The fastigial pressor response

Case report

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A distinct vasomotor and cardioregulatory response first identified experimentally was elicited intraoperatively in a 6-year-old girl by local mechanical stimulation in the vicinity of the fastigial nucleus of the cerebellum. These findings are discussed in the light of current experimental knowledge of the anatomy and physiology of the fastigial pressor response.

KEY WORDS • fastigial pressor response • orthostatic reflex • paramedian reticular nucleus • rostral ventrolateral medulla • cardiodynamic response

The fastigial pressor response was first identified in the cat induced by electrical stimulation in the area of the rostral fastigial nucleus of the cerebellum. The cardiovascular response to this stimulation consisted of an elevation in systemic arterial pressure, tachycardia, and vasoconstriction of arteries in the limbs, abdominal viscera, and kidneys. The fastigial nucleus in humans, as in cats and other mammals, is the most medial of the deep cerebellar nuclei and lies close to the midline adjacent to the roof of the fourth ventricle. Fastigiobulbar fibers terminate in the vestibular nuclei and reticular formation of the medulla and pons. Little experience exists with this distinctive pressor-tachycardia response in humans.

This report describes the characteristic response obtained by mechanical stimulation in a 6-year-old child of a discrete region in the cerebellar medullary center near the midline and immediately above the fourth ventricular roof. The experimental background of this pressor-tachycardia response and its probable function are reviewed.

Case Report

This 6-year-old girl was admitted to the neurosurgical service at Victoria Hospital with a 3-year history of progressive gait ataxia and left-hand clumsiness. She was brought to the emergency department after experiencing several falls, always to the left side. Developmental history was normal and there had been no head injury.

Examination. The general physical examination was normal. The patient was alert and intellectually appropriate for her age. Cranial nerve examination revealed bilateral horizontal gaze nystagmus, most severe on the left. Extraocular movements were full and funduscopic examination was normal. Although gross motor and sensory testing were within normal limits, there was marked dysmetria and dysdiadochokinesia involving the left arm and leg. Gait was wide-based and ataxic, with a tendency to fall to the left. Reflexes were brisk and symmetrical. Plantar responses were flexor.

Routine hematological and biochemical screening tests were normal. Computerized tomography (CT) demonstrated a 4-cm area of lucency within the left cerebellar hemisphere which only partially enhanced with intravenous contrast material (Fig. 1 left). The lesion was poorly demarcated from the surrounding cerebellar tissue. Magnetic resonance (MR) imaging suggested a deeply situated neoplasm, consistent with a low-grade glioma.

First Operation. The patient was taken to the operating room where, through a left suboccipital craniectomy, the left cerebellar hemisphere was explored. A poorly demarcated grayish mass was found approximately 1 cm deep to the cerebellar cortex. Pathological diagnosis of multiple biopsies was consistent with low-grade cerebellar astrocytoma. Because a definite plane between tumor and normal cerebellar tissue could not be established, complete removal was not attempted.
was resumed from a different approach; however, when
the same location was reached the heart rate quickly
rose again to 150 beats/min, accompanied by multi-
ple premature ventricular contractions. Systolic
arterial pressure rose to 180 mm Hg but was again quickly
brought under control by withdrawing from the site.
All grossly identifiable tumor was removed from within
the cerebellum and the remainder of the procedure was
completed uneventfully. Blood loss was limited to less
than 60 cc and total time of anesthesia was 5.25 hours.

Postoperative Course. The patient awoke in the rec-
covery room and was oriented and able to converse,
demonstrating good strength in all limbs. Her pupils
were briskly reactive and there was no restriction of
extracranial movement. The preoperative left-sided dys-
metria remained. Approximately 6 hours postopera-
tively, she suddenly sat upright, vomited, and became
extremely bradycardic, followed by complete cardiac
arrest. Both pupils became dilated and unreactive; no
limb response could be elicited.

Following intubation and cardiopulmonary resuscita-
tion, normal sinus rhythm was restored. Both pupils
became sluggishly reactive and minor bilateral sponta-
neous arm movement was observed. An urgent CT scan
showed no evidence of hemorrhage or hydrocephalus.
The patient remained deeply comatose and serial CT
scans showed progressive diffuse supratentorial edema
and ischemic changes. Electroencephalography and
brain-stem auditory evoked potential recording were
consistent with severe, diffuse hypoxic-ischemic en-
cephalopathy. Cerebral death was declared on the 6th
postoperative day.

Pathological Examination. Examination of the tu-
mor fragments under light microscopy with hematoxy-
lin and eosin (H & E) staining revealed moderately
cellular areas containing astrocytes with small dark
nuclei against a fibrillary background. A few Rosenthal
fibers were present. This section of the tumor was
consistent with cerebellar astrocytoma. In addition,
there were areas with tumor cells arranged in rows
mixed with small calcifications. The cells had dark
round nuclei with perinuclear halos showing the classic
“fried egg” appearance characteristic of oligodendro-
glioma. The final diagnosis was mixed cerebellar astro-
cytoma and oligodendroglioma.

Postmortem examination identified microscopic is-
lands of residual tumor in the bed of the resection
within the cerebellum (Fig. 2). The medulla oblongata
and cerebellar peduncles were free of surgical artifact.
The cerebellar nuclei, in particular the fastigial nucleus,
were not clearly identified with H & E staining.

Discussion
In a variety of species, experimental electrical stimu-
lation of the cerebellar cortex, the deep cerebellar
nuclei, and the adjacent white matter has long been
known to alter systemic arterial pressure, heart
rate, and regional blood

* Ultrasonic surgical aspirator manufactured by Cavitron
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flows. The fastigial pressor response elicits a powerful activation of both vasomotor and cardioregulatory mechanisms by sympatheoxcitatory pathways relayed through medullary centers directly to the intermediolateral cell column of the spinal cord, which is the origin of sympathetic preganglionic neurons. Two medullary centers, in particular, have been identified by neuroablative and electrophysiological studies as targets of these fastigio bulbar projections: the paramedian reticular nucleus and the rostral ventrolateral medulla. The paramedian reticular nucleus lies in the medial reticular formation at the level of the midportion of the inferior olivary nucleus and is bordered by the raphe medially, the hypoglossal nucleus superiorly, the descending hypoglossal fibers laterally, and the corticospinal fibers comprising the pyramid ventrally. In addition to fastigio bulbar fibers, the paramedian reticular nucleus receives projections from the carotid sinus nerve, solitary nuclear complex, and the vestibular nuclei among others. This site has long been known to influence vasomotor tone, and both vasopressor and vasodepressor actions have been demonstrated here. More recently, a distinct inhibitory interaction between projections from the region of the fastigial nucleus and carotid sinus nerve influencing systemic arterial pressure has been demonstrated. Individual neurons have been shown to receive either or both projections and to project directly to spinal cord sympathetic centers. The ventrolateral medulla is also a well-known vasomotor center which sends fibers to the intermediolateral nucleus. Although no direct fastigio bulbar projection to this site has been demonstrated, lesions of the rostral ventrolateral medulla have resulted in significant reductions in the amplitude of vasopressor and cardiac responses to stimulation of the region of the fastigial nucleus.

A fastigial depressor response has also been identified by stimulation of local neurons and not axons-of-passage in the fastigial nucleus by excitatory amino acids. The fastigial depressor response is characterized by a marked hypotension and bradycardia and appears also to depend on the integrity of the rostral ventrolateral medulla. Like the fastigial pressor response, it does not tonically contribute to maintaining resting arterial pressure but appears to be involved in phasic control and mediates a sympathoinhibition, probably by inhibition of neurons in the rostral ventrolateral medulla.

A vestibular sympathoinhibition elicited by electrical stimulation of the vestibular nerve or of the labyrinth has also been demonstrated with the effect mediated via the vestibular nuclear complex. The fastigial pressor response remains preserved after destruction of intrinsic neurons in the fastigial nucleus by ibotenic acid, indicating that this pressor response is not mediated by local neurons but instead by axons-of-passage or by fibers terminating in the fastigial nucleus.

The pressor-tachycardia pattern associated with the fastigial pressor response simulates that obtained in the orthostatic reflex. The latter results in an elevation of systemic arterial pressure by a well-known widespread vasoconstriction of resistance and capacitance vessels and tachycardia. Although stimulation of baroreceptors in the carotid sinus has been shown to produce these effects, their deafferentation has failed to abolish the orthostatic reflex entirely. Both the region of the fastigial nucleus and the vestibular apparatus have also been implicated in this reflex mechanism.

The fastigial pressor response has been found to have a relatively shorter latency than does the baroreceptor response, suggesting that the initial vasoconstriction of orthostasis is vestibular or cerebellar in origin. Combined experimental lesions of both vestibular nerves and fastigial nuclei were not shown to produce a summated deficit in head-tilt experiments, indicating that they shared a common neuronal pool. Of interest here is the recent finding of a direct reticulospinal projection from the paramedian reticular nucleus to the intermediate gray region of the thoracic cord relaying input from pressor sites in the vestibular nuclear complex.

Anatomical evidence for a vestibulocerebellar input to the fastigial nucleus exists, and the same input relaying postural information has been suggested electrophysiologically. There is evidence, therefore, of a vestibulocerebellar circuit the nodal points of which share the same target action — a vasopressor action — mediated by one or two medullary centers whose targets are the sympathetic centers of the intermediate gray matter of the thoracic cord.

The occurrence of this rather distinctive pressortachycardia event by stimulation of a discrete area of the cerebellum in the region of the fastigial nucleus in

Fig. 2. Whole-mount photograph of an axial section of the cerebellum and medulla showing the intact floor of the fourth ventricle and remaining cerebellum. H & E.
our patient suggests the existence of a fastigial pressor response in the human. Multiple premature ventricular contractions or other cardiac irregularities are hitherto undescribed in this response. Whether interference with this response was a factor in accentuating the profound bradycardia witnessed in our patient by a lack of appropriate sympathoexcitation during her sudden upright posture is a matter for speculation.

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