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The medullary vascular syndromes revisited

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Abstract There are two major vascular syndromes of the medulla oblongata: the medial and the lateral. The medial medullary syndrome is characterized by the triad of ipsilateral hypoglossal nerve palsy with contralateral hemiparesis and loss of deep sensation. Lateral medullary infarction commonly presents with Horner's syndrome, ataxia, alternating thermoanalgesia, nystagmus, vertigo and hoarseness. Combinations of the two major syndromes occur as bilateral medial medullary, hemi-

medullary and bilateral lateral medullary syndromes. Each of these syndromes frequently manifests with incomplete or atypical findings depending on the extent of the lesion. Magnetic resonance imaging has been useful in the clinical diagnosis of medullary infarctions. The site of the lesion may help predict the arteries involved.

Key words Medullary syndrome · Magnetic resonance imaging · Brainstem infarction

Introduction

The medulla has a compact arrangement of structures, and hence a single lesion usually gives a constellation of signs that constitute a syndrome. Over the years, many syndromes of the medulla oblongata have been reported and the original descriptions were reviewed by Currier in 1969 [10]. In this review, we present a simplified concept of classifying the medullary vascular syndromes to enable clinicians to readily recognize them.

There are two basic ischemic vascular syndromes of the medulla: the more common lateral medullary infarction and the rare medial medullary infarction (Fig. 1). The other even less common medullary syndromes, namely bilateral medial medullary, bilateral lateral medullary, and hemimedullary infarctions, are combinations of these two basic patterns. Furthermore, each of these syndromes may be "complete" or "incomplete".

Medial medullary syndrome

Medial medullary infarction is a rare event characterized by the clinical triad of contralateral hemiparesis with loss of proprioception and vibration sense, and ipsilateral hypoglossal nerve palsy. These findings are due to the involvement of the pyramidal tract and the medial lemniscus above the medullary decussation and the hypoglossal nucleus or fibers that lie just adjacent to them (Table 1, Fig. 1). To our knowledge, fewer than 20 cases of this syndrome have been described [8, 9, 11, 18, 24, 26, 34]. Although Spiller in 1908 [35] first deduced the clinical findings in medial medullary infarction, his case report is not typical of the syndrome and no histologic correlation is available. Dejerine's excellent review of the brainstem syndromes [13] included the medial medullary infarction, but it is unclear whether he was describing a pathologically verified case. It was during the mid-1970s that autopsy-proven reports of the discrete unilateral syndrome were described by Trelles et al. [38], Chokroverty et al. [9] and Leestma and Noronha [24].

Fig. 1 Structures involved in medial and lateral medullary syndromes (*shaded areas* indicate zones of infarction)

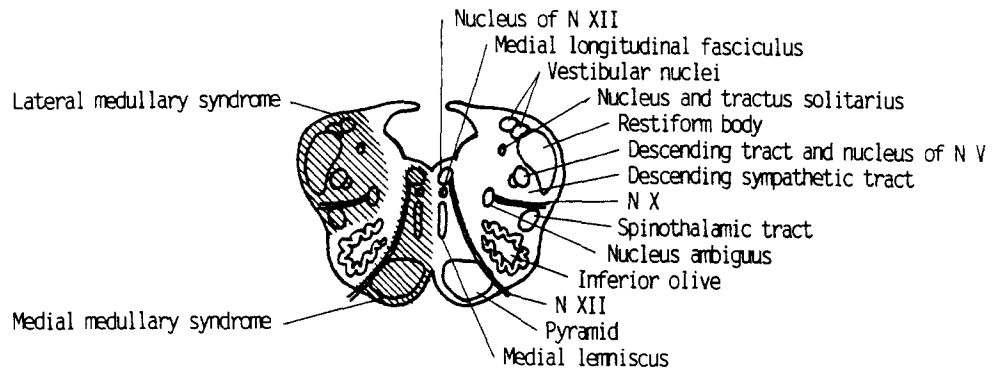


Table 1 Clinical findings in medial and lateral medullary syndrome

Signs and symptoms	Structures involved
Medial medullary	
Ipsilateral	
Tongue paralysis	Hypoglossal nerve or nucleus
Contralateral	
Hemiparesis	Pyramidal tract
Loss of deep sensation	Medial lemniscus
Lateral medullary	
Ipsilateral	
Loss of temperature and pain over half of face	Descending tract or nucleus of V
Horner's syndrome	Descending sympathetic fibers
Ataxia, dysmetria	Spinocerebellar pathways, restiform body, cerebellar hemisphere
Dysarthria, dysphagia, paralysis of vocal cord, diminished gag	Nucleus ambiguus or X cranial nerve
Hiccoughs	Respiratory center in medullary reticular formation
Contralateral	
Nystagmus and vertigo	Vestibular nuclei and connections
Loss of temperature and pain over half of body	Spinothalamic tract

The following description of one of our patients typifies the syndrome:

A 49 year-old right-handed black man recently diagnosed with hypertension presented to the emergency room on 7 July 1993 with left-sided weakness and slurred speech. A day prior to admission, he suddenly felt generally weak and light-headed. He went back to sleep and awoke the next morning with slurred speech, difficulty in swallowing, and paralysis of his left arm and leg. There was no headache or visual symptoms. There was no prior history of trauma or neck manipulation.

On examination, he was alert and oriented. Pupils were equal and reactive to light and accommodation. Fundoscopy was unremarkable. Visual fields were intact with full extraocular movements. He had rotatory nystagmus on right lateral gaze and downbeating nystagmus on primary gaze and upgaze. There was no ptosis or facial asymmetry. Speech was dysarthric. The palate moved symmetrically but the tongue was deviated to the right on protrusion. His left side was hemiplegic, flaccid and hyporeflexic. Plantar responses

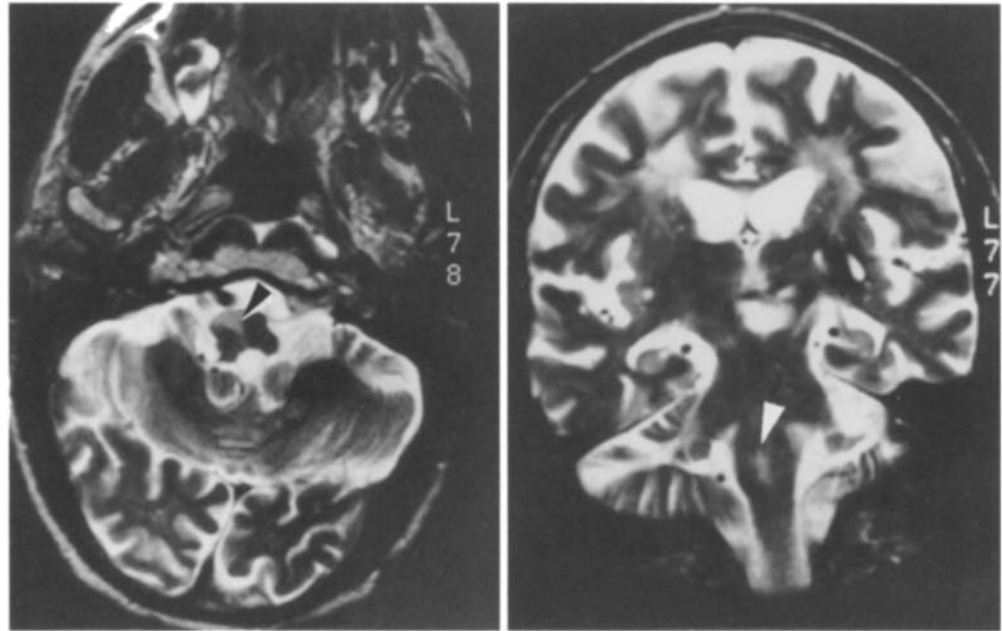
were flexor. Proprioception and vibratory sense were impaired on the left side but pain and temperature sensations were intact.

MRI showed a region of increased signal intensity on T2-weighted images in the right medial medullary area consistent with an infarct (Fig.2). Magnetic resonance angiography (MRA) showed patent basilar and vertebral arteries. Cerebral angiography was essentially normal except for a hypoplastic right vertebral artery without evidence of occlusion or dissection.

During the next 24 h, tone returned, reflexes became active and he developed a left Babinski response. Follow-up examinations over the next 2 weeks did not reveal spasticity.

The presence of the clinical triad facilitated the diagnosis of medial medullary syndrome in this patient. However, recognition of this syndrome may be difficult since partial or "incomplete" manifestations frequently occur (Fig.3). In a recent review, only 4 out of 11 cases presented with all three clinical features of the syndrome [34].

Fig. 2 T2-weighted MRI of patient with medial medullary infarction (*arrows indicate lesion on axial and coronal sections*)



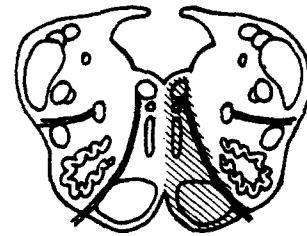
Review of all the case reports shows that pyramidal signs are a constant finding. Patients may present with pure motor hemiparesis with sparing of the face if the infarct is largely confined to the pyramid [9, 24, 31]. It has been suggested that medullary pyramidal infarction results in flaccid paralysis, and that flaccidity differentiates this syndrome from other syndromes of pure motor hemiparesis. This hypothesis is supported by ablation experiments showing that lesions of the pyramid result in flaccidity [5, 24]. However, in some of the reported cases, spasticity set in after days or weeks of flaccid paralysis as with other corticospinal lesions [11, 18, 19, 24, 34].

In one study, only 6 out of 13 patients had hypoglossal nerve palsy [34]. The absence of tongue involvement may have several reasons. The medial medullary lesion may not extend laterally to interrupt the hypoglossal nerve fibers or dorsally to involve the hypoglossal nucleus. Moreover, the hypoglossal nucleus is a long column of cells along the medulla and a lesion in a small portion of it may not cause any detectable deficit. In addition, hypoglossal nerve fibers sometimes appear to be preserved in an area of infarction [11, 18, 34].

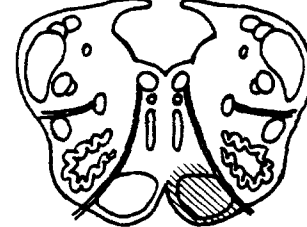
In the absence of tongue paralysis, dissociation of sensory deficits may be helpful in the clinical diagnosis. Contralateral loss of vibration and position sense with intact sensation to other modalities would localize the lesion to the lower brainstem. In contrast, involvement of all sensory modalities usually indicates a lesion in the upper brainstem or higher.

Aside from presenting as “incomplete” syndromes, other neurological findings may be observed and may lead to difficulty in recognizing the diagnosis. In the review by Ho and Meyer [18], 7 of 15 patients had mild and transitory central facial weakness contralateral to the le-

Complete Medial Medullary Syndrome



Medial Medullary Syndrome presenting as Pure Motor Hemiparesis



Medial Medullary Syndrome without tongue paralysis

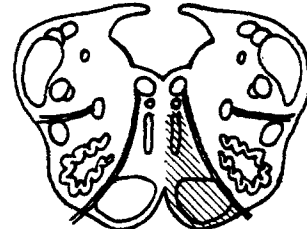


Fig. 3 Variants of the medial medullary syndrome (*shaded areas indicate extent of infarction*)

sion. Facial weakness may result from interruption of the corticobulbar fibers that leave the pontomedullary and midmedullary pyramids to cross to the opposite facial nucleus [11, 18].

Vertical nystagmus is reported to occur in medial medullary syndrome, somewhat surprising for a lesion this low in the brainstem. This may conceivably be due to involvement of the medial longitudinal fasciculus (MLF) which is probably one of the pathways mediating vertical gaze [2, 11, 18]. The caudal portion of the MLF is located just dorsal to the medial lemniscus in the medulla.

Pathological examinations of the case reports reveal, as expected, infarction of the medullary pyramid with varying degrees of extension dorsomedially. Leestma and Noronha [24] reported an unusual feature, that of olivary hypertrophy in one patient with the medial medullary syndrome. They postulated that olivary hypertrophy was a result of deafferentation of the olive from damage to the central tegmental fibers in the vicinity of the inferior olivary nucleus. No clinical effects consequent to this lesion were noted.

Lateral medullary syndrome

Lateral medullary syndrome is due to infarction of the area posterior to the inferior olivary nucleus (Fig. 1). Wallenberg was given credit for the first account of this syndrome when he described his clinical findings in a patient in 1895 and reported the results of autopsy 6 years later [39, 40]. Vieusseux, however, actually provided the earliest account in 1810 when he reported his own stroke to the Medical and Chirurgical Society of London [30]. It is a very striking event and is the most common ischemic syndrome of the medulla. In approximately half of the patients with the syndrome, transient ischemic warnings such as dizziness, headache, double vision, and staggering occur days to weeks before the stroke [7].

Clinical manifestations (Table 1) depend on the extent of the lesion affecting the structures in the dorsolateral medulla. In the recent series of Sacco et al. [33], Horner's syndrome, ipsilateral ataxia, and contralateral hypalgesia were the most common signs in lateral medullary syndrome.

Like other brainstem syndromes, the lateral medullary syndrome presents with "crossed" cranial nerve and long tract findings, specifically "alternating thermoanalgesia" – loss of pain and temperature sensation over half the face and the contralateral hemicorpus. This is accounted for by the involvement of the "crossed" spinothalamic tract and the ipsilateral descending tract and nucleus of the trigeminal nerve. At times, a sensory level along the trunk can be detected and this is probably due to sparing of the medial laminations of the spinothalamic tract that subserve the arm and trunk. Sensory loss over the ipsilateral half of the face is maximal in the V-1 distribution (forehead) probably because its tract is located more ventrally. If the le-

sion extends more medially, the fibers crossing from the opposite trigeminal nucleus to the thalamus may also be involved, leading to bilateral facial hypesthesia with more dysesthesia ipsilaterally. Facial pain, usually located in the forehead, eye or cheek, may represent ischemia of the descending nucleus of V [7].

Horner's syndrome usually includes ptosis and miosis, and less commonly facial anhidrosis. This is due to involvement of the descending sympathetic fibers.

Ataxia of the ipsilateral limbs and falling to the side of the lesion may be related to interruption of the spinocerebellar pathways and involvement of the restiform body. Cerebellar infarction accompanying lateral medullary syndrome is not uncommon and may contribute to the observed ataxia.

Dysarthria, dysphagia, hoarseness, paralysis of the vocal cord, and diminished gag reflex are the result of involvement of the nucleus ambiguus and exiting fibers of the vagus nerve.

Nystagmus is frequently horizontal and rotatory. It is usually worse when looking to the side opposite the lesion [7, 15]. Vertigo, often with nausea and vomiting, is a prominent symptom. This symptom complex is due to involvement of the vestibular nuclei and their connections. However, the direction of the nystagmus may be altered by the involvement of the cerebellum.

Hiccough has been attributed to involvement of the respiratory centers in the medullary reticular formation. The inspiratory and expiratory centers may be affected differentially, but this hypothesis remains to be proved [10, 15].

The following case illustrates the findings in lateral medullary infarction:

An 85-year-old hypertensive right-handed black man was brought to the emergency room after being found on the floor. Over the previous week, he had been unsteady on his feet, leaning more to the right side, and had fallen several times. The night prior to admission, he fell out of bed and was unable to get up. He did not have headache, diplopia, or swallowing difficulty, but complained of hoarseness.

On examination, he was alert, oriented, and had frequent hiccoughs. There was ptosis of the right eye. The right pupil was smaller than the left, but both were reactive to light. Extraocular movements were full but there was mild nystagmus on right lateral gaze. He had mild right central facial paresis. Speech was hoarse and dysarthric. Gag reflexes were poor bilaterally. Tongue was in the midline. Muscle strength was normal, but the right side was severely dysmetric, ataxic, and dysdiadochokinetic. Pain and temperature perception was impaired on the right side of the face and left side of the body. Proprioceptive and vibratory sensations were intact.

Laryngoscopy showed paralysis of the right vocal cord. Chest radiographs revealed infiltrates in the right upper lung field. Computed tomography on admission showed a right cerebellar infarct. Magnetic resonance imaging (MRI) later revealed areas of increased signal intensity on T2-weighted images in the right cerebellar hemisphere and lateral portion of the medulla (Fig. 4).

The presence of frequent hiccoughs in this patient was a clue to the diagnosis; the findings of Horner's syndrome, ipsilateral ataxia and alternating thermoanalgesia was quite typical. Like the medial medullary syndrome, lateral

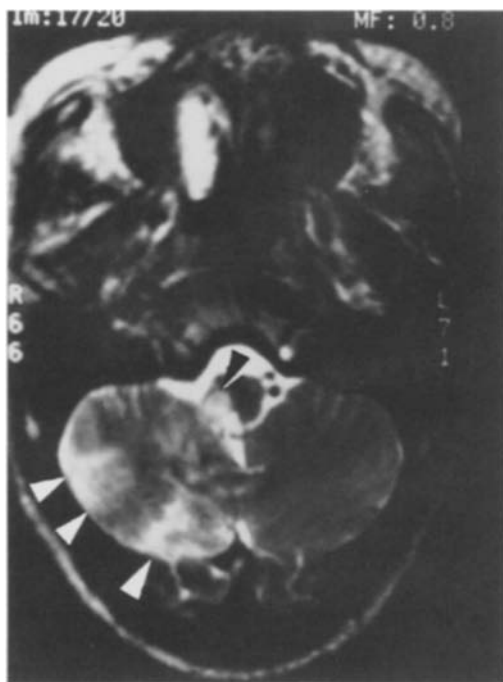


Fig. 4 T2-weighted MRI of patient with lateral medullary infarction (dark arrow; white arrows point to associated cerebellar infarction)

medullary infarction may manifest with the classical findings (Table 1), but “incomplete” or “atypical” presentations are common.

Central facial paresis may be seen in lateral medullary infarction. In contrast to the medial medullary syndrome, the facial weakness is ipsilateral to the lesion and is thought to be due to interruption of the rostral fibers swinging from the opposite corticobulbar tract to the facial nucleus [10, 15, 33].

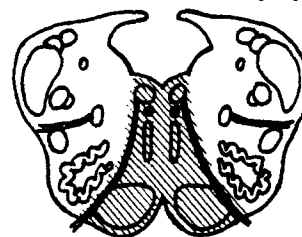
Loss of taste is an infrequent finding that may result from involvement of the nucleus and tractus solitarius. Autonomic dysfunction has been reported including tachycardia, lightheadedness, blood pressure lability, and respiratory failure [7].

Dorsal medullary syndrome presenting with vomiting, unsteadiness, and intractable hiccoughs has been described by Fisher et al. [15]. On autopsy, a dorsal infarction in the medulla was found involving the area underlying the lateral portion of the floor of the fourth ventricle. This syndrome should probably be considered as a dorsal “fragment” of the lateral medullary syndrome.

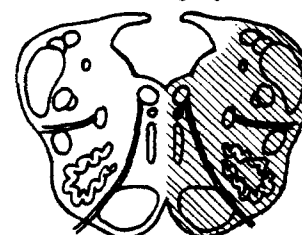
Other medullary syndromes

Aside from the two major syndromes discussed, there are other ischemic vascular syndromes of the medulla, namely bilateral medial medullary, hemimedullary, and bilateral lateral medullary syndromes (Fig. 5). These syn-

Bilateral Medial Medullary Syndrome



Hemimedullary Syndrome



Bilateral Lateral Medullary Syndrome

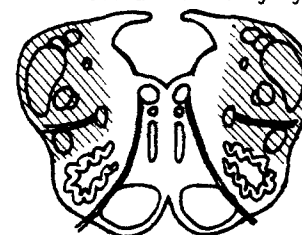


Fig. 5 Combined medullary syndromes (shaded areas indicate location of lesions)

dromes are better viewed as combinations of the major syndromes occurring either simultaneously or during separate episodes. Rare as medial medullary syndromes are, these combination syndromes are even more uncommon.

Bilateral medial medullary syndrome was among the cases described by Dejerine in his book in 1914 [13]. Davison provided the first clinicopathological description of this syndrome in 1944 [12]. In the review of Ho and Meyer [18], bilateral involvement occurred in 7 of 15 cases of medial medullary syndrome. Clinically, it presents with flaccid quadriplegia sparing the face, bilateral disturbance of deep sensation, hypoglossal nerve palsy, and respiratory failure. Combinations of complete and atypical medial syndromes may give slightly different findings in bilateral medial medullary infarctions (Table 2) [19, 20, 21, 26, 37].

In *hemimedullary infarction*, the clinical diagnosis depends on recognizing features of both the medial and lateral medullary infarctions. Reinhold [29] reported the earliest clinicopathologically proven case of hemimedullary syndrome in a physician in 1894, a year before Wallenberg's description of the lateral medullary infarction. Babinski and Nageotte in 1902 [1] described a medullary

Table 2 Presentations in bilateral medial medullary syndrome

Medial medullary syndrome	Findings
1. Bilateral complete syndrome	Quadriplegia, total paralysis of tongue, bilateral impairment of deep sensation
2. Unilateral complete syndrome + contralateral syndrome without tongue paralysis	Quadriplegia, tongue deviation to one side, bilateral impairment of deep sensation
3. Unilateral complete syndrome + contralateral syndrome of pure motor hemiparesis	Quadriplegia, tongue deviation to one side, with contralateral impaired deep sensation
4. Bilateral syndrome without tongue paralysis	Quadriplegia, tongue midline on protrusion, bilateral impairment of deep sensation
5. Unilateral syndrome without tongue paralysis + contralateral syndrome of pure motor hemiparesis	Quadriplegia, tongue midline on protrusion, unilateral impairment of deep sensation
6. Bilateral syndrome of pure motor hemiparesis	Quadriplegia, tongue midline on protrusion, no deep sensation impairment

syndrome that bears their names and presents with lateral medullary syndrome and contralateral hemiparesis or an extensor plantar response. This is essentially a combination of lateral medullary infarction and “incomplete” medial medullary syndrome presenting as pure motor hemiparesis. In one recent review of hemimedullary syndromes [27], only six autopsy-proven cases were collected and one more added by MRI correlation. The term “hemimedullary syndrome” should probably be reserved for cases in which the lateral and medial infarctions occur simultaneously.

In the pathologically examined *bilateral lateral medullary syndromes* of Devereaux et al. [14] and Caplan [6], the patients developed a new lateral medullary infarction in the setting of a pre-existing infarction in the opposite lateral medulla. Failure of automatic respiration, previously observed in bulbar polio, high cervical lesions and brainstem tumor, has been reported in bilateral infarctions of the lateral medulla. This loss of respiratory automatism is presumably due to differential involvement of the voluntary and automatic respiratory centers and has been called Ondine’s curse, after the mythical sea nymph whose curse on her unfaithful lover resulted in his death because of failure to breathe when asleep [6, 14]. Bilateral brainstem involvement, however, is not necessary for respiratory failure. It appears that unilateral lesions in the pontomedullary paramedian reticular formation, nucleus tractus solitarius, nucleus ambiguus, nucleus retroambiguus, dorsal motor nucleus of the vagus, region of medial lemniscus, and spinothalamic tract may result in hypoventilation. Furthermore, while voluntary respiration has been localized to the pyramidal tracts, a lesion sparing the corticospinal pathway can also impair voluntary respiratory movements [3, 25].

Role of MRI

Medullary infarctions that do not present with the classical syndromes may be difficult to diagnose. MRI offers visualization of medullary infarcts as small as 2 mm. MRI

may also help reveal coexisting lesions such as cerebellar infarcts associated with the lateral medullary syndrome that may not be readily seen on CT scans [16, 32]. In a series of lateral medullary syndromes, CT was abnormal only when cerebellar infarction was present (3 of 33 cases), while MRI was abnormal in 20 of 22 cases [33].

The brainstem is evaluated best by using both T1- and T2-weighted axial images. Since the changes on MRI depend on tissue water content, infarcts are seen as approaching the intensity of CSF rather than that of surrounding brain tissue [32]. Thus, infarctions appear dark on T1-weighted images and bright on the T2 sequence. Coronal sections help demonstrate the extent of the lesion rostrocaudally. The detection of signal abnormalities in medullary infarctions has been reported as early as 9 h after the ischemic event [21].

Recently, Bogousslavsky et al. [4] attempted to assess infarct location and etiology using both MRI and MRA. They were able to show a high frequency of severe large-artery disease in posterior circulation infarctions. However, the number of medullary infarcts studied in this series was low.

Pathological and vascular considerations

Different causes of medullary infarction have been documented including thrombosis, artery to artery embolism, talc or fibrocartilaginous embolism [20, 26], syphilitic arteritis [15], fibromuscular dysplasia [28], and dissection of the arterial wall [8]. In young individuals, dissection of the vertebral artery may develop spontaneously or after chiropractic manipulation, yoga, or trauma to the neck [8, 33]. The presence of severe headache or neck pain should raise the suspicion of vertebral artery dissection.

The studies of Stopford [36], Krayenbuhl and Yasargil [22], Lazorthes et al. [23], Fisher et al. [15], and Gillilan [17] have contributed to our understanding of the vascular supply of the brainstem.

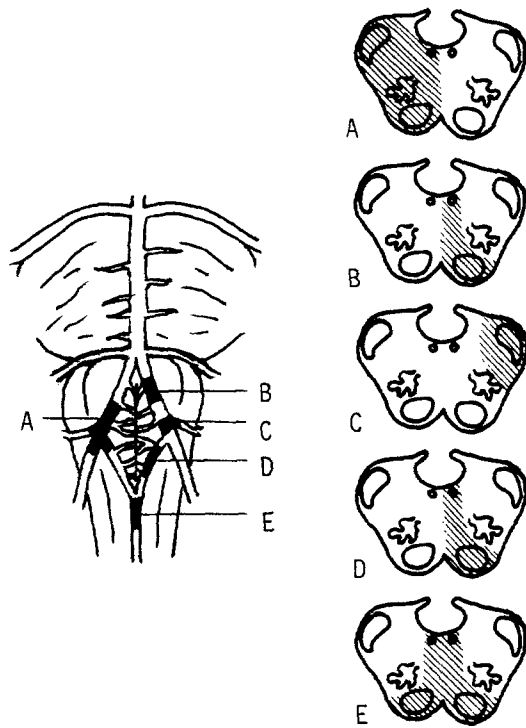


Fig. 6 A–E Vascular involvement in medullary syndromes. **A** Hemimedullary syndrome; **B, D** medial medullary syndrome; **C** lateral medullary syndrome; **E** bilateral medial medullary syndrome

Lateral medullary infarction was previously known as “the syndrome of the posterior inferior cerebellar artery (PICA).” However, the seminal work of Fisher et al. in 1961 [15] showed that most of their cases were due to vertebral artery occlusion with or without extension to the PICA, and less commonly to occlusion of the PICA alone. In bilateral lateral medullary infarction, the vessels involved are the same as those in the unilateral syndrome [6, 14].

Bilateral involvement in medial medullary infarction is usually a consequence of occlusion of the unpaired ante-

rior spinal artery that supplies the lower two-thirds of the medial medulla [18, 21, 24, 34]. On the other hand, the rostral third of the medial medulla is irrigated by the vertebral artery and the branching of the anterior spinal artery, and unilateral medial medullary infarction may result from their occlusion. Short and long penetrating paramedian branches arise from the vertebral and anterior spinal arteries. “Incomplete” medial medullary syndromes may be attributed to differential involvement of the short and long branches [11, 24].

Hemimedullary syndrome frequently follows occlusion of the vertebral artery, probably a consequence of decreased flow in both the PICA and the paramedian penetrating branches [10, 15, 27].

Based on these studies, we present a schematic figure correlating the location of the medullary infarction with the arteries involved (Fig. 6).

Prognosis

The lateral medullary syndrome is usually thought to carry a benign prognosis. However, in Fisher’s series of 18 cases, 12 patients died within 12 h to 29 days of the stroke, mostly from respiratory failure, atherosclerotic disease elsewhere, or infection; the other deaths remained unexplained [15]. When cerebellar infarction is present, coma may occur from cerebellar edema and subsequent herniation [6, 7]. Moreover, Caplan drew attention to propagation of a clot from the vertebral to the basilar artery resulting in progression of neurologic deficits [6].

In the medial medullary syndrome, the outcome is favorable when the lesion is limited to the upper third of the medulla. A higher incidence of respiratory compromise has been reported in medial infarcts occurring in the lower two-thirds of the medullary pyramid. Furthermore, bilateral involvement is more common in the lower medulla and has a poorer prognosis [34].

The combined syndromes generally have a more complicated clinical course and carry a graver prognosis [6].

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