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HEAD MOVEMENTS SUGGEST CANAL AND OTOLITH PROJECTIONS ARE ACTIVATED DURING GALVANIC VESTIBULAR STIMULATION

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Abstract—Three-dimensional changes in the angular orientation of the head were monitored during galvanic vestibular stimulation (GVS) delivered through electrodes implanted bilaterally in the tensor tympani muscle of the guinea-pig middle ear. Bilateral GVS was delivered by passing current between both ears with the anode situated in one ear and the cathode in the other ear. Unilateral GVS was also delivered between one ear and an indifferent electrode on the skull. Constant-current stimulation caused the head to tilt predominantly within the roll and yaw planes toward an ear stimulated with anodal current and away from an ear stimulated with cathodal current. No significant head tilt in the pitch plane was observed with either bilateral or unilateral GVS. Bilateral GVS was found to induce significantly greater roll head tilt (RHT) and yaw head tilt (YHT) than the same intensity of unilateral anodal or cathodal GVS, but not the sum of responses induced by the two polarities of unilateral GVS. Significant asymmetries were observed in the responses of YHT and RHT for unilateral anodal and cathodal GVS; unilateral cathodal stimulation generated greater head deviation compared with the same intensity of unilateral anodal stimulation. These asymmetric responses are consistent with activation of irregularly discharging afferents, which have been shown previously to exhibit asymmetric responses for anodal and cathodal GVS (Kim and Curthoys, 2004). Together with the observations of previous guinea-pig studies, the results suggest that head movements induced by GVS may be mediated by irregularly discharging afferents innervating the otoliths, and possibly the horizontal semicircular canals.

Key words: vestibular, galvanic, stimulation, posture, guinea pig.

INTRODUCTION

Mammals rely on vestibular signals from the inner ear to maintain the upright posture of their head relative to gravity. These signals are naturally generated through the mechanical transduction of gravitational forces into neuronal potentials by vestibular receptors located in the inner ear. Delivery of galvanic vestibular stimulation (GVS) stimulates primary vestibular neurons artificially (Goldberg et al., 1982, 1984), and has been shown to induce eye movements (Zink et al., 1998; Watson et al., 1998; MacDougall et al., 2002, 2003) and postural responses in humans (Coats, 1972; Hlavacka and Njiokiktjien, 1985; Johansson and Magnusson, 1991). The current study aimed to better understand the end organ(s) of origin that actually mediate postural movements of the head during GVS.

Human studies with GVS have found that surface galvanic stimulation applied bilaterally between the mastoids induces postural changes within the roll plane (Hlavacka and Njiokiktjien, 1985; Johansson and Magnusson, 1991; Johansson et al., 1995; Cathers et al., 2004). These postural changes consist of a bias in body-sway measured as a change in center-of-pressure (CoP) toward the ear receiving anodal stimulation and away from an ear receiving cathodal stimulation. Postural sway asymmetries have also been observed during trapezoidal pulses of GVS delivered unilaterally to one ear when passing current between electrodes placed over one mastoid and the back of the neck (Coats, 1972). These body-sway asymmetries appear to be induced by distributed vestibulospinal projections activated by GVS. This is because passing currents between the mastoids produced a cumulative lateral flexion of the spinal column, including the cervical and thoracic vertebrae (Fitzpatrick and Day, 2004). Also, passing currents between surface electrodes placed over the neck and the arm produced no postural responses, whereas passing currents between the two mastoids produces postural sway asymmetries (Magnusson et al., 1990).

Studies with animals have shown that GVS stimulates activity among primary vestibular neurons non-specifically. Galvanic currents applied to the inner ear cause a change in the membrane potential of primary vestibular afferents. Cathodal current causes membrane depolarization, resulting in an influx of positive charge within the cell and an increase in firing rate. Anodal current causes membrane hyperpolarization, resulting in an accumulation of positive charge outside the cell and a decrease in firing rate (Goldberg et al., 1982). Although it is possible that GVS may stimulate activity at the level of sensory hair cells, GVS appears to bypass hair cells to stimulate primary vestibular neurons at their spike trigger zones directly. This view is supported by the observation that anodal currents applied to the endolymph can increase afferent

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Abbreviations: GVS, galvanic vestibular stimulation; LED, light emitting diode; MSNA, muscle sympathetic nerve activity; PHT, pitch head tilt; RHT, roll head tilt; SCC, semicircular canal; VOR, vestibulo-ocular reflex; YHT, yaw head tilt.

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discharge because they cause a distal increase in positive charge at the spike trigger zone of hair cell receptors (Goldberg et al., 1984). Irregularly discharging primary vestibular afferents tend to be more sensitive to galvanic currents than are regularly discharging primary vestibular afferents (Goldberg et al., 1982, 1984), a result that was shown in other mammalian species (Baird et al., 1988; Kim and Curthoys, 2004). In guinea pigs, GVS delivered to the tensor-tympani of the middle ear – external to the inner ear – was shown through extracellular recordings from Scarpa's ganglion to stimulate primary afferents innervating the two otoliths and the three semicircular canals (SCCs) (Kim and Curthoys, 2004).

Although we know precisely what is stimulated when GVS is delivered in and around the inner ear, it still remains unclear what central projections are 'activated,' and actually contribute to induced behavioral responses, such as ocular and postural responses. This has largely been the topic of debate in the most recent literature. Cohen et al. (2012) have argued, on the basis of the static nature of postural sway asymmetries to GVS, that galvanic stimulation preferentially activates output projections of the otolith system. This was also supported by the pattern of eye movement responses observed during GVS in some human studies, which were predominantly tonic and torsional (Zink et al., 1998; Watson et al., 1998; MacDougall et al., 2005), and relatively free of nystagmus indicative of a significant SCC contribution. Subjective reports of linear changes in roll-plane body orientation (Hammam et al., 2012) are also consistent with otolith activation and the direction of these GVS-induced eye movements. In addition to subjective sensations of self motion, GVS has also been shown to modulate muscle sympathetic nerve activity (MSNA) in humans (Bent et al., 2006; Grewal et al., 2009). This modulation of MSNA by GVS has been attributed to recruitment of otolith projections, because natural angular head accelerations do not generate sympathetic nerve activity in humans (Ray et al., 1998). Also, GVS has been shown to modulate changes in arterial pressure in rats exposed to linear gravitation forces that primarily stimulate the otoliths (Abe et al., 2007).

Other researchers have argued that the behavioral responses induced during GVS are caused by activation of both otolith and semicircular projections (Curthoys and MacDougall, 2012). This view is primarily supported by the finding that eye movements induced by GVS tend to contain significant horizontal nystagmus when human subjects are seated in complete darkness without fixation (MacDougall et al., 2002, 2003). Even when fixation is presented to suppress horizontal eye movement, the induced torsional eye movements contain significant nystagmus (Watson et al., 1998; Zink et al., 1998). Consistent with these observations in humans, Kim (2009) found that GVS generated horizontal nystagmus at moderate intensities of GVS passed bilaterally between the tensor-tympani muscles of guinea pigs (>40 μA). Lower current intensities of bilateral GVS (5–30 μA) have been found to generate purely tonic changes in eye position without nystagmus (Kim, 2009, 2013), consistent with preferential activation of otolith-ocular projections. However, intensities of GVS as low as 20 μA have been shown to affect the gain of the vestibulo-ocular reflex (VOR) for whole body rotations in head-free guinea pigs (Shanidze et al., 2012). Although this would suggest that both SCC and otolith projections induce eye movements at low to moderate intensities of GVS, the relative contributions of these projections to postural control during GVS remain unclear.

Some insight into to the potential end-organ origins of GVS-induced postural responses may be gained from the observations of previous lesion studies with animals. Similar to high-intensity anodal GVS, peripheral vestibular lesions silence the activity of all primary vestibular afferents, including irregularly and regularly discharging afferents. It has been shown that selective unilateral otolith damage in guinea pigs is known to cause a maintained roll head tilt (RHT) toward the lesioned ear (de Waele et al., 1989; Vidal et al., 1993), which is not observed after selective unilateral lesion of the vertical SCCs. Any maintained RHT observed during GVS would be consistent with the activation of otolith-spinal output projections, and any unbalanced vestibular inputs to the insterstitial nucleus of Cajal (Fukushima et al., 1987). Moreover, when unilateral horizontal SCC lesions in guinea pigs were performed, ipsilateral yaw head tilt (YHT) toward the lesioned canal was observed (de Waele et al., 1989; Ris et al., 1999). This maintained YHT response was not observed following selective lesion of any other vestibular end organ. Therefore, maintenance of postural equilibrium in yaw appears to depend on activation of the horizontal SCC system during upright stance. Any maintained YHT observed during GVS may indicate activation of canal-spinal output projections.

Unlike peripheral lesions however, low to moderate intensities of anodal GVS have the potential to selectively silence only the activity of more irregularly discharging primary vestibular afferents (Minor and Goldberg, 1991; Chen-Huang et al., 1997; Kim and Curthoys, 2004), which may influence the activity of central neurons involved in the vestibulospinal reflex (Boyle et al., 1992). In support of this view, Shanidze et al. (2012) found that similar intensities of GVS modulated the vestibulocollic reflex in guinea pigs. However, it remains unclear to what extent the lower intensities of GVS generate RHT and YHT responses in these animals, as there exist no behavioral data on the 3D angular changes in head orientation induced during GVS in these animals. Parker (1970) found that direct electrical stimulation of the vestibular nerve in rabbits induced RHT directed contralateral to the excited nerve. However, the RHT was recorded using a force transducer attached to apparatus restraining the head, and did not characterize head movements in the yaw or pitch planes (i.e., YHT and pitch head tilt (PHT)). To determine which end-organ pathways may contribute to postural responses induced by GVS, the present study characterized the precise 3D angular changes in head