Linear acceleration-evoked cardiovascular responses in awake rats

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Zhu H, Jordan JR, Hardy SP, Fulcher B, Childress C, Varner C, Windham B, Jeffcoat B, Rockhold RW, and Zhou W. Linear acceleration-evoked cardiovascular responses in awake rats. J Appl Physiol 103: 646–654, 2007. First published June 7, 2007; doi:10.1152/japplphysiol.00328.2007.—It has been well documented that vestibular-mediated cardiovascular regulation plays an important role in maintaining stable blood pressure (BP) during postural changes. But the underlying neural mechanisms remain to be elucidated. In particular, because the vestibular stimulation employed in previous animal studies activated both semicircular canals and otolith organs, the contributions of the otolith system has not been studied selectively. The goal of the present study was to characterize cardiovascular responses to natural otolith stimulation in awake rats that were subjected to pure linear motion. In any of the four directions tested, transient linear motion produced a short-latency (~520 ms) increase in mean BP with a peak of 8.27 ± 0.66 mmHg and was followed by a decrease in BP. There was an initial small biphasic response in heart rate (HR) that was followed by a longer duration increase. The short-latency increase in BP was absent in rats that were pentobarbital sodium anesthetized or that were labyrinthectomized bilaterally, but it was unaffected by baroreceptor denervation, indicating that it was of otolith origin. The increase in BP was linear acceleration intensity dependent and was not affected by absence of visual cues. Furthermore, the BP response was attenuated by inactivation of the medial and inferior vestibular nuclei by microinjections of muscimol, indicating that the otolith-driven cardiovascular responses are mediated by the neurons in these areas. These results not only demonstrate the otolith specific influences on the cardiovascular system but also they establish the first rodent model for examining the neural mechanisms underlying the otolith-mediated cardiovascular regulation.

BECAUSE OF PLANETARY GRAVITATIONAL influence, changes in posture are associated with changes in the length of the vascular orthostatic column. For example, going from a supine to a standing position results in an increase of the orthostatic column by sixfold, which puts a great challenge to the return of blood to the heart from the lower extremities (29). Failure to make necessary adjustments in vasoconstriction and cardiac tone during posture changes will result in orthostatic hypotension with serious consequences, such as decreases in cardiac filling, cardiac output, and perfusion of the brain. Several mechanisms may act to prevent the postural-related hypotension. Among them, the most important short-latency mechanism is to produce increased sympathetic outflow to selected arterial beds, which shuts down blood flow to unnecessary regions and reserves blood for the systematic circulation (8). Considerable evidence suggests that changes in sympathetic outflow elicited by the vestibular system are involved in preventing orthostatic hypotension (for review, see Refs., 26, 35–37, 39, 41, 42, 45). First, anatomic and electrophysiological studies have demonstrated the existence of neural pathways connecting vestibular and brain stem autonomic nuclei (23, 28, 31, 38, 47). Second, lesions of the vestibular nerves or vestibular nuclei significantly compromised the ability of cats to compensate for orthostatic hypotension produced by nose-up tilt (4, 12, 19). Third, electrical stimulation of the vestibular nerves (33) or natural vestibular stimulations resulted in changes in sympathetic nerve activity and blood pressure (BP) (9, 16, 17, 32, 40).

Posture changes and movements are detected by two sets of vestibular end organs, semicircular canals and otolith. Angular head turns are detected by the semicircular canals, while linear translation and tilt with respect to gravity are detected by the otolith organs (34). The otolith system has been suggested to play a primary role in the vestibular-sympathetic reflex during changes in posture with regard to gravity (3, 11, 15, 20, 24, 26, 30, 41). Deficits in otolith function result in orthostatic intolerance. For example, microgravity in spaceflight has been shown to elicit marked morphological and physiological changes to the otolith system, and astronauts often experience severe orthostatic intolerance on their return to the normal gravitational environment (2, 21, 27, 48). Understanding of the otolith influence on autonomic control will improve the understanding of the etiology of orthostatic hypotension in patients with vestibular disorders and in astronauts exposed to microgravity, and it will provide insights into developing effective therapeutic treatments.

Earlier animal studies of the vestibular-sympathetic reflex were often conducted in anesthetized or decerebrate animals and employed vestibular stimulations (for review, see Ref. 35) that activated both semicircular canals and the otolith organs. The contribution of the otolith system to the vestibular-sympathetic reflex has not been studied selectively. In the present study, we developed an awake rat model in which pure linear accelerations were employed to selectively activate the otolith system. We found that linear acceleration elicited characteristic cardiovascular responses in awake rats. Furthermore, using a chemical lesion method, we functionally identified the regions in the central vestibular nuclei that mediate the linear acceleration-evoked cardiovascular responses.

MATERIALS AND METHODS

Animals

Male Sprague-Dawley rats weighing 300–500 g (Harlan Sprague-Dawley, Indianapolis, IN) were used in this study. The animals were
maintained in a laboratory animal facility at 22°C under a 12:12-h light-dark cycle with food and water available ad libitum. All procedures were approved by the Institutional Animal Care and Use Committee at University of Mississippi Medical Center.

Surgical Procedures

Head holder or injection cannula implantation. All surgical procedures were performed aseptically. A head holder was surgically implanted on the skull to allow for stabilization of the rats. Animals were anesthetized with pentobarbital sodium (50 mg/kg ip), and the head was fixed on a stereotaxic instrument. A midline dorsal cranial skin incision was made, soft tissues were cleared, and the head was leveled between the skull suture landmarks, bregma and lambda. The head holder is a small stainless steel cylinder that was rigidly attached to the skull. The head holder was secured in place with three stainless steel machine screws trepanned through the skull and adhered with dental acrylic. In another group of rats, in addition to head holders, injection guide cannulas were implanted for microinjection of muscimol for chemical lesions. Burr holes were made in the skull over coordinates corresponding to the medial and inferior vestibular nuclei (MIVN). The coordinates are estimated from the atlas of Paxinos and Watson (22). A 26-gauge cannula (Plastic One, Roanoke, VA) was placed 1 mm above the MIVN bilaterally or unilaterally. The head holder and the guide cannula were secured in place with four stainless steel machine screws and dental acrylic. Rats were housed individually after the surgery and permitted to recover for at least 7 days before surgical catheterization and tests.

Surgical catheterization. An abdominal aortic catheter was chronically implanted via the femoral artery to allow for measurement of BP. Rats were anesthetized with halothane (2–5% in medical-grade oxygen). A heparinized saline (10 units/ml) filled polyethylene catheter (PE 50) was inserted into the femoral artery and advanced 3–5 cm to the abdominal aorta. The catheter was tied in place with two 5-0 silk sutures, and the free ends were passed through a subcutaneous tunnel to exteriorize at the nape of the neck. Rats were housed individually and permitted to recover for 48 h before tests.

Sinoaortic baroreceptor denervation. Rats were anesthetized with halothane. A midline incision was made in the neck to expose the carotid bifurcation bilaterally. To denervate the aortic baroreceptor zones, all visible aortic nerves were severed where they branched from the superior laryngeal nerve, and the superior laryngeal nerve was cut at the junction with the vagus nerve near the caudal end of the node ganglia. In addition, the superior cervical ganglia and 1 cm of the cervical sympathetic truck were removed. To denervate the carotid region, the adventitia of the carotid bifurcation was raised above the surrounding tissue and painted with a solution of 10% phenol in absolute ethanol. Following this procedure, the region was rinsed twice with sterile saline. Sinoaortic baroreceptor denervation leads a transient increase in arterial pressure and a Horner's-like syndrome. To minimize the risk as result of this transient hypertension, animals were anesthetized with pentobarbital sodium anesthesia (50 mg/kg ip) and placed in the prone position from moving. The rat's head was stabilized by attaching a stainless steel rod to the surgical implanted head holder. The linear motions were in four directions (i.e., forward, backward, left, and right) and consisted of an acceleration phase of 200 ms (1–3 m/s²) followed by a deceleration phase of 200 ms (1–3 m/s²). Directions of linear motion were randomized throughout the recording sessions so that the animals could not predict the direction at the onset of each movement. Both pulsatile BP and average BP were measured using a disposable pressure transducer connected to the femoral artery catheter. BP signals were displayed on a previous calibrated blood pressure display unit (Stemtech, Milwaukee, WI) and were digitized at 2 kHz with 16-bits resolution and saved on a hard disk for offline analysis (Cambridge Electronic Design, Cambridge, UK). Before the data collection, 30 linear motions were delivered to habituate startle responses. After this, 80 trials (20 trials for each direction) were tested for each rat each day for 3 consecutive days. Most of the trials were conducted under “light-off” condition; i.e., visual cues were available during linear motion (12, 19). Because linear head motions not only activate the vestibular system but also activate the visual system by generating retinal slips, the role of visual cues in the linear acceleration-evoked cardiovascular responses was tested. Some of the trials were conducted under “light-off” condition (without visual cues). For the rats with labyrinthectomy or baroreceptor denervation, changes in BP were measured 2–3 days after the surgery.

Chemical lesions of the MIVN. After collecting control data, a 32-gauge stainless steel injector (Plastic One, Roanoke, VA), which is 1 mm longer than the chronically implanted guide cannula, was placed into the MIVN through the guide cannula. A 1-µl Hamilton syringe driven by an adapted micrometer screw (Stoeling, Wood Dale, IL) was inserted into the guide cannula. A 1-µl Hamilton syringe driven by an adapted micrometer screw (Stoeling, Wood Dale, IL) was connected to the injector through a piece of Tygon tubing (0.38 mm inner diameter, 10 cm long) for pressure injection. Muscimol hydrobromide solution (5 nM, 180 nl, dissolved in saline) was injected over 60 s into the MIVN. Rats received either unilateral or bilateral injections of muscimol. To limit the effects of drug diffusion, a single injection (either single unilateral or bilateral) of muscimol was made in the present study. Twenty minutes after injections, linear acceleration-evoked BP changes were remeasured. Control animals received equal volumes of saline injection. To identify the center of injection site in the brain tissue postmortem, the muscimol solution was tinted with the dye, fast Evans blue. After recording, the animals were anesthetized with Nembutal and perfused with saline followed by a solution of 10% formaldehyde. Coronal serial sections of brain stem were examined to check the sites of injections.
Trials in which BP were unstable within 10 s of motion onset were rejected (~1/10 trials rejected). Trials in the data stream were sorted, aligned on the onset of the head motion, and averaged (~20 trials per condition) to obtain low-noise estimates of average BP and instantaneous HR (derived from the pulsatile BP record) as a function of time.

Data Analysis

The difference between the groups was analyzed by t-test or one-way ANOVA.

RESULTS

Linear Acceleration-Evoked Cardiovascular Responses in Normal Rats

Figure 1 shows raw cardiovascular responses during a single linear motion. The top trace shows the acceleration profile (left motion with peak acceleration of 0.2 g). The bottom two panels show typical HR and BP responses to a left linear motion in awake (lower panel) and pentobarbital anesthetized (top panel) conditions. When the rat was awake, the linear motion-evoked BP response was biphasic with a short-latency (~520 ms) increasing phase (peak at ~2 s) followed by a decreasing phase (peak at ~5 s). The linear motion-evoked HR response had an increasing phase that peaked at ~5 s after motion onset. When the rat was anesthetized, neither the initial increase in BP nor the increase in HR was observed (Fig. 1, top).

Figure 2 summarizes the linear motion-evoked changes in BP and HR on day 1 from a group of 11 rats. Each panel shows the BP (top traces) and HR (bottom traces) responses to linear motion in one of the four directions (forward, top panel; backward, bottom panel; left, left panel; right, right panel) in awake (black traces) and anesthetized (gray traces) conditions. Because there were no significant differences in BP and HR changes evoked by linear motions in the four directions (ANOVA, P > 0.05), data from all directions were pooled. In awake rats, the linear acceleration-evoked BP response had an initial increasing phase with a peak of 8.27 ± 0.66 mmHg (~7% of the baseline BP of 115.08 ± 2.74 mmHg; Student’s t-test, P < 0.001) at 1.97 ± 0.02 s after linear motion onset, followed by a decreasing phase with a peak of −3.23 ± 0.56 mmHg (P < 0.001; Student’s t-test) at 4.64 ± 0.19 s after linear motion onset. For anesthetized rats, the linear motion did not produce a significant increase in BP (P > 0.05, Student’s
t-test), but it induced a decrease in BP with a peak of $-3.33 \pm 1.48$ mmHg at $4.84 \pm 0.06$ s after the linear motion onset. When the rats were awake, the linear motion induced an initial small biphasic response (~2 s duration, an increasing phase with a peak of $5.00 \pm 1.55$ beats/min and a decreasing phase with a peak of $5.05 \pm 1.64$ beats/min) in HR followed by an increase with a peak of $15.97 \pm 1.38$ beats/min (~4% of baseline HR, $384.25 \pm 14.18$ beats/min; $P < 0.001$, Student’s t-test) 5.3 s after linear motion onset. When the rats were anesthetized, the linear motion did not produce significant changes in HR (bottom gray traces in each panel).

The linear motion-evoked BP increases were positively correlated with intensity of linear acceleration ($R^2 = 0.688$, $P < 0.05$; Fig. 3A). However, neither the decrease in BP ($R^2 = 0.04$, $P = 0.70$; Fig. 3B) nor the increase in HR ($R^2 = 0.40$, $P = 0.18$) were significantly correlated to linear acceleration intensity (Fig. 3C).

Two additional control experiments were conducted to demonstrate the specificity of the linear motion-evoked BP and HR responses. First, we measured linear motion-evoked BP and HR responses in 3 consecutive days (Fig. 4). There were no significant differences in linear acceleration (0.2 g)-evoked BP and HR changes among the 3 days ($P > 0.05$, ANOVA). Because on each day the linear motion-evoked BP and HR responses were measured after 30 repeated stimulations, these results indicate that the linear motion-evoked BP and HR responses also did not habituate after multiple day stimulation. Thus it was unlikely the BP and HR responses observed in Fig. 2 were due to startle responses. Second, we examined the role of visual cues in the linear acceleration-evoked cardiovascular responses by conducting some trials under light-off condition (without visual cues). We found no significant difference in the linear acceleration-evoked BP and HR responses under light-on or light-off condition ($P > 0.05$, paired t-tests).

Taken together, the above results indicate that linear motion evokes a well-defined short-latency increases in BP and HR that 1) are not due to body fluid movement associated linear motion, 2) are not due to startle response, and 3) are not mediated by vision, and the results indicate that 4) the increase in BP is positively correlated with linear acceleration intensity. The next two experiments were conducted to establish the otolith origin for the linear motion-evoked short-latency increase in BP.

Fig. 3. Relationships between linear acceleration intensity and changes in BP and HR. A: significant correlation between linear acceleration intensity and the increase in BP ($R^2 = 0.688$, $P < 0.05$, $n = 6$). B: nonsignificant correlation between linear acceleration intensity and the decrease in BP ($R^2 = 0.04$, $P = 0.70$). C: nonsignificant correlation between linear acceleration intensity and the maximal increase in HR ($R^2 = 0.40$, $P = 0.18$).

Fig. 4. Summary of linear acceleration-evoked BP and HR changes in awake rats for 3 consecutive days. The onset of linear motion was at time 0. B/m, Beats/min.
Effects of Baroreceptor Denervation on the Linear Acceleration-Evoked Cardiovascular Responses

Baroreceptor-denervation resulted in higher baseline HR (442.77 ± 6.60 vs. 384.25 ± 14.18 beats/min; t-test, P < 0.05), but did not induce significant change in baseline BP (122.52 ± 2.27 vs. 115.08 ± 2.74 mmHg; t-test, P > 0.05). Figure 5 illustrates the effects of baroreceptor denervation on the linear motion (0.2 g)-evoked BP and HR responses. The rats with baroreceptor denervation showed similar BP and HR changes in response to linear motions in the four directions tested. Thus the responses of BP and HR in all directions were pooled. Figure 5 reveals two findings. First, the short-latency increase in HR but not the short-latency increase in BP. These results indicate that baroreceptor reflex mediated the longer latency increase in HR but not the short-latency increase in BP. Because the study was to examine the otolith-driven cardiovascular responses, in the rest of the paper we focused on the linear motion-evoked short latency BP response.

Effects of Unilateral or Bilateral Labyrinthectomy on the Linear Acceleration-Evoked Initial BP Changes

The effects of labyrinthectomy on linear acceleration-evoked BP responses were studied to establish vestibular origin for the linear motion-evoked short-latency increase in BP. Bilateral labyrinthectomy did not result in significant differences in baseline BP (116.15 ± 1.20 vs. 115.08 ± 2.74 mmHg; t-test, P > 0.05). Figure 6 shows that the maximal increase in BP elicited by linear motions (0.2 g) was significantly less in bilateral labyrinthectomized rats than that of normal rats (2.64 ± 1.83 vs. 8.27 ± 0.66 mmHg; P < 0.01, t-test). Unilateral labyrinthectomy did not result in significant changes in linear motion-evoked BP increase (P > 0.05; n = 2; data not shown).

Effects of Chemical Lesions of the MIVN on the Linear Acceleration-Evoked Initial BP Change

To identify the central sites that mediate the linear motion-evoked BP response, we examined the effects of inactivation of the MIVN by microinjection of muscimol on the linear acceleration-evoked BP changes. The linear acceleration-evoked cardiovascular changes were measured before and after bilateral or unilateral muscimol injections. To limit the diffusion of muscimol to other brain areas, a single injection of muscimol was made on each side. Muscimol injection did not induce significant changes in baseline BP (108.25 ± 1.85 vs. 108.55 ± 2.36 mmHg; n = 4; paired t-tests, P > 0.05) and baseline HR (419.93 ± 9.55 vs. 394.84 ± 29.68 beats/min; n = 4; paired t-tests, P > 0.05). A total of 10 rats received muscimol injections. The injection sites of eight rats were within the MIVN, which were confirmed by histology after the tests. Figure 7 summarizes the centers of muscimol injection sites (effective injection sites in filled symbols and noneffective injection sites in open symbols). Bilateral injections of muscimol into the MIVN resulted in smaller linear motion-evoked changes in BP in four of four rats (Fig. 8, left). Unilateral injections, however, resulted in smaller changes in BP in two of four rats (Fig. 8, right). Sham injections did not affect the linear acceleration-evoked BP changes (data not shown). For the other two rats, the injections missed the target; one was found in cerebellum, and the other was in nucleus tractus solitarius. The injections on these sites did not induce significant changes in the linear motions-induced maximum increase in BP and HR (data not shown). These results indicate that the linear motion-evoked BP increase was mediated by neurons in the MIVN.
DISCUSSION

The present study demonstrates for the first time that linear motions, which selectively stimulate otolith system, evoke well-defined short-latency cardiovascular responses in conscious rats. These results not only demonstrate the otolith specific influences on the cardiovascular system but also provide an effective rodent model for investigating the neural mechanisms underlying vestibular mediated cardiovascular regulation. A series of experiments were performed in the present study to reveal the following characteristics of the linear motion-evoked BP and HR responses in rats. First, in awake rats, all of the four directions of linear accelerations (forward, backward, left, and right) produced a biphasic response in mean BP, i.e., a rapid increase (4 s) followed by a rapid decrease (4 s) in mean BP. When the rats were anesthetized by pentobarbital sodium, i.e., lost reflex and sensory responses, the short-latency increase in BP and increase in HR were abolished. Linear motion only induced a decrease in BP. These results suggest that the decreasing phase was not reflex driven and could result from body fluid movement. Previous work in humans with dynamic linear acceleration by Cui et al. (3) has also shown this biphasic pattern of BP response (Fig. 6B, third trace from the top). Second, the linear acceleration-evoked increase in BP was attenuated by bilateral labyrinthectomy, indicating the BP change was predominantly due to the activation of vestibular receptors. Third, baroreceptor denervation did not affect the linear motion-induced BP response, but it significantly attenuated the HR responses. The results suggest that the linear motion-induced BP response was not driven by baroreceptor reflex. Fourth, the linear acceleration-induced increasing phase of BP response but not the decreasing phase was linear acceleration intensity dependent. Fifth, the observed
BP and HR changes could not be due to a startle response to the linear motions because the cardiovascular responses did not habituate over repeated otolith stimulations. Furthermore, visual information did not play an important role in the linear acceleration-evoked cardiovascular responses. Taken together, these results indicate that the linear motion-induced short-latency increase in BP was of otolith origin. The decreasing phases in BP and the HR responses, however, were not primarily driven by vestibular stimulation. Thus the short-latency increase in BP is the focus of our study to investigate the neural mechanism underlying the vestibular-mediated cardiovascular regulation.

It is worth noting that bilateral labyrinthectomy attenuated but not did not abolish the linear motion-induced BP changes. This could be due to incomplete the labyrinthectomy, which was achieved by inner ear implantations of streptomycin pellets. Another possibility is that nonlabyrinthine signals may substitute the otolith signals and lead to a partial function recovery. Although the labyrinth provides a large input to vestibular nucleus neurons, the central vestibular nuclei receive inputs from nonlabyrinthine sensory signals that can provide information regarding body position in space, such as somatosensory and proprioceptive inputs (13, 14). Recent studies suggest that nonlabyrinthine inputs to the central vestibular system may be important in controlling blood pressure during movement, particularly following vestibular dysfunction (44–46). In this study, we also found that unilateral labyrinthectomy did not significantly affect the linear acceleration-evoked BP response. Whether the lack of effect of unilateral labyrinthectomy results from a rapid vestibular compensation by the intact side remains to be determined.

In the present study, we further identified the central sites that mediate the linear acceleration-induced cardiovascular responses. The MIVN have been suggested to be the primary central vestibular nuclei for mediating vestibular-autonomic reflexes. Electrolytic or chemical lesions of these areas have been shown to abolish sympathetic nerve responses to natural or electrical vestibular stimulation (33, 38, 40). Anatomic studies have identified the direct connections between the MIVN and brain stem autonomic regions (1, 23, 28). Thus, in the present study, we examined whether the MIVN mediate the linear acceleration-evoked increase in BP. We found that inactivation of the MIVN by microinjection of muscimol significantly attenuated the linear motion-evoked short-latency increase in BP. These results provide evidence that neurons in the MIVN mediate the otolith-cardiovascular reflex. It has been proposed that vestibular neurons that participate in generating vestibular-sympathetic responses integrate a number of sensory signals and modulated by inputs from Purkinje cells in the posterior cerebellar vermis (13, 14, 44–46). In future studies, it will be important to determine whether the otolith-driven cardiovascular responses are modulated by other brain areas that provide inputs to the vestibular nuclei.

The linear acceleration-evoked cardiovascular response in conscious rats was similar to that observed in human studies that employed different behavioral paradigms to stimulate the otolith system. A series of studies by Ray and colleagues first demonstrated the involvement of the otolith system in the static head-down rotation in the prone positions, which activates the otolith organs, elicits increases in muscle sympathetic nerve activity and limb vasoconstriction (11, 20, 24, 30), whereas yaw head rotation, which stimulates the horizontal semicircular canals, does not affect sympathetic outflow (25). They also found that visual inputs and baroreceptor reflex did not contribute to the head-down neck flexion-induced changes in sympathetic nerve activity (30). Yates et al. (43) demonstrated that transient horizontal linear acceleration induces a rapid increase in BP and the response was much smaller in patients...
with vestibular dysfunction. Our results are also consistent with previous animal studies by Gotoh and colleagues (9, 32) that found BP changes during altered gravity, which is also sensed by the otolith system.

Modest linear accelerations (0.2 g) induced a mean increase in systolic BP of ~8 mmHg in rats as well as in human subjects (43), which was only 7% of the average baseline BP. It is unlikely that such small changes in BP would have any significant physiological effects. Nevertheless, this small but reliably measurable increase in BP reveals two important features of the otolith-mediated cardiovascular regulation. First, there exist specific neural pathways that channel otolith afferent signals to the central regions that regulate blood pressure. We performed a series of control experiments and demonstrated that the linear motion-evoked short-latency increase in BP is not only of otolith origin but also is mediated by neurons in the MIVN. Second, we suggest that neural signals carried in otolith afferents cannot be directly channeled into the cardiovascular reflexes because these signals are ambiguous; i.e., they may result from either translation that results no change in orthostatic column or tilt that results in changes in orthostatic column (6, 7). It is the change in orthostatic column that challenges stable BP and needs to be rapidly compensated. Because there is no change in orthostatic column during the horizontal linear motion in the present study, there is no need for adjustments in BP. The small BP change observed in this study is consistent with this analysis. We propose that the central vestibular system must discriminate tilt from translation to generate proper cardiovascular responses. In an ideal situation, the central tilt-translation discrimination mechanism should completely turn off the otolith afferent input during translation and does not evoke any changes in BP. Because a small increase in BP was observed during translation, we suggest that in rats the central tilt-translation discrimination mechanism works adequately but not perfectly. How the central vestibular system resolves the ambiguity in otolith afferent signals remains the central issue of the vestibular physiology (10). Recent single unit recording from monkeys suggests one possible solution, in which a group of vestibular neurons receive inputs from both canal and otolith afferents but its firing rates encode only tilt (49). These neurons exhibit strong sensitivity to static and dynamic tilt but minimum sensitivity to translation. If these neurons exist in a rat’s MIVN, their strong modulation during tilt may contribute to the maintenance of stable BP when orthostatic column is altered, and their small modulation during translation may contribute to the small BP changes observed in the present study. We propose that these neurons may play an important role in transforming the raw otolith afferent signals into signals that encode head tilt, which are then channeled to drive the otolith-cardiovascular reflexes.

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GRANTS

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