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V. INTEGRATIVE FUNCTIONS

EVIDENCE FROM SPACE ON THE INDEPENDENT CONTROL OF OTOLITH-GENERATED EYE MOVEMENTS

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Binocular studies of torsional eye movements were performed on two astronauts during a six-month mission on the Russian Space Station Mir. Preceding the flight, numerous baseline studies provided a reliable indication of the torsional position of the two eyes in the normal 1G of Earth, and these positions were used as the baselines against which the studies in space were compared. In both g conditions, the subjects sat quietly upright.

In both space and on Earth, the head was held by an apparatus consisting of a pre-molded bitebar rigidly attached to a mask in a solidly fixed position. The mask had two video cameras and a clear field of view. On Earth, a line throngh the center of both pupils was verified to be Earthhorizontal. Video-taping both eyes provided 25 synchronized images per second using the videooculography system on SensoMotoric Instruments.

Eight or nine studies were interspersed during the 180-day mission. In both subjects, the torsional positions of the eyes differed from the positions on Earth and each eye differed from the position of its mate. In one astronaut, torsional position was 3° to 4° counterclockwise from baseline. In the other astronaut, the left eye was torted counterclockwise and the right eye clockwise. The offset of eye torsion during the entire mission persisted for several days postflight, as did the difference between the two eyes. The next tests, performed several months later, found the eyes had reverted to their preflight baseline positions.

In contrast to the torsional conjugacy observed on Earth-based testing, the independent behaviour of the two eyes in space suggests that a partial decoupling between the otoliths and eye muscles may occur in novel gravitational states. A sudden change in otolith signal may act to decouple the multisynaptic connections from each labyrinth to the separate neuronal pathways going to each eye.

A third astronaut was examined monocularly during a 30-day mission. Torsional eye position in this subject was similarly offset from baseline, persisting for many days postflight. Althongh only one eye was examined, his findings are consistent with the observations in the binocular studies.

IMPAIRED ADAPTATION TO CORIOLIS FORCE PERTURBATIONS OF REACHING MOVEMENTS IN INDIVIDUALS WITHOUT LABYRINTHINE FUNCTION

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Inertial Coriolis forces are generated when resching movements are made during passive body rotation in a fully enclosed rotating room. Such Coriolis forces lead to disruptions of movement trajectory and endpoint. Normal subjects can adapt fully to the Coriolis force. without visual feedback on the basis of correlating efferent commands and proprioceptive feedback and somatosensory contact cues providing information about movement tonchdown. In the present experiment, we evaluated the ability of five profoundly labyrinthine-defec ti ve subj ects and five age-matched, normal control subjects to adapt to Coriolis force perturbations of their reaching movements. The subieots pointed in complete darkness 40 times to the location of a just extinguished visual target before, during, and after constant velocity rotation at 10 rpm CCW in our slow rotation room. The initial per-rotation reaching movements of both groop s were deviated in the direction of the transient Coriolis forces present during the movements and ended about 4 cm rightward of the baseline position. The control subjects showed complete adaptation of endpoint and curvature within 15 reaches so that they again resched in straight lines to the targd position. The labyrinthine-defective subjects showed impaired adaptation of movement endpoint coupled with complete adaptation of movement curvature, albeit with a longer time constant than the control subiects. After asymptotic adaptation, the labyrinthine loss subiects reached in straight lines to an endpoint 2.5 cm right of baseline. The labyrinthine-defective subjects also showed significantly greater variability of movement endpoint (but not curvature nor peak velocity) relative to the control subjects in their pre- and post-rotation movements. In other experiments, the same pattern of full trajectory adaptation and partial endpoint was seen in subjects with normal vestibular function exposed to transient Coriolis forces in a novd tonic background force environment. These findings indicate that vestibular function is implicated in adaptive limb movement control. They are inconsistent with alpha equilibrium point models of movement control and models positing vision as the sole or primary source of information for updating motor control.

TACTILE CUEING MODEL FOR G-SEAT USE IN FLIGHT SIMULATION

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Tactile cueing using G-seats hes been employed to provide normal acceleration and roll and pitch cues in fixed and moving base flight simulators. Althongh the addition of tactile cueing has been shown to improve pilot performance in fixed base simulation, its transfer value and training benefit remain unclear. A aseat drive algorithm based on a validated model of motion perception from tactile cues is likely to be more useful in training than a drive algorithm that is based solely on simulator pilot performance. The contribution of tactile cueing to motion perception is less well understood than the contribution of visual and vestibular cues to motion perception. An experiment was conducted to quantify the frequency dependent contribution of tactile cueing to linear motion perception using a modified NASA Langley Research Center pneumatic G-seat. Eight blindfolded subjects participated in a horizontal linear motion nulling experiment, lying on their backs during z-axis motion on the MIT sled. Sum of sines disturbances (.06 Hz - 0.5 Hz) were provided in both sled velocity and G-seat pressure in one half of the trials and only sled velocity in the other half. Subjects attempted to null the sled velocity using a hand controller. Transfer functions from sled velocity to subject command response (vestibular transfer function) and from G-seat pressure to subject command response (tactile transfer function) were computed using cross-correlation methods for each of the two conditions (G-seat on and G-seat off). A significant response to G-seat pressure, fit with a differentiator transfer function, was observed. Differences between the G- seat on and G- seat off vestibular transfer function were significant but smaller than expected. Differentiation in the tactile transfer function agrees with previous research on tactile receptors in the skin and supports the use of G- seats to cue acceleration on set rather than acceleration magnitude. A normal acceleration-based drive algorithm for a pneumatic G-seat and a simulation concept using a pneumatic G-seat and a helmet-mounted display are proposed.

THE VESTIBULAR SYSTEM AND MOTION SICKNESS

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Deferring to the dictum that definitions should be expressed in terms of laboratory criteria, ie observational criteria, motion sickness can be defined as “the signs and symptoms of the sickness caused by motion”.

The signs and symptoms of motion sickness constitute a poison response, and they consist of two different kinds of phenomena: stomach emptying phenomena and stress response phenomena. The evidence indicates that both these kinds of phenomena are in appropriate to the stimulus. The stomach is emptied in the absence of a poison, and the stress response occurs in the absence of a stress.

Evidence suggests that the motion triggers a vestibular-related poison detector in the brain and the brain then runs programs that produce the signs and symptoms of a poison response, including conscious sensations such as nausea and distress. It is especially interesting that a feeling/emotion such as distress can be produced this way.

THAT SICKENING LURCH: GRAVICEPTIVE CONTROL OF BLOOD PRESSURE IN MAN

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Many readers may be familiar with the malaise which can be provoked rapidly when in a car by making a head movement at the same time as the driver unexpectedly accelerates or brakes. A possible mechanism of this sickness is suggested by recent findings in the cat of direct projections from the otolith 'graviceptive' organs to neural centres of autonomic regulation. Here we present evidence for a direct graviceptive control of autonomic function in man. Subjects were seated in an electrically powered car and tasked with making transient head movements in flexion, or extension, guided by an up-front visual display so that head tilt would approximate tilts in uprightuess induced by car acceleration. Some head movements were accompanied by an unexpected acceleration of the car, backwards or forwards, from rest, at 2m/s2 for ls, thereafter coasting gently to rest. During an acceleration pulse, the direction of uprightness (vector sum of gravity plus pulse) tilted by approximately 12°. If the head tilted in the same direction ('sympathetic') it remained inertially 'upright' and gave little otolithic stimulation. However, if the head tilted in the opposite direction to motion ('anti-pathetic') the otolith organ would signal a tilt of approximately 22°. Isolated head tilts (control condition), and car accelerations combined with symp- or anti-pathetic head tilts were dispensed to 2 groups of 4 normal subjects in a balanced, cross over design in which each stimulus condition was given 8 times with 40s between trials. ECG, tonometric BP transduced from the left radial artery and finger plethysmography were recorded. Within 10s of motion isolated head movements provoked small increases in systolic BP (1-3 mmHg). Car motion coupled with sympathetic head tilt provoked peak increases in diastolic and systolic BP of 4-6 mmHg. Antipathetic head tilt provoked highly significant peak increases in systolic (7.6-9.4 mmHg) and diastolic (5-6 mmHg) and the average BP over 10s after motion onset was significantly raised in the car forwards - head jerking backwards condition. Heart rate was unaffected little by stimulation. Isolated head movement caused little change in peripheral blood flow but all car-coupled motion induced a vasoconstriction. Subjects reported little sensation of motion during sympathetic tilts with no malaise. A marked sensation of motion accompanied antipathetic head movement and despite the motion being gentle as compared with a motor car, 3 subjects reported mild symptoms of discomfort, malaise, headache and sleepiness; although none of these rated themselves susceptible to motion sickness on a questionnaire. The results demonstrate a marked increase in BP mediated by backwards head tilt occurring with forwards body motion and subjective reports suggested that such manoeuvres provoke malaise. The findings pertain to motion sickness and are relevant to transporting patients with autonomic distress whose condition is worsened by accelerating and braking in the ambulance.

VESTIBULAR INFLUENCES ON CIRCULATION AND RESPIRATION: RECENT INSIGHTS

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Considerable evidence suggests that the vestibular system influences the control of blood pressure and respiration to provide for homeostasis during movement and changes in posture. Selective stimulation of vestibular afferents produces changes in sympathetic nerve activity, blood pressure, and activity of respiratory muscles. In particular, pitch vestibular stimulation elicits cardiovascular and respiratory responses. Vestibular-cardiovascular and vestibular-respiratory responses are sustained during static pitch vestibular stimulation, and the gain of these responses remains relatively constant across stimulus frequencies, saggesting that they are the result of stimulation of otolith organs. Furthermore, vestibular nerve transection in anaesthetized animals compromises the ability to adjust blood pressure during unexpected tilts in the pitch plane. The brainstem circuitry that produces vestibular-sympathetic responses includes the medial and inferior vestibular nuclei, the lateral medullary reticular formation, and neurons in the rostral ventrolateral medulla that relay the labyrinthine signals to sympathetic preganglionic neurons in the spinal cord. The pathways that mediate vestibular-respiratory responses are currently unknown, as medullary neurons that generate the respiratory rhythm and relay this activity to the spinal cord are not necessary for vestibular stimulation to elicit effects in the diaphragm or abdominal muscles.

This presentation will highlight our observations concerning vestibular-cardiovascular and vestibular-respiratory responses made during the past year. We have discovered that multiple somatic inputs regarding body position in space, including visual, vestibular, and somatosensory signals, are integrated to produce changes in blood pressure during unexpected alterations in posture. As a result, in alert animals deficits in correcting blood pressure during tilts are only apparent when several somatic sensory cues regarding position in space (e.g., visual and vestibular inputs) are eliminated. In addition, the ability to correct blood pressure during changes in posture recovers over a period of several days following bilateral vestibular nerve transection. Current studies are examining the possibility that the caudal cerebellar vermis, which has been shown to integrate multiple sensory inputs and to powerfully affect the control of circulation, may participate in the adaptive plasticity in vestibular-cardiovascular responses in awake animals.

A second line of research has shown that the influences of the vestibular system are predominantly limited to those components of the sympathetic nervous system that regulate blood pressure, as opposed to sympathetic efferents that influence motility in the gut or bladder. Because the responsiveness of sympathetic efferents innervating vascular smooth muscle is powerfully influenced by baroreceptor inputs, we found that the magnitude of vestibular-sympathetic reflexes is inversely proportional to blood pressure, and that these responses are completely abolished when mean blood pressure exceeds 150 mm Hg.

A third group of experiments has attempted to determine which premotor neurons in the brainstem may relay vestibular signals to spinal respiratory motoneurons. As discussed above, neurons in addition to those involved in producing breathing must play a prominent role in generating vestibular-respiratory reflexes, although the locations of these neurons is currently unknown. Anatomical studies involving the transneuronal transport of pseudorabies virus were thus undertaken to determine the locations of all brainstem neurons that project to phrenic (diaphragm) or abdominal motoneurons. These studies have shown that many neurons in the medial reticular formation make connections with spinal respiratory motoneurons, raising the possibility that these neurons mediate vestibular-respiratory responses. Medial reticular formation neurons may also be responsible for producing other behaviors involving contractions of respiratory muscles, including emesis. This possibility awaits to be examined.

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