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General review

Vestibular-ocular reflex dysfunction following mild traumatic brain injury: A narrative review

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ABSTRACT

Mild traumatic brain injury (mTBI) is a prevalent injury which occurs across many populations, including children and adolescents, athletes, military personnel, and the elderly. mTBI can result in various subjective symptoms and clinical deficits, such as abnormalities to the vestibulo-ocular reflex (VOR). Over 50% of individuals with mTBI are reported to have VOR abnormalities, which strongly contribute to feelings of dizziness and unsteadiness. Dizziness is a strong predictor for prolonged recovery following mTBI and is additionally linked with mental health difficulties and functional limitations affecting likelihood of return to work. Early diagnosis, and subsequent treatment, of VOR deficits following mTBI may greatly improve recovery outcomes and a patient's quality of life, but a thorough comprehension of the related pathophysiology is necessary to understand the assessments used to diagnose VOR abnormalities. Therefore, the purpose of this article is i) provide readers with an introduction on the VOR physiology to facilitate understanding about mTBI-related abnormalities, and ii) to discuss current assessments that are commonly used to measure VOR function following mTBI. As the VOR and oculomotor (OM) systems are heavily linked and often work in tandem, discussion of the relevant aspects of the OM system is also provided.

1. Introduction

Each year, an estimated 69 million individuals will suffer a traumatic brain injury [1] with an estimated 80-85% classified as mild (mTBI) [1–3]. The forces involved in the traumatic events responsible for mTBI can cause the brain to rotate and/or collide against the skull (through what is often referred to as a coup-contrecoup mechanism) [4], transmitting and reflecting pressure waves through the brain [5], and resulting in mTBI's focal and/or diffuse pathology [6–8]. The resulting pathophysiology is dictated by the primary injury and secondary responses that occur [5,9], involving complex reactions and a neurometabolic cascade [5,10]. The primary injury is the immediate result of the traumatic event and the mechanical forces applied, which can cause distortion, shearing, impact to the cell membranes, compression, rotation, translation, laceration, and vascular injury affecting certain brain structures [5,9,11]. These disturbances dictate the extent of the secondary response post mTBI [5,12], which is non-mechanical in nature, factors into the overall heterogeneity of mTBI [13], and consists of the neurochemical and neurometabolic events as well as their consequences [5,11].

When considering the difference between the focal and diffuse injuries that may occur, the former are generally caused by a direct impact to one's head [6], while the later injuries (such as concussion) are often caused by inertial loading, rotational, acceleration, and deceleration forces [6,13]. These diffuse injuries cause stretching, shearing, and/or tearing of the axons (diffuse axonal injury, DAI) [9,12,14], leading to damage to axonal membranes and causing local transport impairment and subsequent detachment over time [15–17]. These damages prompt what is often called the neurometabolic cascade, causing dysregulation in neurotransmitter release and ion fluxes that result in temporary changes to cellular pathology during the acute and sub-acute period following mTBI [18,19]. Broadly, DAI can lead to widespread changes, compromising neuronal synchrony, firing rates [20], and affecting neural pathways [21]. While white matter abnormalities are predominant

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following mTBI and are identified in many cortical and subcortical structures [19], gray matter and cerebral vascular tissue changes can also result from diffuse injuries [22,23].

1.1. Visuo-vestibular implications of mTBI

The diffuse nature of mTBI can cause abnormalities in a wide array of clinical domains. Deficits to the visual and vestibular systems are of particular concern due to their important contributions, both separately and together, to support one's overall daily functioning and ability to interact with their surrounding environment. These two systems are highly interconnected and particularly vulnerable following mTBI due to the wide range of neural pathways, cortical and subcortical structures involved in their functioning [24–26]. More specifically, abnormalities or impairments to the vestibulo-ocular reflex (VOR) and oculomotor (OM) system are frequently reported in mTBI populations with rates ranging from 29-69% [27–29].

The VOR allows us to maintain gaze stability when our head is in movement, and thus impairments to this reflex following mTBI can lead to an inability to steady one's gaze during head movement causing blurred vision, dizziness, vertigo, swaying sensations, and balance-related repercussions [30,31]. The OM system supports VOR function by helping to produce the necessary eye movement responses. Individuals demonstrating symptom provocation induced by VOR and OM tasks following mTBI have been shown to experience a longer time to recovery [28,32,33] and be at higher risk for re-injury [34]. With regards to daily functioning, a compromised VOR and/or OM system can lead to difficulty reading, writing, driving, concentrating, and performing daily tasks [35–39]. Unsurprisingly, these deficits can have further psychological implications, negatively affecting an individual's quality of life.

Understanding the physiology of the VOR and OM systems is necessary to grasp both how VOR deficits can occur following mTBI and how clinical and computerized assessments can detect these. Therefore, the purpose of this article is:

- to provide readers with an introduction of the VOR physiology to facilitate understanding mTBI-related abnormalities;
- to discuss current assessments and tools that are commonly used to measure VOR function following mTBI.

As the VOR is heavily tied with the OM system, an overview of the relevant aspects of the OM system is also provided below.

2. How do the OM-VO systems work?

2.1. Vestibulo-ocular reflex

The vestibular and visual systems work alongside one another to optimize vision. The vestibular system consists of a peripheral sensory apparatus, a central integration center, and a motor output (Fig. 1) [40,41]. It can be divided into two subsystems:

- the vestibulo-ocular system responsible for visual/gaze stabilization, acuity, and the development of visual spatial and perception abilities [41];
- the vestibulo-spinal system responsible for maintaining the body's orientation in space and contributing to postural tone [41,42].

The peripheral sensory apparatus lies in the bony labyrinth of the inner ear and is composed of 3 semi-circular canals (lateral, anterior, and posterior), and 2 otolith organs (the utricle and the saccule). The three semi-circular canals and their neural elements detect angular acceleration in the three planes of motion, while the otolith organs detect linear acceleration and gravitational changes [42,43]. The central vestibular system receives and processes the majority of these afferent signals in the vestibular nuclei [41,42,44], with the cerebellum receiving the remaining signals [42]. The integration of vestibular, visual, and somatosensory inputs occurs in the vestibular nuclei allowing interactions with the oculomotor nuclei, the spinal cord, the cerebellum, the autonomic system, the thalamus, and the contralateral nuclei [42,45]. The resulting motor output addresses posture, locomotion, spatial orientation, and gaze stabilization [42,44]. This motor output results from vestibular stimulation and is integrated into three reflexes: the vestibulo-ocular reflex (VOR, helps maintain gaze stability while body is in motion), vestibulo-spinal reflex (VSR, helps maintain posture and orientation of the body in space through the myotatic reflex), and vestibulo-collic reflex (VCR, helps to stabilize the head and neck) [42,43].

The VOR has an important role in eliciting the responses that enable humans to stabilize images on the fovea, keeping vision clear during rapid head movements [46]. Damage to the peripheral vestibular system can result in impaired VOR function as the activation of the sensory organs within it, the semicircular canals and otolith organs, produce a signal that is sent to the eye muscles through the VOR [31]. Damage to the semicircular canals affect the VOR more strongly than damage to the otolith organs as the direct pathways for compensatory eye movements from rotational and angular acceleration, detected by the semicircular canals, are greater than those for linear acceleration, detected by otoliths [46]. The sensory input received from the vestibular system through the VOR is combined with OM eye movements in gaze control to maintain clear vision [26,47]. A lack of harmonious communication between visual inputs and vestibular inputs is often the cause of the vestibular symptoms being experienced. In this instance, the VOR will not produce signals for an eye movement equal and opposite to the head movement sensed in the peripheral apparatus.

2.2. Oculomotor system

The visual pathways span the length and all 4 lobes of the brain, while approximately 70% of the sensory input to the brain stems from visual inputs [48]. There are 32 cortical areas [49] and various subcortical structures involved in vision and linked through myelinated axon (white matter) tracts [50,51]. The breath of pathways and structures contributing to visual function explain its impressive precision, while also highlighting its great vulnerability to injury.

In order for visual function to remain intact, sensory input (afferent pathway), central integration, motor output (efferent pathway), and higher order visual processes must all function optimally. Visual function contains two pathways: afferent and efferent. The afferent pathway receives and processes visual inputs (i.e. visual acuity, color, contrast sensitivity), while the efferent pathway moves the eyes, anticipates visual stimuli, and produces outputs accordingly [52–54]. Efficiency between afferent and efferent neural interconnections in cortical and subcortical structures of the brain and with cranial nerves is necessary for optimal vision [52].

OM contributions supporting VOR function can come from version and vergence eye movements which are facilitated by the six extraocular muscles around each eye required for OM function [48,51]. These are organized into three agonist/antagonist pairs allowing for abduction/adduction, elevation/depression, and intorsion/extorsion [51,55]. Version eye movements are conjugate eye movements that keep an image stable on the fovea while tracking and/or glancing back and forth between targets [56]. These include saccades (rapid ballistic eye movements that allow one to change fixation from one target to another) and smooth

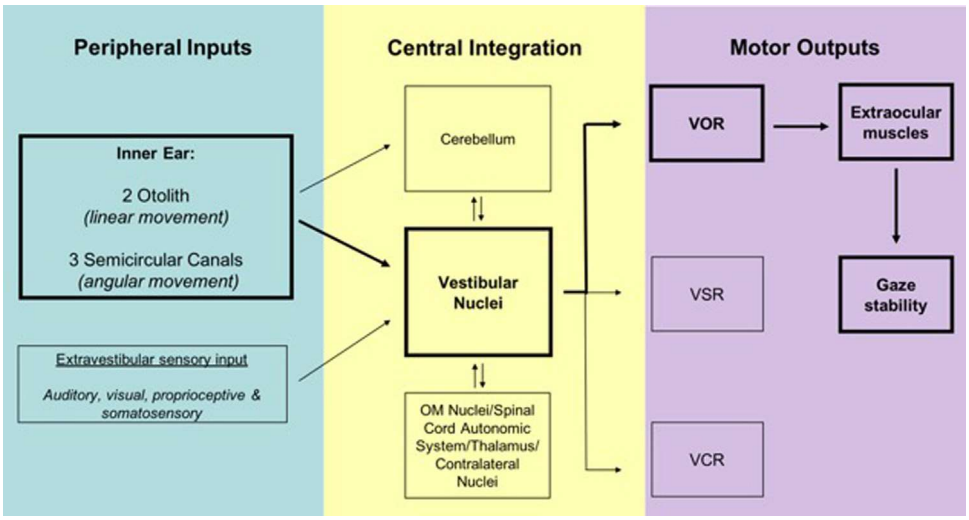

Fig. 1. A simplified depiction of the pathophysiology driving the visual-vestibular systems, with the Vestibulo-Ocular Reflex (VOR) pathway highlighted in bold. Inputs from the peripheral sensory apparatus (otolith organs, semicircular canals, and extravestibular contributions) are integrated primarily by the vestibular nuclei, with most remaining signals sent to the cerebellum. Constant feedback between various structures and the vestibular nuclei is necessary to integrate visual, vestibular, and somatosensory feedback (called central integration), and produce appropriate motor responses. Motor outputs are integrated into three reflexes: the Vestibulo-ocular reflex (VOR), vestibulo-spinal reflex (VSR), and vestibulo-collic reflex (VCR). The motor outputs of the VOR communicate with the extraocular muscles to produce eye movements of equal and opposite direction to head movement, a process called gaze stability.

pursuit eye movements (slow eye movements allowing one to track moving objects smoothly by focusing the image on the eye's fovea) [57,58]. Vergence eye movements are disconjugate movements of the eyes that require simultaneous adduction or abduction of the eyes to maintain a binocular fusion on near targets [25,54]. These include convergence, divergence, and contributions from the accommodation system, together driving the eyes inward or outward in order to keep appropriate fixation and focus on objects [48]. Visual function relies on the integrity of a series of cranial nerves, with cranial nerves III, IV, and VI in specific involved in fine oculomotor control [35]. Eye movements are generated through these relevant cranial nerves which innervate the twitch and non-twitch muscle fibers that control the extraocular muscles [59]. OM movements only represent the first steps in a complex process required for overall visual function.

3. How is the VOR and the OM system affected post-mTBI?

Numerous clinical deficits can emerge following a mTBI and each patient presents with a unique combination of subjective symptom complaints and objective abnormalities. The heterogeneous injury responses following mTBI are linked to the mechanism of injury [9,14], injury biomechanics [9,11], the diffuse vs. focal nature of the injury [13], individual anatomical differences [19,60], and the susceptibility of certain structures to injury [7,61]. These factors lead to highly variable manifestations of visual-vestibular dysfunctions following mTBI, reflecting the potential global and far-reaching effects of mTBI on visual and vestibular function.

3.1. Vestibulo-ocular reflex abnormalities/impairments

Abnormalities in the VOR following mTBI have been known to contribute feelings of dizziness, vertigo, disequilibrium, visual motion sensitivity and overall unstable sensations [26,30,31]. Moreover, problems of oscillopsia [45,48], impaired fixation, visual tracking, instability and blurred vision have been commonly associated with VOR pathologies [45,31,62]. Due to the multiple structures involved in integrating afferent signals and creating the motor output of the vestibular system, the source of the impairment for vestibular pathologies as a whole is not restricted to the peripheral vestibular apparatus but rather can come from deficits in any interconnected structures [63–65]. In addition, the peripheral vestibular apparatus is challenging to evaluate as it is located in such a small space as the labyrinth of the inner ear [44].

3.2. Oculomotor system abnormalities/impairments

Frequent OM abnormalities post-mTBI occur in: accommodation [53,66], saccades [53,66,25], smooth pursuits [53,66,25], convergence [53,66,25], extraocular mobility [66–68], and pupillary function [54,66]. In addition, impairments or abnormalities to cranial nerves, visual acuity, pupillary response time, strabismus, stereopsis, nystagmus, the visual field, contrast sensitivity, color vision, various cortical and subcortical structures, and the visual pathway can occur following mTBI [53,69,70], with the *potential* to affect OM function. Specifically, areas *involved* in the OM functions known to be affected by mTBI include: the primary and extrastriate visual cortices, dorsolateral prefrontal cortex, thalamus, cerebellum, brainstem, posterior parietal cortex, frontal supplementary eye fields [69–71]. As the OM system supports VOR function, the eye movements driven by this system must be considered when evaluating for VOR abnormalities following mTBI.

4. How is the VOR evaluated following mTBI?

Understanding the physiology of the VOR and the OM system is important as the complex interconnections they share render it challenging to isolate the pathophysiological mechanisms underlying visuo-vestibular abnormalities or impairments following mTBI. Assessments following such injuries have traditionally included stationary balance tests, focused on the vestibulospinal component of the vestibular system, and only recently evolved to include measures of vestibulo-ocular components [72,73]. As the ability to maintain gaze stability stems from oculomotor function working in combination with the VOR [48,74], the ideal battery of measures should evaluate OM function and VOR in order to locate the source of the impairment, determine its severity, and inform one's rehabilitation. However, as the focus of this article centers on the VOR, assessments that solely assess OM function will not be discussed. While progress has been made recently in the development

Table 1
Popular clinical and technology-based assessments used to measure VOR and OM functioning.

Assessment	Methodology	Advantages	Limitations
Caloric Testing	Cold or warm water/air is irrigated into the external auditory canal, creating a conductive current in the endolymph of the semicircular canals. Reactive eye movement would be the expected response, thus if absent, it is indicative of vestibular hypofunction of the horizontal semicircular canals	"Gold standard" study for detecting unilateral vestibular loss Determines which side is contributing to vestibular hypofunction Effective for testing very low frequencies (< 0.01 Hz)	Intensity of caloric stimulation depends on anatomy and technique of irrigation Requires equipment and trained administrators Less sensitive and specific than rotational chair testing for bilateral vestibular loss Vertical semicircular canals are not assessed Rarely used in children < 6 yrs
Rotational Chair Testing	Participant is situated in a rotating chair so that the head and body move in unison with the chair. The chair rotation is controlled by a computer, such that multiple velocity and frequency combinations may be tested in a single session. Eye and head movement are compared to calculate VOR gain and phase shift	"Gold standard" study of detecting bilateral vestibular loss Determines the extent of the central nervous system compensation to vestibular hypofunction Effective for testing low frequencies (0.01–1.0 Hz)	Not widely available/requires expensive equipment Less sensitive than caloric testing for unilateral vestibular hypofunction Vertical semicircular canals are not assessed Can test sinusoidal harmonic acceleration OR step testing, not both Restrictions in equipment and administration with younger children
Dynamic Visual Acuity	Clinical: Patient reads the lowest possible from the Snellen eye chart while static. Depending on the protocol, the examiner or patient than rotates the patient's head while the/she again reads the lowest possible line. A positive test result is 3+ lines difference between static and dynamic conditions Technology: The letter "E" is placed on a computer screen and the direction of the letter's orientation is randomly changed. While the head is rotated between 120 to 180°/sec, the patient identifies the orientation of the "E"	Effective for testing mid-range frequencies (2 Hz) Can identify both unilateral and bilateral vestibular loss More ecologically valid Reliability is well-established	Does not differentiate between central and peripheral causes Requires administration by a trained professional (clinical) and/or expensive equipment (technological) Little data available for younger children and adolescents. Unable to exclude the contributions of non-vestibular mechanisms
Head Thrust Test	Clinical: Patient is asked to fix their gaze on a target while their head is rapidly rotated in each direction of the semicircular canals (i.e. left/right horizontal, anterior, and posterior canals). If the VOR is impaired, a loss of gaze fixation will occur when the head is rotated towards the affected canal followed by a corrective saccade back to the fixation point Technology: The clinical head thrust test is completed while wearing video-oculography goggles, which allows for more precise quantification of VOR gain and corrective saccades	Effective for testing high frequencies (5 Hz) Can detect both bilateral and unilateral semicircular canal problems Measures all 3 pairs of semicircular canals, including the vertical canals Does not invoke dizziness in patients More appropriate for children than caloric or rotary chair testing	Unable to exclude the contributions of non-vestibular mechanisms Requires administration by a trained professional While unilateral function can be evaluated, may not be as sensitive to deficits as caloric testing Technology-based assessment requires expensive, although compact and portable, equipment Vertical canals are more difficult to assess
Scleral Search Coil	A narrow-gauge coil wire is embedded in a pliable plastic lens and placed on the patient's eye. Patients are asked to focus on a target while head impulses are applied. The coil records electric currents induced by a magnetic field during testing	"Gold standard" for high velocity eye and head movements Can quantify the "pathophysiological signature" of OM deficits Highly reliable	Invasive May cause patient discomfort Ophthalmic anesthetic are required Requires administration by a trained professional Recording time is limited to 30 minutes Not suitable for children
VOMS	Participant completes a series of OM (smooth pursuits, saccades, and convergence) and VOR (VOR test, visual motion sensitivity) assessments. Before and after each test, the participant ranks symptoms of headache, dizziness, nausea, and fogginess from 0 (not present) to 10 (severe)	Low cost (does not require equipment) Feasible (can be done in virtually any setting) Requires no/little equipment Individuals can be easily trained to administer the test Contains both VOR and OM assessments	Outcome is subjective (based on participant's self-reported symptoms) Does not quantify actual eye movement (outcomes are based on symptom provocation) Subtle deficits in OM functioning are easily missed No research available in young children
Electronystagmography	Electrodes are placed around the nose and eyes. Participants are asked to complete a test battery with the electrodes in place, typically including the caloric exam, measures of oculomotor function (gaze, stability, smooth pursuit, and saccades), and positional testing	Helps to determine whether dizziness is central or peripheral in origin. Quantitative assessment of eye movement Contains both VOR and OM assessments	Cannot measure torsional eye rotations, absolute eye position, or rapid eye movements Requires expensive equipment and trained examiners Difficult to calibrate in children under 3 years old due to attention span and physical capabilities

of newer measurement tools to evaluate VOR, the gold standard to objectively measure the VOR remains the same as it has for decades, and is a measure of broader vestibular function [75]. A description of common tests to evaluate VOR function are discussed in following paragraphs, with their advantages and limitations outlined in Table 1.

4.1. Gold standard tests

As the VOR signals originate primarily in the semicircular canals with small contributions from the otolith organ signals [46], a particular emphasis is put on semicircular canal function when evaluating the VOR. Currently, the gold standard tests for adults and children remain the caloric test and rotational chair test [76,77]. Caloric testing is important in determining unilateral vestibular hypofunction [77,78]. The rotational chair test is useful in determining bilateral peripheral vestibular loss [77]; however, a major downfall is that it does not detect unilateral vestibular hypofunction [77,78]. The challenge with these tests is that they are performed at low velocities and thus do not stimulate the vestibular system at an amplitude comparable to activities of daily life [46,79]. While the caloric test and the rotational chair test are considered the gold standard, they do not have the ability to conclusively define individuals who have a vestibular disease as they do not test all components of vestibular function [80] or evaluate the interconnected OM system. One measure that does assess both eye movements and VOR function is the scleral search coil [81,82]. However, while addressing a major downfall of the rotational chair test and caloric test by measuring high velocity/acceleration vestibular eye movements [82], as well as measuring horizontal, vertical, and torsional eye movements [81], this assessment requires extensive training to administer and is highly invasive for the patient.

4.2. Clinical tests

Overall, many vestibular tests focus on the horizontal semicircular canals as it is the most accessible arm of the vestibular system [42,77]. In the last decade, progress has been made surrounding measures of otolith function (such as the vestibular evoked myogenic potential test) [65,78]. However, fewer assessment tools are generally available for clinicians to evaluate the otolith organs and the vertical semicircular canals [65,83]. While this is understandable for VOR testing as the semicircular canals are more heavily involved in signaling to the extraocular muscles in order to maintain gaze stability, such tools are still important in order to be able to detect specific dysfunctions within the peripheral labyrinth and differentiate between central and peripheral contributions to the dysfunction. Appropriately combining results from existing tests can provide clinicians with a picture of the vestibulo-ocular component of the vestibular system as a whole.

VOR-specific clinical tests that are commonly used in mTBI populations are the head thrust test, the clinical dynamic visual acuity test, and the vestibular/oculomotor screen. The head thrust test can be used to evaluate unilateral peripheral dysfunction of vestibular system through the individual's ability to stabilize their vision during the head thrust maneuver [73]. The clinical dynamic visual acuity test detects peripheral vestibular dysfunction by measuring the visual suppression of VOR by comparing the static visual acuity to the dynamic visual acuity [79,84,85]. While the dynamic visual acuity test can detect peripheral vestibular dysfunction using higher frequencies more comparable to activities of daily life [31,47], a limitation exists in that positive results may be indicative of problems with central integration rather than a dysfunction in the peripheral system [79]. Additionally, the dynamic visual acuity test's sensitivity and specificity are highly variable as the methodology used in its administration is not uniform [46]. The vestibular/oculomotor screening tool (VOMS) measures smooth pursuit, horizontal and vertical saccades, near point of convergence distance, horizontal and vertical VOR, and visual motion sensitivity [86]. While the VOMS evaluates both VOR and OM components, it predominantly assesses only subjective symptom provocation induced by these tasks [86]. In general, the downfall to the clinical tests described here is that they lack precision, quantifiable changes, comprehensiveness, and are variable depending on their administration and the methodology used.

4.3. Computerized tests

To address many of these downfalls, measures quantifying eye-movements have recently evolved. To improve the clinical head thrust test, the video head impulse test has emerged recording overt and covert saccades, VOR gain, and asymmetry [46,87]. The device uses high-speed cameras and inertial sensors to calculate horizontal, vertical, and torsional eye movements [88]. A technological parallel to the clinical dynamic visual acuity test recently developed is the computerized dynamic visual acuity tests that provide both a quantifiable dynamic visual acuity component and a gaze stability component [85]. The dynamic visual acuity component assesses dynamic visual acuity at a constant velocity in both the yaw and pitch planes [85,89] and the gaze stability component estimates the maximum velocity at which one can maintain gaze stability [85]. Videonystagmography would be the computerized complement to the VOMS to quantify performance during the VOR and OM eye movements [90]. While these more recent measures add objectivity and decrease the influence of human error or experience when administering the tests, limitations still remain as the protocols followed when administering such tests are heterogeneous and psychometric properties are still lacking in certain age-groups. Overall, not one measure comprehensively assesses all elements contributing to VOR function. This highlights the need to select complementary measures when assessing VOR function to ensure it is comprehensively evaluated.

5. Conclusion

The visual and vestibular systems are vulnerable to injury following mTBI due to the diffuse and multifactorial nature of the injury along with the vast interconnections between these systems. More specifically, as the VOR's ability to maintain gaze stability is heavily reliant on its ability to communicate with the extraocular muscles, deficits at any point along the pathways supporting this communication can result in VOR dysfunction that can greatly affect a patient's quality of life. It is crucial for clinicians to gain an understanding of the complexity of the VOR, along with its supporting OM system, to appreciate the necessity for comprehensive assessments of VOR function following mTBI. The current state of practice in evaluating VOR function following mTBI is heterogeneous and requires increased uniformity and standardization across clinical practice. Several tools are currently available to assess VOR function following mTBI, all of which possess a number of advantages as well as limitations. Future directions should further establish the psychometric properties of existing and emerging measures, which will allow clinicians to confidently interpret results. Additionally, a focus should surround establishing a battery of tests that will allow the assessment to:

- differentiate central from peripheral dysfunction in the vestibular system;
- differentiate unilateral from bilateral peripheral dysfunction;

- specify the structure within the peripheral labyrinth or VOR pathway that has been compromised;
- describe how the physiological deficit is affecting the VOR and OM response.

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