

Circularvection: Visually-induced illusion of self-rotation

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The visual system exerts a powerful subcortical influence over neurons in the brainstem vestibular nucleus. Consequently, visual stimulation by a horizontally rotating peripheral visual field ('optokinetic stimulation'), in the absence of head movement, results in an illusion of self-motion known as 'circularvection' (CV). The aim of the present experiment was to extend the description of human CV using an optokinetic drum. Most subjects exposed to a 50°/sec optokinetic stimulus experienced Stage 2 CV (ie. 'egocentric + exocentric motion perception') within 10-15 secs of the onset of the stimulus. The latencies to Stage 3 CV (ie. 'exocentric motion perception') were significantly greater than those to Stage 2 CV ($p < 0.01$); however, the CV latency did not change significantly with increasing velocity (24 - 92°/sec). A significant decrease in CV latency occurred with increasing exposure to the optokinetic stimulus ($p < 0.0001$).

Spatial orientation relies upon the integration of sensory information from both the visual system and the vestibular system in the inner ear (see Howard, 1981 for a review). Eye movement reflexes such as the optokinetic reflexes (OKRs) and vestibulo-ocular reflexes (VORs) are essential in order to stabilize gaze and to perceive the visual world as stationary, during head movement (Howard, 1981; Young et al. 1973). Visual information converges on brainstem vestibular nucleus neurons via the accessory optic tract, the inferior olive and the cerebellar flocculus (see Baloh & Honrubia, 1990 for a review). This integration of visual input with vestibular information conveyed from the vestibular labyrinths, allows the OKRs and VORs

to generate compensatory eye movements in response to a broad range of head movement velocities (see Howard, 1981 for a review). Because the stabilization of gaze during head movement relies on the integrity of these reflexes, lesions within the OKR and VOR pathways can result in the apparent movement of the visual world during head movement, a condition known as oscillopsia (eg Zee, Yee & Robinson, 1976; McConnell, Darlington, Smith, Sturge, Thomson, Nukada & Mair, 1990; see Baloh & Honrubia, 1990 for a review).

Due to the modulation of vestibular nucleus neurons by optokinetic stimulation and the fact that the OKR and the VOR normally work together to provide adequate gaze stabilization during head movement, the occurrence of peripheral visual field movement in the absence of head movement results in a powerful illusion of self-motion which is indistinguishable from true motion (see Howard, 1981 for a review). This illusion, which in many ways is the perceptual opposite of oscillopsia, has been exploited in many amusement park rides and cinemas around the world. When the visual field movement is rotation in the horizontal or vertical planes (e.g horizontal rotation about the vertical axis using an 'optokinetic drum'), the illusion is known as 'circularvection' (CV) and is usually associated with optokinetic nystagmus (Brandt, Dichgans & Koenig, 1973). Neuronal studies have shown that optokinetic stimulation in the absence of head movement can induce firing patterns in vestibular nucleus neurons similar (but not identical) to those induced by actual head movement (Henn, Young & Finlay, 1974). Thus, CV can be viewed as a perceptual assay of the effects of optokinetic stimulation on vestibular nucleus neurons, and it is used clinically for this purpose (Baloh & Honrubia, 1990; Zee et al., 1976).

Most neurologists, ophthalmologists and otolaryngologists use a portable, hand-held 'rolling pin'-type optokinetic stimulus for the purpose of inducing optokinetic nystagmus in clinical settings. Although this kind of optokinetic stimulus is convenient and cheap, it has several disadvantages: 1) unless it is held at the correct distance from the patient, it will not fill the peripheral visual field and therefore the optokinetic stimulus will be inadequate; 2) there is no control over the velocity of the stimulus. Because such portable devices can be used anywhere, there is a tendency not to give the patient formal instructions. Since the frequency of optokinetic nystagmus can change depending upon whether or not the patient attempts to 'follow the stripes' (Howard, 1981; Baloh & Honrubia, 1990), different results can be obtained depending on the nature of the instructions and the patient's interpretation of them. By contrast, optokinetic drums (see below) offer a well-controlled environment in which to test patients OKRs and their corresponding perceptions (e.g. Zee et al., 1976). Although such drums require a reasonable amount of space (e.g. 3 x 3 metre room), they need not consist of anything more sophisticated than black and white plastic draped from a wheel with spokes extended to achieve a drum diameter of about 2 metres. A motor can be fitted; however, it is not strictly necessary provided that someone is prepared to push the drum and a tachometer is available to confirm the rotation velocity.

A distinction has been made between several different stages of CV (Brandt et al., 1973). In the first stage ('egocentric motion perception'), the subject perceives the visual field as moving and him/herself as stationary. In the second stage ('egocentric + exocentric motion perception'), the subject experiences self-motion in the opposite direction to the moving visual field. In the third and final stage of CV ('exocentric motion perception'), the subject experiences self-motion but perceives the visual stimulus to be stationary. In the context of the current experiment, the latter two stages will be referred to as Stage 2 and Stage 3 CV, respectively. The main aim of the present experiment was to obtain normative data on the latency to experience CV using a large number of subjects, with a view to future use of the data in the diagnosis of vestibular disorders.

Methods

A total of 92 subjects were used in the present experiment. Sixty of these were used to obtain data on latency to Stage 2 CV from a large sample of subjects in response to 1 velocity of drum rotation (50°/sec).

The remaining 32 subjects were used to investigate the latency to Stage 2 and Stage 3 CV as a function of drum velocity and the number of trials (see below). All experiments were carried out in accordance with University of Otago Ethics Committee guidelines.

CV was induced by seating subjects in an optokinetic drum which was 2 metres in diameter and 2 metres high. The interior of the drum was covered in black opaque plastic with vertical white stripes; the white stripes subtended 4°, and the black stripes 20°, of visual angle. Thus, the black and white vertical stripes completely filled the visual field. The drum was rotated around the subject in an anticlockwise direction. A tachometer attached to the drum enabled the experimenter to check drum velocity continuously during the experiment. In the initial experiments, the drum was rotated by hand and the velocity held constant by continuously checking the tachometer; this was simple in practice because the drum was attached to a modified bicycle wheel and consequently there was very little friction during rotation. It was determined that, at a drum velocity of 50°/sec, most people experienced Stage 2 CV within 5-15 secs (Fig. 1). Therefore, a trial interval of 30 secs was chosen for all subsequent experiments. In the subsequent experiments, the drum was rotated using a Heron systems DC motor and drum velocity could be selected (20-100°/sec) using a dial in the control room.

Subjects were seated in the centre of the drum with their head resting against a back bar and their feet on a foot rest. They were read instructions indicating that they were to 'stare straight ahead at the stripes without attempting to follow them' and should press a button at the onset of Stage 2 CV or Stage 3 CV. Subjects were instructed not to move their head under any circumstances. In the second set of experiments, each subject ($n = 32$) received 3, 30 sec trials, corresponding to a high (92°/sec), medium (63°/sec) or low (24°/sec) drum velocity for Stage 2 or Stage 3 CV (but not both). Each subject received only 3 trials in order to control for the effects of previous optokinetic experience on reaction time. The order of presentation of the 3 velocities was randomized. Before the beginning of the first trial and during each 1 min intertrial interval, the subject sat in darkness. Earphones delivered white noise during the experimental trials in order to mask auditory distractions. At each trial onset, the drum light, the earphones and a digital timer (Lafayette) were activated simultaneously for 30 sec. The subject's button press automatically stopped the digital timer, yielding a reaction time in secs.

For some subjects, optokinetic nystagmus (OKN) was also recorded in order to ensure that the visual stimulus was sufficient to evoke OKN. In these cases,

Beckman electrodes were attached to the outer canthus of each eye and a ground electrode was placed in the centre of the forehead. The output from the electrode leads was amplified and displayed on a Grass polygraph.

The reaction time data were subjected to a logarithmic transformation and 2-factor analyses of variance with linear regression were performed. The significance rate was set at 0.05.

Results

When a large number of subjects ($n = 60$) were tested with a drum velocity of $50^\circ/\text{sec}$, latencies to CV were variable but most subjects experienced Stage 2 CV within 10-15 secs (Fig. 1A). The CV illusion was so powerful that subjects who placed their feet on the floor after the onset of CV, reported that they felt as if they were 'jumping from a spinning chair'.

Using a smaller group of subjects tested at multiple velocities, the latency to Stage 3 CV was consistently greater than that to Stage 2 CV ($p < 0.01$; Fig. 1B). Despite a small reduction in mean latency to Stage 2 CV with increasing velocity, drum velocity had no significant effect on latency to CV. Latency to Stage 2 and Stage 3 CV decreased significantly with increasing number of trials ($p < 0.0001$). Optokinetic nystagmus appeared normal in those subjects from whom recordings were taken.

Discussion

Most subjects in the present experiment experienced Stage 2 CV within 10-15 secs of the onset of a $50^\circ/\text{sec}$ optokinetic stimulus. This result is generally consistent with those of previous studies (Brandt et al., 1973) and suggests that shorter or longer latencies (e.g < 5 and > 20 sec) might be indicative of an altered sensitivity to CV, for example, as a consequence of bilateral vestibular impairment caused by vestibulotoxic aminoglycoside antibiotics, or as a result of lesions within the optokinetic pathways (respectively) (Zee et al., 1976; see Halmagyi, Fattore, Curthoys & Wade, 1994; Wersall, 1995 for reviews).

As would be expected from the previous documentation of CV (Brandt et al., 1973), the latencies to Stage 3 CV, in which the visual field appeared stationary during apparent self-motion, were significantly greater than those to Stage 2 CV, in which apparent self-motion was experienced in the opposite direction to the moving visual field. However, the latency to CV did not change significantly within the stimulus velocity range used in the current experiment ($24\text{-}92^\circ/\text{sec}$).

Figure 1: (A) distribution of Stage 2 CV latencies for 60 subjects exposed to a $50^\circ/\text{sec}$ optokinetic stimulus;

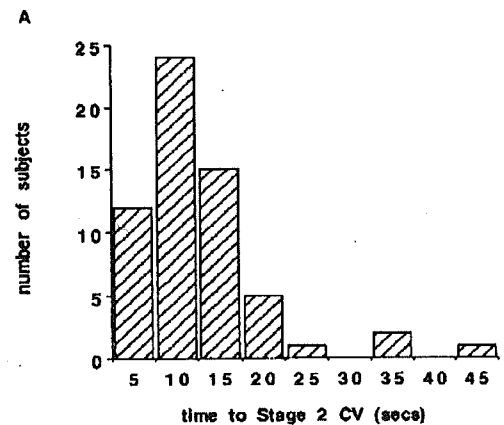


Figure 1: (B) latency to Stage 2 ($n = 16$) and Stage 3 CV ($n = 16$) for subjects exposed to a low ($24^\circ/\text{sec}$), medium ($63^\circ/\text{sec}$) or high ($92^\circ/\text{sec}$) velocity optokinetic stimulus;

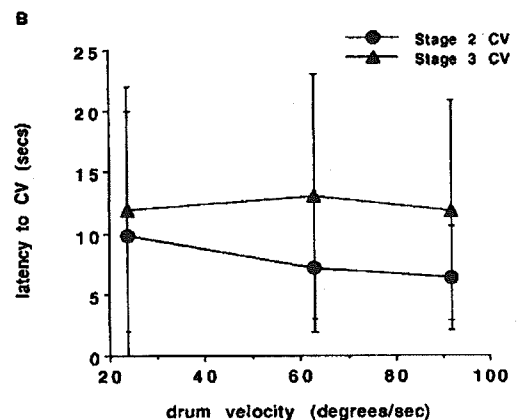
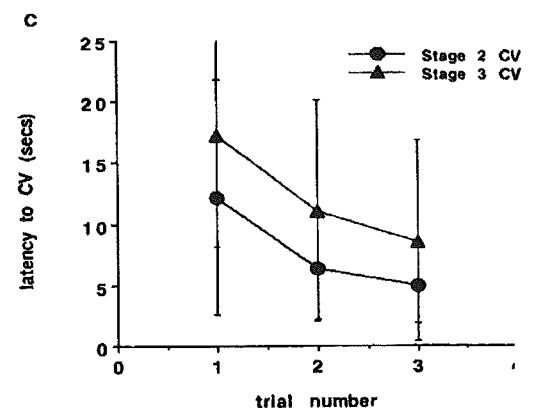


Figure 1: (C) latency to Stage 2 ($n = 16$) and Stage 3 CV ($n = 16$) as a function of trial number. For (A) and (B), symbols represents means and bars represent ± 1 standard deviation of the mean.



An interesting finding from the present study was the large and significant decrease in CV latency that occurred with increasing exposure to the optokinetic stimulus over the 3 trials. This may have been due to the subjects' increasing understanding of the CV illusion during the 3 trials. However, it is unlikely to be due simply to practice at the button-pressing task; in other studies we have found that reversing the direction of drum rotation after the first 3 trials can increase the CV latency to the initial values (Darlington, Smith, Howse & Munro, 1988). The reduction in CV latency over trials may represent an increased understanding of the nature of the illusion, or increased sensitivity to CV, or both. Either way, this order effect suggests that caution should be exercised in exposing subjects to 'practice' trials or a large number of trials in which repeated exposure could confound results. This result also suggests that, where CV and OKR measurements are being used for diagnostic purposes (e.g. Zee et al., 1976), care must be taken to ensure that the patient has not adapted to the optokinetic stimulus before the critical data have been collected.

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