Role of the Cerebellum and the Vestibular Apparatus in Regulation of Orthostatic Reflexes in the Cat

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ABSTRACT

The contribution of the fastigial nucleus and the vestibular nerves (eighth cranial nerves) to the orthostatic reflexes in anesthetized, paralyzed cats was studied. Bilateral lesions of the rostral fastigial nucleus resulted in impairment of the reflex changes in blood pressure, femoral arterial flow, and resistance evoked by head-up tilting to 30° or 60°. The deficit consisted of an increase in the magnitude of the initial fall in blood pressure during tilting. The effects on blood pressure were paralleled by decreased vasoconstriction in the femoral artery. Extracranial lesions of the vestibular nerves produced comparable deficits which were not enhanced by subsequent lesions of the fastigial nucleus. Denervation of the baroreceptors impaired the reflexes, and subsequent lesion of the fastigial nucleus increased this deficit. The pressor response evoked by electrical stimulation of the rostral fastigial nucleus also reversed the deficit in orthostasis produced by hemorrhage. Small doses of sodium pentobarbital which did not alter the resting blood pressure or the pressor response to carotid occlusion impaired the responses to stimulation of the fastigial nucleus and tilting. Therefore, the rostral fastigial nucleus, which might be triggered by the vestibular apparatus, appears to participate in concert with the baroreceptors in the initiation and possibly the maintenance of the orthostatic reflexes.

KEY WORDS sympathetic nervous system blood pressure posture cerebellar nuclei blood flow brain stimulation baroreceptor reflexes

The orthostatic reflexes consist of a patterned activation of sympathetic neurons in response to the assumption of an upright posture (1). The preponderant effect is a widespread vasoconstriction of resistance and capacitance vessels and tachycardia (1, 2). The vasoconstriction tends to resist the hydrodynamic action of gravity which forces the blood into the dependent extremities and viscera; the response thereby maintains blood pressure and protects circulation to the brain. It is widely assumed that the orthostatic responses are triggered by baroreceptors, particularly those in the carotid sinus (1, 3) and possibly those in the low-pressure circulation (4-6). According to this view, the reduction in stretch of the sinus during standing results in a decrease in discharge from the baroreceptors, a release of pre-ganglionic sympathetic neurons from inhibition, and thus an increase in sympathetic nerve activation and vasoconstriction. There are, however, several studies (7, 8) in which deafferentation of baroreceptors failed to abolish the orthostatic responses. Such findings suggest that receptors other than the baroreceptors may participate in initiating these postural reflexes.

It has recently been discovered (9-11) that electrical stimulation of a highly restricted region of one of the deep nuclei of the cerebellum, the fastigial nucleus, can elicit a powerful activation of the cardiovascular system. The cardiovascular events associated with the fastigial pressor response consist of an elevation in systemic arterial blood pressures, tachycardia, and vasoconstriction of arteries in limbs, kidney, and abdominal viscera (11-14); this cardiodynamic pattern simulates the orthostatic reflex. On this basis we have suggested (14) that the cerebellum may participate in the regulation of orthostatic mechanisms. Moreover, since the cerebellum receives neural input from the vestibular apparatus (15-17), the vestibular apparatus may also participate in the cardiovascular responses to upright posture.

In the present study we attempted to determine whether the cerebellum and the vestibular apparatus participate in initiating orthostatic reflexes and whether they interact with baroreceptors in performing this function by studying the effects of selected lesions or of electrical stimulation of the fastigial nuclei, the vestibular nerves, and the
baroreceptors on the reflex cardiovascular responses to tilting in the cat. Our findings supported the view that cerebellar and vestibular mechanisms are involved in initiating and sustaining orthostatic reflexes. Preliminary reports of this study have been published (12, 18).

Methods

Forty-two adult cats were anesthetized with alpha-chloralose (40-50 mg/kg, iv). All cats were paralyzed with gallamine triethiodide (5 mg/kg, iv) and artificially ventilated through a tracheal cannula. End-tidal CO₂ recorded by an infrared gas analyzer (Beckman L1), was maintained at 2-3%. Rectal temperature was maintained at 37°C by a thermostatically regulated infrared lamp.

A polyethylene catheter was placed in the aorta via the femoral artery, and the tip was positioned at the approximate point where the diaphragm is attached to the posterior body wall. This level corresponds to the hydrostatic indifferent point (1). The location of the catheter tip was confirmed at the end of each experiment. The systemic blood pressure was recorded from the aortic catheter by a pressure transducer (Statham P23Db) and the heart rate was computed from the blood pressure pulse by a cardiotachometer (Beckman type 9857). These records were simultaneously displayed on channels of a polygraph (Beckman Dynograph type 504A). Blood flow was recorded from the femoral artery by an electromagnetic flowmeter as previously described (14), and the vascular resistance was computed by dividing the mean arterial blood pressure by the mean arterial flow. The mean arterial blood pressure was derived from the aortic catheter by a pressure transducer (Statham P23Db), and the heart rate was computed from the blood pressure pulse by a cardiotachometer (Beckman type 9857). These records were simultaneously displayed on channels of a polygraph (Beckman Dynograph type 504A). Blood flow was recorded from the femoral artery by an electromagnetic flowmeter as previously described (14).

The mean arterial blood pressure was derived from the formula \( P_a = \frac{P_s + 2P_d}{3} \), where \( P_s \) is systolic pressure and \( P_d \) is diastolic pressure. After attaching flow probes to the appropriate arteries, the cat was placed in a stereotactic frame with the head flexed to 45°. Two electrodes consisting of Teflon-coated steel wires (diameter 0.006 inches) bared at the tip for 1 mm and carried in no. 28 stainless-steel hypodermic tubing (14) were placed separately into each fastigial nucleus through a small burr hole in the calvarium just above the nuchal ridge. To identify the optimal placement in the fastigial nucleus, the electrodes were lowered to be the cerebellum initially in 0.5-mm steps, and monopolar stimulation was applied; the indifferent electrode was a copper clip placed on the temporalis muscle. The brain was stimulated with square-wave pulses (0.1–0.2 ma, 50 Hz, 0.1-msec pulse width) generated by a constant-current stimulator until a site was reached from which an optimal pressure response was evoked (9). The electrodes were then firmly secured to the skull with dental cement. Later in the experiment, electrolytic lesions were made through these electrodes.

After implantation of the electrodes, the cat was placed on a tilt table with a suitable saddle and secured by ligatures tied to the forelimbs. The head and neck were firmly fixed to the table thereby preventing their movement during tilting. The axis of tilt of the table passed through the approximate point of the tip of the catheter in the aorta. The zero reference line of a pressure transducer was placed at the level of the axis of tilt. Thus, the zero level was constant at all times during tilting. Pressure recorded in this way represents central perfusion pressure (1). The cat was then tilted to 30° and 60° in a head-up position off the horizontal over 2–3 seconds. Within this time there was no appreciable alteration in systemic blood pressure (7). The upright posture was maintained for exactly 1 minute, and then the cat was returned to the horizontal position over 2–3 seconds. After obtaining base-line observations on the cardiovascular responses to tilting, the cat was subjected to various experimental procedures including placement of electrolytic lesions in the fastigial or other deep cerebellar nuclei, electrical stimulation of the fastigial nuclei, denervation of the vestibular nerves or peripheral baroreceptors, and intravenous administration of drugs. A minimal interval of 30 minutes was allowed following placement of lesions and retesting of the cardiovascular responses.

Electrolytic lesions of the cerebellar nuclei were made by passing a d-c anodal current (5 ma) for 30–45 seconds from a constant-current d-c source through the implanted electrodes. In 11 cats, lesions were placed bilaterally in the fastigial nuclei at the pressor sites. In 8 cats, extrafastigial cerebellar lesions were produced in or near the adjacent interpositus nuclei. In 8 cats, the vestibular nerve was denervated extraebrainially. The tympanic bullae were exposed ventrally through the neck; the mastoid processes were exposed by midline cervical incision and opened with an electric drill. With careful dissection, the peripheral branches of the vestibular nerve innervating the semicircular canals and the utricle would be visualized along with part of the basal portion of the cochlea. These branches were transected with small iris scissors. In 6 cats, the peripheral baroreceptors were denervated by transecting both the carotid sinus nerves and the vagi at the midcervical level. In some experiments, the response to carotid occlusion was determined by occluding the common carotid arteries bilaterally with a small arterial clamp for 12 seconds. When electrical stimulation was combined with the tilt procedures, the fastigial nucleus was stimulated with a 1-2-minute train of 0.1-msec square-wave pulses at a stimulus frequency of 10 Hz.

At the end of each experiment, the cat was killed by perfusing the heart with 10% formalin (w/v). The brain was removed and fixed, and frozen sections were stained for cells by Nissl's method to identify the lesion sites. The significance of changes in cardiovascular parameters was estimated by a paired t-test; \( P < 0.05 \) was significant.

Results

**CARDIOVASCULAR RESPONSES TO HEAD-UP TILTING IN THE ANESTHETIZED, PARALYZED CAT**

The reflex cardiovascular responses to head-up
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Cardiovascular responses to a head-up tilt in the anesthetized, paralyzed cat. a: Typical tracing showing changes in heart rate, systemic arterial blood pressure, femoral arterial resistance, and femoral arterial flow during 1 minute of head-up tilting to 30° or 60°. The onset of tilting is signified by the upward arrow, the termination is signified by the downward arrow. Note that the vascular responses are graded. b: Schematic representation of the phases of the blood pressure response to tilting. See text for details.

Blood pressure

Femoral arterial resistance

Femoral arterial flow

Figure 1

The typical cardiovascular responses to tilting were seen in 42 of 67 cats. In the remaining 25 cats, the blood pressure did not return to control levels during the 1-minute tilt or when tilting was repeated. These cats were not included in this study. In most of the cats for which the data were discarded, however, the diminished orthostatic response appeared to be the consequence of a reduction in blood volume, because infusion of 10-15 ml of normal saline or plasma expander (Rheomacrodex) resulted in the return of a well-developed, maintained blood pressure during head-up tilting.

EFFECTS OF PARALYSIS ON ORTHOSTATIC REFLEXES

To eliminate any effects of muscle contraction on the orthostatic reflexes, the cats were paralyzed with gallamine triethiodide (5 mg/kg, iv) and artificially ventilated. Paralysis did not produce any significant differences in the blood pressure responses to tilting to 30° but did slightly increase the magnitude of the initial fall in blood pressure during tilts of 60°. This increased fall in blood pressure could be averted by firmly binding the abdomen, indicating that it probably resulted from increased pooling of blood in the splanchnic bed as a consequence of paralysis of the abdominal muscles. These findings indicate that proprioceptors probably do not participate significantly in triggering postural cardiovascular reflexes in the anesthetized cat.

EFFECTS OF BILATERAL LESIONS OF THE FASTIGIAL NUCLEUS ON ORTHOSTATIC REFLEXES

Bilateral electrolytic lesions were focally placed within the area of the fastigial nucleus from which a pressor response was evoked by electrical stimulation (Fig. 2). Such lesions significantly impared the cardiovascular response to tilting; a representative experiment is illustrated in Figure 3.
Figure 2
Bilateral electrolytic lesions of the rostral and the medial portions of the fastigial nucleus in the cat. This lesion impaired the orthostatic reflex. Bar = 1 mm.

Figure 3
Effects of bilateral electrolytic lesions of the rostral and the medial portions of the fastigial nucleus (FN) on blood pressure (BP), femoral arterial resistance, and femoral arterial flow during tilting in the anesthetized, paralyzed cat. The cat was tilted head-up to 30° or 60° at the upward arrows and returned to the horizontal position at the downward arrows.

Figure 4
Effects of bilateral lesions of the fastigial nucleus (FN) on the mean aortic blood pressure during 1 minute of head-up tilting to 30° or 60° in 11 anesthetized, paralyzed cats. Open circles represent prelesion values and solid circles represent values after lesions had been made. Each point represents the mean ± se. Note the impairment in blood pressure responses produced by fastigial lesions.

Control • FN lesions
Diffs from control: * p<.01, ** p<.001

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and pooled data is presented in Figure 4. The deficits included (1) an augmentation of the initial fall in blood pressure (Figs. 3 and 4), (2) a delay in the onset of the early, compensated phase (Figs. 3 and 4), and (3) a failure during the late, compensated phase of blood pressure to return to control values (Fig. 4), particularly with a tilt of 60°.

The collective impairments in the blood pressure responses to tilting after fastigial lesions were probably the result of a decrease in vasoconstriction (Fig. 3) which was reflected by a delay in the onset of arterial resistance, a slower rise to peak, and a failure in the maintenance of the reflex increase in arterial resistance. The changes in vascular resistance produced by the brain lesion were consistent and surprisingly similar in magnitude from cat to cat (Fig. 5). Despite the impairment of vascular responses, the lesions did not alter the reflex tachycardia.

**FIGURE 5**

Effects of bilateral lesions of the fastigial nucleus (FN) on the changes in femoral arterial resistance elicited by head-up tilting to 30° and 60° in 11 anesthetized, paralyzed cats. The resistance was measured at the nadir of the initial hypotensive response to tilting.

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**FIGURE 6**

Effects of bilateral extracranial denervation of vestibular nerves (VIIIth nerve) on mean aortic blood pressure response to graded tilting in eight anesthetized, paralyzed cats. The control responses represent the responses to tilting after opening of the tympanic bullae but prior to transection of the vestibular nerves and do not differ from the responses in other unoperated controls (see Fig. 3).

EFFECTS OF EXTRAFASTIGIAL LESIONS OF CEREBELLAR NUCLEI ON ORTHOSTATIC REFLEXES

To determine the anatomical specificity of fastigial lesions, bilateral lesions were placed in the interpositus nucleus and adjacent deep cerebellar nuclei in eight cats. Such lesions had no effect on the blood pressure response to tilting, thereby demonstrating the anatomical specificity of the rostral fastigial nucleus in the regulation of the orthostatic reflexes.

EFFECTS OF DENERVATION OF THE VESTIBULAR NERVES ON ORTHOSTATIC REFLEXES

Because the fastigial nucleus receives information from the vestibular apparatus (15-17), the fastigial mechanisms involved in orthostatic reflexes might be triggered by vestibular stimuli. To test this possibility, the effects of denervating the vestibular nerves on the blood pressure responses to tilting in eight cats were examined. As seen in Figure 6, bilateral extracranial lesions of the vestibular nerves significantly impaired the responses to tilting. The deficits were most evident during the initial phase of a 30° tilt and throughout all phases of a 60° tilt.

The impaired blood pressure responses to tilting produced by vestibular nerve lesions did not differ.
from those produced by fastigial nucleus lesions alone. Moreover, in three cats, the subsequent placement of bilateral lesions in the fastigial nucleus after bilateral vestibular nerve transection did not produce any further deficit. Thus, it appears that fastigial and vestibular projections involved in the orthostatic reflexes share some common neuronal mechanism.

**INTERACTION OF BARORECEPTORS WITH FASTIGIAL MECHANISMS**

To examine the effects of denervation of baroreceptors on responses to tilting, either alone or in combination with lesions of the fastigial nuclei, the carotid sinus and the vagus nerves were bilaterally transected in six cats. Such denervation resulted in a sustained elevation of blood pressure (Fig. 7) as previously described (13). Head-up tilting to 30° resulted in a fall in blood pressure which failed to show any compensation over the 1-minute period. However, the mean blood pressure which was maintained during the tilt in baroreceptor-denervated cats was the same as that observed prior to buffer nerve transection. With a 60° tilt, the fall in blood pressure was greater, reaching levels that were slightly lower than control levels.

Bilateral fastigial nucleus lesion in cats whose buffer nerves were sectioned did not alter the elevated mean blood pressure but did cause an impairment in the compensatory response to tilting greater than that produced by a buffer nerve transection alone (Fig. 7). This finding indicates that the baroreceptor and fastigial mechanisms are additive in initiating or sustaining orthostatic reflexes, suggesting that they are subserved, at least in part, by separate neuronal mechanisms.

Even after the transection of baroreceptors and the placement of bilateral lesions in the fastigial pressor areas, blood pressure during tilting was sustained within the physiological range. The subsequent administration of phentolamine (1 mg/kg, iv), however, resulted in a prompt fall in mean blood pressure and the virtual disappearance of any compensatory responses to tilting (Fig. 7). This finding suggests that, in addition to baroreceptor and vestibular-fastigial control, mechanisms possibly of spinal origin participate in mediating the reflex blood pressure responses to tilting.

**EFFECTS OF ELECTRICAL STIMULATION OF THE FASTIGIAL NUCLEUS ON THE RESPONSES TO BILATERAL CAROTID OCCLUSION**

To determine the interaction between the fastigial pressor response and the orthostatic reflexes the effect of electrical stimulation of the pressor area of the fastigial nucleus on the pressor response to bilateral carotid occlusion was ex-
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Blood pressure (mm Hg)

FIGURE 9
Effects of electrical stimulation of the fastigial nucleus (FN) on an impaired orthostatic reflex response produced by hypovolemia in the anesthetized, paralyzed cat. a: Control tilt. b: Tilt response 15 minutes after withdrawal of 15 ml of blood. c: Combined stimulation of fastigial nucleus (FN) with tilting immediately after withdrawal of 15 ml of blood. The fastigial stimulus was a train of square-wave pulses (50 Hz at 0.4 mA). Note that the impaired orthostatic response induced by hypovolemia is corrected by simultaneous stimulation of the fastigial nucleus.

amined. The pressor responses to both carotid occlusion and stimulation of the fastigial nucleus summated (Fig. 8). This observation further indicates that the fastigial and the baroreceptor mechanisms involved in orthostatic control are, in part, distinct.

EFFECTS OF ELECTRICAL STIMULATION ON DEFICIENT ORTHOSTATIC REFLEXES

The previous studies have demonstrated that lesions of the fastigial nucleus will impair the orthostatic reflexes. We also attempted to determine whether electrical stimulation of the fastigial nucleus at the pressor region could reinforce an impaired orthostatic reflex and revert it to normal. The orthostatic reflex was decompensated in four cats by acute withdrawal of 10-15 ml of blood. A typical experiment is illustrated in Figure 9. Following the withdrawal of blood, the usual orthostatic reflex response elicited by a head-up tilt of 60° (Fig. 9a) was converted to an uncompensated response (Fig. 9b). The abnormal response was entirely restored to normal (Fig. 9c) by coupling the hemorrhage with concurrent fastigial stimulation. The intensity of stimulation of the fastigial nucleus required for compensation was always above threshold for a pressor response in the supine cat. However, the observations demonstrate that fastigial excitation can serve to reinforce and compensate deficient orthostatic reflexes.

EFFECTS OF SODIUM PENTOBARBITAL ON THE BLOOD PRESSURE RESPONSES TO TILTING

Finally we attempted to determine whether the fastigial pressor mechanisms participate in the tilting responses in the otherwise intact cat by examin-
tilting response parallels the reduction of the fastigial pressor response produced by a barbiturate that has no effect on the reflex pressor response to carotid sinus occlusion. We conclude, therefore, that in the intact cat the fastigial mechanisms participate in initiating and possibly sustaining the orthostatic reflexes in association with the baroreceptors.

Discussion

The present study has demonstrated that small bilateral electrolytic lesions of the rostral fastigial nucleus can impair the cardiovascular response to head-up tilting in the anesthetized, paralyzed cat (7). The deficit consists of a prolongation of the onset and a reduction in the magnitude of the reflex vasoconstriction. The disorder of vasoconstriction is reflected as a prolongation and enhancement of the transient fall in blood pressure at the moment of tilting and a failure of the blood pressure to return to control levels when the tilt is maintained.

The impairment in the orthostatic responses produced by the lesions cannot be attributed to nonspecific effects of brain damage, because comparable lesions placed in adjacent cerebellar sites fail to influence the response. It is also unlikely to be due to an irritative effect of the lesion, because stimulation at the same site facilitates the response. Therefore, impairment of the orthostatic response to tilting produced by lesions of the cerebellum is probably the result of damage to cells or fibers in the ventromedial portion of the rostral fastigial nucleus. These findings in conjunction with the observations that (1) electrical stimulation restricted to the same area of the fastigial nucleus produces a patterned cardiovascular response simulating orthostatic reflexes (14) and (2) such stimulation will reinforce and possibly correct the deficiency in orthostasis produced by hypovolemia strongly support the hypothesis that the fastigial nucleus modulates the reflex cardiovascular response to posture.

The precise mechanism by which the fastigial nucleus acts in response to tilting is uncertain. If one assumes that the nucleus is reflexly activated by tilting, the question then arises as to the source of the afferent information initiating the response. Since the cats in this study were paralyzed and their heads were not moved on the neck during tilting, it is unlikely that proprioceptive or cutaneous information was important.

One possibility examined in this study was that information rising in the vestibular apparatus may serve as a stimulus. This contention has gained support from the observations that bilateral extracranial transection of the vestibular nerves impairs the tilting response to the same extent as do lesions of the fastigial nucleus alone. The fact that combined lesions of both the vestibular nerves and the fastigial nucleus did not produce a summated deficit indicates that they probably share a common neuronal pool. Whether this finding means that the presumed neurons in the rostral fastigial nucleus which excite sympathetic preganglionic neurons are activated by the vestibular apparatus remains to be established. There is, however, evidence that information arising in the vestibular organs is conveyed into the fastigial nucleus, although the pathways are not clearly known (15-17). Moreover, it remains to be determined if the necessary receptors reside in utricle or semicircular canals. The static deficit in the orthostatic reflex produced by vestibular nerve lesions suggests that at least a part of the deficit can be attributed to gravitational receptors. The fact that stimulation of the vestibular apparatus will evoke the autonomic pattern of orthostasis, however, contrasts with the usual autonomic effect of intensive stimulation of this end organ, namely the autonom ic concomitants of motion sickness (19). Possibly, only a very limited part of the input from portions of the vestibular apparatus may be involved in orthostatic control.

A second input to the fastigial nucleus of importance for orthostasis might be from the baroreceptor nerves themselves. Baroreceptor afferents from the carotid sinus nerve project into the paramedian reticular nucleus (20, 21) which, in turn, projects in a reciprocal manner to the fastigial nucleus (22, 23). Electrical stimulation of the fastigial nucleus at the pressor site excites neurons in the paramedian reticular nucleus (24), inhibits the depressor response to baroreceptor stimulation (11, 12, 24), and facilitates the response to carotid artery occlusion. The fact that baroreceptors might trigger the fastigial pressor response, however, seems unlikely, for the responses produced by denervation of baroreceptors combined with lesions of the fastigial nucleus are additive, thereby suggesting that they involve separate pathways. Further support for the distinction between fastigial and baroreceptor mechanisms acting on tilting is provided by the experiments demonstrating a differential sensitivity of the two systems to small doses of barbiturates.

It is evident that, despite partially distinct neural
networks, the baroreceptors act in concert with fastigial and vestibular mechanisms in regulating orthostatic reflexes. Not only do lesions of both inputs impair the orthostatic responses, but the interaction between the fastigial nucleus and the baroreceptor reflexes favors compensatory mechanisms protecting against the hydrodynamic effects of posture by facilitating the pressor responses initiated by withdrawal of baroreceptor stimulation while simultaneously inhibiting the pressor response to baroreceptor stretch (11, 12).

After a combined denervation of baroreceptors and lesions of the fastigial nucleus, the blockade of alpha-receptors by phentolamine produces an even greater impairment of the hypotension resulting from head-up tilting. This finding suggests that some residual reflex activation of sympathetic nerves produced by tilting is preserved in the absence of baroreceptor and vestibular inputs. The pathways mediating this component of orthostatic responses are unknown. It is possible that spinal mechanisms might also participate. The persistence of some orthostatic reflexes in spinal man (25) and the presence of reflex pressor responses to small distortions of the spinal cord transected at the first cervical segment (26) make this possibility viable.

One of the principal deficiencies in the circulatory response to tilting after bilateral lesions of the fastigial nucleus is a delay in the onset of the compensation of the reflex vascular responses to tilting. This fact suggests that the fastigial nucleus response has a relatively shorter latency than does the response of the baroreceptor. This inference is borne out by the studies of Scher and Young (27) and Warner (28) who found that the time for maximal activation of the carotid baroreceptor reflex responses to a square-wave change in blood pressure (27) or electrical stimulation of the carotid sinus nerve (28) was about 15-20 seconds. Based on their calculations the onset time for the reflex vasoconstriction resulting from tilting, if initiated by baroreceptors alone, would be 7-9 seconds, a figure corresponding to the onset of the response in the cats in this study after bilateral lesions of the fastigial nucleus. The vestibular and the fastigial effects on orthostasis, therefore, are preponderant in the very early phases of tilting. The biological utility of these mechanisms in orthostasis is that they provide a short-latency input to the blood vessels. Thus, the first movements of the head during postural changes will excite the vestibular apparatus leading to an initial patterned activation of preganglionic sympathetic fibers to the heart and the blood vessels. As the movement progresses to a more upright posture, the hydrodynamic changes will excite baroreceptor mechanisms; as these mechanisms begin to participate, they will reinforce and sustain the reflex. The relative contribution of these mechanisms in the alert, unanesthetized cat and their relevance to orthostatic control in man remains to be determined.

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