR gain of the contralesional sides of all 3 semicircular canals was significantly correlated with head impulse velocity. AC, anterior canal; c, contralesional; HC, horizontal canal; i, ipsilesional; PC, posterior canal; VOR, vestibulo-ocular reflex.

This study conducted a retrospective review of the medical records of 32 patients diagnosed with VN. It is important to note that this study has certain limitations. First, the small sample size makes it difficult to draw definitive findings. Second, the retrospective nature of the study does not completely rule out bias based on patients' selection criteria. Third, the recruited patients were mostly of the superior or total type of VN, and isolated inferior VNs were not included. This may have led to relatively preserved iPC gains in this study, which may be a confounding factor in the analysis. Fourth, the study included 5 patients who underwent vestibular function testing in the subacute phase,[26] 3 days after symptom onset.

In conclusion, this study provides insights into the relationship between head impulse velocity and VOR properties in patients with VN using vHIT. Head impulse velocity may have a differential impact on VOR gain, depending on the degree of deficit. These findings highlight that current head impulse velocities may provide adequate sensitivity for detecting VOR deficits; however, in the situation of a relatively normal VOR, increasing head velocity in vHIT may be considered to identify subtle deficits on the contralesional side of patients with VN. Standardizing head velocity during the vHIT may enhance the accuracy and clinical utility of this test for evaluating vestibular function in patients with VN. Further research is warranted to expand our understanding of optimal head velocity specifications and their impact on VOR assessment in various vestibular disorders.

Ethics Committee Approval: This study was approved by the Ethics Committee of Chosun University Hospital (Approval No: CHOSUN 2023-07-020; Date: July 20, 2023)

Informed Consent: Verbal informed consent was obtained from the patients who agreed to take part in the study.

Peer-Review: Externally peer-reviewed.

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Declaration of Interests: The authors have no conflicts of interest to declare.

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