Research Paper

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Autonomic response to real versus illusory motion (vection)

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This study explored the cardiovascular responses to illusions of self-motion (vection) induced in normal subjects according to the hypothesis that vection may be a model for vertigo in vestibular disease. Responses were obtained from 10 men who were exposed to rapid tilts of 20° and 30° rolling from the upright position down to the right or left shoulder. These responses were compared with those evoked during the illusion of roll-tilt vection provoked by a torsionally rotating visual field. Comparisons were made between 10-second data epochs before and after stimulus onset. In response to vection, blood pressure (BP) in the radial artery rose consistently in six subjects, and in all of these, a pressor response to real tilt was also observed. The remaining four subjects consistently had decreased BP in response to vection, and their BPs were affected little by tilt. Subjects whose BP increased with vection and tilt may have been dominated by tendency to arousal, whereas those whose BP decreased may reveal the more appropriate response to tilt from the upright position, which is a decrease in BP. This may reflect individual stereotypes and differences in the relative contributions of somatosensory and vestibular control of autonomic regulation.

Key words: vertigo, vection, tilt, blood pressure, optokinetic.

The profound vasovagal consequences of vestibular vertigo are ample evidence that the labyrinth has significant implications for autonomic function. However, it is only recently that animal studies have demonstrated possible mechanisms in the form of direct projections from vestibular nuclei serving cardiac and respiratory regulation [1]. The present study intended to gain insight into the autonomic responses accompanying vertigo by using vection in normal subjects as a model of vertigo caused by vestibular disease.

Vertigo is illusory self-motion caused by disordered vestibular activity, which wrongly signals that the head is moving. Vection is an illusory self-motion induced by a moving visual field, which is familiar to readers in the "railway carriage" illusion of self-motion provoked by seeing a train go by. Vection is attributed to a summation of visual motion signals and vestibular signals in vestibular nuclei type I neurones. These neurones give an output which the brain may interpret as self-motion [2–4], even though their input may be only from visual motion. Therefore, both vection and vertigo arise through activity in the vestibular nuclei, which causes a false perception of self-motion that is at odds with other sensory inputs. *A priori* one would expect individual differences in the autonomic responses to vection because normal subjects' reactions to induced dizziness, on fairground rides or during caloric irrigation, for example, vary from exhilaration to fear.

To provoke substantial autonomic responses, we studied roll-tilt, which is tilting from the upright position down to either shoulder. Illusory roll-tilt is provoked in the form of a roll-tilt vection by a torsionally rotating visual field. While viewing the rotation, the observer first sees object motion and then, after a delay, may experience the illusion of rotating and being tilted in the direction opposite to field rotation.

Material and methods

Apparatus and tilting

Subjects were seated with head upright in a flight simulator (SEGA, Tokyo, Japan) that executed discrete tilts from the upright position by rolling subjects about an anteroposterior, horizontal axis aligned through the midline of the trunk at the level of the heart (Fig. 1). Head, trunk, and limbs were supported and restrained with foam padding. Peak velocities of tilt were 20° per second with settling times of 2.5 and 3.0 seconds. Tilts were maintained for 30 seconds, after which the machine returned to the upright position with a similar velocity profile. Subjects closed their eyes during tilts.

Optokinetic stimulus inducing vection

A motorized cone with a diameter of 58 cm and a depth of 25 cm was mounted in the flight simulator at a distance of 28 cm from the nasion (Fig. 1). The subjects looked into the

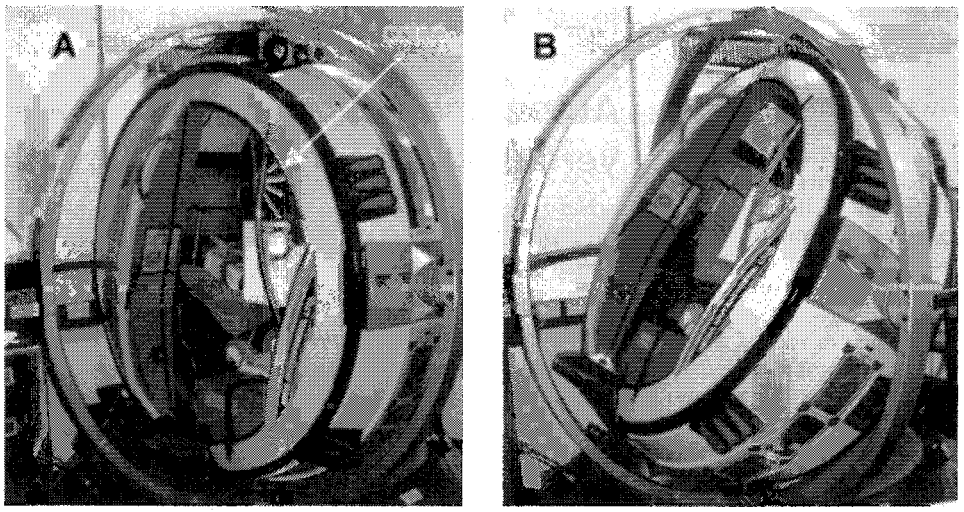

Figure 1. (A) SEGA flight simulator in upright position with optokinetic cone visible within the cockpit. **(B)** SEGA tilted 30° rightward.

cone, fixating the apex. The inside was matte black with radially oriented stripes of charged fluorescent tape 20 mm wide. The stripe/background contrasts were 0.89 at the beginning of testing and decayed to 0.67. To provoke vection, the cone rotated about its principle axis at 40° per second. Subjects indicated the onset and offset of vection with a foot switch. During and immediately prior to this procedure, the cockpit was in darkness.

Autonomic recordings

The electrocardiogram (ECG) was recorded by a BP-508 (Colin Corporation, Komaki, Japan), which also recorded continuous tonometric arterial blood pressure (BP) from a robotic sensor positioned over the left radial artery, approximately 20 mm from the wrist line. The forearm was fully supported and restrained to exclude mechanical shocks, and it was semi-abducted so that the BP sensor was on the axis of tilt. Pulsatile blood flow measure was obtained by photoelectric plethysmography (Model PPS; Grass, Boston, MA, USA) from the index finger of the right hand and high pass filtered with a time constant of 3 seconds. Galvanic skin resistance (GSR) was obtained from solid state electrodes (ARBOTM Neonaten, Hamburg, Germany) mounted on the second and third fingers of the right hand. Respiration was recorded as the measure of airflow from a thermocouple in the left nostril. Instantaneous heart rate (HR) and R-R interval time (RRt) were derived in analog form from the raw signal with a Grass tachograph. Signals were sampled for processing at 125 Hz, and averages were taken of HR, RRt, diastolic arterial blood pressure (DAP), systolic arterial blood pressure (SAP), and peak-to-peak beats of the plethysmograph. All measurements were taken for 10 seconds before and 10 seconds after the onset of stimuli (tilts, vection, and cone motion). GSR amplitude was measured from baseline to peak in ohms attained during the 10 seconds after stimulus.

Background information questionnaire

Subjects completed a validated questionnaire that probed headache, ear and eye diseases, and optical correction; susceptibility to motion sickness; susceptibility to startle, shock, blushing, and fainting; introversion–extraversion; physical activity and attitude to physical risk and amusement parks; and use of social and prescribed drugs. Relevant medical history was sought by questioning.

Experimental design

Ten healthy adult men (age range, 29–52 y; mean, 36.2 y; standard deviation, 6.4) consented to the study according to the guidelines of the local ethics committee. None were smokers. None had unusual ingestion of drugs or unusual life events over the previous 24 hours. Testing was performed midmorning or midafternoon.

Stimuli were given in a balanced design: five subjects were exposed to real tilts followed by optokinetic stimulation with a rest period of 10 minutes between tests. The remainder underwent the inverse sequence.

Tilts of 20° and 30° to the right and left shoulders were given in a balanced design with two trials at each amplitude. Timing of tilts was varied to be unpredictable, and at least 30 seconds elapsed before and between tilts.

Vection stimuli included alternating 1 minute of rotation to the right or left shoulder (two times each way) followed by a 1-minute pause and stimulation in the opposite direction. During testing, the door of the simulator was closed.

Data analysis

Data were collected continuously for at least 30 seconds before and after any stimulus to decide whether the baseline was stable enough to identify responses (Fig. 2).

Responses were often small, so we adopted the following tactic: first, responses that were visible in stable baselines were identified. Thereafter, measurements were taken on all records with similarly stable baselines. This procedure included both obvious and minimally sized responses.

Vection occurred intermittently. The first onset of vection could be as early as 10 to 20 seconds after cone motion onset in susceptible subjects and could come and go thereafter. Because cone motion onset induced transient responses that could last up to 10 seconds, we decided that, as

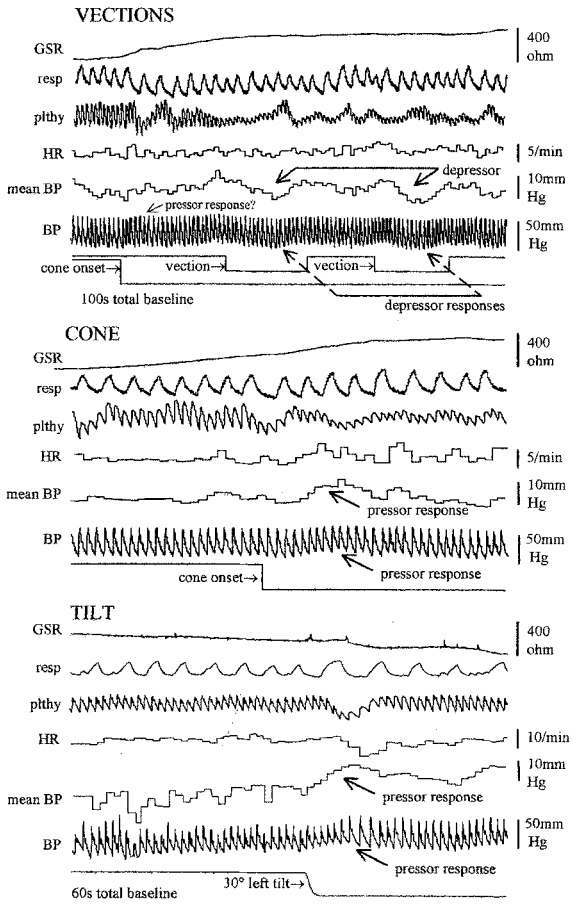

Figure 2. Extended data records showing baselines for responses to vection (upper traces with two examples of the depressor response), pressor response to cone onset (middle traces), and pressor response to tilt (lower traces). GSR = galvanic skin response; resp = respiration; plthy = plethysmograph; HR = heart rate; BP = blood pressure.

a precaution against collecting overlapping responses, we could only accept vection responses that occurred more than 20 seconds after cone motion onset or that were spaced 20 seconds after the offset of the last episode of vection (Fig. 2).

After failures to achieve vection and rejection of overlapping responses and unstable baselines, data from three vection trials were obtained for each subject. Tilting occasionally produced artifacts (usually a spontaneous arm movement) that had to be rejected.

Because of the temporal constraints on vection, comparative measurements and statistical analyses were performed on 10-second epochs before and after stimulus or vection onset.

Results

General characteristics of responses

On inspection of the records, we found that SAP and DAP increased in nine subjects on real tilt down (Figs. 2,3). HR appeared to initially increase and then decrease. In response to the onset of illusory tilt (vection), six subjects appeared to have increased DAP and/or SAP, whereas four subjects appeared to have decreased DAP and/or SAP (Fig. 3). RRt did not change systematically with illusory tilt. Both the return to the upright position and the onset of cone rotation gave more variable responses in RRt, DAP, and SAP with no consistent pattern. The plethysmograph showed decreased peripheral volume of variable magnitude in most subjects in response to all of the stimuli.

For each of the six subjects who appeared to have increased DAP and/or SAP (of which two had short peaks in BP and four had rises in BP sustained over 10 seconds), *t* tests showed significant rises in the 10 seconds after vection onset in comparison with the 10 seconds before ($p < 0.05$). For subjects who appeared to have decreased DAP and/or SAP after vection, the results of *t* test results on the 10 seconds before vection versus the 10 seconds after vection were significant ($p < 0.05$). Of most importance, the distribution of rising and falling BPs was unequivocally bimodal. When responses were visibly evident, subjects were consistent in showing either a rise or fall, never both (see Fig. 2, raw records, and Fig. 4, DAP and SAP). Absolute amplitude of increases in diastolic BP in type 1 ranged from 0 to 7.5 mm Hg across the six subjects, and absolute amplitude of systolic BP ranged from 0 to 14 mm Hg. In type 2, diastolic BP decreased by up to 8 mm Hg, and systolic BP decreased by up to 8.5 mm Hg.

Using χ^2 goodness-of-fit tests, we rejected the null hypothesis of normally distributed diastolic BP changes for n = 30 observations at $\chi^2 = 7.2$ ($df = 1$, $p < 0.01$). Similarly, systolic BPs were not normally distributed ($\chi^2 = 26.1$, $df = 3$, $p < 0.001$). Because BP responses to vection were continuously distributed and their behavior was consistent within subjects, we tentatively classified subjects as type 1 (BP rising during illusion or pressor response) and type 2 (BP falling during illusion or depressor response) (see also Fig. 3).

Averages of subjects' responses were taken and grouped as types 1 or 2. These are shown in Figure 4 for 10 seconds preceding and 10 seconds following stimuli (averaging epochs are short because vection comes and goes). The averages included all vection over 10 seconds in duration. The latencies of vection after onset of visual stimulus were 20.3 ± 6.6 seconds in type 1 subjects and 18.2 ± 4.1 seconds in type 2 subjects, with durations of 30.5 ± 12.3 seconds and 39.0 ± 16.2 seconds, respectively. Mean ages of the two groups were 34.0 ± 7.0 years in type 1 and 37.6 ± 5.6 in type 2 (mean ± standard deviation). There were no differences between the two groups in latency, duration of vection, or age (analysis of variance, $p > 0.05$).

Repeated analyses of variance were performed on RRt, DAP, SAP, plethysmographic responses, and respiratory frequency seen in the 10 seconds before and 10 seconds after stimulation (or onset of vection) using the factors before and after stimulus (or vection) onset, trial number (first, second, third), and group (type 1, type 2). For tilt down, DAP and SAP increased by 4.8 and 8.9 mm Hg in type 1 subjects ($p < 0.01$), for whom there were also significant plethysmographic responses. For type 2 subjects as a group, there were no significant changes in BP or plethysmographic response, although individual subjects' BP did increase on

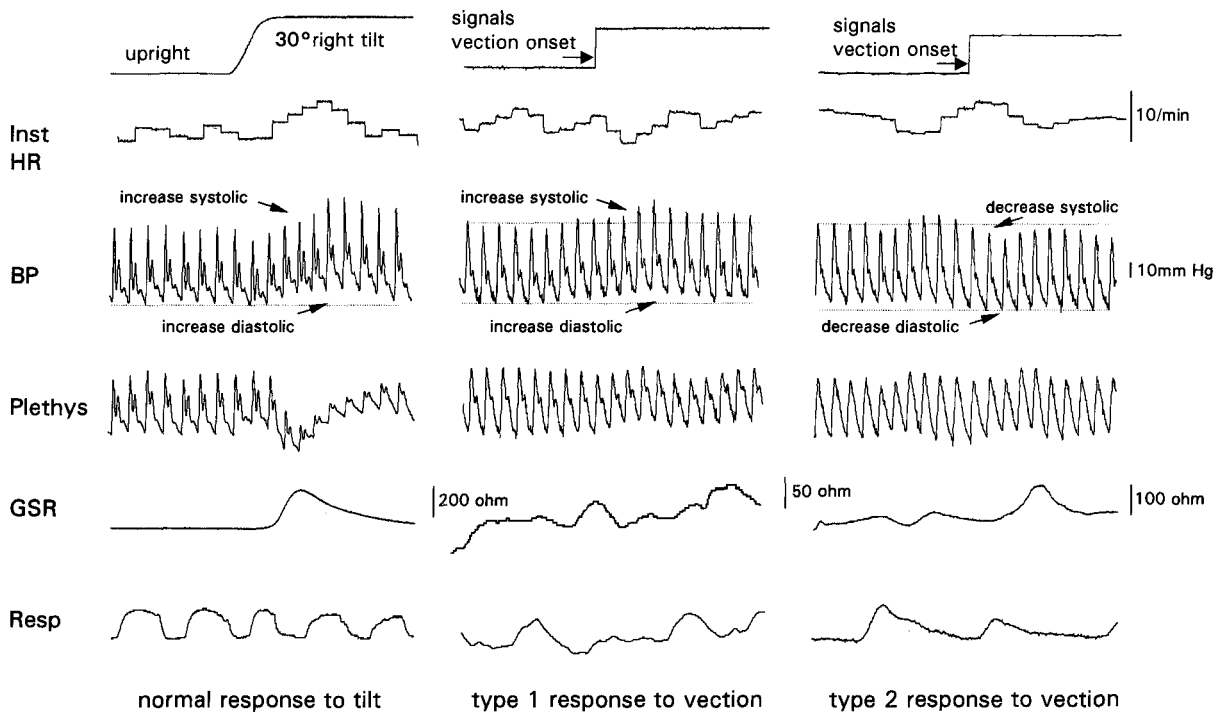

Figure 3. Examples of autonomic responses to real tilt and illusory tilt comparing 10 seconds before and after stimulus onset. Inst HR = instantaneous heart rate; BP = blood pressure; Plethys = plethysmograph; GSR = galvanic skin resistance (shown in Siemens, units of conductance); Resp = respiration. Note similar profiles for the increases in BP in real tilt and in the type 1 response to vection. Note that the absence of marked GSR responses suggests that there is little startle.

some tilts. There were no consistent changes in RRt in either group. Onset of vection provoked an increase of 3.5 mm Hg in SAP in type 1 subjects and a decrease in DAP (-2.8 mm Hg) and SAP (-2.2 mm Hg) in type 2 subjects ($p < 0.05$, Fig. 4). For tilt up, there were no systematic autonomic responses in either subject type. Onset of cone motion provoked only a decrease in plethysmographic response in type 2 subjects ($p < 0.05$).

Respiratory frequency in type 2 subjects was significantly higher in response to cone motion onset and significantly lower during vection ($p < 0.05$). However, based on averages, respiratory responses were unrelated to cardiovascular responses.

There were no differences in GSR response between stimulus conditions (analysis of variance, $p > 0.05$). During vection, type 1 subjects had a weak increase in conductance, whereas type 2 subjects had a marked increase in conductance (Fig. 4).

Latencies of response

Based on type 1 subjects whose BP increased during tilt, the latency of the increase in BP was 2.5 seconds. Plethysmographic changes had a latency of 3.0 seconds. Latencies of responses to illusory tilt are impossible to estimate because the onset of the illusion is difficult to pinpoint. Latencies to the peak of the GSR responses to tilt down, recorded as mean ± standard deviation, were 4.3 ± 0.4 seconds for type 1 and 4.5 ± 0.4 seconds for type 2. Latencies of response to cone motion were 3.8 ± 0.5 seconds for type 1 and 3.8 ± 0.5 seconds for type 2 (no differences between subject types).

Estimates of response novelty, adaptation, or startle components

Decreases in the magnitude of plethysmographic response were found on the first three trials (analysis of variance, $p < 0.05$) for type 1 subjects but only on the first trial for type 2 subjects.

Repeated analyses of variance on the peak amplitudes of GSR responses in the 10 seconds after tilt, cone motion, or vection onset showed no differences ($p > 0.05$) in response among the first three trials.

Response consistency

A type 1 subject and a type 2 subject have recently been retested 6 months after the original study and have produced pressor and depressor responses respectively to vection, as they did originally.

Discussion

The key finding in this study was that rapid roll tilt provoked a pressor response that one might expect as an arousal-readiness response to significant spatial reorientation, whereas with vection, some subjects' BP increased (type 1) and others' BP decreased (type 2). These responses to vection persisted with repeated exposure. Type 1 subjects' BP also significantly increased over the 10 seconds after tilting the body. Bigger GSR and plethysmographic response were observed in real tilt, and plethysmographic response in vection was variable and weak. No relationships were found between BP and HR in any stimulus condition.

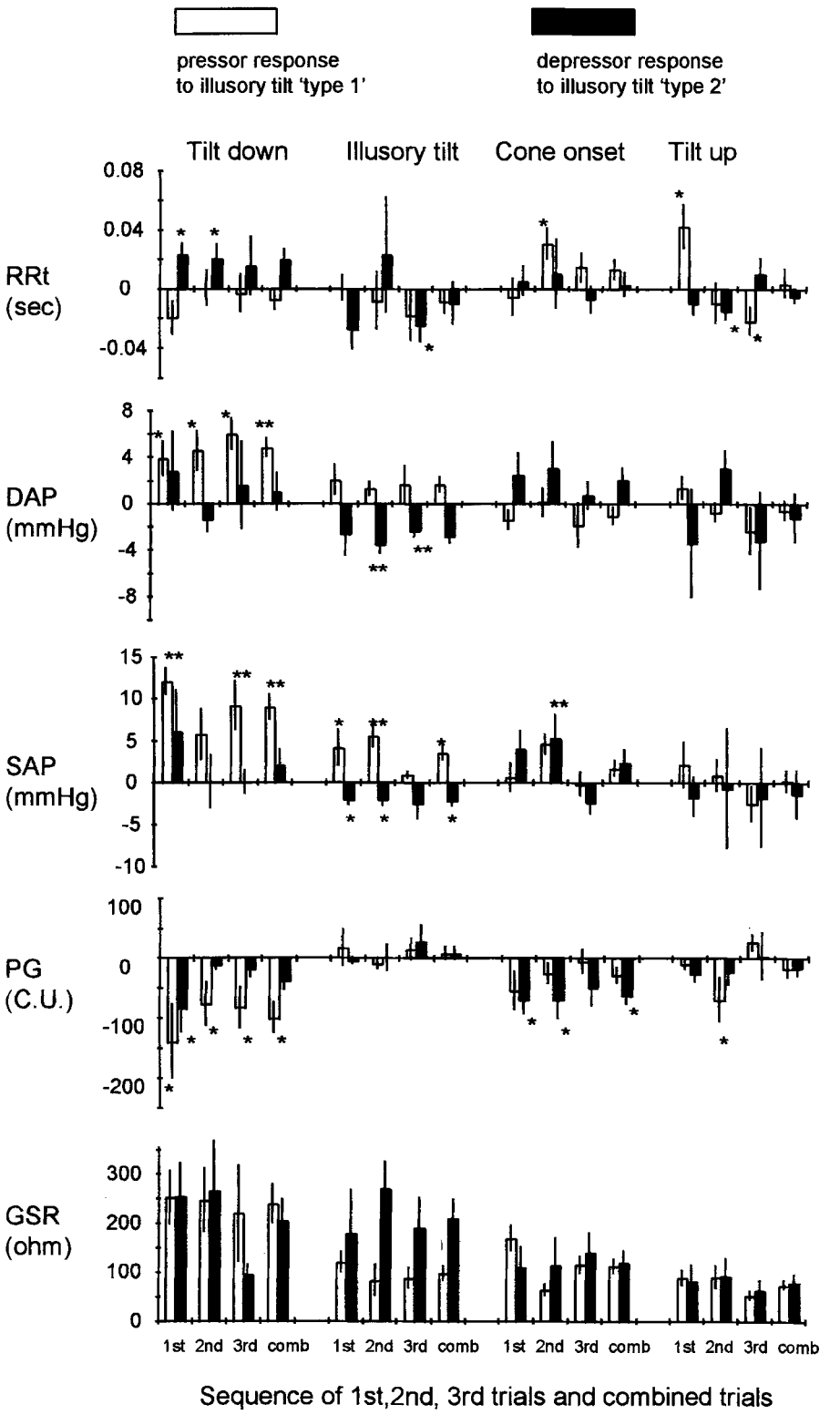

Figure 4. Autonomic responses to tilt down, illusory tilt, cone motion onset, and tilt up showing the changes in averages over 10 seconds pre-stimulus as compared with 10 seconds post-stimulus. The first three successful trials and their average are shown. Type 1, unfilled columns; type 2, filled columns. **, *, significant differences from 10-second pre-stimuli recording at $p < 0.01$ and $p < 0.05$, respectively. RRt = R-R interval time; DAP = diastolic arterial blood pressure; SAP = systolic arterial blood pressure; PG = plethysmograph; C.U. = computer unit; GSR = galvanic skin resistance. Each column shows mean ± standard error of mean.

Our attempt to classify subjects according to whether they had pressor or depressor responses to vection is in accord with a recent study on a visually induced illusion of tilt from the supine position toward the upright position, which found two classes of subjects, those who failed to respond and those whose BP increased [5]. It is not surprising that BP responses to illusory motion varied among individuals because vestibular stimulation (eg, calorics or rotation) does not induce stereotypical responses in humans [6]. We have observed that such intersubject variability is also characteristic of patients' responses to vertigo. Furthermore, even when subjects feel motion sickness, autonomic responses are not systematic [7,8]. However, in the present study, none of the subjects reported any discomfort as a

consequence of the experiment, which would indicate that motion sickness had been provoked, so it is unlikely that the types of responses we observed were related to motion sickness susceptibility.

In absolute terms, the observed changes in BP, either to tilt or to vection, were small. Effects of similar magnitude in humans have been reported for other forms of vestibular stimulation, for example, tilt of the head [9] or inertial force vector [10], typically below 5 mm Hg. Although they may have statistical significance, one might query the functional significance of such diminutive changes. The reason vection provokes small responses is probably that it inputs to normal closed loop autoregulation so that its effect is minimized by other regulatory mechanisms that fail to corroborate that a major spatial reorientation has taken place.

In respect to the mechanisms mediating the responses observed, animal studies have demonstrated that somatosensory inputs, including body movement, initially increase BP. For vestibular-canal input (calorics or rotation), some studies have shown a decrease in BP [11,12], whereas others have shown the opposite pressor effect [13]. Otolithic [14] stimulation in humans appears to raise BP (note the profile of the BP response to linear acceleration is identical to our type 1 response to rapid tilt). Hence, we may speculate on the mechanisms of response variability. For example, the increased BP response to real tilt could be derived from somatosensory or vestibular otolithic inputs. The depressor response during the vection in type 2 subjects may be induced by activation of the vestibular system by the visual flow (*nota bona,* no somatosensory input in vection). The pressor response during vection in type 1 may be associated with the cortical sympathetic output, which is induced by onset of vection. This suggestion is supported by the finding that during vection there was a significant increment of systolic arterial BP without a change in HR. Seen from another perspective, it may be that subjects whose BP increased with vection and tilt may be dominated by tendency to arousal, whereas those whose BP decreased may reveal the more appropriate response to tilt from the upright position, which is a decrease in BP. In sum, individual subjects' responses to real or illusory reorientations may reflect the relative weightings of somatosensory and vestibular modes of autonomic control and/or their preferred response tactic.

Although the central mechanism of sympathetic outflow is still unclear, tilt could excite sudomotor neurons through the vestibular and/or somatosensory systems [15]. The higher GSR response during real tilt (higher than during visual stimulation or vection) might be because of the congruent multisensory input driving the response. Animal studies show the importance of the medullary reticular formation as well as influences from midbrain, hypothalamus, and limbic structures [16]. With respect to the GSR responses in both tilt and vection, it is known that vestibular input directly activates the sudomotor nerve in animals and may therefore do the same in humans [17].

No relationship could be found between subjects' autonomic responses and their profiles from questionnaires, which suggests the possibility that individual responses to illusion represent a relatively independent idiosyncratic factor, as is apparently the case with motion sickness susceptibility, for example [18,19]. It remains to be shown what these individual types of response in normal subjects imply for symptoms associated with pathological vertigo.

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