Intracranial Atherosclerosis

Edited by

Jong S Kim
Department of Neurology
University of Ulsan College of Medicine
Asan Medical Center
Seoul
South Korea

Louis R Caplan
Department of Neurology
Beth Israel Deaconess Medical Center
Boston
Massachusetts
USA

KS Lawrence Wong
Department of Medicine and Therapeutics
The Chinese University of Hong Kong
Prince Wales Hospital
Hong Kong
People's Republic of China

Foreword by

Geoffrey A. Donnan
Director, National Stroke Research Institute, Melbourne; and
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111 River Street, Hoboken, NJ 07030-5774, USA

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List of contributors

Dr Andrei V Alexandrov MD
Department of Neurology
University of Alabama at Birmingham
Birmingham
Alabama
USA

Dr Pierre Amarenco MD
Department of Neurology and Stroke Centre
Bichat Hospital
Paris
France

Dr Juan F Arenillas MD, PhD
Stroke Unit
Department of Neurology
Hospital Clinico Universitario
University of Valladolid
Valladolid, Spain

Dr Oh Young Bang MD, PhD
Department of Neurology
Sungkyunkwan University
Samsung Medical Center
Seoul
South Korea

Dr Thomas G Beach MD, PhD
Sun Health Research Institute
Arizona
USA

Dr Louis R Caplan MD
Department of Neurology
Beth Israel Deaconess Medical Center
Boston
Massachusetts
USA

Dr Xiang-Yan Chen PhD
Department of Medicine and Therapeutics
The Chinese University of Hong Kong
Prince Wales Hospital
Hong Kong
People's Republic of China

Dr Christopher Chen MD
Department of Pharmacology
Yong Loo Lin School of Medicine
Singapore

Dr Marc I Chimowitz MBChB
MUSC Stroke Center
Department of Neurosciences
Medical University of South Carolina
Charleston
South Carolina
USA

Dr Harry J Cloft MD, PhD
Department of Radiology
Mayo Clinic
Rochester
Minnesota
USA

Dr Geoffrey A. Donnan
National Stroke Research Institute
Department of Neurology
Austin Health
Professor of Neurology
University of Melbourne
World Stroke Organization

Dr Edward Feldman MD
Department of Clinical Neurosciences
Brown University School of Medicine
Providence
Rhode Island
USA

Dr Mark Fisher MD
Department of Neurology
University of California Irvine Medical Center
Orange
California
USA

Dr Philip Gorelick MD, MPH
Department of Neurology and Rehabilitation
University of Illinois College of Medicine at Chicago
Chicago
Illinois
USA

Dr Jinghao Han PhD
Department of Medicine & Therapeutics
Chinese University of Hong Kong
Prince of Wales Hospital
Hong Kong
People's Republic of China

Dr Qing Hao PhD
Department of Medicine & Therapeutics
Chinese University of Hong Kong
Prince of Wales Hospital
Shatin
Hong Kong
People's Republic of China

Dr Yining Huang MD
Department of Neurology
Peking University First Hospital
Beijing
People's Republic of China

Dr Jiann-Shing Jeng MD, PhD
Stroke Center and Department of Neurology
National Taiwan University College of Medicine
Taipei
Taiwan

Dr Wei-Jian Jiang MD
Department of Neurology and Interventional Neuroradiology
Beijing Tiantan Hospital
The Capital University of Medical Sciences
Beijing
People's Republic of China

Dr Dong-Wha Kang MD, PhD
Department of Neurology
University of Ulsan College of Medicine
Asan Medical Center
Seoul
South Korea

Dr Jeong Eun Kim MD, PhD
Department of Neurosurgery
Seoul National University College of Medicine
Seoul
South Korea

Dr Jong S Kim MD, PhD
Department of Neurology
University of Ulsan College of Medicine
Asan Medical Center
Seoul
South Korea

Dr Sun U Kwon MD, PhD
Department of Neurology
University of Ulsan College of Medicine
Asan Medical Center
Seoul
South Korea

Dr Jae-Hong Lee MD, PhD
Department of Neurology
University of Ulsan College of Medicine
Asan Medical Center
Seoul
South Korea

Dr Thomas W Leung MD
Department of Medicine and Therapeutics
The Chinese University of Hong Kong
Prince of Wales Hospital
Hong Kong
People's Republic of China

Dr David S Liebeskind MD
UCLA Stroke Center and Department of Neurology
Los Angeles
California
USA

Dr Min Lou MD
Department of Neurology
The Second Affiliated Hospital of Zhejiang University
School of Medicine
Hangzhou
Zhejiang
People's Republic of China

Dr Aevan McLaughlin MD
Department of Clinical Neurosciences
Brown University School of Medicine
Providence
Rhode Island
USA

Dr Kazuo Minematsu MD, PhD
Cerebrovascular Division
Department of Medicine
National Cardiovascular Center
Suita City
Osaka
Japan

Dr Susumu Miyamoto MD, PhD
Department of Neurosurgery
National Cardiovascular Center
Suita
Osaka
Japan

Dr Fadi B Nahab MD
Emory Stroke Center
Department of Neurology
Emory University School of Medicine
Atlanta
Georgia
USA

Dr Mai Nguyen-Huynh MD
Department of Neurology
University of San Francisco
San Francisco
California
USA

Dr Chang Wan Oh MD, PhD
Department of Neurosurgery
Seoul National University Bundang Hospital
Seongnam
Gyunggi-do
South Korea

Dr Alex E Roher MD, PhD
Longtine Center for Molecular Biology
and Genetics
Sun Health Research Institute
Sun City
Arizona
USA

Dr Dae Chul Suh MD, PhD
Department of Radiology
University of Ulsan College of Medicine
Asan Medical Center
Seoul
South Korea

Dr Jun C Takahashi MD, PhD
Department of Neurosurgery
National Cardiovascular Center
Suita
Osaka
Japan

Dr Toshiyuki Uehara MD
Department of Medicine
National Cardiovascular Center
Suita City
Osaka
Japan

Dr Yongjun Wang MD
Department of Neurology
Beijing Tiantan Hospital
Beijing
People's Republic of China

Dr KS Lawrence Wong MD
Department of Medicine and Therapeutics
The Chinese University of Hong Kong
Prince Wales Hospital
Hong Kong
People's Republic of China

Preface

It reads in the literature that "extracranial atherosclerosis is an important cause of strokes while intracranial atherosclerosis is uncommon occurrence, accounting for 8–10% of strokes. However, it is more often seen in certain ethnic groups such as Asians, Blacks and Hispanics." Whenever we read this, we cannot but feel weird. How many Asians, Black people and Hispanics are there in the world? As of March 2008, the world population had reached an estimated 6.65 billion, of which Asia and Africa accounted for over 60 and 12%, respectively, while Europe and North America accounted for only 11 and 8%, respectively. Although the location of cerebral atherosclerosis has not been reliably investigated in some parts of Asia, the dominance of intracranial atherosclerosis is well established in China, which alone comprises 20% of the world population. Therefore, our view is that the quote above should be revised as follows: "Intracranial atherosclerosis is the major cause of strokes worldwide. However, extracranial atherosclerosis is more often seen in certain ethnic groups, such as Caucasians."

Nevertheless, the literature has focused predominantly on extracranial atherosclerosis, largely neglecting intracranial atherosclerosis. Perhaps, there may be no other disease for which there is a greater discrepancy between the coverage of the literature and the real-world incidence than this example. To be sure, there are several reasons for this unusually large discrepancy. First, the literature in modern times has been dominated by scientists from North America and Northern Europe, who naturally focused on their main interest, extracranial atherosclerosis, while information from other countries has received less notice, especially when written in a local language. Second, extracranial carotid atherosclerosis has gained extra interest because endarterectomy has been a gold mine for vascular surgeons. By contrast, intracranial atherosclerosis remains technically inaccessible, attracting little attention from physicians. Finally, while diagnostic tools such as duplex scan can reliably assess the status of extracranial atherosclerosis, there have been technical limitations to evaluate intracranial vessels until recently.

With advances in imaging technologies such as magnetic resonance angiography, computed tomogrpahic angiography and transcranial Doppler, intracranial atherosclerosis is now more easily detected. In addition, advanced magnetic resonance imaging techniques, including diffusion-weighted magnetic resonance imaging, allow us to investigate stroke patterns and pathogenic mechanisms in patients with intracranial atherosclerosis. With the advent of these technologies, there has been a rapid accumulation of research papers that investigate pathology, risk factors, stroke mechanism, clinical syndrome, diagnosis and medical management of intracranial atherosclerosis. Furthermore, there have been remarkable developments in materials and technologies of stenting and angioplasty, while tools that help assess the cerebral perfusion, such as perfusion magnetic resonance imaging, single photon emission computed tomography or positron emission tomography, now enable us to select the patients who would benefit from bypass surgery.

The aim of this book is to provide our readers with this ever-increasing knowledge on so far underinvestigated areas of intracranial atherosclerosis. This book, written by more than 30 experts in the field, is the first comprehensive textbook devoted to intracranial atherosclerosis. As such, we expect that this book will be of interest to physicians and researchers in diverse medical fields, including neurology, neurosurgery, radiology and rehabilitation medicine, who take care of stroke patients. Residents and students should also find this book interesting and stimulating.

Although we extensively reviewed the currently available knowledge, we think that many important questions still remain to be investigated: Is the pathology of atherosclerosis really different between extra- and intracranial arteries? Are the ethnic differences in the location of atherosclerosis related to differences in risk factors, genetic factor or still unknown factors? What would be the best medical therapy for patients with intracranial atherosclerosis? Who are the

optimal candidates for revascularization therapy, such as angioplasty/stenting or bypass surgery? What is the ultimate clinical outcome in patients with intracranial atherosclerosis? Our hope is that this book will not only guide readers in their clinical practice but also stimulate them to be interested in and to perform research in the field of intracranial atherosclerosis aimed at solving these problems. By doing so, this book will ultimately contribute to the care of patients with intracranial atherosclerosis, the major cause of stroke worldwide.

Finally, we express our sincere appreciation to all the contributors who took time out of their busy schedules to send us manuscripts, and also to Wiley-Blackwell for seeing this book through production. Without their help, this book could not have been brought to light.

Jong S Kim
Louis R Caplan
KS Lawrence Wong
September 2008

Foreword

The editors have produced a superb book that aggregates for the first time all the information pertaining to intracranial atherosclerosis ranging from anatomy, pathology, and mechanisms through to epidemiology. This is particularly timely, given the recent increase in our understanding of this fascinating aspect of stroke medicine. Because it is a relatively infrequent cause of stroke in the West, probably only about 10%, compared to its major contribution to stroke in Asia (probably 30–60% of all strokes), it has occupied only a minor place in stroke medicine. However, with the emergence of Asia as a major academic driving force, the true place of intracranial atherosclerosis in the pantheon of stroke medicine worldwide is now being appreciated.

Increasingly, stroke is being considered as a global problem that needs to be addressed by collaborative initiatives across racial and geographical borders. This book is a good illustration of this approach, with contributions from many different parts of the globe, both East and West. This is very much the philosophy of the World Stroke Organization that has a similar global approach to stroke as a clinical problem together with an awareness that more than half of the worlds' strokes are occurring in Asian countries. To reduce the unacceptable burden of stroke one of the major thrusts must be towards understanding stroke mechanisms in Asia so that adequate preventative measures can be instituted. This volume certainly contributes to this endeavor.

Interestingly, the extent of the problem of intracranial atherosclerosis and a better understanding of its mechanisms have come up with the more recent advances in neuroimaging, including ultrasound. This book nicely puts this in context and should be a useful reference for those wishing to enter this cutting-edge area of research. No stone is left unturned with a nice chapter on rarer causes of intracranial arterial disease for those confronted with difficult individual cases. Indeed, the book is such that it should be accessible by all those interested in stroke ranging from medical students through to experts in the field.

I wish you well in reading this volume. I found it interesting, informative, and addressing an important and emerging area of stroke. I am sure you will find the same.

Geoffrey A. Donnan
President, World Stroke Organization
October 2008

PART ONE

Epidemiology and risk factors

1 Anatomy of intracranial arteries

David S Liebeskind and Louis R Caplan

Comprehensive knowledge of intracranial arterial anatomy forms the basis for consideration of intracranial atherosclerosis. Anatomy defines the location of such neurovascular lesions, delineates the extent and involvement of branching perforators, and consequently determines the effects on downstream perfusion. Anatomy is also intertwined with pathophysiology, as vessel morphology influences hemodynamic variables that promote plaque growth and vessel wall constituents may predispose to atherosclerotic involvement. Once an atherosclerotic plaque has formed, the arterial territories within the brain may shift, reflecting diminished perfusion beyond a stenosis and compensatory collateral flow via anastomoses from adjacent arterial sources.[1] Simply stated, the anatomical features of these arteries or pipes and their perforators determine perfusion, penumbra, and the parenchymal consequences of brain ischemia. These intracranial vessels differ in anatomy from other circulatory beds in the heart or periphery, with only limited correlates noted in the comparative anatomy of intracranial arteries across species. Arterial anatomy adds to the complexity of neurological localization, providing a unique classification of neurovascular disorders. Consideration of intracranial arterial anatomy is most germane to clinical management where recognition of particular stroke syndromes influences treatment decisions. Identification of a culprit atherosclerotic lesion also hinges on anatomical details of the case.

The historical perspective on characterizing anatomy of the intracranial arteries includes an ironic twist where only marginal advances regarding pathology of these arterial segments have been made since the autopsy series performed hundreds of years ago, and angiography reigns as the definitive modality for defining these structures almost a century after its introduction. Pathology related to atherosclerotic involvement of the major intracranial arteries has largely eluded modern imaging techniques because of the small size of these vessels and the orientation of these segments that defy conventional imaging planes. Numerous noninvasive methods have been developed to image intracranial arterial anatomy,[2] yet these modern vascular imaging techniques including transcranial Doppler ultrasound (TCD), computed tomographic angiography (CTA), and magnetic resonance angiography (MRA) are not as accurate as the gold standard of conventional or digital subtraction angiography (DSA).[3] Recently, the advent of angioplasty and stenting for intracranial atherosclerotic disease has reinforced the importance of DSA, since arterial access is needed for treatment. Noninvasive imaging modalities such as TCD, CTA, and MRA each provide differing information regarding a balance of anatomical information, such as measures of the arterial lumen versus physiological data reflecting flow through a specific arterial segment and distal perfusion. DSA remains the prevailing method for evaluating vascular anatomy in the brain, although modifications such as three-dimensional rotational angiography (Fig. 1.1) have allowed for expansion across numerous frames of reference.

This introductory chapter considers the vascular anatomy of the major intracranial arterial segments supplying blood flow to the brain, emphasizing

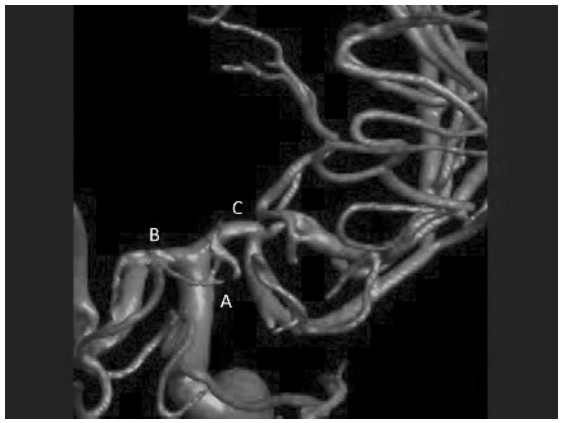

Fig 1.1 Three-dimensional rotational angiography illustrating the proximal segments of the anterior circulation, including bifurcation of the internal carotid artery (A) into the anterior carotid artery (B) and middle cerebral artery (C).

proximal segments where atherosclerotic lesions or stenoses are often noted. The extracranial segments of these vessels are not discussed and only marginal attention has been devoted to distal branches beyond the primary or secondary intracranial arterial divisions. For each artery, the chapter reviews embryologic development, basic morphology such as orientation and luminal dimensions, functional aspects such as perforators, territories, and collateral anastomoses, and common variants encountered in standard anatomy.

Embryology

The arteries of the central nervous system originate from mesenchymal elements that coalesce to form channels that cover the surface of the neural tube. Over time, certain channels persist and enlarge to become principal conduits whereas others involute. A single ventral median artery forms, with paired or symmetrical branches that spread out in a circumferential pattern over the surface. A segmental pattern of blood flow predominates the 4- to 12-mm human embryo stage, arising from the branchial arches. Intracranial blood flow at this stage is distributed by the primitive trigeminal, otic or acoustic, and hypoglossal arteries. Early arterial blood flow is centripetal, extending from the periphery to the center. Beyond the 12-mm stage, longitudinal connections develop, including the vertebral arteries that form with involution of the cervical intersegmental arteries. The embryologic development of the circle of Willis is also important to consider when these segments are recruited to later shunt blood flow due to stenosis or occlusions in the anterior or posterior circulations. Previously hypoplastic segments may be recruited and may progressively enlarge over time, whereas others involute because of disuse. The specific events characterizing the embryologic development of particular intracranial arteries are detailed below.

Arterial wall

The majority of anatomical descriptions consider the cerebral vasculature as a mere conduit to supply and return blood through the brain, yet these vascular channels play an active role in the regulation of blood flow in the brain. The proximal segments of the intracranial arteries distribute flow to specific areas of the brain to match metabolic demand during development and for years thereafter. Cerebral perfusion depends on intraluminal pressure and downstream resistance. Because arterial blood pressure is so readily measured and commonly employed as a principal vital sign, the presumption is that cerebral blood flow is principally mediated by blood pressure. Most of the pressure head or arterial pressure gradient is lost before blood flow reaches the terminal branches feeding the cortical surface and deep regions of the brain. Resistance is directly modulated by these proximal arterial circuits and their vessel wall constituents, in addition to other biophysical factors and metabolic orchestration within the intracranial compartment. Unlike the peripheral vasculature, where precapillary sphincters mediate pressure gradients, the cerebral circulation lacks such structures and pressure gradients are modified in the arteries and arterioles of the brain. Flow is also readily shunted or equilibrated via unique anastomotic structures such as the circle of Willis. These features underscore the importance of recognizing the unique role of the proximal arterial circulation in the brain, not just as pipes for flow distribution, but as active physiological elements in metabolic homeostasis. The structural characteristics in the vessel wall that enable such functional capacity are therefore an important anatomical aspect to consider.

Several features distinguish intracranial arteries from arteries of similar caliber elsewhere in the body. Arteries in the brain have a well-developed internal elastic lamina with only a minimal degree of elastic fibers scattered in the media.[4] Unlike arteries elsewhere throughout the body, the intracranial arteries do not have an external elastic lamina. Other distinctive features of the intracranial arteries include the presence of tight endothelial junctions with a relative paucity of pinocytic vesicles, and differing distribution of enzymes within the vessel wall. The adventitial layer is typically thin compared with the systemic arteries. In general, the cerebral arteries have a smaller wall-to-lumen ratio than arteries elsewhere in the body.[5] Overall, the intimal layer accounts for about 17% of total vessel wall thickness, with the media constituting 52% and adventitia only 31%.[6,7]

The arterial lumen is defined by the adjacent architecture of the vessel wall. Cerebral endothelial cells with tight junctions form a critical element of the blood–brain barrier.[8] These endothelial cells are not fenestrated and the tight junctions bestow only selective permeability to this boundary, preventing exchange of numerous substances. This boundary is often referred to as the "blood–brain barrier." Under pathophysiologic conditions, this selective permeability boundary may be deranged.[9] The number of endocytotic vesicles is also limited compared with the endothelial lining of other vascular beds. Cerebral endothelial cells have a high concentration of mitochondria, denoting their active metabolic role and, possibly, their vulnerability to ischemia.[10] Endothelial cells in cerebral arteries and arterioles play an active role in the regulation of hemodynamics. This capacity is partially related to the expression of a wide array of vasoactive substances, including endothelin and nitric oxide.[11] The internal elastic lamina of intracranial arteries is fenestrated, with holes that vary in size according to the arterial segment.[12] Beyond the endothelial layer, the cerebral arteries have protuberances at distal branching sites that also modulate flow. These structures have been variably defined as intimal cushions, bifurcation pads, or subendothelial protuberances. Underneath the luminal surface, these structures contain groups of smooth muscle cells arranged in irregular fashion, with intertwined collagenous fibrils and are encompassed by the split internal elastic membrane.[13] Although the exact role of these structures in titration of arterial pressure has not been fully elucidated, it appears that these structures help alter flow via fluid shear stress mechanisms. Fluid shear stress is a critical physiological variable both in the development of atherosclerosis and in compensatory arteriogenesis.[14,15] A circumferential orientation of the smooth muscle cells at branching sites may be related to titration of arterial inflow resistance by acting via a sphincter-like mechanism.

In normal intracranial arteries, smooth muscle cells make up 72% of the media, whereas this composition is radically altered under pathophysiologic conditions such as intracranial atherosclerosis or chronic hypertension.[4] Age-related changes are found in the composition of the media. Autonomic nerves located in the tunica adventitia have connections with these subendothelial structures via intercellular smooth muscle cell contacts. Within the media, smooth muscle cells are generally oriented in a pattern circumferential to the lumen except at bifurcations.[6] Adjacent collagen and elastin fibers run perpendicular to the smooth muscle layer or in parallel with the long axis of the vessel. The thin medial layer of intracranial arteries compared with systemic vessels is thought to be related to compliance differences associated with surrounding cerebrospinal fluid. The number of smooth muscle cell layers within the media diminishes distally. A basement membrane associated with the adjacent smooth muscle cells forms the framework for adjoining layers of the intima and adventitia. Nerve fibers approach the media from the adventitial layer. Within the adventitia, loose connective tissue surrounds autonomic nerve fibers and all vessel wall structures are enclosed by spindle-shaped fibrocytes. Once beyond the dura mater, the intracranial arteries have no vasa vasorum. The external surface of the intracranial arteries in these regions is in direct contact with surrounding cerebrospinal fluid. A rete vasorum in the adventitia is permeable to large proteins, allowing ingress or exchange with cerebrospinal fluid in the subarachnoid space.[16]

Characteristics of the intracranial arterial wall in humans typically consider the proximal intracranial arteries such as the middle cerebral artery (MCA) separately from much smaller intracerebral or pial arterioles. As the internal carotid artery (ICA) courses distally, there is progressive disappearance of the external elastic lamina. The MCA is a terminal continuation of the ICA with a gradual change in blood vessel wall characteristics of histopathology. The relative

amounts of intima, media, and adventitia in the MCA are less than the equivalent amount per vessel size in the more proximal ICA. The MCA internal elastic lamina is thicker and partially fenestrated. Compared with similar sized extracranial arteries, the MCAs have less adventitia with less elastic tissue and few perivascular supporting structures, including an absence of vasa vasorum.[17]

Internal carotid artery

Each ICA supplies approximately 40% of total perfusion to the brain. The ICA develops from the third primitive aortic arch. The distal cervical segment of the ICA arises from the junction of the distal aspect of this third primitive aortic arch with the dorsal aorta. The ICA arises from the common carotid artery in the neck, extending into the head at the skull base via the carotid canal (Fig. 1.2). There are three named segments of the intracranial ICA, including petrous, cavernous, and supraclinoid segments (Fig. 1.3). The petrous ICA extends for about 25–35 mm anteromedially from the skull base to the cavernous sinus.[18] The shape of the petrous ICA varies depending on the development of the surrounding bony structures skull. Along this course, it bends anterior to the tympanic cavity near the apex of the petrous bone and traverses the posterior aspect of the foramen lacerum. The ICA crosses the membranes of the cavernous sinus, winding anteriorly and superomedially, then ascending vertically in a groove along the sphenoid bone and then passing along the medial aspect of the anterior clinoid process. On exiting the cavernous sinus, the ICA extends through the meninges to become the supraclinoid segment. The cavernous ICA typically averages 39 mm in length. The supraclinoid or cerebral ICA bends posteriorly and laterally between the oculomotor (III) and optic (II) nerves. Because of this sinuous course of the ICA, the cavernous and supraclinoid segments are often collectively referred to as the carotid siphon. Beyond the supraclinoid segment, the ICA terminates at the bifurcation into the anterior carotid artery (ACA) and MCA. This bifurcation is often referred to as the "carotid T" because of its shape or the "top-of-the carotid" because of its location.

Along the course of the intracranial ICA, branching progressively increases with more distal locations.[19]

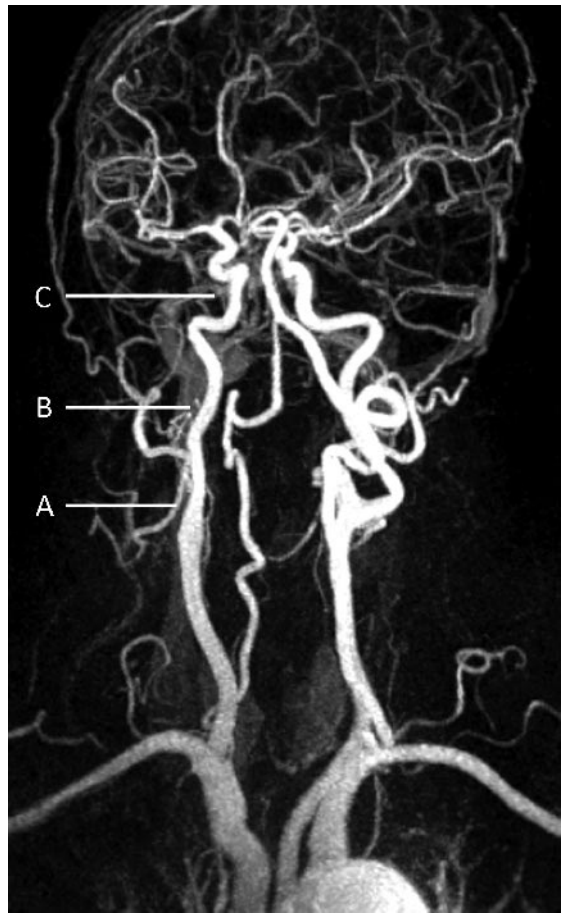

Fig 1.2 Gadolinium-enhanced magnetic resonance angiography depicting the course of the right internal carotid artery (ICA) from its extracranial origin at the carotid bifurcation (A), through the carotid canal at the skull base (B), to become the intracranial ICA (C).

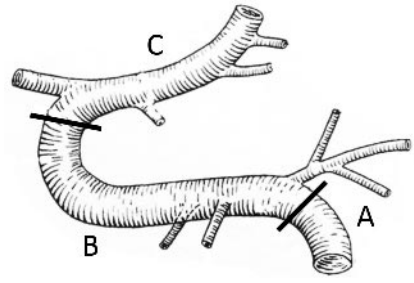

Fig 1.3 Line drawing of the intracranial internal carotid artery, depicting the petrous (A), cavernous (B), and supraclinoid (C) segments.

The petrous segment gives rise to the caroticotympanic artery, supplying the tympanic cavity, and the pterygoid or vidian branch passing through the pterygoid canal.[18] This vidian artery anastomoses with the internal maxillary artery. On occasion, the persistent stapedial branch of the petrous segment traverses a bony canal and continues as the middle meningeal artery.[18] The cavernous portion, however, has far more tributaries including the meningohypophyseal trunk, the anterior meningeal artery, the artery to the inferior portion of the cavernous sinus, and the ophthalmic artery. The meningohypophyseal trunk further subdivides into diminutive branches that include the basal and marginal (artery of Bernasconi and Cassinari[20]) tentorial arteries, the inferior hypophyseal artery, and the dorsal meningeal artery. The inferolateral trunk arises from the inferolateral aspect of the cavernous ICA, supplying many small branches to the tentorium and trigeminal (V) nerve divisions. Collateral anastomoses between the ICA and the external carotid artery (ECA) are formed by the inferolateral trunk extending to the internal maxillary artery. The supraclinoid ICA also has numerous branches including the superior hypophyseal perforators to the anterior pituitary and stalk, posterior communicating artery (PCoA), and anterior choroidal artery (AChA) before bifurcating into the ACA and MCA.[21]

The two ACAs connect through the anterior communicating artery (ACoA) thus joining the left and right carotid circulations. The PCoA extends posteriorly to connect with the primary segment of the posterior cerebral artery, allowing collateral flow to pass between the anterior and posterior circulations.[1] This vascular network, referred to as the circle of Willis (Fig. 1.4), plays a critical role in shunting blood flow between adjacent territories in the brain. At its origin, the PCoA often has a widened segment referred to as the infundibulum. The PCoA passes ventral to the optic tract, with perforators that supply the optic tract, posterior aspect of the chiasm, posterior hypothalamus, and anterior and ventral nuclei of the thalamus. In 15% of individuals, this vessel continues distally as the posterior cerebral artery.[22,23] Great variability may be noted in the caliber of the PCoA, ranging from less than 1 mm to greater than 2 mm. The anatomy of the PCoA differs in various populations and in clinical conditions associated with ischemia.[23,24] Hypoplasia or absence of the PCoA is found in a minority of cases at autopsy, with bilateral hypoplasia in only 0.25% of individuals.[22] The configuration and size of the PCoA also differs between rates gleaned from autopsy studies and angiography series.

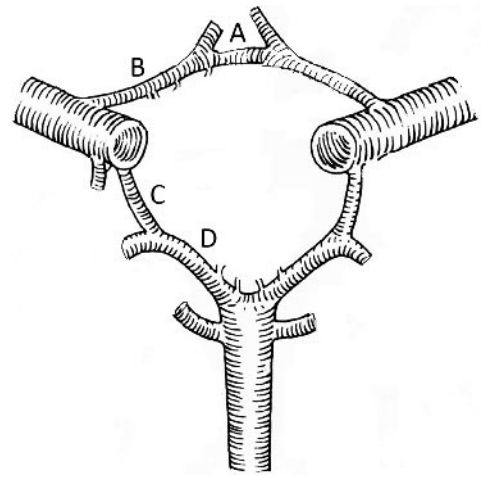

Fig 1.4 Line drawing of anastomotic connections at the circle of Willis, including the anterior communicating artery (A), the proximal or A1 segment of the anterior carotid artery (B), the posterior communicating artery (C), and the proximal or P1 segment of the posterior cerebral artery (D).

There are numerous variant configurations of the ICA, including its rare absence or hypoplasia. The amount of blood volume supplied to distal structures can vary depending on the caliber of the terminal ICA. The course of the ICA sometimes varies, coursing through the middle ear or bending towards the midline in a configuration termed kissing ICAs at the cavernous segments. Anomalous origins of the posterior fossa arteries from the ICA, including the superior cerebellar artery (SCA), anterior inferior cerebellar artery (AICA), or posterior inferior cerebellar artery (PICA), may also occur. Persistent fetal connections to the posterior circulation may involve the PCoA, trigeminal, otic or acoustic, hypoglossal, and proatlantal intersegmental arteries. The persistent trigeminal artery is the most common persistent embryonic connection (85%), arising from the cavernous ICA and joining the upper basilar artery.[25] The persistent otic artery is very rare, connecting the petrous ICA with the basilar artery inferior to AICA. The persistent hypoglossal connects the distal cervical ICA with

the distal vertebral artery. Intercavernous ICA collaterals may also allow for blood to flow laterally to either hemisphere.

Anterior choroidal artery

The AChA arises from the posterior aspect of the ICA, about 2–4 mm distal to the origin of the PCoA and about 5 mm proximal to the carotid terminus.[26] The AChA is relatively small, yet serves as an important landmark in delineating important structures at angiography.[27] There are two segments of the AChA, including the cisternal and plexal segments. The AChA may have a single origin or may consist of several smaller vessels (4% of individuals).[26,28] The AChA arises from the MCA or PCoA in 2–11% of individuals.[28] Complete absence of the AChA has also been reported.[27] The external diameter of this vessel is often only 0.5–1 mm, although a reciprocal relationship has been noted in the caliber of this vessel with the ipsilateral PCoA. The cisternal segment passes posteriorly from the lateral to medial aspect of the optic tract in close proximity to the posterior cerebral artery (PCA), extending for about 12 mm, extending to a total length of about 26 mm. The AChA gives off penetrating branches to the optic tract in this segment. As the AChA courses posteriorly it gives off penetrating branches to the globus pallidus and the genu and posterior limb of the internal capsule. Subsequent branches extend laterally to supply the medial temporal lobe cortex, hippocampal and dentate gyri, caudate, and amygdala. Medial branches supply the cerebral peduncle, substantia nigra, red nucleus, subthalamus, and ventral anterior and lateral nuclei of the thalamus. The AChA is the only branch of the ICA that supplies a portion of both the anterior and posterior circulation, although the midbrain and thalamic supply is very variable. More distally, the AChA extends through the choroidal fissure to become the plexal segment. The juncture of the AChA at the choroidal fissure is often referred to as the plexal point. The plexal segment then enters the choroid plexus near the posterior aspect of the temporal horn. Arterial supply of this segment includes the lateral geniculate body, optic radiations, and posterior limb of the internal capsule. The AChA anastomoses with lateral branches of the posterior choroidal artery, PCoA, PCA, and MCA.[27,29] Variants include AChA origin from the PCoA or MCA.

Middle cerebral artery

The MCA provides arterial blood flow to the largest extent of the intracranial circulation. The MCA is typically 75% of the caliber of the parent ICA.[30] After diverging from the terminal ICA below the anterior perforated substance, it courses horizontally and slightly anteriorly to reach the sylvian fissure, where branches perfuse the frontal, parietal, and some extent of the temporal and occipital cortices (Fig. 1.5). The proximal or horizontal segment of the MCA averages around 15 mm in length yet may be as long as 30 mm.[30] At younger ages, the M1 segment rises obliquely but this segment tends to course more inferiorly or anteriorly with increasing age later in life.[31] Between the 7 and 12 mm (7 weeks) embryonic stage, small perforators that are precursors of the MCA may be seen arising from the ICA. The MCA is smaller than the AChA at these early stages and then grows larger. During the second month of fetal life, the sylvian

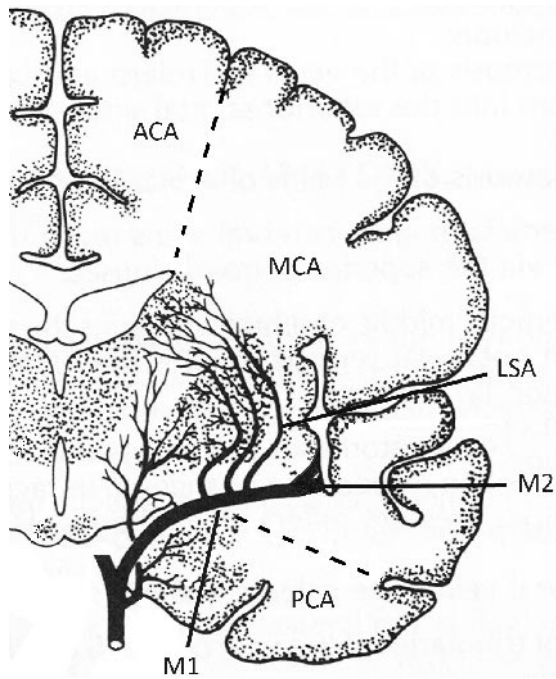

Fig 1.5 Schematic of the middle cerebral artery, illustrating the proximal segment or M1, lenticulostriate arteries (LSA), and bifurcation into M2, with downstream territories delineated between adjacent anterior carotid artery and posterior cerebral artery regions.

fissure develops as a groove over the cerebral hemisphere and the MCA grows within this depression. The MCA becomes enveloped in the sulcation of the cerebral cortex, following the growth of each specific brain region.

The proximal or M1 division of the MCA provides lenticulostriate arteries that feed the globus pallidus, putamen, internal capsule, corona radiata, and caudate nucleus. This segment is typically around 2.5 mm in internal diameter.[30] These end arteries originate from the M1 segment in almost perpendicular fashion to penetrate the brain parenchyma. The lateral lenticulostriates ascend for 2–5 mm posteromedially from the M1 and then course laterally and superiorly for an additional 9–30 mm to penetrate the internal capsule. The medial lenticulostriates generally arise from more proximal segments of the MCA or from distal reaches of the terminal ICA and proximal ACA.[32–35] There is considerable variation in the relative distribution and origins of medial versus lateral lenticulostriate perforators. The arterial diameter of lateral lenticulostriates is typically greater than the medial lenticulostriate perforators. Overall, there are typically 5–17 lenticulostriate arteries, although all are barely identifiable at angiography.[30] There are three principal patterns that have been described for the anatomy of the lenticulostriates.[32–35] Grand *et al.*[33] described a pattern where either one or more of the larger lenticulostriates arise just beyond the MCA bifurcation (49%), all arise proximal to the major bifurcation (39%), and a minority of cases where some of the larger perforators arise from the medial portion of the stem. According to Jain,[34] 54.1% originate from the MCA trunk, 25.6% from the division point, and 20.3% from one of the branches of the MCA. The lateral lenticulostriates supply the lateral portion of the anterior commissure, the putamen, the lateral segment of the globus pallidus, the superior half of the internal capsule, the adjacent corona radiate, and the body and head of the caudate nucleus. The medial lenticulostriates arise in perpendicular fashion to the parent MCA or ACA, yet bend in mesial fashion. The areas supplied by the medial lenticulostriates, including the prominent recurrent artery of Heubner, and the AChA are adjacent to the territories of the lateral lenticulostriates. The relative territorial extents are reciprocal in size and depend on the development of each of these arterial groups.[32–35]

The largest branch of the proximal MCA is the anterior temporal artery, extending from the middle of the proximal MCA and winding anteriorly and inferiorly. Although the configuration of the proximal MCA often varies, the vessel most often splits into two or more principal divisions near the sylvian fissure. Although prior studies have suggested symmetry in the morphology of bilateral MCAs, no clear correlations exist. The anterior and posterior divisions of the MCA extend into the sylvian fissure and spread out over the hemisphere. These cortical branches include the temporopolar, frontobasal, operculofrontal, precentral, postcentral, posterior parietal, angular, anterior temporal, middle temporal, and posterior temporal arteries. As the MCA branches loop over the insula in the sylvian fissure, they form the sylvian triangle, a landmark classically used to identify mass lesions on angiography. Terminal branches of the MCA form collateral anastomoses with the ACA and PCA.[1]

Variation in MCA anatomy is less common than variants in other intracranial arteries. Fenestration of the M1 segment occurs, and duplicated M1 segments may also arise from the ICA.[36] Angiographic demonstration of MCA fenestration may be evident in approximately 0.26% of individuals.[37] Yamamoto *et al.*[38] described 14 accessory MCAs and seven duplicated MCAs in a series of 455 bilateral carotid angiographies. The M1–M2 junction is characterized by a bifurcation in 64–90% of individuals, trifurcation in 12–29%, and complex branching in isolated individuals.[30] Some controversy has surrounded specific landmarks and associated classification of the MCA segments. Whereas many identify the segments of the MCA based on each successive branch point, others use a nomenclature that relates each of these MCA segments with a specific adjacent anatomical structure. For instance, some refer to the M2 origin at the initial bifurcation of the proximal or M1 MCA, whereas others identify the M2 segment as the arterial segment that overlies the insula.

Anterior cerebral artery

The ACA develops from residual elements of the primitive olfactory artery at the terminus of the ICA. The paired primitive olfactory arteries from each side form a plexus in the midline that gives rise to the ACoA. During development, the ACA extends superiorly and

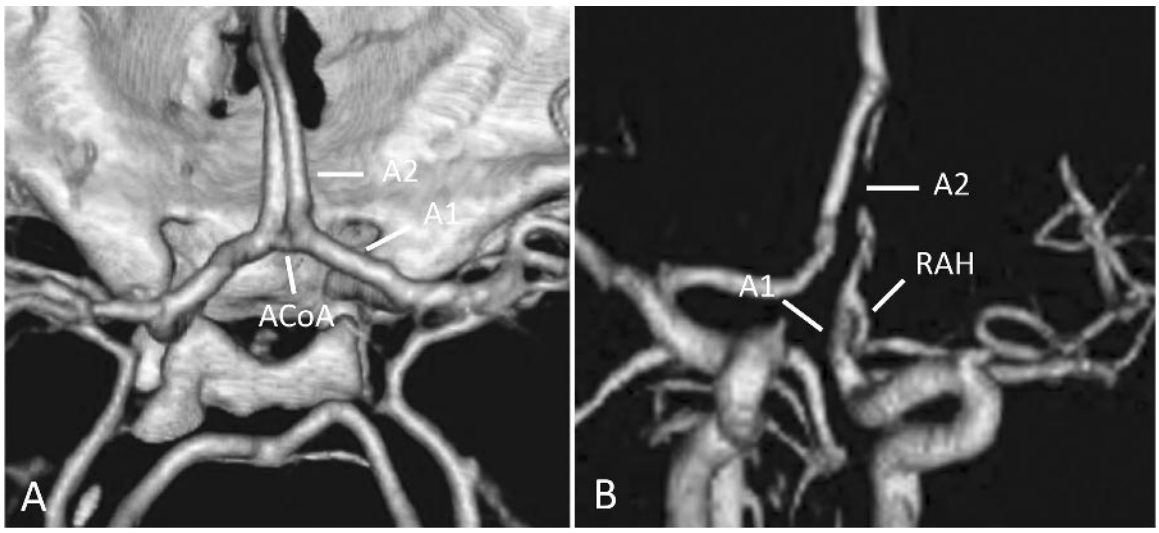

Fig 1.6 CT angiography illustration of two different configurations in the anterior carotid artery (ACA) complex and anterior communicating artery (ACoA) anastomosis. (A) A patent ACoA provides interhemispheric flow between left and right ACAs. (B) ACoA is absent, yet a prominent recurrent artery of Heubner (RAH) is demonstrated.

then posteriorly over the hemispheres in the midline whereas the remainder of the primitive olfactory artery regresses to become a small perforating vessel. The ACA is typically 50% of the caliber of the parent ICA.[39] The internal diameter of the A1 is usually 0.9–4 mm, with hypoplasia defined as a diameter less than 1 mm. The A1 segment measures 7–18 mm, with an average span of 12.7 mm.[39] The ACA extends anteromedially between the optic chiasm (70% of individuals) or optic nerve (30% of individuals) and the anterior perforated substance to join the contralateral ACA through an anastomosis via the ACoA.

The ACoA forms the anterior aspect of the circle of Willis, a critical route for collateral flow between the cerebral hemispheres. The ACoA is the shortest cerebral artery, measuring only 0.1–3 mm in length.[39] The anatomy of the ACA–ACoA is variable (Fig. 1.6) with hypoplasia of different segments, including absence of the ACoA. Accessory routes, fenestrations, and other complex azygous connections between the proximal ACAs are also described.

The proximal ACA or A1 segment gives off numerous perforating arteries that supply the adjacent optic nerves and chiasm inferiorly, and the hypothalamus, septum pellucidum, anterior commissure, fornix, and corpus striatum. These mesial lenticulostriate vessels often include a prominent recurrent artery of Heubner that supplies the caudate head, putamen, and anterior limb of the internal capsule.[40] The A2 segment begins at the juncture of the ACA with the ACoA and extends to the genu of the corpus callosum. The recurrent artery of Heubner arises from the A2 segment in 49–78% of individuals.[39] The recurrent artery of Heubner may be a single vessel or can be represented by a number of parallel arteries. Beyond the proximal segment of the ACA, azygous connections allow for shunting of flow between the cerebral hemispheres. The ACAs course over the cerebral hemispheres in the interhemispheric fissure as paired vessels, with their distal extent typically determined by the corresponding anatomy of the PCAs. Subsequent divisions including the pericallosal and callosomarginal arteries divide to provide arterial supply to the corpus callosum and anteromesial cortices. Several variations in distal ACA anatomy have been described, including the observation that the left pericallosal artery is located more posteriorly than the corresponding right-sided vessel in 72%.[41] Similarly, absence of the callosomarginal artery has been noted in 18–60% of cases studied.[41]

Cortical branches of the ACA include the orbitofrontal, frontopolar, callosomarginal, and pericallosal arteries. As the terminal portion of the ACA travels along the corpus callosum, its anterior pericallosal

branches form anastomoses with the posterior pericallosal branches of the PCA.[41]

Variant anatomy of the ACA most commonly includes hypoplasia or absence of the A1 segment (10% of individuals).[39] Other variations may include anomalous origin of the ACA from the ICA, agenesis or accessory branches, direct connection of bilateral A1 segments, or other combinations that involve azygous orientation of distal ACA segments.

Vertebral artery

The vertebral artery enters the skull at the level of C1 through the foramen magnum. The intracranial or intradural (V4) segment of the vertebral artery ascends anteriorly to the medulla, approaching the midline at the pontomedullary junction, where it meets the contralateral vertebral artery to form the basilar artery. The paired longitudinal arteries that form the arterial supply to the posterior circulation during early fetal development retain their proximal course as the vertebral arteries. The left vertebral artery is larger than the right 42% of the time, whereas the right is larger than the left 32% of the time. In the remainder of individuals, the vertebral arteries are equivalent in caliber. Vertebral artery hypoplasia is fairly common, often involving the right side. The frequency of this finding depends largely on the modality used to image the vessel, the size threshold used to define hypoplasia, and the study population, including healthy subjects or patients with ischemic stroke. Defining hypoplasia as $\leq$2 mm by ultrasonography, one group reported a frequency of 1.9% in 451 subjects.[42] Among healthy subjects with a threshold of $<$3 mm, another group noted a frequency of 6% in 50 healthy subjects.[43] Others have recently noted a frequency as high as 35.2% in 529 patients with ischemic stroke.[44] Utilizing a luminal diameter threshold of 2.2 mm, prominent asymmetry in vertebral artery hypoplasia has also been described (7.8% on the right and 3.8% on the left) in 447 subjects.[45] Differentiation of hypoplasia from a diseased arterial segment may be difficult to define on the basis of luminal dimensions alone. The configuration or compensatory enlargement of neighboring segments may provide clues to this distinction. Furthermore, definitions may vary considerably depending on the imaging technique employed. The vertebral artery may also terminate in the PICA rather than extend to the junction with the basilar artery. In such cases, the vertebral artery is generally smaller than the contralateral vertebral artery.

The terminal vertebral artery yields several branches that supply the rostral end of the spinal cord and posterior inferior aspect of the cerebellum. Anterior and posterior spinal arteries extend from this segment. Each anterior spinal artery fuses with its counterpart, supplying the ventral medulla and rostral spinal cord. The posterior spinal arteries do not pair in the midline, but descend the spinal cord at the level of the dorsal roots. The posterior inferior cerebellar artery branches from the vertebral artery to supply the inferior aspect of the cerebellum.

Posterior inferior cerebellar artery

The largest tributary of the vertebral artery is the PICA, arising 10–20 mm before the vertebrobasilar junction (Fig. 1.7).[46] In 20% of individuals, the PICA arises from below the foramen magnum.[47] During embryogenesis, the PICA may be evident as a larger branch of numerous arteries that extend posteriorly from the hindbrain in the 20–24 mm embryo stage.

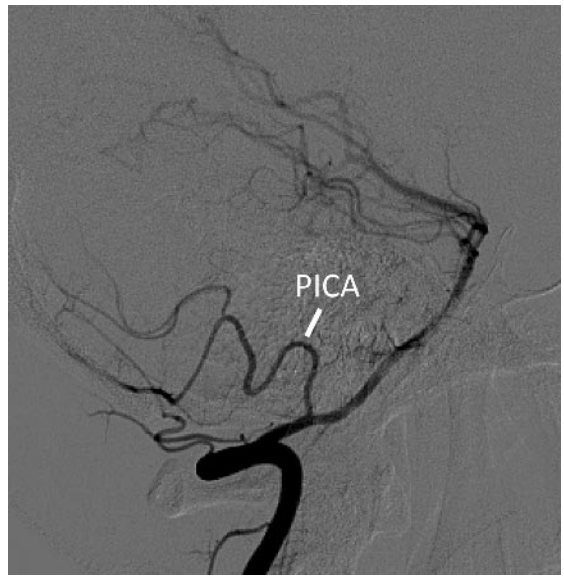

Fig 1.7 Digital subtraction angiography of the posterior inferior cerebellar artery (PICA), showing proximal medullary segments and hemispheric tributaries.

At later stages, this vessel continues to predominate, growing into the largest arterial offshoot. There are four segments of this vessel, including the anterior, lateral, posterior medullary, and supratonsillar PICA. The anterior medullary segment travels laterally near the inferior aspect of the olive of the medulla oblangata, continuing in a loop that courses between the cerebellum and medulla. Numerous perforating arteries extend from the first three segments of the PICA to supply anterior, lateral, and posterior aspects of the medulla. The PICA then extends posteriorly in the tonsillomedullary fissure adjacent to the glossopharyngeal (IX) and vagus (X) nerves. Beyond this point, the PICA curves over the cerebellar tonsil to become the supratonsillar segment extending further across the cerebellum as the medial and lateral terminal PICA branches. At the juncture of the posterior medullary and supratonsillar segments of the PICA, perforating vessels arise to feed the choroid plexus of the fourth ventricle. This choroidal point is used as a landmark to identify masses within the posterior fossa.

Variations in PICA anatomy include hypoplasia or absence of this branch (10–20% of individuals), typically accompanied by a prominent ipsilateral anterior inferior cerebellar artery. Absence of the PICA is also accompanied by numerous medullary perforators that arise directly from the vertebral artery. Duplication of the PICA and double origin with distal arterial convergence of the PICA[48] may occur. PICA–AICA connections and other anomalies may be seen, yet the specific frequencies of such anatomic configurations remain unclear. On occasion, both inferior cerebellar territories may be supplied by a bihemispheric PICA originating from one vertebral artery.[49]

Basilar artery

The basilar artery extends from the confluence of the vertebrals near the pontomedullary junction to the terminal bifurcation as the PCAs at the level of the midbrain. During embryologic development, paired vessels on the ventral surface of the hindbrain fuse to form the basilar artery. Distal segments of these paired basilar arteries have connections with the ipsilateral ICA. Over time, the plexus formed by the paired basilar arteries fuses with progressive disappearance of fenestrations. The basilar artery is often tortuous or serpentine, with a straight course noted about 25% of the time. Curvature of the basilar as it ascends has been associated with the presence of a wider contralateral vertebral artery, suggesting hemodynamic modeling. The length of the basilar artery is consistently 25–35 mm, irrespective of body size.[50] The diameter is about 2.7–4.3 mm at the proximal portion. The luminal diameter of the basilar artery tends to taper towards the distal end.

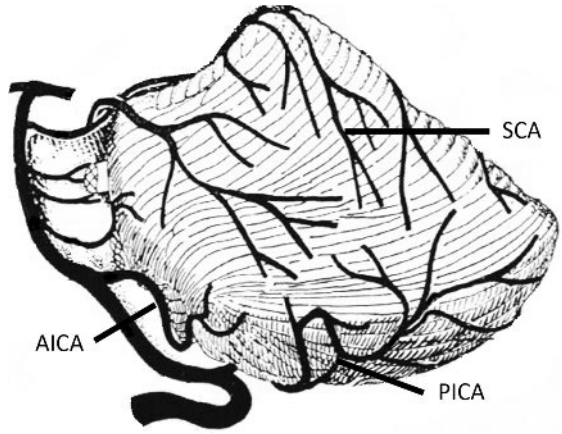

Fig 1.8 Schematic of posterior circulation, demonstrating cerebellar arteries including superior cerebellar artery (SPA), anterior inferior cerebellar artery (AICA), and posterior inferior cerebellar artery (PICA).

The largest branches of the basilar artery include the anterior inferior cerebellar artery (AICA) and superior cerebellar artery (SCA) (Fig. 1.8). Numerous smaller perforators embrace the brainstem, coursing from the midline ventral aspect around the surface to the lateral dorsal surface, and diving deep into the substance of the brainstem between fiber tracts. These pontine perforators are grouped into medial and lateral subdivisions, often referred to as paramedian and circumferential arteries. Lateral pontine perforators extend to also supply the ventrolateral surface of the cerebellum, whereas the medial perforators perfuse midline structures of the midbrain. There is a somewhat downward trajectory of brainstem perforating arteries so that the most rostral portion of the basilar artery supplies penetrators to the pontine tegmentum. The internal auditory or labyrinthine artery may arise from the basilar to provide arterial blood flow to the cochlea, labyrinth, and facial (VII) nerve.[51] Alternatively, this artery may arise as a branch of the AICA. Owing to the paired structure of posterior circulation arteries,

asymmetries or relative dominance of one artery such as the PICA, AICA, or SCA may occur. Contralateral cerebellar infarction may therefore result from occlusion or disease of one cerebellar artery. The frequencies of such patterns vary and are difficult to estimate, although the advent of MRI and MRA allows for systematic evaluation of such relatively subtle features. Terminal branches of the PICA, AICA, and SCA form anastomoses that allow for collateral flow to easily shift between their arterial territories.[1,52]

Anterior inferior cerebellar artery

The AICA extends off of the basilar artery approximately one-third to halfway through its course.[52] The AICA arises from the caudal third of the basilar 74% of the time.[52] This artery is the smallest of the principal cerebellar arteries. During development, the AICA appears as one of the larger perforatoring vessels extending to the posterior aspect of the hindbrain. The AICA extends laterally and inferiorly, in close proximity to the abducens (VI) nerve. Similar to the lateral pontine perforators, it courses around the brainstem and then enters the cerebellopontine angle cistern along with the facial (VII) and vestibulocochlear (VIII) nerves. The AICA crosses the anteroinferior aspect of the cerebellum to supply the middle cerebellar peduncle, flocculus, and adjacent cerebellum. The AICAs supply a rather small variable portion of the anterior inferior cerebellum.[53] The supply to the brachium pontis and flocculus is consistent. Numerous pontine perforators may arise from its proximal segment. The lateral branch runs across the cerebellum in the horizontal fissure. The medial branch of the AICA courses inferiorly to supply the biventral lobule. Similar to absence of the PICA, the AICA may be absent or hypoplastic and is typically accompanied by a prominent PICA. Variable infarct patterns may be noted in these regions of the posterior circulation, likely reflecting arterial configurations of the principal cerebellar arteries that may involve hypoplastic segments or anomalous anastomoses.[53,54] Combined or multiple territorial infarcts such as the PICA and AICA or AICA and SCA may reflect dominant patterns of arterial supply originating from the vertebral and basilar arteries. Similarly, collateral anastomoses between these territories may provide sufficient arterial inflow to spare distal segments of a particular arterial territory. As individual anatomy is often studied only after the onset of stroke, the original arterial configuration and mechanistic events may only be surmised. Similarly, the frequency of specific arterial supply patterns may be difficult to ascertain in the healthy population as individuals are most often studied after presentation with potential neurovascular disorders.

Superior cerebellar artery

The SCA also extends from the basilar in symmetric fashion, just proximal to the terminal bifurcation of the basilar into the proximal PCAs. SCA morphology is more consistent across individuals than other cerebellar branches.[55] The SCA courses laterally below the oculomotor (III) nerve, passing around the cerebral peduncles and below the trochlear (IV) nerve.[28] Numerous perforators extend from the proximal or ambient SCA to supply the adjacent pons and midbrain, whereas distal segments split into the lateral marginal and superior vermian branches. These divisions may also arise independently from the basilar artery or even the PCA. Duplication of the SCA is noted in 28% of individuals, with bilateral duplication in 10%.[56] The SCA variably divides into medial SCA and lateral SCA branches. The lateral marginal SCA supplies the anterosuperior cerebellum, superior cerebellar peduncle, middle cerebral peduncle, and dentate nuclei. The superior vermian SCA supplies the superior cerebellar peduncle, tentorium, inferior colliculi, cerebellar hemispheres, and dentate nuclei. Anastomoses between this branch and the inferior vermian branch of the PICA allow for robust collateral perfusion across the cerebellar hemispheres.[57]

Posterior cerebral artery

The PCA develops from fusion of several vessels that supply the mesencephalon, diencephalon, and choroid plexus in the fetus.[58] These vessels stem from the terminal aspect of the PCoA at the distal end of the carotid circulation. The PCA most often then extends posteriorly to spread over the ipsilateral cortex, whereas the proximal connection with the PCoA regresses. In this common scenario, the primary arterial supply shifts to a source from the terminal basilar artery. In the remainder of individuals, the PCA supply continues

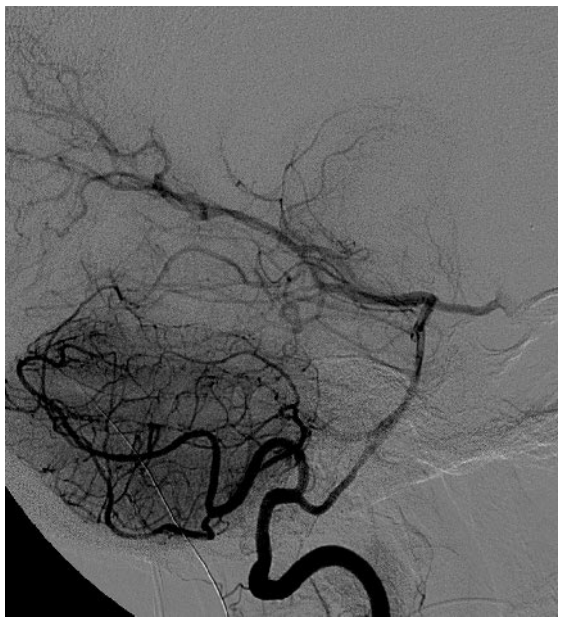

Fig 1.9 Digital subtraction angiography illustrating posterior cerebral artery anastomosis with posterior communicating artery, thalamoperforators, and distal cortical branches.

from what has been termed a fetal PCoA. Variants of PCoA anatomy include a diverse range of caliber in this segment, complete agenesis, and anomalous origins of other vessels from this arterial segment.[59]

The PCA extends from the terminal portion of the basilar artery in the interpeduncular cistern, passing above the oculomotor (III) nerve to circle the midbrain above the tentorium (Fig. 1.9). As it passes through the peduncular, ambient, and quadrigeminal cisterns, numerous perforators supply adjacent structures.[60] This pattern of arterial limbs includes paramedian perforators, short circumferential and long circumferential branches that typify the general structure of the major arterial territories in the posterior circulation. The perforating arteries from these segments range from 200 μm to 800 μm in diameter.[60] The artery of Davidoff and Schechter extends from the P1 segment to supply part of the inferior surface of the tentorium. The midbrain receives arterial blood from the peduncular or P1 segment before posterior thalamoperforators arise. In the successive ambient segment, the thalamogeniculate arteries diverge to supply the lateral geniculate and pulvinar nuclei. Medial and lateral branches of the posterior choroidal arteries extend from this portion of the PCA to supply the pineal gland, third ventricle, dorsomedial thalamus, pulvinar, lateral geniculate body, and choroid plexus.[61] These distal posterior choroidal arteries form anastomoses with the AChA.

These deep arterial territories composed of perforatoring arterioles that encompass the thalamus are often difficult to comprehend because of their complex configuration (Fig. 1.10).[62] The P1 or proximal PCA serves as an important arterial segment in this configuration, with variable contributions from the basilar artery, PCoA, and AChA.[63] These vessels have perforators that supply these critical structures at the juncture between the anterior and posterior circulations. The anterior thalamoperforating arteries consist of about 7–10 branches that arise from the superior and lateral surfaces of the PCoA.[63] A larger branch, the premamillary artery, is often noted.[64] This vessel courses from the posterior aspect of the PCoA, penetrates the hypothalamus and subsequently terminates in branches that supply the anterior and ventroanterior nuclei of the thalamus. Further posteriorly, the thalamus is

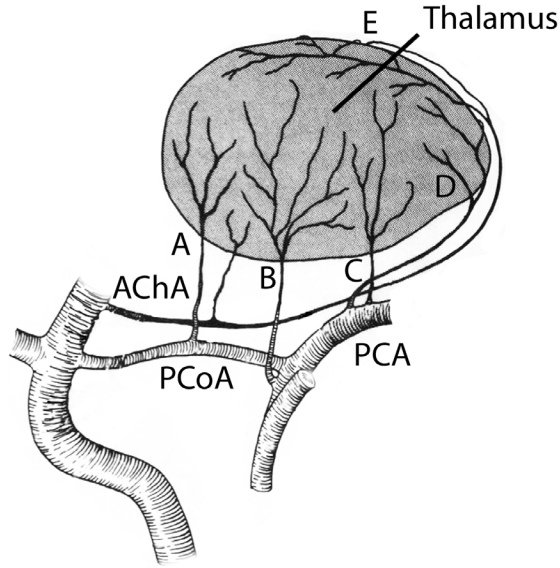

Fig 1.10 Schematic of the arterial supply to the thalamus, illustrating premamillary arteries (A), interpeduncular perforators (B), thalamogeniculate arteries (C), posterior choroidal arteries (PCoA) (D), and anterior choroidal artery (AChA) (E). PCA, posterior cerebral artery.