

dynamic and unpredictable physical motions be visually presented, and how can they be integrated into any virtual scene without reducing the immersion of the VR content.

This paper examines VR HMD use in-car, in-motion for the first time. Through a user study, we investigate whether consuming stationary 360° VR content in-car and in-motion induces motion sickness. We examine this both for existing consumer VR HMDs that interpret rotations of the car as rotations of the HMD, and future positionally-tracked HMDs that do not. We also examine to what extent motion sickness can be rectified by conveying motion peripherally, allowing for physical motion cues to be combined with any VR content. New baselines for VR motion sickness in-car are established and guidelines for future experimentation are provided, before we finally explore the further research required to enable comfortable VR HMD use in-motion.

## RELATED RESEARCH

### Motion Sickness

Motion sickness refers to illness arising from a person being within a motion environment, such as a moving vehicle [59]. Symptoms of motion sickness typically include cold sweat, dizziness and nausea/vomiting [52]. The predominant theory is that it arises due to sensory conflicts (also sensory re-arrangements or sensory mismatch), as first discussed by Reason and Brand [59]) between what Bertolini *et al.* describes as motion-sensitive input signals [3]. For example, if the motion perceived by the visual system conflicts with that perceived by other sensory systems there is a likelihood of motion sickness being induced. Reason and Brand [59] (as summarised by [5]) described there being two major categories of motion sickness, being derived from conflict between angular and linear vestibular systems (Canal-Otolith mismatch), and conflict between visual and vestibular systems (visual-vestibular mismatch).

The vestibular system (or inner ear) is essentially a human inertial motion sensor, able to detect rotational changes (equivalent to a gyroscope, sensed via the semicircular canals) and acceleration (equivalent to an accelerometer, sensed via the Otolith organs). This information is used alongside cues from the visual system and the somatosensory system (e.g. motor actions and proprioception) to determine a perception of self-motion. Reason and Brand suggested there were two types of conflict: type 1, where both systems signal contradictory motion information, and type 2, where one system signals motion whilst the other does not. The greater the discrepancy between the sensory information and the expected sensory information, the greater the chance of motion sickness occurring, and the greater the severity of the sickness [53, 5].

There are however other theories regarding the origins of motion sickness. The subjective vertical conflict theory [5] suggests that motion sickness arises from “situations where the determination of the subjective vertical, the internal representation of gravity, is challenged”, meaning movements where the reference point of gravity changes, e.g. roll and pitch movements when seated, but not yaw movements (side-to-side). For example, “driving uphill at night along a winding road may provoke car sickness in the passengers in the back seat. The

continuously changing gravito-inertial force vector, together with the inability of the semicircular canals to appropriately signal the angular motion because of the stable visual interior of the car... will subsequently provoke motion sickness.” [5]. The ecological theory of motion sickness suggests that it occurs due to motion causing postural instability:

“Animals become sick in situations in which they do not possess (or have not yet learned) strategies that are effective for the maintenance of postural stability (p. 195), and that postural instability... is necessary to produce symptoms” [60] from [8]

Indeed, studies have demonstrated that motion sickness can occur due to postural sway even without visual cues [54]. Postural sway has been considered as an indicator of the onset of motion sickness [12]. However, the fundamental causes of motion sickness are not yet fully understood, with suggestions that “an underlying central mechanism... driving both our posture and motion sickness symptoms” exists [8, 12].

Regardless of the underlying theory, it is well known that specific movements play significant roles in motion sickness. For example, Bles *et al.* noted that “linear acceleration and deceleration without appropriate view of the road ahead” induced sickness [5]. Lateral (bumps and undulations in a car ride) and vertical oscillations (at low frequencies, between 0.1 and 0.5Hz, peaking at 0.16Hz, e.g. the motion of a boat) both induce motion sickness [32]. The Coriolis or cross-coupling effect [30] is when nausea is provoked by head movements during yaw motion (i.e. where a conflict arises between the Canal-Otolith systems). Consider a car turning a corner, whilst a passenger additionally rotates their head. Depending on the directions of the rotation, the perceived rotational velocity may be very different to the actual rotational velocity. This is one of a number of effects experienced, particularly by pilots [55].

Finally, it is important to note that perception of motion is not uniform with respect to the field of view of the viewer:

“Peripheral vision is relatively better at detecting motion than form. A moving object seen in the periphery is perceived as something moving, but it is more difficult to see what that something is... A person’s ability to detect slow-moving stimuli decreases with eye eccentricity... For faster-moving stimuli, however, the ability to detect moving stimuli increases with eye eccentricity.” [34] from [1] and [14]

Indeed, Keshavarz *et al.* [38] noted the impact of peripheral vision on perception of motion, showing that having peripheral vision of a projection screen displaying vehicle motion caused greater visually-induced motion sickness.

### Occurrence and Prevalence

Motion sickness has three components: the characteristics of the stimulus, the susceptibility of the person, and the total time of exposure [59]. The result is that “anyone with a functional vestibular system can suffer from motion sickness, given the right prerequisites and if the exposure is continuous over a long period” [16] with studies showing that “virtually anyone with normal vestibular function when exposed to provocative physical body motion, disruption of vestibulo-ocular reflexes, or optokinetic stimulation can to some extent be made motion sick”. [40]. Thus prevalence tends to be categorised by severity of affliction. It has been suggested that approximately one-third of the population are highly susceptible to motion