NEUROLOGICAL SURGERY

THIRD EDITION

VOL
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VOLUME THREE

NEUROLOGICAL SURGERY

A Comprehensive Reference Guide to the Diagnosis and Management of Neurosurgical Problems

THIRD EDITION

Edited by

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Diagnosis and Treatment of Head Injury in Adults

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chapter 47

Pathophysiology and clinical evaluation of ischemic vascular disease

J. F. Toole and D. D. Burrow

In the United States and other developed countries, cerebrovascular disease is the most important cause of chronic disability and the third most frequent cause of death in adults.

The term "cerebrovascular disease" denotes a pathological condition of the central nervous system attributable to a disturbance in its blood supply. Ischemic cerebrovascular disease excludes conditions in which hemorrhage is the primary event and, in common usage, is restricted to those conditions in which the ischemia results in a focal neurological deficit. Both focal and diffuse ischemic events of the cerebral hemispheres, brain stem, and cerebellum are discussed in this chapter. Ischemia of the spinal cord is not considered.

Definitions and Classification

Cerebral ischemic events are classified by their site, mechanism, and course.

SITE

Localization may, in many instances, be deduced from the clinical features. Given the anatomical site and its extent, an attempt must be made to place the lesion in the territory of a single artery, at the border zone or watershed of contiguous arteries, or in an area of venous occlusion.

A *single territory infarct* is the lesion most frequently observed in patients presenting with ischemic stroke.[147] Collateral flow can be expected to restrict the volume of the infarct to something less than the region normally perfused by the occluded vessel. Since embolic occlusion and anterograde extension of thrombus from a more proximal occlusion account for most cerebral infarcts, the continuing use of the term "cerebral thrombosis" as a synonym for single territory cerebral infarcts is inappropriate.[99]

If the perfusion pressure is simultaneously reduced in adjacent arteries, the tissue of the *border zone* between the arterial territories will be the site of maximal ischemic damage. Sudden and profound systemic hypotension is the most common cause of *border zone,* or *watershed, infarcts* of the brain.[147]

Diffuse hypoxic encephalopathy follows either circulatory arrest or widespread disturbances of the microvascular circulation.

Venous infarction is relatively uncommon but may develop when cortical veins and their smaller radicles are occluded.

VASCULAR MECHANISM

A single territory infarct may follow occlusion of a cerebral vessel by an embolus, by the extension of anterograde thrombus from a more proximal arterial occlusion, or by intrinsic disease of the cerebral vessel. The mech-

anisms of border zone and venous infarction and diffuse ischemic encephalopathies have already been commented upon. In Tables 47–1 and 47–2, ischemic cerebrovascular events are classified by their initiating mechanisms.

TIME COURSE

The recognition of the significance of transient ischemic attacks was an important advance in the field of cerebrovascular disease.[65,157]

Transient ischemic attacks are episodes of temporary and focal cerebral dysfunction of vascular origin commonly lasting from 2 to 15 minutes but occasionally lasting as long as 24 hours.[157] If the signs or symptoms persist for more than 24 hours but subsequently completely resolve in less than 3 weeks, the episode is designated a *reversible ischemic neurological deficit.*

When the neurological deficit has been progressive for 6 hours or more, cerebral hemorrhage, tumor, and subdural hematoma are diagnostic possibilities. Because of their urgent diagnostic and therapeutic considerations these conditions are designated as *stroke in evolution* or *progressing stroke.*

If the lesion is stable for 18 to 24 hours, further progression is unlikely, and it should no longer be categorized as a "progressing stroke." With vertebrobasilar lesions, however, up to 72 hours should elapse before they are designated as "completed stroke."

Table 47–1. *Causes of Focal Cerebral Ischemia Classified by Site of Primary Disease*

Site	Condition	Mechanism
Precardiac	Pulmonary consolidation	Emboli from pulmonary veins
	Deep venous thrombosis and atrial septal defect	Paradoxical embolism
	Clearing a Scribner shunt	Retrograde arterial embolism
Cardiac	Myocardial infarction	Emboli (arrhythmia, hypotension)
	Valvular heart disease (particularly mitral)	Emboli
	Cardiomyopathy	Emboli
	Arrhythmia (particularly chronic and paroxysmal atrial fibrillation)	Emboli
	Less common: hypertensive heart disease, infectious endocarditis, marantic endocarditis	Emboli (septic or sterile)
Great vessels	Aortic dissection	Extension (thrombosis, emboli)
	Aortitis: Takayasu's, syphilitic, giant cell, rheumatoid, ankylosing spondylitis	Emboli, branch occlusion
Carotid artery	Atherosclerotic plaque (stenotic or ulcerated)	Emboli
	Carotid occlusion	Anterograde thrombosis, emboli (hemodynamic)
	Less common: fibromuscular hyperplasia, kinking, arteritis	Emboli (hemodynamic)
Vertebral artery	Distal occlusion	Anterograde thrombus, emboli (frequently results in cerebral infarction)
	Proximal occlusion	Emboli (infrequent cause of cerebral infarction)
	Proximal subclavian occlusion	Subclavian steal syndrome
	Neck manipulation	Intimal injury at C2 level
Intracranial (arterial)	Atherosclerosis of major cerebral vessels (particularly vertebral and basilar arteries)	Occlusion
	Arteritis	Occlusion
	Subarachnoid hemorrhage	Spasm and vessel rupture
	Arteriolar sclerosis (hypertension)	Occlusion
	Transtentorial herniation	Compression of posterior cerebral arteries
Intracranial (venous)	Cortical vein thrombosis	Venous infarction
	Occlusion of deep venous system	Venous infarction
Uncertain site	Erythrocytosis (PCV greater than 55%)*	Thrombosis (arterial and venous)
	Thrombocytosis	In situ thrombosis, emboli
	Macroglobulinemia (with hyperviscosity)	Sludging
	Oral contraceptives	Thrombosis (arterial or venous)

*PCV = packed cell volume.

Table 47–2. *Causes of Diffuse Cerebral Ischemia*

Circulatory arrest
Cardiac arrest
Impaired circulation
Strangulation
Profound hypotension
Extensive extracranial vascular disease
Impaired energy substrate supply
Hypoxemic hypoxia
Severe anemia
Hypoglycemia
Carbon monoxide poisoning
Diffuse small-vessel disease
Hypertension
Eclampsia
Small-vessel arteritis, cerebral lupus, polyarteritis nodosa, noninfectious granulomatous angiitis
Thrombotic thrombocytopenic purpura
Microembolic disease
Fat embolism
Disseminated intravascular coagulation
Cardiopulmonary bypass procedures

Epidemiology of Ischemic Disease

The diversity of clinical manifestations and the difficulty of determining the pathological changes in and the mechanism of individual cases restrict the usefulness of epidemiological studies.[47] However, we may note that:

1. The incidence of cerebral infarction increases greatly with *age*. In the Mayo Clinic study of the population of Rochester, Minnesota, the incidence increased from 72 per 100,000 population per year among persons 45 to 54 years of age to 1786 per 100,000 population per year among those of more than 75 years of age.[153]

2. Most studies have shown the incidence of cerebral infarction to be a little less in women than in men of the same age.[153]

3. The presence of *hypertension* (even an elevation of the systolic pressure taken as a "casual reading") increases the risk of subsequent cerebral hemorrhage or infarction. This risk increases over the entire range of observed pressures, and there is no critical blood pressure above which this risk component commences.[118] The prognosis for an ischemic stroke is a little worse and the risk of reinfarction is also greater in the person with hypertension.[108,190,202]

4. Cardiac disease of all forms is associated with an increased risk of cerebral infarction (usually embolic). Atrial fibrillation increases the risk of cerebral infarction sixfold, and ischemic heart disease increases it fivefold.[65,117]

5. A history of recent transient cerebral ischemia markedly increases the probability of cerebral infarction. Whisnant and co-workers observed 16.5 times the expected incidence of stroke in the first year after a transient ischemic attack.[31,233]

6. Increased serum cholesterol and possibly triglyceride levels are associated with an increased risk in men before the age of 55 years.[233]

7. In younger persons diabetes mellitus is a risk factor, particularly for discrete small brain stem infarcts.[117]

8. Obesity, cigarette smoking, and decreased physical activity have been suggested as risk factors.[124,166]

9. Retinopathy, cervical bruit, brachial blood pressure differences, and evidence of peripheral vascular disease are indicators of underlying arterial disease that is related to mechanisms of stroke.

10. The evidence that there are major differences in the incidence of cerebral infarction among geographical boundaries and racial groups is clear.[130] The relative frequency of cerebral hemorrhage, compared with cerebral infarction, is greater in Japan and China.[131]

11. Comparison of the results of observations made upon groups of patients with recent brain infarcts with those of grouped control persons indicate that the viscosity and rheological characteristics of blood, particularly at low flow rates, may influence the size and frequency of cerebral infarcts.[123,173,197] Circulating platelet aggregates and hypercoagulability are found with greater frequency in stroke patients.[239]

Pathophysiology of Cerebral Ischemia

CELLULAR EVENTS

When the brain is rendered ischemic, electrical activity disappears within 10 to 20 seconds; the sodium-potassium pump fails within 30 seconds; and glucose levels fall rapidly. Water passively follows the influx of sodium, causing intracellular edema within 3 minutes of the initiation of ischemia. In 5 to 10 minutes intracellular lactate levels have risen fivefold and cellular glucose is exhausted.[145,230] To this point, however, all changes are reversible.

The persistence of ischemia causes progressive and ultimately irreversible failure of the cellular organelles. Mitochondrial injury has the most direct influence upon the survival of the cell. For many tissues (except for nerve cells), ultrastructural changes have been followed through varying durations of ischemia. The consensus is that the appearance of clumped electron-dense bodies within the mitochondria is a reliable "marker" of cell death.[226] For tissues other than those of the nervous system, 6 to 24 hours of complete ischemia may elapse before cell death occurs. It is interesting to note that the ultramicroscopic criteria of cell death are not observed in the cat brain even after 5 hours of *total* ischemia but will rapidly appear upon reestablishment of circulation.[67] This important phenomenon has its counterpart in myocardial ischemia.[145,226]

What explanations can be offered for this observation? The reperfusion of tissues with their accumulated energy deficit, nonfunctional membrane pumps, and increased cellular permeability allows the cell to be flooded with ions that are normally excluded (e.g., calcium and sodium ions). The calcium ion, by activating phospholipase enzymes, may initiate further injury to the mitochondrial and cellular membranes.

CONSEQUENCES OF ARTERIAL OCCLUSION

Arterial occlusion lowers the perfusion pressure in the distal vasculature, thus summoning the available collateral flow through channels that rapidly become maximally dilated by the metabolic effects of ischemia.[212] If the flow into the occluded vasculature is sufficient, the patient is spared ischemic injury. Studies indicate that the achieved flow is often close to this "sufficient" or "critical" flow; therefore, clipping the middle cerebral artery of the dog, for example, does not cause cerebral infarction unless the dog's blood pressure is simultaneously reduced. It should be noted that flow through the dysautoregulated collateral vasculature is proportional to the perfusion pressure, i.e., the difference between mean arterial and tissue pressures. Cerebral arteriosclerosis, insufficient to initiate an ischemic event, may play a critical role in determining the outcome by limiting the collateral flow.

The low flow rates distal to occluded cerebral vessels increase the effective viscosity of blood, particularly in the higher "physiological" hematocrit range. This complex interrelationship of shear rate, relative viscosity, and hematocrit is graphically presented in Figure 47–1.[238] As the shear rate is closely related to the blood velocity, the dramatic effect of higher hematocrit values in limiting flow rate and oxygen transport is apparent. The hematocrit that will provide optimal oxygen transport in such pathological vascular beds is often in the range 30 to 40 per cent.[102] This theoretical consideration is reflected as increased infarct size with increased hematocrit.[93]

A cerebral infarct is a rapidly expanding lesion that reaches a maximum volume between the second and fourth days. Swelling is life-threatening in 20 per cent of patients with large cerebral infarcts and 50 per cent of patients with large cerebellar infarcts.[5,180]

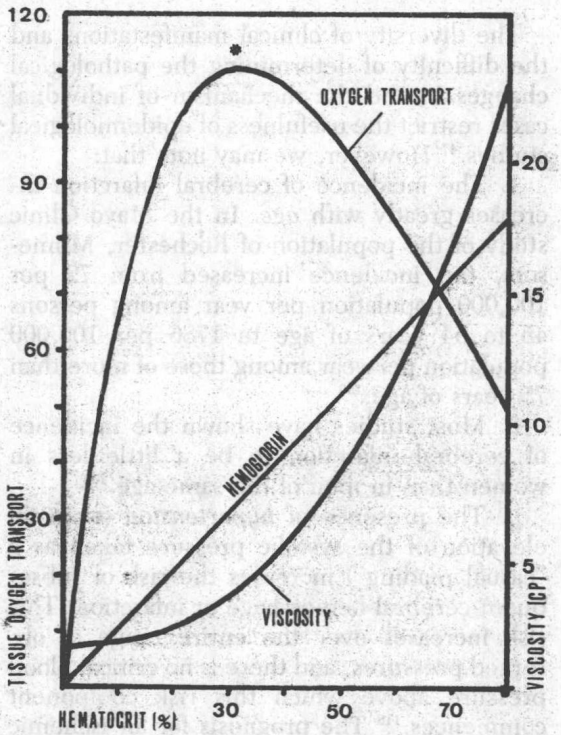

Figure 47–1. Hemorrheology witin the slow-flow vasculature in the vicinity of a cerebral infarct. In this composite graph, the viscosity curve shows a steep increase with increasing hematocrit. The curves marked "Hemoglobin" and "Oxygen Transport" indicate that although the oxygen-carrying capacity rises with hematocrit, the oxygen transport reaches an optimum near 30 per cent as a result of the reduced blood flow.

Ischemic cerebral swelling is a complex reaction. Vascular dilatation within the zone of ischemia makes a minor contribution to the swelling. Intracellular edema commences within minutes and progresses rapidly, taking water from the extracellular space, which in turn is replenished from the vascular compartment. Perfusion of the ischemic brain causes vasogenic or extracellular edema, particularly when the subsequent breakdown of the blood-brain barrier allows the release of osmotically active substances into the extravascular space. Ischemic cerebral edema is, however, usually maximal before the blood-brain barrier has been significantly disrupted.[169] The early and clinically most significant edema is largely metabolic or cytotoxic in type and intracellular in site and results directly from ischemic injury of the sodium-potassium cell membrane pump.[170]

Ischemic brain swelling may further compromise the perfusion of ischemic brain by increasing both the local tissue hydrostatic pressure and the overall pressure of its intracranial compartment.[125]

INFLUENCE OF ANASTOMOSES

Effective arterial anastomoses may form at three levels in the vasculature of the brain: extracranially, in the circle of Willis, or in the leptomeninges.

The most common extracranial anastomotic channels are those between the external and the internal carotid arteries. When the internal carotid artery is obstructed below the origin of the ophthalmic artery, rich collaterals develop from the maxillary branch of the external carotid to the ophthalmic artery, through which blood flows retrograde to the distal internal carotid artery and thence to the brain.[73] In certain situations, the ophthalmic artery may carry most of the blood to a hemisphere whose internal carotid artery is occluded. Other anastomoses may develop through the caroticotympanic artery, the artery of the pterygoid canal, or the trigeminal artery.

Congenital variations in the size of the vessels of the circle of Willis are frequently encountered. In more than 50 per cent of normal persons the circle of Willis has one or more hypoplastic vessels, most commonly the posterior communicating arteries.[213] These anomalies appear to influence the location of cerebral infarctions.[14]

Important anastomotic channels exist between the major cerebral arteries. Because these occur over the surface of the brain, they are commonly described as the leptomeningeal anastomoses. Their potential capacity is less than that of the anastomoses within the circle of Willis, and they are seldom able to protect the brain from infarction when a major cerebral artery is occluded.[227]

Within the substance of the brain there are few significant anastomoses. While there are unquestionably capillary and supracapillary anastomoses throughout the brain, the caliber of these is such that they are of unknown importance. Therefore, the penetrating arteries can be considered end-arteries. In certain cases of cerebral infarction, the parenchymal microvascular anastomoses may appear as a "tumor-like" angiographic blush.

MECHANISMS OF ARTERIAL OCCLUSION

Embolism

Embolism is the most frequent mechanism of brain infarction.[147] Lhermitte and co-workers concluded that two thirds of middle cerebral artery infarcts were embolic.[141] The heart is the most frequent source and may account for half of the infarcts within the anterior circulation (Table 47–3).[19] Careful postmortem examination of the brain revealed that 54 per cent of 340 persons dying of heart disease had cerebral infarcts compared with 12 per cent of 100 patients free of heart disease.[229]

Table 47–3. *Frequency of Cardiac Lesions Causing Cerebral Embolization**

Cardiac Lesion	Per Cent
Myocardial infarction (recent and old)	50
Rheumatic heart disease	26
Congestive cardiomyopathy (and myocardial disease of undetermined causation)	15
Infective endocarditis	7
Thrombotic endocarditis	2
Atrial myxoma	<1

*Data from: Blackwood, W., et al.: Atheromatous disease of the carotid arterial system and embolism from the heart in cerebral infarction: a morbid anatomical study. Brain, *92*:897–910, 1969. McCall, A. J., and Fletcher, P. J. H.: *In* Hutchinson, E. C., and Acheson, E. J., eds.: Strokes: Natural History, Pathology and Surgical Treatment. Philadelphia, W. B. Saunders Co., 1975.

Ischemic heart disease is an important cardiac source of cerebral emboli. The risk of embolism is maximal during the first 14 days after myocardial infarction, but persists indefinitely, particularly after large anterior myocardial infarcts. Atrial fibrillation has become the most common source of cerebral emboli.[66,237] Mitral valve prolapse, a common disorder with a low absolute risk of cerebral embolism, is an important cause of stroke in the young. It is a disorder with many causes, and the risk of embolism is notably greater when prolapse is due to significant underlying heart disease associated with significant regurgitation or regional myocardial or papillary muscle dysfunction.[23,26] Patients with valvular heart disease, particularly mitral stenosis, are at risk for cerebral embolism, and this risk is substantially increased by association with arrhythmia or heart failure. The highest absolute risks of cerebral embolism are associated with bacterial endocarditis.[115]

A less frequent cause is cardiac myxoma, an uncommon benign tumor, usually in the left atrium, that may present at any age with obstruction of the left ventricular inflow tract, peripheral embolism, and constitutional symptoms.[241]

Paradoxical emboli usually enter the systemic circulation through a foramen ovale guarded only by a left atrial flap. The flap is opened by the transient increase in the right atrial pressure that occurs with pulmonary embolism.[218] Embolic occlusion of only 30 per cent of the pulmonary vascular bed is sufficient to produce a reversal of atrial pressures, and 20 per cent of the population have the required atrial septal "defect."[224] As one patient in every five with major pulmonary embolism is at risk, it is not surprising that 4 per cent of all cerebral infarcts may be due to paradoxical embolism.[147]

Pulmonary consolidations may initiate phlebothrombosis within the pulmonary veins and generate either septic or sterile emboli.

Emboli of sufficient size to be arrested within a major cerebral artery enter the anterior circulation 10 times as frequently as they enter the posterior and then commonly proceed into the middle cerebral artery. Embolic arrest occurs at bifurcations or major branches because it is at these sites that the arterial cross section takes a stepwise reduction. The balance between intravascular fibrinolysis and thrombosis will determine the fate of the impacted embolus. That fibrinolysis ultimately clears the occluded vessel is suggested by the observation that 80 to 90 per cent of cerebral infarcts are associated with radiologically demonstrable occlusion of the perfusing vessel if studied less than 12 hours after the event, but only 20 per cent demonstrate occlusion when studied at a delayed interval.[200] The consequence of this delayed perfusion is often accelerated ischemic swelling and hemorrhagic infarct with significant edema.[57,95,170] Thrombosis of the stagnant vessel distal to the occlusion may be initiated by products from the embolus. This process will occlude the distal vasculature, further reduce the retrograde collateral flow, and increase the size of the cerebral infarct.

The nature of the embolus will influence this balance. Mature thrombus, particularly if partially organized, is more slowly and incompletely cleared than recent thrombus. Aggregations of platelets are loosely bound by fibrin and therefore may be rapidly and completely cleared from sites of impaction. Such emboli are the most frequent cause of transient ischemic attacks. Many other materials may form occasional emboli: cholesterol, debris from an ulcerated atheroma, calcium from cardiac valves, fat, air, dislodged arterial catheters, gun shot, and intravenous cannulae.

It is the opinion of the authors that many cerebral infarcts not associated with demonstrable arterial occlusion are examples of cerebral embolism in which there has been subsequent lysis of the embolus.

Microemboli are distributed throughout the brain with a frequency of impaction proportional to the regional blood flow; however, the pathological consequences are greatest in deep cerebral white matter because of the paucity of parenchymal collaterals within this tissue.

Anterograde Extension of Thrombus

Vessel occlusion may be followed by thrombosis and extension of this thrombus distally.[147] Extension becomes significant when it continues past branches bringing significant collateral flow. When the internal carotid artery is occluded at its origin, it is expected that anterograde thrombosis will extend through the unbranched section to a point just short of the ophthalmic artery. In 50 per cent of cerebral infarctions that follow carotid occlusion, thrombus extends beyond the ophthalmic artery, beyond the circle of Willis,

and into the middle or anterior cerebral arteries or both.[32,224] Similarly, thrombus may extend from the distal vertebral into the basilar artery.[33]

Hemodynamic Infarction

The frequency of asymptomatic carotid and vertebral artery occlusion suggests that the hemodynamic consequences of even complete occlusion of vessels proximal to the circle of Willis are usually insufficient to cause cerebral infarction unless complicated by embolism or anterograde thrombosis.[32,33] When cerebral infarction develops as a result of reduction of cerebral perfusion pressure, it occurs in the border zone between the areas of supply of major cerebral vessels. Such infarcts may follow profound systemic hypotension or occlusion of an internal carotid artery when important collateral flow is restricted by congenital deficiencies of the circle of Willis.

Intrinsic Disease of Cerebral Vessels

Severe atherosclerotic stenosis of intracranial vessels is surprisingly infrequent in white persons, but may be encountered in the distal portions of the vertebral and internal carotid arteries and in the basilar artery. Battacharji and co-workers found that only 4 per cent of middle cerebral artery occlusions could be attributed to intrinsic disease.[14] In contrast, the majority of occlusions in penetrating vessels of the hemispheres, pons, and cerebellum are the result of hypertension-related arteriolar sclerosis.

Nonatheromatous arterial pathological conditions are considered later in this chapter.

Clinical Evaluation

HISTORY

The clinical interview is conducted to gain adequate historical data to define the neurological symptoms and time course. At a clinical level, the opinion that a patient has ischemic cerebrovascular disease is based almost exclusively upon the temporal profile of the onset and the evolution of symptoms. When the patient is aphasic or comatose, even a fragmentary history of the ictus from a direct witness may be of critical importance and be only as far away as the telephone.

The patient should be asked to begin his or her story with the first episode rather than with more recent events. This orientation will often cause patients to recount symptoms they considered inconsequential. If multiple episodes have occurred, a description of the first, the most recent, the worst, and any episodes in which the symptoms were substantially different should be reviewed and noted. In each, the symptoms must be adequately reviewed to allow conclusions about the site of ischemia, the time course of events, and whether precipitating factors were operative.

Although atherosclerosis, ischemic heart disease, and hypertension are the most frequent causes of ischemic cerebrovascular disease, the patient's entire medical and neurological history must be utilized to exclude the many "nonatheromatous" initiating disorders and to assess the coincidental disease.

NEUROVASCULAR EXAMINATION

The neurovascular examination assesses: (1) cardiac rhythm, (2) pulses of arteries supplying the cerebral circulation, (3) auscultation of the heart and the great vessels, (4) arterial blood pressure recordings, (5) tests of specific precipitants of cerebral ischemia (posture-related hypotension, carotid sinus massage, Valsalva maneuver, head posturing), (6) retinal vasculature, and (7) peripheral vasculature. These techniques are briefly considered but have been more fully described elsewhere.[223]

Cardiac Rhythm

The opportunity to assess the rhythm occurs during the bimanual radial pulse examination. Dysrhythmia demands electrocardiographic evaluation, and if a paroxysmal rhythm disorder is suspected, prolonged electrocardiographic monitoring is required.[36]

Pulses

As the technique for palpating the internal carotid artery with the finger introduced into the oropharynx is difficult and has not gained wide acceptance, the internal carotid and vertebral arteries must be considered inaccessible to any direct examination. The remaining cranial or cervical arteries are of much less value to the examiner; however, a bilateral simultaneous examination of the radial arteries should be performed to seek evidence of sub-

clavian artery disease. In addition, branches of the external carotid artery (the superficial temporal and facial arteries) may be palpated to assess the common and external carotid arteries.

A frequent source of confusion is the prominent, redundant common carotid artery low in the neck. This finding typically results from elevation and unfolding of the aortic arch and may be mistaken for an aneurysmal dilatation of the vessel.

Auscultation

Auscultation is used to check sites in the extracranial vasculature with a propensity for atheroma. The sequence of examination is illustrated in Figure 47–2. Because of the many bruits noted in the carotid vessels that are conducted to this site from the heart or great vessels, examination begins at the heart and follows the aortic outflow. Proximal internal carotid or bifurcation bruits are best heard over the carotid artery at the level of the upper border of the thyroid cartilage. An orbital bruit suggests either arterial disease or increased flow of the ipsilateral carotid siphon.

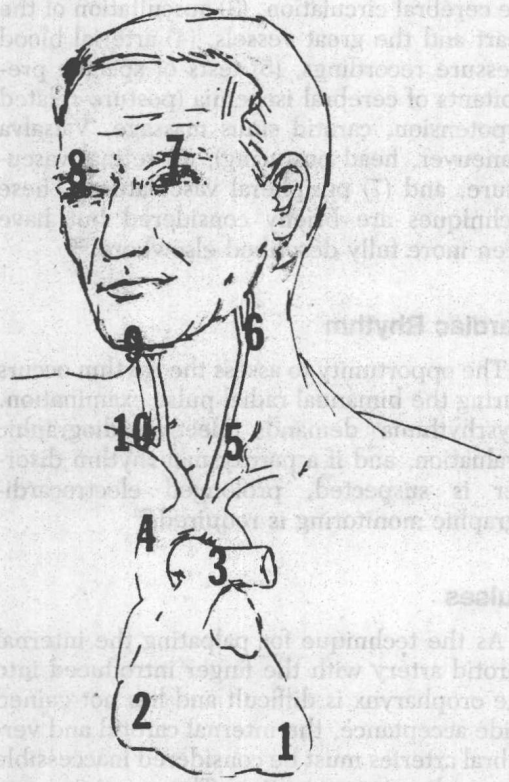

Figure 47–2. A logical sequence for auscultation of the heart, the great vessels in the neck, and the orbits. These are the sites where murmurs are heard most frequently.

The significance of arterial murmurs can be summarized by the following general rules: (1) murmurs become audible after the lumen has been reduced about 50 per cent; (2) with increasing stenosis the pitch becomes higher; (3) with increasing stenosis the intensity increases until the lumen is reduced by at least two thirds, at which point the intensity of the murmur decreases until it finally becomes inaudible when occlusion is complete; and (4) as the degree of stenosis increases, the duration of murmurs becomes more prolonged. Murmurs that are continuous into diastole suggest at least 90 per cent reduction in the cross-sectional diameter of the lumen of the vessel. Therefore, the following general points can be made: (1) murmurs of low pitch and of high intensity or volume are generally associated with large orifices through which a large volume of blood is flowing; (2) murmurs of low intensity, of high pitch, and of long duration suggest blood flow high in velocity through a small orifice; and (3) the intensity of the murmur does not correlate well with the severity of the disease that causes it. Consequently, the tightest stenoses are generally accompanied by a high-pitched soft murmur that is accentuated during systole but that continues into diastole.[111]

The finding of a bruit in a patient over 40 years of age indicates arterial disease in three out of four instances; such a bruit would, however, be far less significant if found in a child or young adult. It has been reported that 90 per cent of children under the age of 5 years have bruits and that as many as 30 to 40 per cent of healthy young adults have neck bruits.[88]

Blood Pressure Recording

The level of arterial blood pressure is of profound importance to an assessment of the patient with cerebrovascular disease. A valuable screening device for the subclavian steal syndrome is the recording of blood pressure in both arms. This recording and examination for postural hypotension should be routine in patients with syncope, dizziness, and vertebrobasilar symptoms. A difference of 20 mm Hg between the systolic pressures recorded in the right and left arms is significant. The