Review

Brainstem Blood Supply and Clinical Manifestations in Brainstem Vascular Lesions

Noboru GOTO and Chen ZHANG

Abstract: Advances in neuroradiological diagnostic equipment have made even the diagnosis of brainstem vascular lesions much easier. To understand neurological symptomatology more precisely, however, it is necessary to know the microanatomy of brainstem blood supply in relation to brainstem structures and clinical manifestations of brainstem vascular lesions. Currently, several incorrect classifications of brainstem blood supply are used throughout the world. Such classifications have been proposed without the study of brainstem structures and vascular distribution by serial sections of the brainstem. We propose that it is best to classify clinical manifestations of brainstem vascular lesions according to the anatomical sites of the lesions and precise knowledge of blood supply. This paper discusses this point and shows that intracerebral vascular syndromes should be named by the vascular supplying areas, not by the subarachnoid vessels.

Key words: symptomatology, brainstem, blood supply, serial sections, cerebrovascular disturbance

Introduction

Disorders of the brainstem may differ from patient to patient because of differences in anatomical structures involved in the lesions¹⁾. It has long been a matter of interest to examine the blood supply of the brainstem and clinical manifestations in patients with brainstem vascular disturbances in relation to local diagnoses. Such attempts have been made by several researchers²⁻⁴⁾. However, correlations between anatomical brainstem structures and precise arterial tributary areas of the brainstem are still not clearly understood. The brainstem is often understood to include only the mesencephalon, pons and medulla oblongata, although it has had several interpretations for a long time. In this article, the above correlations of the brainstem are reviewed and discussed in detail from the clinico-anatomic point of view.

Methodology

Several methods have been introduced to examine the vascular supply to the human brainstem: 1) histological observation and reconstruction after making serial sections of the normal brain; 2) sectional or translucent (Spalteholz's technique) observation after injection of dye into the vascular system⁴⁾; 3) vascular cast observation after intravascular injection of corrosion⁵⁾; 4) soft X-ray observation after intravascular injection of contrast medium; 5) autopsy

Department of Anatomy, Showa University School of Medicine, 1-5-8 Hatanodai, Shinagawa-ku, Tokyo 142, Japan.

examination of patients with cerebrovascular infarctions in the brainstem; 6) miscellaneous methods including various combinations of the above-mentioned methods.

Our conclusions are based mainly on the observation of serial sections of four normal brainstems, macroscopic examinations of several hundred normal brains, and macroscopic and microscopic examinations of many autopsy brains (more than 300) with cerebrovascular diseases.

Historic Considerations

Research on the blood supply of the brainstem has been conducted by many researchers^{2-4,6)} for more than a century. Among those studies, two prominent reports can be pointed out. Foix and Hillemand (1925)²⁾ classified arteries supplying the brainstem into three kinds of subarachnoid branches: artères paramédianes, artères circonférentielles courtes et artères circonférentielles longues—paramedian arteries, short circumferential arteries and long circumferential arteries, respectively. This epochal new idea was supported by many. However, it is not a pertinent classification for the reason discussed in the next section; i.e. abundant variations of the subarachnoid vessels. Moreover, it has since transpired that their view is not correct because they did not examine the inside of the brainstem three-dimensionally. Many researchers have been guided into pitfalls by incorrect views due to the surface observations of Foix and Hillemand (1925)²⁾. We believe it is not scientifically responsible to advocate their views. The other prominent study was reported by Gillilan (1964)⁴⁾ who described the intrinsic arterial patterns determined from contrast-medium-injected serial sections of the brainstem, and found four zones of distribution of the intrinsic arteries: median, paramedian, lateral and dorsal. Apart from using different names, many researchers have accepted the idea of vascular supply areas related to clinical brainstem vascular lesions.

As a final historical remark, we would like to call special attention to the vascular or areal naming of the brainstem. Technical terms such as medial, median and paramedian have been used in different ways by different authors^{2,4,6-8)}. Apart from such different names, in this paper we will discuss the vascular supply of the brainstem and its clinical symptomatology on the same basis.

Main Trunks and Branches Supplying the Brainstem

The main subarachnoid arterial trunk supplying the brainstem is the vertebrobasilar system. Even in this main trunk, there are many variations that include a duplication or fenestration of the basilar artery, an ipsilateral anomaly of the duplication, and hypogenesis or agenesis of the vertebral artery, among others.

The main subarachnoid branches from the vertebrobasilar trunk are the anterior spinal artery, posterior inferior cerebellar artery, anterior inferior cerebellar artery, labyrinthine artery, superior cerebellar artery, posterior cerebral artery, etc. Regarding the primary branches mentioned above, however, there are many combinations of branching sites from the main trunk. For example, the labyrinthine and anterior inferior cerebellar arteries may branch off from the basilar artery or vertebral artery separately, or may have a common branch from the basilar or vertebral artery. They sometimes branch off from a loop of the inferior cerebellar artery which is occasionally formed on the anterior inferior surface of the cerebellum as a common branch instead of the posterior and anterior inferior cerebellar arteries. In this case, the labyrinthine artery is usually supplied from the loop. The superior

cerebellar artery shows three types of branching: one for each side, two for one side and one for the other side, or two for each side.

In conclusion, it is difficult to classify even primary branches into groups, much less secondary branches. Arteries supplying the brain can be divided into three categories: 1) the extracranial main arterial source supplying the cranial cavity, 2) the subarachnoid arteries, and 3) the intrinsic arteries within the brain. It is very rare to observe variations of the intrinsic arteries and extracranial sources, while there are abundant variations regarding the subarachnoid arteries. This is a very important point when classifying brainstem blood vessels.

Principle of the Arterial Supply of the Brain

According to Goto's observations, which consisted of macroscopic superficial findings in normal brains, and microscopic findings of histological serial sections of normal brains and autopsied brains with cerebrovascular disturbances, the arterial supply of each subdivision (telencephalon, diencephalon, mesencephalon, metencephalon and myelencephalon) of the brain can principally be divided into three (or sometimes four) tributary areas and their supplying branches: median, lateral, dorsal and medial (in part) areas and homonymous branches (Fig. 1). The brainstem blood supply is discussed below in detail in relation to clinical manifestations.

Vascular Supply of the Mesencephalon

Intrinsic arteries supplying the mesencephalon can be classified into three groups: median, lateral and dorsal mesencephalic branches (Figs. 1–3).

The median mesencephalic branches

The median mesencephalic branches are divided into two further groups: superior and inferior. Both median mesencephalic branches from the tip (or top) of the basilar artery supply the homonymous areas through the posterior perforating substance between the cerebral peduncles. The superior branches supply the red nucleus, oculomotor nucleus, intramedullary oculomotor nerve root, medial longitudinal fascicle, interstitial nucleus of Cajal, posterior commissural nucleus of Darkschewitsch, central gray substance, etc. (Fig. 2).

The inferior branches supply the superior cerebellar peduncle above the decussation (so-called *nucleus albus* which means white nucleus—an incorrect name, of course), intramedullary oculomotor nerve root, trochlear nucleus, medial longitudinal fascicle, central gray substance, etc. (Fig. 3).

The above-mentioned median mesencephalic branches are considered to be part of the small arteries going through the posterior perforating substance and supplying part of the diencephalon, mesencephalon and pons (Goto, 1971)⁹⁾. These branches can be divided into four: 1) thalamoperforating arteries (or posteromedial central branches), 2) superior median mesencephalic branches, 3) inferior median mesencephalic branches, and 4) superior pontine tegmental branches (described later).

The lateral mesencephalic branches

The lateral mesencephalic branches are small arteries branching off from the posterior cerebral or partially from the superior cerebellar arteries. They enter the lateral mesencephalic area from the ventral or lateral surface to supply the crus cerebri, substantia nigra, medial lemniscus, lateral lemniscus, brachium of the inferior colliculus, etc. (Figs. 2, 3).

Fig. 1.

Fig. 2.

Fig. 1. Principle vascular supply of the central nervous system.

DA, dorsal area; DB, dorsal branch; LA, lateral area; LB, lateral branch; MA, medial area; MB, medial branch; MNA, median area; MNB, median branch; VS, ventricular system.

Fig. 2. Arterial supply of the upper mesencephalon.

BA, basilar artery; DMB, dorsal mesencephalic branch; LMB, lateral mesencephalic branch(es); PCA, posterior cerebral artery; PCMA, posterior communicating artery; SMNMB, superior median mesencephalic branch(es).

See reference⁴⁰⁾ for other abbreviations.

The dorsal mesencephalic branches

The quadrigeminal arteries branching off from the posterior cerebral artery in front of the mesencephalon run backward in the ambient cistern, and, at the back of the mesencephalon, give many small dorsal mesencephalic branches which supply the superior and inferior colliculi (Figs. 2, 3).

Clinical Symptomatology of Mesencephalic Lesions

Fog and Hein-Sørensen (1969)¹⁰⁾, summarized the syndrome of midbrain involvement as follows: 1) alternating hemiplegias due to unilateral damage of the nuclei and pertinent fiber tracts, 2) dysfunction of ocular motility caused by lesions of the oculomotor nuclei, of the intrinsic system of oculomotor coordination, or both, 3) disturbances of consciousness due to pathological involvement of the mesencephalic reticular formation, 4) decerebrate rigidity when structures subserving the regulation of muscular tone are affected.

The symptomatology, however, differs from patient to patient according to sites of lesions, stage of disease, pathogenesis, etc. In the present article, clinical manifestations of the brainstem will be discussed in relation to the blood supply.

Median mesencephalic syndromes

The important vascular syndromes of the tributary areas by the superior and inferior median mesencephalic branches correspond to the syndromes of Benedikt and of Claude, respectively.

Fig. 3.

Fig. 3. Arterial supply of the lower mesencephalon.

BA, basilar artery; DMB, dorsal mesencephalic branch; IMNMB, inferior median mesencephalic branch(es); LMB, lateral mesencephalic branch(es); PCA, posterior cerebral artery; PCMA, posterior communicating artery; SCA, superior cerebellar artery. See reference⁴⁰⁾ for other abbreviations.

Fig. 4.

Fig. 4. Median mesencephalic syndromes.

a, Syndrome of Benedikt; b, Syndrome of Claude (modified from Dejerine⁷⁾).

The syndrome of Benedikt (1889)¹¹⁾ represents an ipsilateral paresis of the third cranial nerve combined with contralateral involuntary movement (Fig. 4a). These symptoms are due to a lesion involving the intramedullary oculomotor nerve root, oculomotor nucleus, and red nucleus.

The syndrome described by Claude (1912)¹²⁾ includes an ipsilateral paresis of the third (occasionally together with a slight paresis of the fourth) cranial nerve, and contralateral cerebellar symptoms such as asynergia, ataxia, dysmetria and dysidiadochokinesia. Conjugate reduction of the gaze to the opposite side may be also included (Fig. 4b). These symptoms are caused by a lesion involving the intramedullary oculomotor nerve root and the superior cerebellar peduncle above the decussation, as is occasionally accompanied by a lesion of the trochlear nucleus.

Segarra (1970)¹³⁾ reported two autopsy patients with infarctions of the regions supplied by branches of bilateral thalamoperforating arteries and superior median mesencephalic branches in one, and bilateral thalamoperforating arteries in the other. He proposed the name of the syndrome of the mesencephalic artery for such patients. However, this was obviously incorrect naming, because the thalamoperforating artery supplies part of the diencephalon, not the mesencephalon, as was described above.

Lateral mesencephalic syndromes

There is no independent syndrome reported to involve the whole of the lateral mesencephalic area, because it is supplied by many small branches. However, occlusion of the posterior cerebral artery at the proximal portion after the branching of the posterior communicating artery may involve the whole of this area apart from the disturbance of temporo-occipital circulation.

Although the syndrome of Weber is well known as an alternating hemiplegia (homolateral paresis of the oculomotor nerve to external muscles of the eyeball combined with contra-

lateral hemiplegia), it does not occur very often as a result of single vascular infarction. The blood supplies of regions including the nucleus or intramedullary root of the oculomotor nerve and that of the pyramidal tract are from different sources. The original description of the Weber syndrome was made by Weber (1863)¹⁴⁾ on an autopsy patient with an aneurysm ruptured into the crus cerebri (cerebral peduncle).

It may be more common to observe a pseudo-Weber syndrome which has a lesion outside the mesencephalon compressing the oculomotor nerve and crus cerebri such as intracerebral hemorrhages in the uncus hippocampi, aneurysms from the posterior cerebral artery, etc.¹⁵⁾.

Dorsal mesencephalic syndromes

It is rare to observe a patient with vascular disturbances in this area. The superior and inferior colliculi, however, are important in the pupillary reflex to light, and the transmission of auditory stimuli to the medial geniculate body, respectively. Baloh and colleagues (1985)¹⁶⁾ described the dorsal midbrain syndrome. They presented seven patients with limited upward gaze and convergence nystagmus. Five of them had pineal tumors, and two of them were thought to have intrinsic lesions.

The famous clinical entity called Parinaud's syndrome (1883)¹⁷⁾, which includes paralysis of the upward gaze, areflexic pupils on convergence and to light, occasional inability of the downward gaze, and ocular retraction on upward gaze or convergence, may be considered to be due to lesions involving the tectum mesencephali. The areflexic pupils to light may be due to lesion of the superior colliculus. Symptomatology due to a lesion of the inferior colliculus (for example, the auditory disturbances) is not included in the literature. The syndrome of Parinaud can be observed as a result of compression of the tectum mesencephali from behind, for example by a tumor. It is very rare to observe this condition due to vascular disturbance.

Nothnagel (1879)¹⁸⁾ described a syndrome of unilateral or bilateral ataxia and various palsies of the external ocular muscles. This condition is caused by space-occupying lesions compressing the tectum mesencephali, and the vermis or superior cerebellar peduncle, or both. This is not the case with an intrinsic vascular lesion of the brainstem.

Vascular Supply of the Pons

The study by Böhne (1927)¹⁹⁾ can be considered as a classic. As described in the section on historic considerations, it is rather difficult to understand the fine arterial distribution of the pons without three-dimensional reconstruction of serial brainstem sections. Arteries supplying the pons are classified into three groups: median, lateral and dorsal pontine branches (Figs. 1, 5–8).

The median pontine branches

Peculiar distributions of arteries can be observed in the median pontine area because of the unique and embryologically delayed development of the pontine basis bulging. The complicated arterial distribution of the median pontine area is illustrated in Fig. 5. The upper median pontine basal and tegmental branches supply the upper half of the pontine median area, areas M1 and M2, respectively (Figs. 5, 6). The middle median pontine branch supplies the upper part of the lower half median area, tegmentum and basis, area M3 (Figs. 5, 7). The lower median pontine basal and tegmental branches supply the lower part of the lower half median area, areas M4 and M5, respectively (Figs. 5, 8). Both upper and lower tegmental pontine branches penetrate through the caudal part of the posterior perforating

Fig. 5.

Fig. 6.

Fig. 5. Arterial supply of the median pontine areas.

3N, oculomotor nerve; 4V, fourth ventricle; BA, basilar artery; Cb, cerebellum; M1–M5, see text; Mc, mesencephalon; Mo, medulla oblongata; PCA, posterior cerebral artery; VA, vertebral artery.

Fig. 6. Arterial supply of the upper part of the pons.

BA, basilar artery; DPB, dorsal pontine branch(es); LPB, lateral pontine branch; SBB, superior basilar branch(es); STB, superior tegmental branch(es).

See reference⁽⁶⁾ for other abbreviations.

substance between the bilateral crus cerebri, and the foramen caecum, respectively to areas M2 and M5 (Figs. 5, 6, 8). The rest of median pontine branches, the upper and lower median pontine basal branches and the middle median pontine branch, reach the pons through the pontine basilar sulcus, to areas M1, M3 and M4 (Figs. 5–8).

Area M1 (Fig. 6) includes several structures such as part of the longitudinal fasciculi, or descending pathways including the corticospinal, corticonuclear and corticopontine tracts, the pontine nuclei and transverse pontine fibers; Area M2 (Fig. 6) the decussation of superior cerebellar peduncles, superior central nucleus, predorsal fascicle, medial longitudinal fascicle, part of the reticular formation, corticonuclear fibers to the facial nucleus, lemniscus medialis, corticonuclear (occasionally described as “supranuclear oculomotor”) fibers to the abducent nucleus; Area M3 (Fig. 7) the same structures as M1, medial longitudinal fascicle, and the part of reticular formation; Area M4 (Fig. 8) the same structures as M1 and intramedullary root of abducent nerve; and area M5 (Fig. 8) the medial lemniscus, reticular formation, medial longitudinal fascicle, and nucleus and root of the abducent nerve.

The lateral pontine branches

Arteries to the lateral pontine area branch off from the basilar artery as primary branches:

Fig. 7.

Fig. 7. Arterial supply of the middle part of the pons.

BA, basilar artery; CNB, cerebellar nuclear branch(es); DPB, dorsal pontine branch(es); LPB, lateral pontine branch; MDB, middle branch.

See reference⁽⁴⁰⁾ for other abbreviations.

Fig. 8.

Fig. 8. Arterial supply of the lower part of the pons.

BA, basilar artery; DPB, dorsal pontine branch(es); IBB, inferior basilar branch; ITB, inferior tegmental branch; LPB, lateral pontine branch.

See reference⁽⁴⁰⁾ for other abbreviations.

so-called rami ad pontem, and partially as secondary branches from the superior cerebellar, anterior inferior cerebellar, labyrinthine, inferior cerebellar (*vide ante*) arteries, etc. They reach the pons from the anterolateral surface (Figs. 6–8), and supply the lateral pontine area which includes the following structures: the middle cerebellar peduncle, part of the pontine longitudinal fascicle (or descending pathways) and part of the pontine nuclei in the ventral part of the pons; and the central tegmental tract, the spinothalamic tract, the nucleus and its root of the facial nerve in the dorsal part of the pons.

The dorsal pontine branches

These branches are mainly part of the cerebellar nuclear branches getting into the brain along the surface of the superior cerebellar peduncle branching off from the superior cerebellar artery (Figs. 6, 7). Apart from the above-described main branches, only a few small branches join to supply from the upper point of the posterior inferior cerebellar artery behind the medulla oblongata to the lowest part of the dorsal pontine area (Fig. 8). In the dorsal pontine area, the following structures are included: the superior and inferior cerebellar peduncles, the mesencephalic, motor and principal nuclei of the trigeminal nerve, the superior vestibular nucleus, etc.

Clinical Symptomatology of Pontine Lesions

Trelles (1926)⁽²⁰⁾ published a monograph entitled “Les ramollissements protubérantiels: Etude clinique et anatomique”. His anatomical description, however, was based on the study by Foix and Hillemand (1925)⁽²⁾. Loeb and Meyer (1969)⁽²¹⁾ summarized pontine syndromes

as including various pathologic conditions. Some descriptions are obviously due to intrinsic vascular lesions of the pons. Kubik and Adams (1946)²²⁾ reported a clinicopathological study of basilar artery occlusion. In this section, every vascular syndrome will be discussed according to the vascular supplying areas.

Median pontine syndromes

In the considerable amount of literature regarding pontine lesions, several reports are connected with blood supply of the median pontine area. The basilar thrombosis, on one hand, may produce a bilateral pontine infarction in the pontine basis, areas M1 and M4 (Figs. 5, 6, 8), and in the median pontine area, area M3 (Fig. 7) which includes the basis and tegmentum at the middle level of the pons, or both. Clinically, this condition may cause the locked-in syndrome²³⁾ which is characterized by tetraplegia, pseudobulbar paresis but no interference with consciousness. Such patients may have the capacity to use vertical eye movements and blinking to communicate to others. Actual sites of infarcted areas, however, may differ from one patient to another. On the other hand, a pontine infarction may rarely occur without a thrombosis of the basilar artery. This is usually considered to be due to a thrombosis of branches of the basilar artery. In the case of this condition, each infarcted lesion can be fairly small compared to that of basilar artery thrombosis, and sometimes ipsilateral. Clinical symptomatology of this condition may vary case by case according to the size, number and sites of lesions. In some patients, however, a pontine infarction may occur near the midline of the pontine basis without a demonstrable thrombosis of either the basilar artery or its branches. Each lesion can be small, but multiple infarctions may be present. Clinical manifestations vary from patient to patient including a lack of clinical signs and symptoms.

In the case of a lesion involving the lower median pontine basis, area M4 (Figs. 5, 8), homolateral paralysis of the external rectus muscle occurs, resulting in strabismus externus and diplopia in addition to contralateral hemiplegia. This condition is called the Millard-Gubler syndrome, although their original descriptions (1858, 1858–1859)^{24, 25)} were of hemorrhage and tumor, respectively. If the lesions extends over the midline, which is rather rare in the case of infarction, bilateral abducent palsies and tetraplegia may appear.

In the case of a lesion involving the median upper pontine tegmentum, area M2 (Figs. 5, 6), clinical manifestations may appear with various admixtures of the following: paralysis of conjugate gaze to the side of the lesion, homolateral or bilateral cerebellar signs (ataxia, asynergia and dysmetria), athetotic or ballistic movements of the contralateral limbs, ipsilateral or bilateral facial palsy of a supranuclear type, disorder of position sense and vibration, etc. This is similar to Raymond-Cestan's syndrome²⁶⁾ except for the absence of disturbances of pain, touch and temperature sensation (Fig. 9). This is also called the superior Foville's syndrome in view of the disturbance of conjugate lateral gaze.

If a lesion involves the median lower pontine tegmentum, area M5 (Figs. 5, 8), the syndrome of Foville-Millard-Gubler (or inferior Foville syndrome)²⁷⁾ may occur (Fig. 10). The syndrome is characterized by paralysis of lateral conjugate gaze toward the side of the lesion, homolateral paralysis of the abducent nerve and homolateral paralysis of the facial nerve (peripheral type) when the lesion involves the genu of the facial nerve. In addition, there is a crossed hemiparesis or hemianesthesia including the face, or both, if the lesion extends to the part of the pyramidal fibers, or the spinothalamic and trigeminothalamic tracts.

Fig. 9. Syndrome of upper median pontine tegmentum (partial syndrome of Raymond-Cestan, modified from Dejerine²⁹).

Fig. 10. Syndrome of lower lateral pontine tegmentum (Syndrome of Foville-Millard-Gubler, modified from Dejerine²⁹).

Lateral pontine syndromes

Lesions involving the lateral pontine area produce a clinical picture of cerebellar ataxia: cerebellar asthenia on the side of the lesion with falling to that side, dysdiadochokinesis, dysmetria and impaired coordination in the limb on the affected side. This is sometimes accompanied by a contralateral hemiparesis and hemihypoesthesia for pain and temperature. The name, syndrome of Marie-Foix (or cerebellar hemiparesis of Marie-Foix, 1913)²⁸, is given to this condition, which is commonly caused by cerebral vascular diseases. The following structures may be involved in the lesion: middle cerebellar peduncle, root of the trigeminal nerve, spinothalamic tract and part of the pyramidal tract (Figs. 6–8).

Several months after a lateral pontine lesion, palatal myoclonus, or palato-pharyngo-laryngo-oculo-diaphragmatic myoclonus may appear because of the destruction of the central tegmental tract, or the rubroolivary tract: part of Guillain-Mollaret's triangle²⁹, which connects the nucleus ruber and the inferior olivary nucleus. The myoclonia are usually bilateral, sometimes predominantly ipsilateral, rarely unilateral.

In some patients, the following clinical manifestations may appear: homolateral facial anesthesia for pain and temperature and contralateral anesthesia of the trunk for pain and temperature. This combination corresponds to the syndrome of Grenet (or crossed sensory syndrome)³⁰. The lesion involves the lateral tegmentobasilar region of the middle third of the pons: sensory nucleus of the trigeminal nerve and the spinothalamic tract are involved

Fig. 11. Dorsal pontine syndrome (modified from Dejerine²⁹).

in the lesion. This is sometimes accompanied by a crossed hemiparesis and a homolateral ataxia. The lesion may spread to the pyramidal tract and the middle cerebellar peduncle (Fig. 7).

Some patients may present facial hemispasm, or involuntary hemifacial grimacing on the side of the lesion with the contralateral hemiparesis or hemiplegia. The name, syndrome of Brissaud and Sicard (1908), or syndrome of facial hemispasm and crossed hemiplegia³¹⁾ is applied to this condition, although the original description was in a report on meningo-vascular syphilis of the lateral tegmentobasilar region of the inferior third of the pons (Fig. 8).

Dorsal pontine syndromes

The dorsal pontine syndromes appear usually as part of the superior cerebellar syndrome. However, it rarely happens as an independent vascular syndrome. It is composed of a homolateral paralysis of the muscles of mastication, with or without, wasting, and fasciculation of such muscles, homolateral ataxia and coarse tremor (Fig. 11). The lesion of the syndrome involves the following structures: the motor nucleus of the trigeminal nerve, the medial lemniscus and the superior cerebellar peduncle, which are included in the tributary area of the dorsal pontine branch.

Vascular Supply of the Myelencephalon

Early studies of vascular supply of the myelencephalon (medulla oblongata) were reported by Duret (1873)³²⁾ and Böhne (1927)³³⁾. According to the observation of serial sections, vascular supply of the medulla oblongata can be classified into four branches and tributary areas: median, medial, lateral and dorsal (Figs. 1, 12, 13).

The median medullary branches

These branches are small and reach the median medullary area through the anterior median fissure branching off from the anterior spinal artery and directly from the basilar artery at the upper end of the medulla oblongata. In this area, the following structures are included: the hypoglossal nucleus, the medial longitudinal fascicle, the medial lemniscus, the pyramidal tract, the internal arcuate fibers, etc. (Figs. 12, 13).

The medial medullary branches

Small branches from the vertebral artery supply the inferior olivary nucleus. This area is present only in the upper two thirds of the medulla oblongata (Fig. 12).

Fig. 12.

Fig. 13.

Fig. 12. Arterial supply of the upper myelencephalon.

ASA, anterior spinal artery; DMDB, dorsal medullary branch; LMDB, lateral medullary branch(es); MMDB, medial medullary branch(es); MNMDB, median medullary branch; PICA, posterior inferior cerebellar artery; VA, vertebral artery. See reference⁽⁴⁰⁾ for other abbreviations.

Fig. 13. Arterial supply of the lower myelencephalon.

PSA, posterior spinal artery. See Fig. 12 and reference⁽⁴⁰⁾ for other abbreviations.

The lateral medullary branches

From the vertebral artery, direct small arteries, sometimes after forming a single artery, *artère de la fossette latérale du bulbe*, as named by Foix-Hillemand-Schalit (1925)⁽³⁴⁾, branch off and reach the medulla oblongata through the retro-olivary sulcus to supply the lateral medullary area. Alexander and Suh (1937)⁽³⁵⁾ made an excellent contribution to the description of the blood supply to the lateral medullary area. In this area, several structures are included as follows: the reticular formation, the nucleus ambiguus, the spinal trigeminal tract and its nucleus, the dorsal vagus nucleus, the spinothalamic tract, the internal arcuate fibers, etc. (Figs. 12, 13).

The dorsal medullary branches

These branches diverge from the posterior inferior cerebellar artery to reach the medulla oblongata through a place near the stria medullaris ventriculi quarti to supply the dorsal medullary area. The following structures are included in the dorsal medullary area: the inferior cerebellar peduncle, the medial vestibular nucleus, the cuneate fascicle and its nucleus, the gracile fascicle and its nucleus, etc. (Figs. 12, 13).

Clinical Symptomatology of Myelencephalic Lesions

Currier (1969)⁽⁸⁾ summarized the syndromes of the medulla oblongata. His description is excellent from the view point of vascular supply, except for some of the naming (*vide ante*).

Median medullary syndromes

Dejerine (1914)⁽⁷⁾ described this rare condition in his monograph as *syndrome bulbaire antérieur ou interolivaire* (*hémiplégie alterne inférieure*), i.e. anterior or interolivary bulbar syndrome (inferior alternating hemiplegia, Fig. 14). In this condition, contralateral pyramidal signs and disturbances of deep sensation, and homolateral cerebellar signs and palsy of the

Fig. 14.

Fig. 14. Median medullary syndrome (Syndrome of Dejerine, inferior alternating hemiplegia, modified from Dejerine²⁹).

Fig. 15.

Fig. 15. Lateral medullary syndrome (modified from Dejerine²⁹).

tongue can be observed. Currier (1969)³⁵ classified the so-called Dejerine syndrome under the name of "medial medullary syndrome", into three types: A unilateral lesion with weakness of the tongue due to involvement of the fibers and nucleus of the hypoglossal nerve, in addition to contralateral hemiplegia and loss of posterior column sensations due to involvement of the pyramidal tract and medial lemniscus. The three types include a unilateral lesions, a bilateral lesion, and a combination of median and other part(s) of the medullary areas. The third type includes the syndrome of Babinski-Nageotte (1902)³⁶ which involves the ipsilateral median and lateral medullary areas in the lesion.

Medial medullary syndromes

According to a literature search, there is no case report of a medial medullary vascular lesion that involves only the inferior olivary nucleus (Fig. 12). Actually, it is not known what the symptomatology is if only the inferior olivary nucleus is damaged.

Lateral medullary syndromes

It is well known that the following manifestations appear to be due to involvement of the lateral medullary area: a sudden onset of swallowing disturbance, dysarthria, ipsilateral cerebellar signs, and dissociated alternating hemianesthesia which involves a hemifacial sensory disturbance combined with the crossed sensory disturbance of the trunk and extremities. This is called the lateral medullary syndrome^{34,37} (Fig. 15). According to a literature search, the first report of the lateral medullary syndrome was by Marcet (1817)³⁸ instead of Dr. Gaspard Viessieux who personally experienced manifestations of the syndrome. Senator (1881, 1883)^{39,40} presented autopsy case reports of occlusions of the vertebral artery and of the posterior inferior cerebellar artery. Avellis (1891)⁴¹ reported 150 patients with ipsilateral laryngeal palsy and pointed out only 5 of them with a lesion involving the central nervous system. Wallenberg (1895, 1901)^{42,43} carefully analyzed a patient with the lateral medullary syndrome under the title, "Acute Bulbäraffection: Embolie der Art. cerebellar. post. inf. sinistr. ?". He achieved world-wide fame, as this was called the syndrome of Wallenberg. Stopford (1916)³³, however, opposed that syndrome from the view point of vascular supply. The title of Wallenberg's paper was obviously incorrect. The name, "lateral medullary syndrome"^{34,37}, then, replaced "syndrome of Wallenberg". In addition, Fisher and colleagues

(1961)⁴⁴⁾ drew a definite conclusion. They described 16 autopsy patients with lateral medullary infarction. Among them, 14 had vascular occlusions (12 of the vertebral artery, and 2 of the posterior inferior cerebellar artery). Two other patients had no definite occlusion. No infarctions were found in the cerebellum of any of the autopsies.

Dorsal medullary syndromes

It is rather rare to observe a dorsal medullary syndrome by vascular disturbance. Fisher and colleagues (1961)⁴⁴⁾ reported an autopsy patient with a dorsal medullary infarction that involved the medial vestibular and the lateral cuneate nuclei, and the solitary tract and its nucleus on the left side, because of the thrombosis of the left posterior inferior cerebellar artery and its several branches to the dorsal medullary area (Figs. 12, 13). The patient had headache, unsteadiness, hiccoughs, slight drooping of the left eyelid, and nystagmus. Although Fisher and colleagues did not mention it, such patients probably also had central positional vertigo (Goto, 1983)⁴⁵⁾.

References

- 1) Haymaker W and Kulenbeck H: Disorders of the brainstem and its cranial nerves. In: Clinical Neurology, vol. 3, chap. 40, Joynt RJ (Ed), J. B. Lippincott Co., Philadelphia, pp 1–82 (1990)
- 2) Foix C et Hillemand P: Les artères de l'axe encéphalique jusqu'au diencéphale inclusivement. *Rev Neurol (Paris)*, **II**: 705–739 (1925)
- 3) Stopford JSB: The arteries of the pons and medulla oblongata. *J Anat Psychol*, **50**: 131–164, 255–288 (1916); **51**: 250–277 (1917)
- 4) Gillilan LA: The correlation of the blood supply to the human stem with clinical brain stem lesions. *J Neuropathol Exp Neurol*, **23**: 78–108 (1964)
- 5) Batson OV: Corrosion specimens prepared with a new material. *Anat Rec*, **121**: 425–425 (1955)
- 6) Lazorthes G, Poulhes J, Bastide G et Rouleau J: Les territoires artériels du tronc cérébral: Recherches anatomiques et syndromes vasculaires. *Presse Méd*, **66**: 2048–2051 (1958)
- 7) Dejerine J: Sémiologie des affections du système nerveux. Masson et C^{ie}, Paris (1914)
- 8) Currier RD: Syndromes of the medulla oblongata. In: Handbook of Clinical Neurology, vol. 2, Localization in Clinical Neurology, chap. 10, Vinken PJ, Bruyn GW, Biemond A (Eds), North-Holland Publishing Co., Amsterdam, pp 217–237 (1969)
- 9) Goto N: Morphological studies on blood vessels of the brain: On the distributions of arteries into the posterior perforating substance, and the proposition for "Syndromes of arteries into the posterior perforating substance". *Nihon Univ J Med*, **30**: 983–1000 (1971) (English abstract)
- 10) Fog M and Hein-Sørensen O: Mesencephalic syndromes. In: Handbook of Clinical Neurology, vol. 2, Localization in Clinical Neurology, chap. 12, Vinken PJ, Bruyn GW, Biemond A (Eds), North-Holland Publishing Co., Amsterdam, pp 272–285 (1969)
- 11) Benedikt M: Tremblement avec paralysie croisée du moteur oculaire commun. *Bull Méd (Paris)*, **3**: 547–548 (1889)
- 12) Claude H: Syndrome pédonculaire de la région du noyau rouge. *Rev Neurol (Paris)*, **II**: 311–313 (1912)
- 13) Segarra JM: Cerebral vascular disease and behavior: I. The syndrome of the mesencephalic artery (basilar artery bifurcation). *Arch Neurol*, **22**: 408–418 (1970)
- 14) Weber H: A contribution to the pathology of the crura cerebri. *Med Chir Trans*, **46**: 121–139 (1863)
- 15) Goto N: Pseudo-Weber syndrome (autopsy patient presentation). Anatomy of cerebral vessels, 2nd ed., IV. Vascular disturbances of the central nervous system, Medical Tribune Inc., Tokyo, pp 214–217 (1987) (in Japanese)
- 16) Baloh RW, Furman JM and Yee RD: Dorsal midbrain syndrome: Clinical and oculographic findings. *Neurology*, **35**: 54–60 (1985)
- 17) Parinaud H: Paralysie des mouvements associés des yeux. *Arch Neurol (Paris)*, **5**: 145–172 (1883)
- 18) Nothnagel H: Corpora quadrigemina. Topische Diagnostik der Gehirnkrankheiten. A. Hirschwald,

- Berlin, pp 204–221 (1879)
- 19) Böhne C: Über die arterielle Versorgung des Gehirns. III. Über die arterielle Blutversorgung des pons. *Z Gesamte Anat*, **84**: 776–786 (1927)
 - 20) Trelles JO: Les Ramollissements protubérantiels: Etude clinique et anatomique. G. Doin & C^{ie}, Paris (1926)
 - 21) Loeb C and Meyer JS: Pontine syndromes. In: Handbook of Clinical Neurology, vol. 2, Localization in Clinical Neurology, chap. 11, Vinken PJ, Bruyn GW, Biemond A (Eds), North-Holland Publishing Co., Amsterdam, pp 238–271 (1969)
 - 22) Kubik CS and Adams RD: Occlusion of the basilar artery: A clinical and pathological study. *Brain*, **69**: 73–121 (1946)
 - 23) Plum F and Posner JB: The Diagnosis of Stupor and Coma. F.A. Davis Co., Philadelphia, 1966, 1972 and 1980.
 - 24) Millard A: A propos des foyers apoplectiques de l'encéphale. *Bull Soc Anat (Paris)*, p 206 (1855)
 - 25) Gubler A: Mémoires sur les paralysies alternes en général et particulièrement sur l'hémiplégie alterne avec lesion de la protubérance annulaire (communication 1856). *Gaz Hebd*, **5**: 721–723 (1858); **6**: 3–7, 86–90 (1859)
 - 26) Raymond F and Cestan R: Le syndrome protubérantiel supérieur. *Gaz Hôp Paris*, **76**: 829–834 (1903)
 - 27) Foville A: Note sur une paralysie peu connue de certains muscles de l'œil et sa liaison avec quelques points de l'anatomie et de la physiologie de la protubérance annulaire. *Bull Soc Anat (Paris)*, **23**: 293–414 (1858)
 - 28) Marie R and Foix V: Formes cliniques et diagnostiques de l'hémiplégie cérébelleuse syphilitique. *Sem Méd (Paris)*, **33**: 145–152 (1913)
 - 29) Guillaumin G and Mollaret G: Deux cas de myoclonies synchrones et rythmées vélo-paryngo-laryngo-oculo-diaphragmatiques: Le problème anatomique et physio-pathologique de ce syndrome. *Rev Neurol (Paris)*, **II**: 545–566 (1931)
 - 30) Grenet: quoted by Foville (1858)
 - 31) Brissaud E and Sicard JA: L'hémispasme facial alterne. *Presse Méd*, **16**: 234–236 (1908)
 - 32) Duret H: Sur la distribution des artères nourricières du bulbe rachidien. *Arch Psychiatr Nor Pathol*, **5**: 79–114 (1873)
 - 33) Böhne C: Über die arterielle Versorgung des Gehirns. II. Über die arterielle Blutversorgung der Medulla oblongata. *Z Gesamte Anat*, **84**: 760–776 (1927)
 - 34) Foix C, Hillemand P and Schalit I: Sur le syndrome latéral du bulbe et l'irrigation du bulbe supérieur: L'artère de la fossette latérale du bulbe/Le syndrome dit de la cérébelleuse inférieure/Territoire de ces artères. *Rev Neurol (Paris)*, **I**: 160–179 (1925)
 - 35) Alexander L and Suh TH: Arterial supply of lateral parolivary area of the medulla oblongata in man. *Arch Neurol Psychiatry*, **38**: 1243–1260 (1937)
 - 36) Babinski J and Nageotte J: Lésions syphilitiques des centres nerveux: Foyers de ramollissement dans le bulbe. Hémiasynergie, latéropulsion et myosis bulbaire avec hémianesthésie et hémiplégie croisées. *Nouv Iconog Salpêtrière*, **15**: 492–512 (1902)
 - 37) Merritt HH and Finland M: Vascular lesions of the hind-brain (Lateral medullary syndrome). *Brain*, **53**: 290–305 (1930)
 - 38) Marcet A (according to narration of Gaspard Vieusseux): History of a singular nervous or paralytic affection, attended with anomalous morbid sensations. *Med Chir Trans*, **2**: 217–235 (1817)
 - 39) Senator H: Apoplectische Bulbärparalyse mit wechselständiger Empfindungs lähmung. *Arch Psychiatr*, **11**: 713–726 (1880/1881)
 - 40) Senator H: Zur Diagnostik der Herderkrankungen in der Brücke und dem verlängerten Mark. *Arch Psycniatr*, **14**: 643–668 (1883)
 - 41) Avellis G: Klinische Beiträge zur halbseitigen Kehrkopflähmung. *Berl Klin*, **40**: 1–26 (1891)
 - 42) Wallenberg A: Acute Bulbäraffection: Embolie der Art. cerebellar. post. inf. sinistr.?. *Arch Psychiatr*, **27**: 504–540 (1895)
 - 43) Wallenberg A: Anatomischer Befund in einem als "acute Bulbäraffection: Embolie der Art. cerebellar. post. inf. sinistr.?" beschriebenen Falle. *Arch Psychiatr Nervenkr*, **34**: 923–959 (1901)
 - 44) Fisher CM, Karnes WE and Kubik CS: Lateral medullary infarction: The pattern of vascular oc-

- clusion. *J Neuropathol Exp Neurol*, **20**: 323–379 (1961)
- 45) Goto N, Hoshino T, Kaneko M and Ishikawa H: Central positional vertigo: Clinico-anatomic study. *Neurol Med Chir (Tokyo)*, **23**: 534–540 (1983)
- 46) Goto N: Microanatomy of brainstem blood supply for clinical diagnosis. Proceeding of the 5th Japanese Congress Neurological Surgeons, pp 127–138 (1985)

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