Modern Concepts of CEREBROVASCULAR DISEASE

edited by John Stirling Meyer

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Preface

This is designed to be a practical book written about diagnosis and management of "stroke," or cerebrovascular disease. The subject seemed timely for review since cerebrovascular disorders with resulting disability and impaired higher cortical function (dementia) are becoming an increasingly frequent problem in our aging population. There have been some advances in the non-invasive investigation of strokes which aid in diagnosis (e.g., computer assisted tomography, or the CAT scan, and the non-invasive inhalation of Xe^{133} method for measuring regional cerebral blood flow). The topics covered in the different chapters, together with the authors who were asked to write them, were selected with the practicing physician in mind. The authors have a wide experience from which they write, including practical experience with patient management and/or preventive medicine and public health concerned with the stroke problem. All the authors have experience in teaching and writing and were selected for the clarity of style of their scientific papers and publications relating to the pathogenesis, epidemiology, and management of stroke.

The first chapter, by Dr. C. Miller Fisher, deals with the anatomy of the cerebral circulation and the pathology of cerebral ischemia, infarction, and hemorrhage drawn from his extensive experience at the

Massachusetts General Hospital. Dr. Fisher has devoted a lifetime to clinical evaluation and postmortem correlation of cerebrovascular pathology with neurological symptoms occurring during life. This chapter is well illustrated.

The second chapter, by Drs. James F. Toole and Alan B. Grindal of Bowman Gray School of Medicine, describes tests which are helpful in establishing the diagnosis of stroke. The majority of these tests are simple and can be carried out at the bedside. Dr. Toole is a well-known authority on the subject of neurovascular diagnostic tests.

Dr. John Marshall, of the National Hospital for Nervous Diseases in England, summarizes his exceptional knowledge of the natural history of cerebrovascular disease in Chapter Three. The majority of the information provided is based on his own experience as a neurological consultant to this major teaching hospital in London. He emphasizes recognition of transient cerebral ischemic attacks (TIAs) as one of the most important contributions to present understanding of the pathogenesis and prevention of stroke.

In Chapter Four, Dr. Ninan T. Mathew of Baylor College of Medicine discusses the potential usefulness of non-invasive methods for measuring regional cerebral blood flow and the diagnostic usefulness of this method for the differential diagnosis of transient cerebral ischemic attacks (TIAs), cerebral infarction, cerebral hemorrhage, migraine, multi-infarct dementia and communicating hydrocephalus as a complication of subarachnoid hemorrhage.

In Chapter Five, Dr. K. M. A. Welch, of Baylor College of Medicine, discusses cerebral metabolic changes resulting from cerebral ischemia. Cerebral ischemia reduces oxygen consumption, but glucose consumption and lactate production often increase due to anaerobic glucose metabolism. Impaired cerebral function often correlates poorly with any reduction of high energy phosphates such as ATP and creatine phosphate. Changes in neurotransmitter function resulting from ischemia may play an important part in producing early neurological deficits prior to depletion of high energy phosphates.

Drs. William B. Kannel and Philip A. Wolf, of the Framingham, Massachusetts prospective field trial, discuss risk factors associated with cerebral atherosclerosis and thrombosis with particular emphasis on their own experience with this prospective study of a representative population followed now for over twenty years. In Chapter Six they discuss the broad scope of the stroke problem and relate differences in incidence of stroke with geographical location and associated risk factors such as hypertension, diabetes, and abnormalities of lipid metabolism.

In Chapter Seven, Dr. Victor M. Rivera, of Baylor College of Medicine, discusses the relationship of cerebrovascular disease to the increasing incidence of dementing disorders in our aging population. Although non-vascular causes of dementia (such as neuronal atrophy) may be of relatively greater frequency (i.e. Alzheimer's disease, Pick's disease, Huntington's disease, as well as those of metabolic cause), there is some evidence that multiple infarctions may be an important cause of dementia (multi-infarct dementia). It is of interest that in the natural history of vertebrobasilar ischemia, memory loss, amnesic strokes, and dementia have been reported as late complications.

Finally, in Chapter Eight, medical and surgical treatment of cerebrovascular disease is briefly discussed. The most effective treatment seems to be prevention of stroke by correction of risk factors (i.e., controlling hypertension). Although surgical treatment is discussed, detailed descriptions of neurosurgical and cardiovascular surgical techniques (such as those used for intracranial clipping of aneurysm or carotid endarterectomy) seemed inappropriate in a book of the present scope and design. It should be borne in mind that some of the current concepts of medical treatment of stroke are still under investigation and, hence, are controversial or not widely accepted in daily practice. Nevertheless, with newly available diagnostic methods, such as computer assisted tomography, which provides diagnostic confirmation of strokes with extraordinary accuracy (including exact size and location of edema, hemorrhages, cysts, etc.), as well as non-invasive methods for measuring regional cerebral blood flow, the demand for therapy is likely to increase. Thus, the opportunity now presents itself for adjudication of effectiveness of therapy in stroke.

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CHAPTER ONE

The Anatomy and Pathology of the Cerebral Vasculature

C. Miller Fisher

ANATOMY

The brain is supplied by two internal carotid arteries (ICA) and two vertebral arteries (VA). The ICA arises from the division of the common carotid (CCA), which, on the right side, comes from the innominate artery, and on the left, from the arch of the aorta. The CCA and ICA are rather constant in size and give off no branches in the neck. The external carotid artery (ECA), which also is formed by the division of the CCA, normally supplies no blood to the brain, but in occlusion of the ICA or VA, provides collateral flow of crucial importance. The ICA gives rise to the carot·cotympanic branch in the petrous bone, meningohypophyseal branches in the cavernous sinus region, and the ophthalmic artery immediately distal to the cavernous sinus. About 8 mm distal to the clinoid process the internal carotid gives rise to the posterior communicating artery (PCommA) which passes posteriorly to join the posterior cerebral artery (PCA). The PCA receives its main supply from the ICA, unilaterally in 22% of cases and bilaterally in 7%. About 1.5 cm distal to the clinoid process at the upper arm of the carotid siphon the ICA divides into the middle cerebral artery (MCA)

1. Internal carotid artery
2. Middle cerebral stem
3. Middle cerebral penetrating
4. Middle cerebral superior division
5. Middle cerebral inferior division
6. Lateral orbitofrontal
7. Prerolandic
8. Rolandic
9. Anterior parietal
10. Posterior parietal
11. Angular
12. Posterior temporal
13. Anterior temporal
14. Temporopolar
15. Anterior cerebral stem
16. Recurrent
17. Anterior communicating
18. Anterior cerebral
19. Medial orbitofrontal
20. Frontopolar
21. Callosomarginal
22. Medial prerolandic
23. Medial rolandic
24. Pericallosal
25. Splenial
26. Posterior parietal
27. Posterior communicating
28. Anterior choroidal
29. Posterior cerebral stem
30. Penetrating thalamo-subthalamic paramedian
31. Mesencephalic paramedian
32. Peduncular
33. Posterior cerebral
34. Medial posterior choroidal
35. Anterior temporal
36. Hippocampal
37. Posterior thalamic
38. Lateral posterior choroidal
39. Posterior temporal
40. Splenial
41. Parietooccipital
42. Calcarine
43. Basilar
44. Superior cerebellar
45. Posterior paramedian
46. Short circumferential
47. Anterior inferior cerebellar
48. Vertebral
49. Posterior inferior cerebellar
50. Anterior spinal

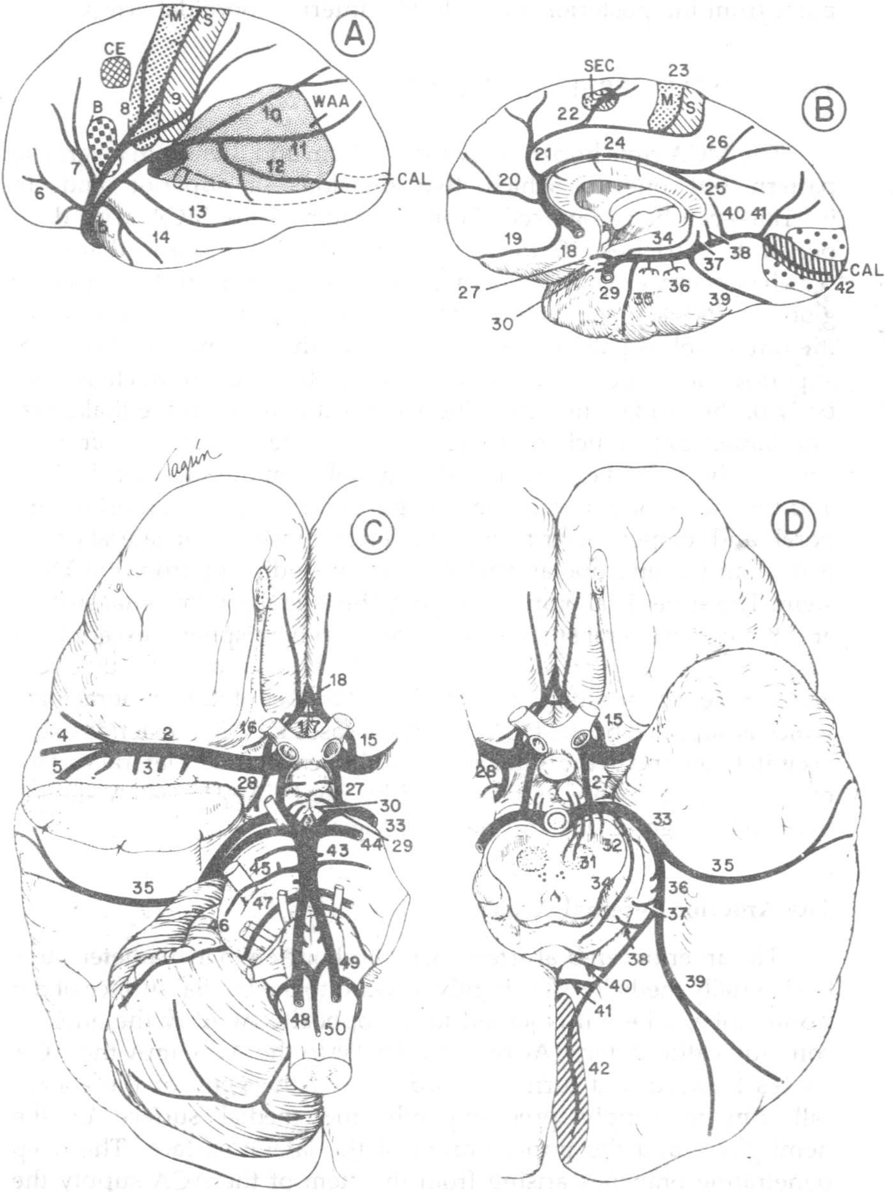

FIG. 1. The cerebral arteries and their branches. B, Broca's area; CE, contraversive eye center; M, motor cortex; S, sensory cortex; WAA, Wernicke's aphasia area; CAL, calcarine; SEC, secondary motor area.

and the anterior cerebral artery (ACA) and gives rise at about the same point from the posterior aspect to the anterior choroidal artery.

THE MIDDLE CEREBRAL ARTERY

The MCA runs horizontally for 1–2.5 cm and divides in a variable pattern but one in which two divisions—a superior and an inferior—can be recognized. From the stem of the MCA arise deep penetrating arteries, 10–15 in number and 0.2–1.0 mm in diameter, which pass upward into the basal ganglia to supply the putamen, outer globus pallidus, posterior limb of the internal capsule above the level of the outer globus pallidus, anterior limb of the internal capsule in its superior half, superior half of the head of the caudate nucleus, and body of the caudate nucleus. The arteries do not reach the thalamus. The superficial branches of the MCA are the lateral orbitofrontal, prerolandic, rolandic, and anterior parietal from the superior division and the posterior parietal, angular, posterior temporal, anterior temporal, and temporopolar from the inferior division. The lateral orbito frontal and temporopolar arteries often arise directly from the MCA stem. The superficial branches supply the lateral orbitofrontal surface and the entire lateral surface of the cerebral hemisphere, except for a strip along the superomedial border, which is supplied by the ACA and a narrow zone along the inferolateral border of the temporal lobe, which is supplied by the PCA. In 50% of cases the MCA extends to the occipital pole and in the other 50%, within 1 cm of it. The lateral surface of the occipital lobe is not supplied by the PCA. The MCA stem is invariably of good size and never vestigial.

The Anterior Cerebral Artery

The anterior cerebral artery, usually about 2 mm in diameter, runs horizontally medially and slightly forward to the medial surface of the frontal lobe, where it is joined to the opposite ACA by the anterior communicating artery (ACommA). Distal to the ACommA the ACA passes forward and turns upward around the genu of the corpus callosum to supply predominantly the medial surface of the hemisphere and the upper border of the lateral surface. The deep penetrating branches arising from the stem of the ACA supply the inferior one-half of the anterior limb of the internal capsule, the inferior one-half of the head of the caudate nucleus, sometimes the inferior one-half of the putamen and the external segment of the global pallidus, the lamina terminalis, the septum lucidum, the medial part of

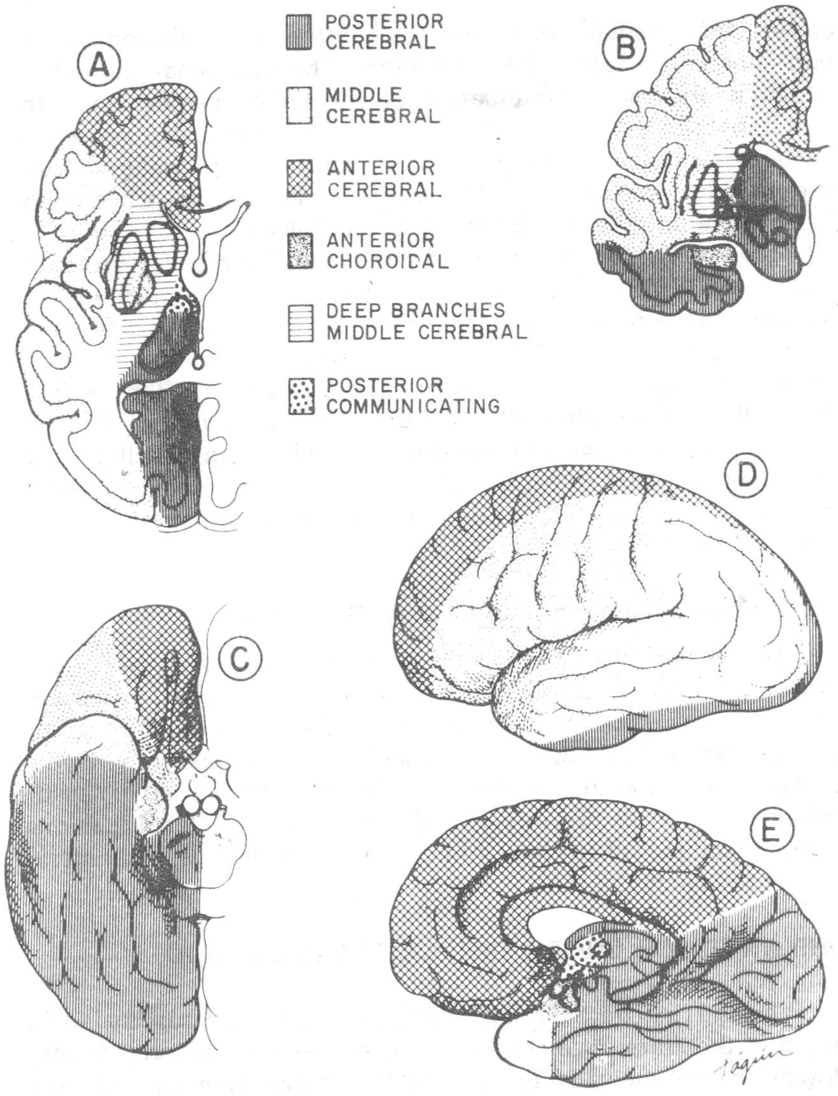

FIG. 2. Regional supply of major cerebral arteries[1].

the anterior commissure, and the upper part of the anterior pillars of the fornix. A prominent branch to the internal capsule called the recurrent artery of Heubner was formerly thought to be associated with a particular contralateral hemiplegic syndrome, but pathological correlation is wanting. The main superficial branches of the ACA are in order, the medial orbitofrontal, frontopolar, callosomarginal, pericallosal, and artery of the splenium. The callosomarginal artery, which is the main continuation of the ACA, passes along the callosomarginal sulcus to reach the marginal sulcus, marking the posterior boundary of the paracentral lobule. The pericallosal artery supplies the precuneus and the corpus callosum, branches to the latter, penetrating to reach the fornix and pillars.

The superficial territory includes the medial one-half of the orbital surface of the frontal lobe, the medial surface of the hemisphere from the frontal pole back to 2.5 cm anterior to the parietooccipital fissure, a strip of the lateral surface of the hemisphere along the superomedial border, broader anteriorly than posteriorly, and the anterior five-sixths of the ipsilateral half of the corpus callosum.

The ACA stems vary in size, one or the other being less than 1 mm in diameter in 14% of cases, both distal ACAs then being supplied from one ACA stem. The ACommA is less than 1 mm in diameter in 20% of cases.

The Posterior Communicating Artery (PCommA)

The posterior communicating artery gives off a series of small branches which supply the optic chiasm, tuber cinereum, hypothalamus, substantia innominata, and anterior one-third of the cerebral peduncle. At times it supplies the subthalamic body, field of Forel, anterior one-third of the posterior limb of the internal capsule below the level of the upper angle of the lateral globus pallidus, anterolateral part of the lateral nucleus of the thalamus, and anterior half of the medial nucleus.

THE ANTERIOR CHOROIDAL ARTERY (A CHOR A)

The anterior choroidal artery (AChorA) arises just proximal to the origin of the anterior cerebral artery and passes posteriorly, slightly downward and medially for 1 cm and then curves laterally for 2 cm to enter the choroidal fissure on the medial aspect of the temporal lobe. In its course it supplies the optic tract as far back as the lateral geniculate body, inferior part of the head of the caudate nucleus, posterior two-thirds of the posterior limb of the internal capsule, as high as the upper angle of the lateral globus pallidus, medial globus pallidus, sometimes posterior inferior part of the putamen, amygdaloid nucleus, uncus and adjacent hippocampus, lateral part of the anterior commissure, choroid plexus in the descending and posterior horn of the lateral ventricle, choroidal membrane in the descending horn of the lateral ventricle, anterior to the plane of the cerebral peduncle, retrolenticular fibers of the internal capsule (optic and auditory radiation), tail of the caudate nucleus and at times middle one-third of the cerebral peduncle (more often supplied by the PCommA,) superior two-thirds of the cerebral peduncle (more often from the PCA), subthalamic body, upper part of the substantia nigra and red nucleus, and most superficial part of the ventrolateral nucleus of the thalamus.

The Posterior Cerebral Arteries (PCA)

The posterior cerebral arteries are formed by the terminal bifurcation of the basilar artery (BA). The stem (the segment between the BA and the PCommA) varies inversely as the size of the corresponding PCommA. When the PCA stem is dominant it runs laterally from its origin, turning around the midbrain to reach the hippocampal and temporal regions on the medial surface of the hemisphere along which it runs to the calcarine cortex and occipital pole. In its proximal 2 cm penetrating arteries (inferior and superior mesencephalic paramedian arteries and the posterior and anterior thalamosubthalamic paramedian arteries) supply the midbrain tegmentum (third and fourth nerves, red nucleus, reticular formation, dentatothalamic radiation), subthalamus, and medial part of the posterior half of each thalamus.[2] One small penetrating artery arising from one PCA may supply the midline territory *bilaterally*. Small branches are supplied to the mamillary bodies and adjacent hypothalamus. As the PCA turns around the cerebral peduncle it supplies the peduncle and gives branches to the tectum of the midbrain. The medial posterior choroidal artery circles the posterior thalamus to reach its superomedial aspect. From the PCA in the region of the pulvinar, penetrating branches pass to the medial and lateral geniculate bodies and the posteroventral thalamus. Still further posteriorly, the lateral posterior choroidal artery traverses the choroidal fissure to enter the choroid plexus in the atrium and body of the lateral ventricle, from which it supplies the superior lateral nuclei of the thalamus.

The superficial branches of the PCA include the hippocampal artery, taking the form of either an arcade with many small branches or a single candelabra supplying the hippocampus, the anterior temporal branch, supplying the anterior part of the inferior surface of the temporal lobe, the posterior temporal branch, supplying the posterior part of the inferior surface of the temporal and occipital lobes, the parietooccipital artery, which ascends in the fissure of the same name to supply the adjacent areas of the medial surface of the posterior parietal and anterior occipital lobes (precuneus and cuneus), and the posterior one-sixth of the corpus callosum and the calcarine artery, which lies in the calcarine sulcus and supplies the superior and inferior banks which comprise the visual cortex. In addition to nourishing the inferior and medial surface of the temporal and occipital lobes, the PCA extends laterally onto the inferior temporal gyrus and the lateral surface of the hemisphere, and in 50% of cases extend up to 1.0 cm onto the lateral surface of the occipital lobe in the region of the occipital pole.

The vessels of the *circle of Willis* become of great importance, in vascular occlusion, in shunting blood from normal into compromised arterial territories—one carotid system to the other, from carotid to basilar or vice versa. The size of the component arteries is highly variable. At least one vessel is less than 1 mm in diameter in 44% of cases. Both PCommA's are 1 mm or less in 33% of cases. The right PCA arises from the ICA in 13% of cases, the left in 9% and both in 7%. The ACommA is small in 20% of cases and one anterior cerebral stem is less than 1 mm in 13% (right 9%, left 4%). When one anterior cerebral artery is small, the ACommA is large. The PCA varies inversely as the size of the corresponding PCommA.

The Basilar and Vertebral Arteries

The vertebral arteries arise from the subclavian arteries, on the right, 2 cm from the origin and on the left, 4 cm from the origin. The left vertebral artery arises from the aorta in 6% of cases. The vertebral artery runs upward in the foramina transversaria of the upper cervical vertebrae, winds medially around the pillar of the atlas, passes forward to pierce the dura and enters the subarachnoid space at the side of the medulla oblongata at the level of the atlantooccipital interspace. It then runs upward and forward onto the anterior surface of the medulla, where, at the pontomedullary level, it joins its mate to form the basilar artery (BA), which then runs upward for approximately 3.0 cm to divide into the posterior cerebral arteries. Variability marks the vertebrobasilar system. The right VA is larger in 26% of cases, the left in 42%, and they are equal in 32%. The VA is less than 2 mm in diameter on the right side in 5% of cases, on the left side in 2%, and both vessels are small in 0.6%. In 9% of cases the BA is largely dependent on one VA, the right in 4%, the left in 5%. One VA fails to reach the BA in 0.6% of cases, ending as the PICA.

The basilar artery supplies the entire pons and the superior and anterior aspects of the cerebellum. The VAs supply the medulla and the inferior surface of the cerebellum. The pattern of blood supply to the pons and cerebellum is commonly regarded as consisting of three sets of branches: *paramedian branches*, supplying the basis pontis and tegmentum on either side of the midline; *short circumferential branches*, running further lateraly to supply a lateral wedge of the pons on either side; and the *long circumferential arteries*, the superior cerebellar artery (SCA), the anterior inferior cerebellar artery (AICA), and the posterior inferior cerebellar artery (PICA), which supply the lateral brain stem on their way to the cerebellum.

The SCA arises a few millimeters from the upper bifurcation of the BA and is extremely constant. It runs laterally, supplying the upper two-thirds of the middle cerebellar peduncle, the posterolateral tegmentum of the pons containing the spinothalamic tract, the medial and lateral lemnisci and the descending sympathetic tract, the superior cerebellar peduncle, and the superior one-half of the cerebellum including the dentate nucleus.

Each AICA arises approximately 6 mm distal to the basilar formation and runs laterally, supplying paramedian branches to the basis pontis and more laterally the lower one-third of the middle cerebral peduncle, the seventh and eighth nerves in their intrapontine course, the cochlear nucleus, the lateral vestibular nucleus, the descending sympathetic tract, the descending tract and nucleus of the fifth nerve, the restiform body, the spinothalamic tract, and the anterior aspect of the cerebellum.

The PICA arises from the VA, 0.5 to 2.0 cm proximal to its junction with the basilar artery. It turns laterally and posteriorly around the lateral aspect of the medulla, looping upward to reach the posterior medulla at the level of the obex, sending a midline branch to the cerebellar vermis and lateral branches to the undersurface of the cerebellum. In the first part of its course it supplies one or two branches to the lateral medullary region, nourishing a wedge of underlying medulla that contains the nucleus ambiguus, the lateral spinothalamic tract, the restiform body, the descending or spinal tracts and nucleus of the fifth nerve, the descending sympathetic tract, the vestibular nuclei, and the dorsal motor nucleus of the vagus. Lateral medullary infarction may be the result of obstruction of either the VA or the PICA.

The paramedian branches of the basilar artery (and the short circumferentials) supply the basis pontis, containing the corticobulbar and corticospinal tracts and the issuing fifth and sixth nerves. The penetrating branches extend posteriorly, or deeply, to reach the tegmentum of the pons, supplying the medial lemniscus, the lateral lemniscus, the medial longitudinal fasciculus, the medial vestibular nuclei, the conjugate lateral gaze centers, the decussation of the trapezoid bodies, and the tegmental reticular formation.

From the upper part oi each vertebral artery a few small branches