10 THE CEREBRAL VASCULAR SYSTEM

CHAPTER OVERVIEW

Cerebrovascular disorders represent one of the major causes of morbidity and mortality in adult populations by directly impacting the functional integrity of the CNS. This chapter will focus on three major aspects of the cerebrovascular system: **anatomy**, **pathology**, and **neurobehavioral syndromes**. As one cannot fully discuss the functional anatomy of the central nervous system without reviewing the vascular system that supplies it, the first priority will be to review the sources and patterns of distribution of the arterial blood supply to the brain. While it is not imperative for most clinicians to be able to name all the vessels, it is important to at least come away with a mental map of the spheres of influence for each of the major arterial groups. As the venous system was covered

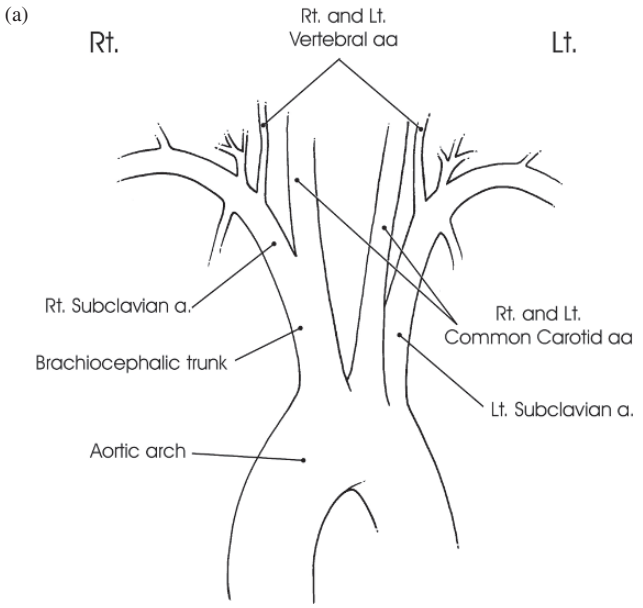
in the preceding chapter in conjunction with the discussion of the meninges, it will not be discussed in the same detail here.

Being under substantially greater pressure than venous blood and not having the benefit of the "filtering" action of the capillaries, the arterial system is prone to a wider range of pathology compared to the venous system. Also being responsible for the constant nourishment and oxygenation of the brain, any disruption of the system will have almost immediate and potentially catastrophic consequences. It is important for the clinician to have a general appreciation of the more common pathological conditions that can affect the cerebral arteries, including their premorbid risk factors, typical clinical presentation, effects on nervous tissue, and expected course over time. To this end, the major types of ischemic deficits, hemorrhagic events, and structural anomalies associated with cerebrovascular disease will be discussed.

Although there is obviously a significant interaction between the nature and severity of the specific pathological condition and the particular arterial system involved (anatomical locus or vascular distribution), the latter is clearly a major determinant of the nature of the neurobehavioral deficits or changes that will likely be manifested. As suggested earlier in this text, with the advances in neuroimaging over the past quarter century, the physical localization of lesions, including vascular, has become much less of a challenge. But as clinical neuroscientists our interest transcends the physical localization of the lesion. We want to understand the potential or probable behavioral consequences of such lesions in order to most effectively manage and care for our patients. Thus, the latter part of this chapter will be devoted to a review of the signs and symptoms or neurobehavioral syndromes commonly associated with lesions of the major cerebral arterial systems.

ANATOMY OF THE ARTERIAL SYSTEMS OF THE BRAIN

There are four ascending arteries that contribute to the cerebral circulation; two **carotid arteries** and two **vertebral arteries**. Although there is occasionally some variation in these vessels, Figure 10–1 represents the most common arrangement. The **left common carotid**



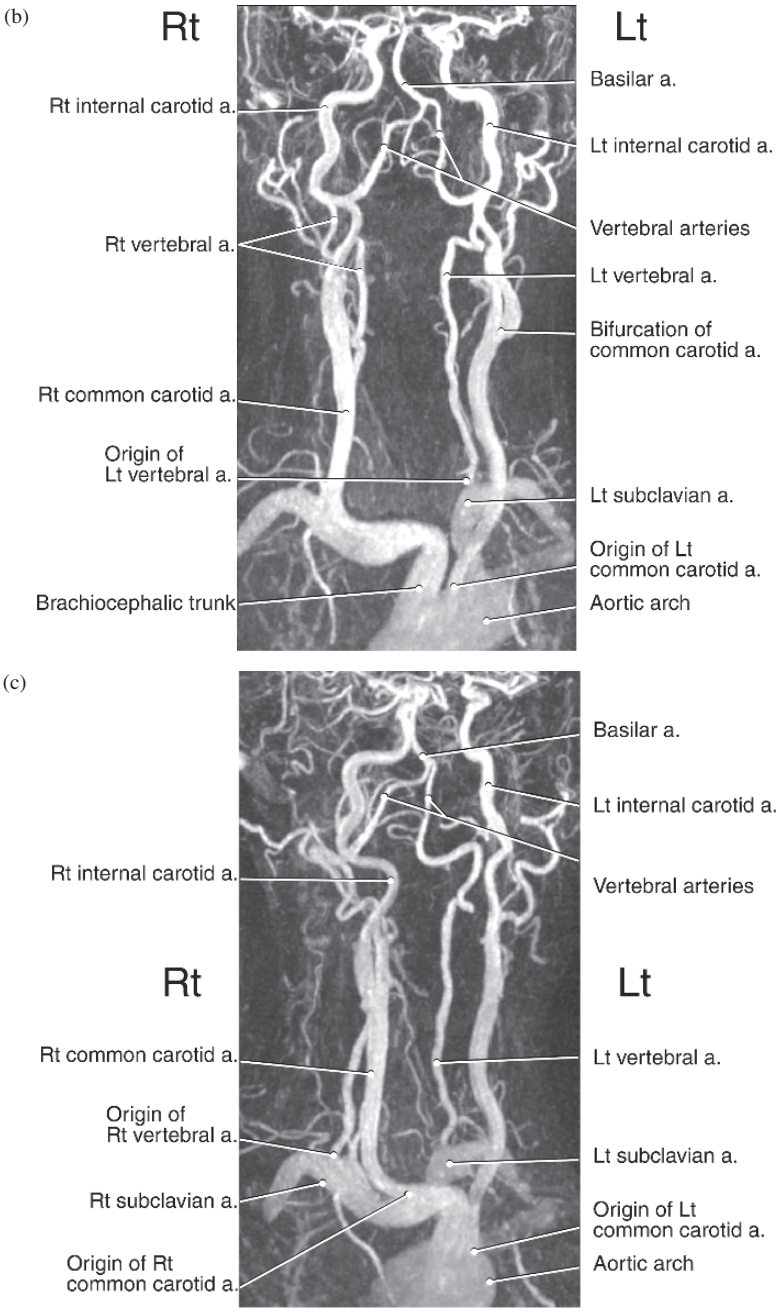


Figure 10–1. (a) Carotid and vertebral arteries coming off the aortic arch and secondary vessels. (b,c) MRAs of carotid and vertebral arteries, showing slightly rotated views from same individual.

artery emanates directly from the **aortic arch**, while the **right common carotid** derives from the **brachiocephalic** artery coming off the aortic arch. Slightly distal to the branching of the right common carotid artery, the brachiocephalic artery also gives rise to the **right vertebral** artery. The third major artery to branch off the aortic arch is the left subclavian artery. The **left vertebral** artery branches off the left subclavian shortly after it exits from the aortic arch.

VERTEBRAL SYSTEM

The vertebral arteries proceed in their cephalic course along the ventral surface of the cervical spine and actually becoming encased in its bony processes (the *transverse foramen*). After entering the foramen magnum, the vertebral arteries are found to lie adjacent to the ventral surface of the medulla in the brainstem. These two vertebral arteries then join to form the singular **basilar** artery at the level of the pontine–medullary junction (Figures 10–2 and 10–3). The basilar artery itself eventually will bifurcate just above the pons (at the level of the midbrain), giving rise to the two **posterior cerebral** arteries (PCA). As can be seen in Figures 10–3 and 10–4, there are several prominent cortical branches of the PCA: the **parietooccipital** artery, the **calcarine** artery (which supplies Brodmann's area 17, the primary visual cortex), and the **anterior** and **posterior** (not shown) **temporal branches** of the PCA (supplying the ventral and medial surfaces of the temporal lobes, including parts of the hippocampus).

In addition to the posterior cerebral arteries, the vertebral and basilar arteries give rise to multiple branches throughout their course along the brainstem. These branches, as seen in Figure 10–3, represent the major source of blood supply both to the brainstem and the

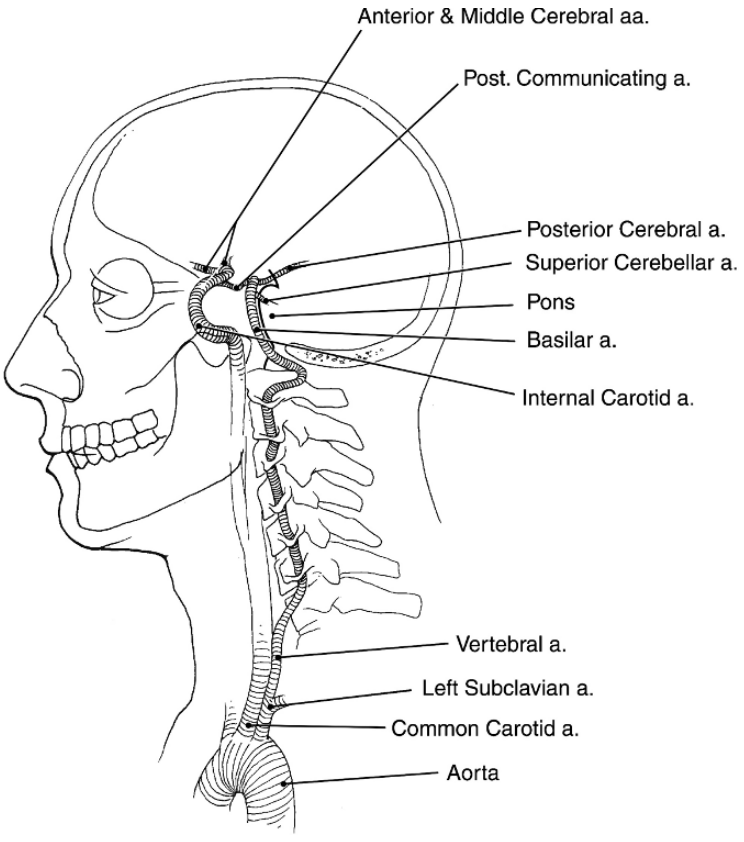


Figure 10–2. Lateral view of the vertebral–basilar system showing its relationship to the internal carotid artery. Figure also illustrates the distal portion of the vertebral artery passing through the transverse processes of the cervical vertebrae before entering the foramen magnum and forming the basilar artery.

cerebellum, as well as to the cervical portion of the spinal cord.[1] The three main branches of the vertebral arteries are (1) the **anterior spinal** artery, (2) the **posterior spinal** arteries, and (3) the **posterior inferior cerebellar** arteries. After the vertebral arteries converge to form the basilar artery, the latter gives rise to two other major vessels supplying the cerebellum and brainstem: the **anterior inferior cerebellar** and the **superior cerebellar arteries**. The anterior inferior cerebellar artery supplies the anterior and inferior portions of the cerebellum and the caudal pons. The superior cerebellar artery supplies the superior aspect of the cerebellum, the rostral pons, and portions of the midbrain. Other smaller arteries (pontine branches) emanate from the basilar artery and serve to nourish brainstem structures, particularly the pons. The areas supplied by the posterior cerebal artery will be discussed below.

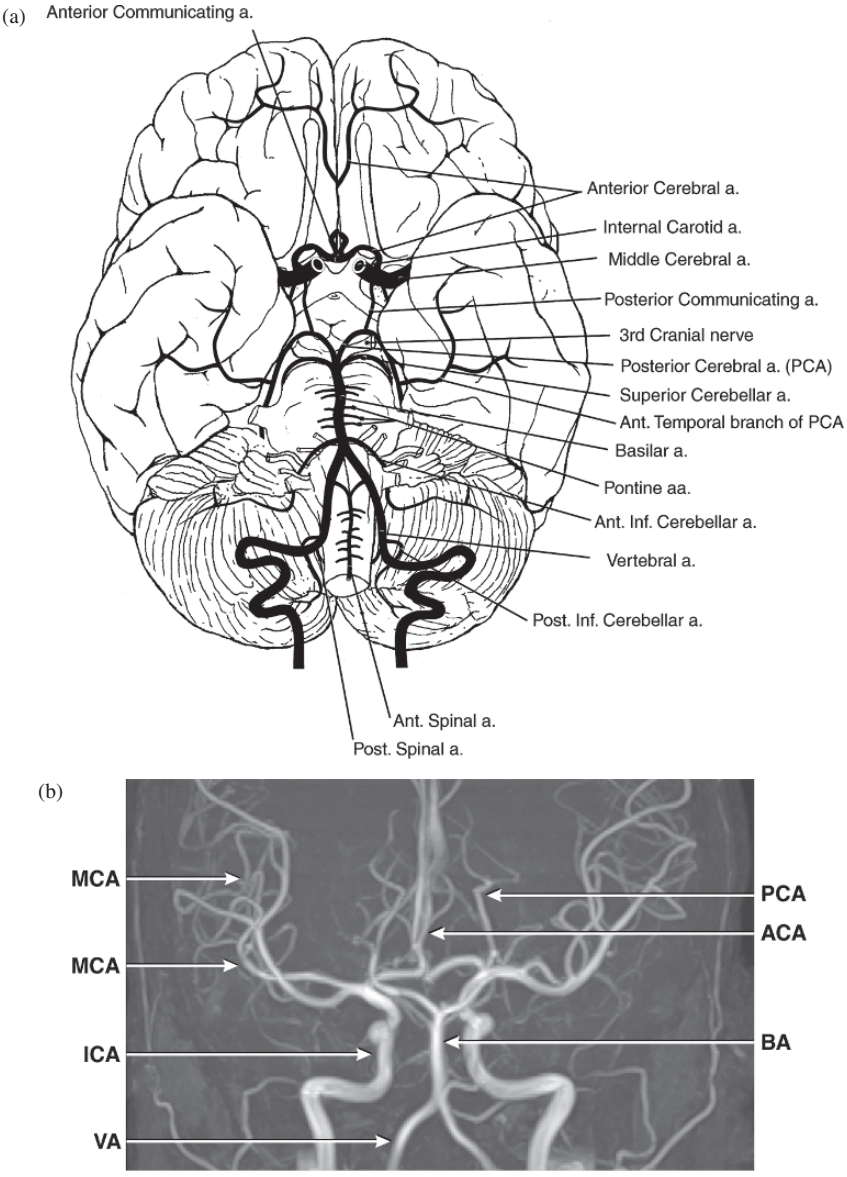


Figure 10–3. (*Continued*)

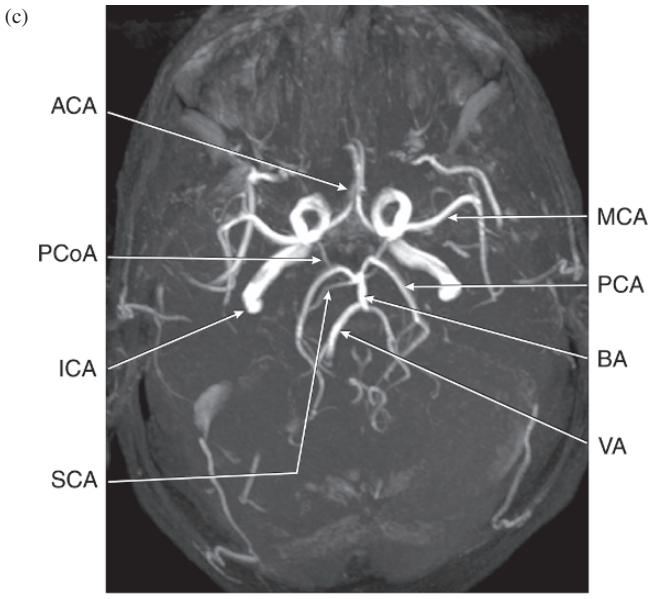


Figure 10–3. (a) Ventral view of vertebral–basilar system and its relationship to the brainstem and carotid circulation. Also illustrated is the "circle of Willis" creating anastomoses between the posterior and carotid circulations. MRA (magnetic resonance angiograms) showing same vessels from (b) A-P and (c) axial perspectives. Abbreviations: ACA, anterior cerebral artery; BA, basilar artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PCoA, posterior communicating artery; SCA, superior cerebellar artery; VA, vertebral artery.

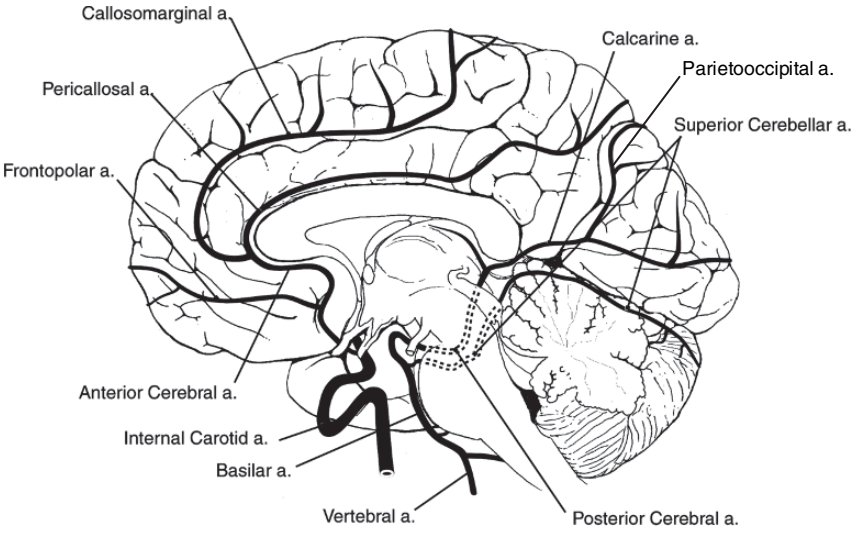


Figure 10–4. Midsagittal view of the cerebral hemisphere showing the general distribution of the main branches of the anterior and posterior cerebral arteries.

CAROTID SYSTEM

The common carotid arteries also proceed in a cephalic direction in a more anterior or ventral position in the neck. These are the arteries one feels as the fingers are placed on either side of the larynx. At approximately the level of the fourth cervical vertebra, the common carotids bifurcate, producing the **internal** and **external carotid** arteries. The latter go on to supply the extracranial tissues of the face and the scalp and most of the dura. The internal carotid arteries continue into the cranial vault where they eventually give rise to the four remaining cerebral vessels, the right and left **anterior** (ACA) and **middle cerebral** (MCA) arteries.

Just prior to entering the circle of Willis (see below) where the internal carotid and vertebral systems anastomose via "communicating" or connecting vessels, the internal carotids give rise to two major vessels, the **ophthalmic** artery and the **anterior choroidal** artery. In addition to supplying other structures in and around the eye and the anterior portion of the dura, one branch of the ophthalmic artery enters the eye along with the optic nerve and supplies the retina. The anterior choroidal artery will be reviewed in greater detail later. As will be seen, as its name implies, it is important in supplying the choroid plexus (lateral ventricles), but also supplies parts of the visual and motor systems and the temporal poles.

Circle of Willis

The internal carotid and vertebral vascular systems interconnect at the base of the brain, anterior to the brainstem and surrounding the optic chiasm (Figure 10–3). This interconnection or anastomosis is known as the ***circle of Willis*** after the 17th century anatomist (Thomas Willis, 1621–1675). The circle of Willis provides a potential diversion for collateral blood supply following the occlusion of one the major cranial arteries feeding into it. However, as we shall see, this potential collateral system is not uniform across individuals and can be influenced by several factors. This "circle" is completed by the presence of "communicating" arteries that connect the right and left internal carotids with the vertebral circulation. Just rostral to the third cranial nerves and slightly posterior to the mammillary bodies, the basilar artery bifurcates, forming the right and left posterior cerebral arteries (PCA). Shortly after their formation, each posterior artery sends off an anterior branch that connects to the ipsilateral internal carotids. These connecting vessels are the **posterior communicating** arteries. The internal carotids in turn divide into the middle and anterior cerebral arteries. The middle cerebral artery (MCA), which basically is the primary extension of the internal carotid, proceeds dorsal-laterally up through the lateral fissure between the temporal and frontal cortices. The anterior cerebral artery (ACA) initially remains more medial as it proceeds anteriorly toward the frontal pole. Just anterior to the optic chiasm, a small branching artery—the anterior communicating artery—connects the two anterior cerebral arteries, thus completing the circle.

The above description of the circle of Willis again represents the more typical pattern, but some individual variations may be noted. Despite the presence of a completed "circle" in the majority of individuals, there may not be much blood flowing around the circle, that is, there is little "communication" between the right and left internal carotids via these communicating arteries. This relative lack of flow around the circle of Willis seems primarily to be a function of the fact that:

1. The communicating arteries themselves are often relatively small.
2. There normally is relatively equal hemodynamic pressure from one arterial system to the other, thus not encouraging flow between the systems.

However, if over time one of the major feeder arteries (eg., one of the internal carotids) becomes gradually stenosed, a pressure gradient develops that encourages the shunting of blood from one side to the other. As a result of this shunting of blood through the posterior and/or anterior communicating arteries, the communicating arteries gradually enlarge, creating a larger lumen, thus facilitating more shunting of blood. Therefore, it is not unheard of to find a fairly complete thrombotic occlusion (see below) of one of the internal carotid arteries with little if any clinical manifestations of compromise of cerebral blood flow. However, if such an occlusion were to take place more acutely, for example, as a result of an embolus, the communicating vessels would not have time to adapt and a major stroke is likely to ensue.

Anterior Cerebral Artery

As noted above, the anterior cerebral artery (ACA) originates at the bifurcation of the internal carotid into the middle and anterior cerebral arteries. The ACA, typically smaller than the middle cerebral artery (MCA), proceeds rostrally (frontally) along the base of the frontal lobe. Shortly after their point of origin, the anterior communicating artery connects the two ACAs. As we will see, aneurysms have a tendency to develop at sites where proximal branching of the arteries occurs, and the ACA is a common site for such aneurysms. Distal to the anterior communicating artery, the main branch of the ACA proceeds anteriorly and dorsally through the interhemispheric fissure. It then curves around the genu of the corpus callosum and follows the corpus callosum posteriorly along its dorsal surface in the callosal sulcus, which lies between the corpus callosum and the cingulate gyrus (Figure 10–4). This portion of the ACA is known as the **pericallosal** artery. Typically, a second more dorsally positioned branching of the ACA follows the cingulate sulcus. This second branch is the **callosomarginal** artery. The anterior cerebral artery also sends off secondary arteries that supply the orbital (**orbital branch**) and polar (**frontopolar branch**) frontal cortices and the medial frontal and parietal cortices (including most if not all of the cingulate gyrus). Branches of this artery also supply the genu and body (more or less the anterior two thirds) of the corpus callosum, as well as parts of the anteroventral striatum and the anterior limb of the internal capsule (the **recurrent artery of Heubner**). As will be seen in Figure 10–5, the distal branches of this anterior system also tend to overlap slightly onto the dorsal–lateral surface of the frontal and parietal lobes.

Middle Cerebral Artery

The larger middle cerebral artery is a more direct continuation of the internal carotid artery. This, combined with its larger lumen, and hence, greater hemodynamic flow, increases the probability that emboli emanating from the heart or carotid vessels will affect the distribution of the MCA rather than going up through the ACA. After separating from the terminal end of the internal carotid, the MCA proceeds dorsolaterally into the inferior aspect of the lateral fissure. Within the frontal–parietal operculum in the region of the insular cortex, the MCA divides into various cortical branches (varying to some extent from one individual to another) that exit from the superior surface of the lateral fissure. As shown in Figure 10–5, these MCA branches (which include the **orbitofrontal**, **prefrontal**, **central**, **postcentral**, **anterior** and **posterior parietal**, **angular**, and the **posterior, middle, anterior,** and **polar temporal** arteries) supply almost the entire lateral convexities of the frontal, parietal, and temporal lobes. This amounts to most of the lateral surface of the brain. The MCA also supplies the cortex of the insula and the claustrum. Smaller, penetrating arteries (**medial** and **lateral lenticulostriate** arteries), coming off the MCA, supply other internal subcortical structures. These will be reviewed separately below. The terminal branches of both the anterior and posterior cerebral arteries, which primarily supply the medial cortical

surfaces, extend slightly onto the dorsolateral and ventrolateral surface of all four lobes where they overlap with the terminal branches of the MCA. This region of overlap is referred to as the ***watershed*** or ***borderzone*** areas, and as we shall see may become important in certain hypotensive states or in some cases infarction of the internal carotid artery and hypertension.

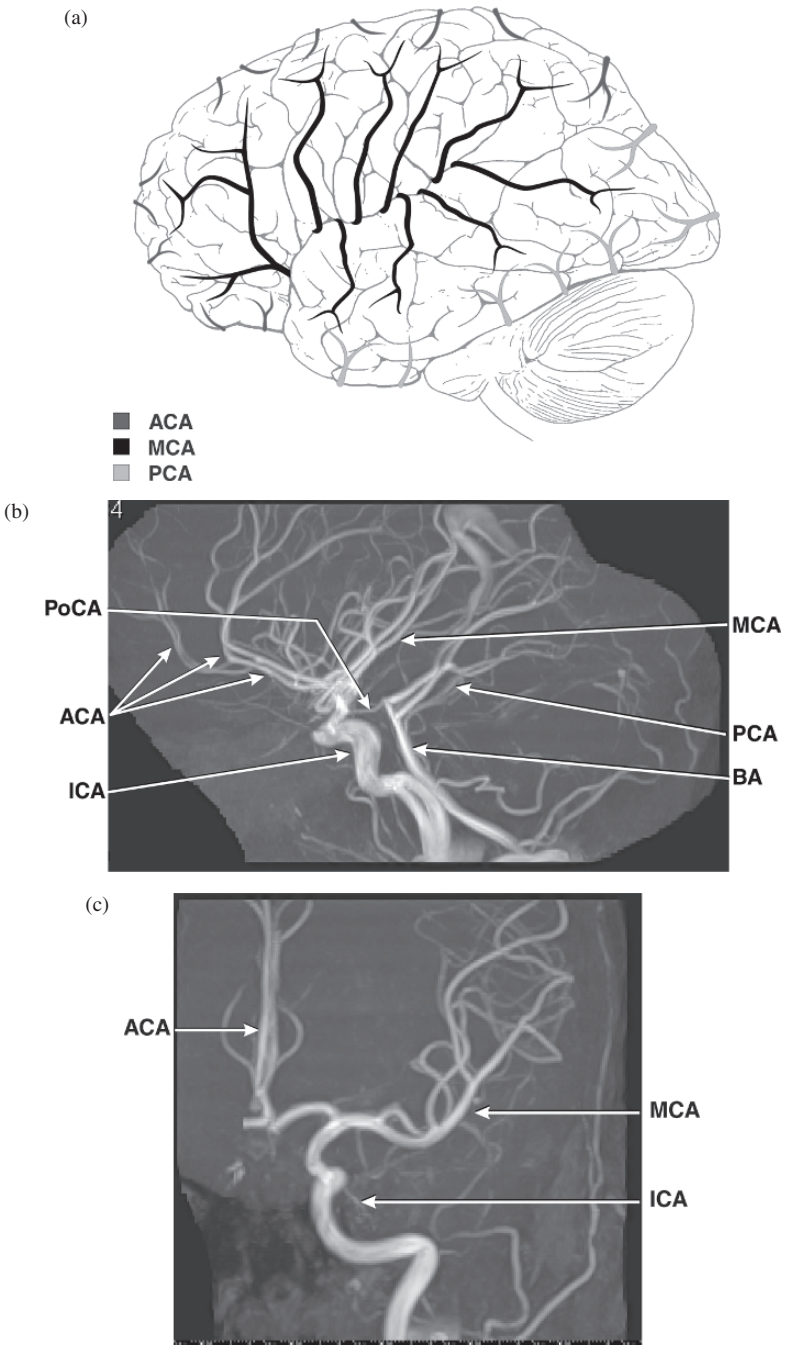


Figure 10–5. (*Continued*)

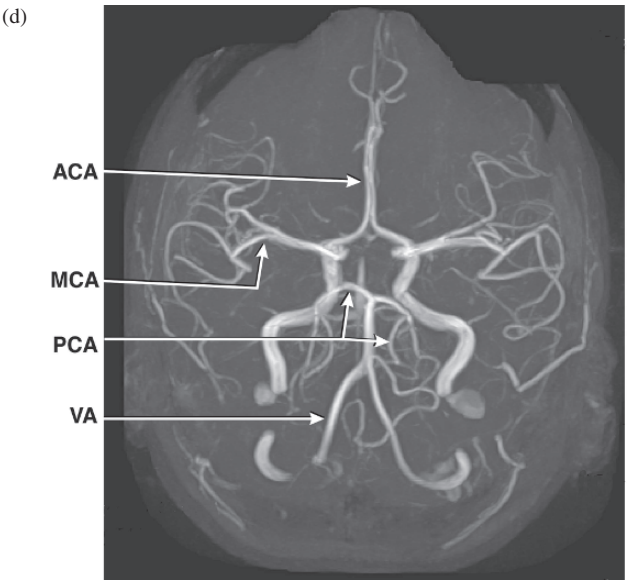


Figure 10–5. (a) Lateral view illustrating the distribution of the middle cerebral artery. The main trunk (M-1) of the MCA comes off the internal carotid and reaches the surface of the hemispheres by passing through the lateral (Sylvian) fissure. As shown in Figure 10–4, the anterior and posterior circulations are most prominent on the medial surface of the cerebral hemispheres, but also extend slightly onto the lateral surface of the hemispheres where they anastomose with the terminal branches of the MCA. The MRAs in figures (b), (c), and (d), respectively, show the lateral, A-P, and oblique axial views of these major vessels. Abbreviations: ACA, anterior cerebral artery; BA, basilar artery; ICA, internal carotid artery; MCA, middle cerebral artery; PCA, posterior cerebral artery; PcoA, posterior communicating artery; VA, vertebral artery.

POSTERIOR CEREBRAL ARTERY

As noted earlier, the posterior cerebral arteries are part of the vertebral system and are formed from the bifurcation of the basilar artery. After their origination at the level of the midbrain, they curve posteriorly around the midbrain with the main trunk remaining on the medial surface of the occipital–temporal cortices (see Figure 10–4). Branches of the PCA supply the inferior and medial portions of the temporal lobe, except for the temporal pole. This includes at least part of the hippocampal gyrus and the parahippocampal and occipitotemporal (*fusiform*) gyri (part of the hippocampus is supplied by the anterior choroidal artery). As can be seen in Figure 10–4, occipital branches of the PCA supply the medial portions of the occipital lobe, the lingual gyrus and cuneus (which include the primary visual cortex), and parts of the medial superior parietal lobule. The splenium of the corpus callosum also is supplied by the PCA system. Again, the terminal branches of the PCA tend to overlap and anastomose with the terminal branches of the ACA and the MCA both on the margins of the lateral convexities as well as on the medial surfaces of the hemispheres.

CENTRAL PERFORATING ARTERIES

The ACA, MCA, PCA, and the posterior communicating artery also give off smaller perforating vessels that supply core brain structures. One group of these vessels derives from the area of the ACA. These vessels supply the hypothalamus, the optic chiasm, and the medial

structures dorsal to it, including the preoptic area, the septum pellucidum, and the rostrum of the corpus callosum. The genu and body of the corpus callosum are supplied by the pericallosal branch of the ACA itself. Other more anterior perforating arteries supply the rostral portions of the head of the caudate nucleus, part of the putamen, and the anterior limb of the internal capsule.

The middle cerebral artery is the source for the **medial** and **lateral lenticulostriate** arteries (Figure 10–6). They emanate from the MCA shortly after its origination as it begins to penetrate the inferior portion of the lateral sulcus. These small vessels supply the "lenticular" nucleus (putamen and globus pallidus), the more posterior portions of both the caudate nucleus (except for the tail), and the anterior limb of the internal capsule, as well as the dorsal portion of its posterior limb. The more ventral aspects of the posterior limb of the internal capsule are supplied by the anterior choroid arteries. As we shall discuss later, the lenticulostriate arteries appear to be particularly vulnerable to hypertensive disease and are often involved in subcortical lacunar infarctions.

The PCA and the posterior communicating arteries also are the source of numerous small penetrating arteries. One portion of the larger **posteromedial group** proceeds rostrally to supply the mammillary bodies and hypothalamic structures, while other portions of this (the ***thalamoperforating*** and ***inferior thalamic*** branches) supply the anterior and medial thalamus, as well as other midbrain structures such as the red nucleus. A **posterolateral group**, arising either from the PCA or posterior communicating arteries, supplies the remainder of the thalamus, including the lateral and posterior nuclear groups and the geniculates. These latter arteries are known as the ***thalamogeniculate*** branches.

The **anterior choroidal** artery, which derives from the posterior surface of the internal carotid just distal to the branching of the posterior communicating artery, already was mentioned. In addition to supplying the choroid plexus of the lateral ventricles, it also may supply a number of other fiber pathways and nuclear groups. These include the optic tract, the tail of the caudate, the amygdala and portions of the hippocampal formation, and posterior portions of the internal capsule. It also may supply parts of the lateral

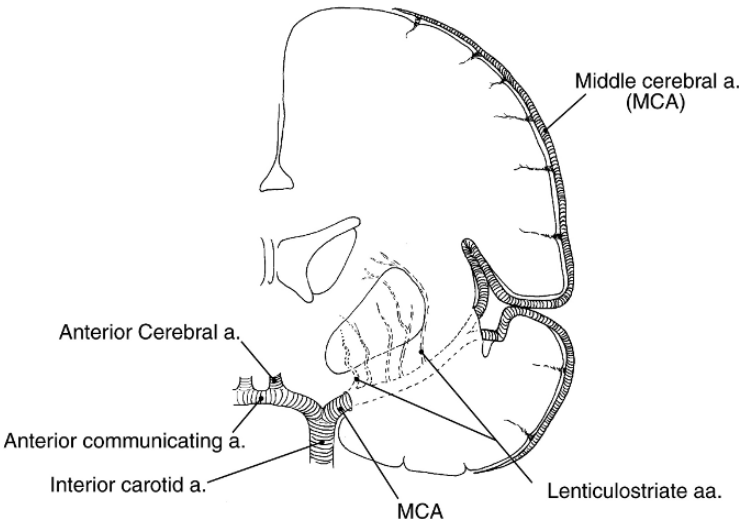


Figure 10–6. The lenticulostriate arteries, one of several groups of smaller penetrating arteries, branch off the MCA within the fold of the lateral fissure to supply portions of the basal ganglia and internal capsule.

geniculates, globus pallidus, and posterior putamen, and other subthalamic nuclei, including the substantia nigra.

The **posterior choroidal** artery, which arises from the PCA, supplies the choroidal plexus of the third ventricle, the tectum or inferior and superior colliculi, and probably contributes to the dorsal medial nucleus of the thalamus.

SUMMARY OF THE ARTERIAL BLOOD SUPPLY TO THE BRAIN

The **brainstem** (midbrain, pons, and medulla) and **cerebellum** as well as the upper part of the **spinal cord** primarily are supplied by vessels arising from the vertebral and basilar arteries. Certain midbrain structures also are supplied by the anterior (e.g., substantia nigra) and posterior (e.g., tectum) choroidal arteries

The **lateral convexity of the cerebral hemispheres** is generally supplied by the branches of the MCA, which along with the ACA is derived from the bifurcation of the internal carotid arteries. However, both the ACA and PCA overlap the distribution of the MCA on the margins of the lateral surface of the hemispheres.

The **medial** and **inferior surfaces of the hemispheres** are supplied by the ACA and PCA. The ACA supplies the orbital and medial surface of the frontal lobes, including the sensorimotor regions for the lower limbs and most of the remaining medial parietal cortices, the cingulate gyrus, and most of the anterior two thirds of the corpus callosum. The PCA supplies the medial surface of the occipital cortex (includes the primary visual areas), parts of the medial superior parietal lobule, most of the medial and inferior temporal lobe, and the posterior third (splenium) of the corpus callosum.

The **basal ganglia** primarily are supplied by the lenticulostriate arteries that emanate from the proximal portion of the MCA. However, parts of these structures also are supplied by the anterior choroidal artery and small vessels coming off the posterior communicating artery.

The **thalamus** largely is supplied by the smaller vessels that branch off the PCA and posterior communicating arteries, including the thalamoperforating, inferior thalamic, thalamogeniculate, and posterior choroidal arteries. The anterior choroidal arteries also may make a minor contribution. The **anterior hypothalamus** largely is supplied by the small penetrating (anteromedial) vessels originating from the ACA and/or anterior communicating artery in the vicinity of the circle of Willis, while the more posterior portions of the hypothalamus are supplied by the posterior penetrating arteries.

Most of the anterior and posterior limb of the **internal capsule** also is nourished by the lenticulostriate arteries. Small arteries that originate directly from the internal carotid may supply the genu. The anterior choroidal artery generally supplies some of the more ventral portions of the posterior limb, as well as the retrolenticular portions.

The **visual system** is supplied by multiple vessels. The retina of each eye separately is supplied by its corresponding ophthalmic artery from the internal carotid. The optic nerves, optic chiasm, and the initial segment of the optic tracts largely are supplied by the small penetrating arteries derived from the ACA and the anterior communicating artery. Small vessels from the internal carotid or middle arteries primarily supply the more anterior portions of the optic tract. The more posterior portions of the tract, along with parts of the lateral geniculates, are supplied by the anterior choroidal arteries. The lateral geniculates also are supplied by the posterolateral penetrating arteries of the posterior system. The superior optic radiations are supplied by the posterior cortical branches of the middle cerebral artery, while the inferior radiations are nourished by the posterior cerebral artery (PCA). The primary visual cortex and parts of the secondary visual cortex are supplied by the PCA.

The **motor system** also is subserved by a variety of vessels. The disruption of any of these vessels can produce a weakness or paralysis. The primary motor cortex that mediates the

face, hands, and trunk is served by the MCA, whereas the legs (especially the lower legs and feet) are represented on the medial surface of the hemispheres, and thus are supplied by the ACA. The MCA–ACA watershed territory supplies the motor cortex that mediates control of the proximal arm and proximal leg. The basal ganglia and internal capsule primarily are supplied by the lenticulostriate arteries (branches of the MCA), although some input is derived from the anterior choroidal and posterior penetrating arteries. The corticospinal tracts are supplied by various small vessels deriving both from the internal carotid and posterior cerebral artery systems at the level of the midbrain and by branches of the basilar and vertebral arteries at the level of the pons and medulla. The cerebellum, the disruption of which can lead to difficulties with balance, coordination, and weakness, is supplied by the superior cerebellar artery, the anterior and posterior cerebellar arteries that derive from the vertebral system.

PATHOLOGY OF THE VASCULAR SYSTEM

Thus far, the focus of this text has been primarily on functional neuroanatomy rather than on neuropathology. However, with the vascular system, it seems prudent to review, at least in general terms, the types of vascular problems that most frequently affect the brain and some of the more common syndromes that can result from specific vascular lesions. A detailed discussion of the pathophysiology of strokes will not be presented here.[2]

For present purposes, cerebrovascular disorders can be classified into three general types: (1) **ischemic lesions**, which can be either hypoxic or occlusive in origin, (2) **hemorrhagic lesions**, and (3) **blood vessel anomalies**. It should be noted, however, that these disorders are not mutually exclusive. For example, an ischemic infarct or a vascular anomaly, such as an aneurysm, may eventually hemorrhage, producing a hemorrhagic lesion. Ischemic lesions are the most common causes of stroke, accounting for about 80% of all strokes. Hemorrhagic lesions account for the remainder of all strokes, and most often are produced by ruptured berry aneurysms or hypertensive hemorrhages.

Ischemic Disease

Occlusive Lesions

Ischemic lesions can result from a (1) blockage of the vessel itself, (2) lack of sufficient oxygen in the blood, or (3) systemic circulatory problem that results in poor perfusion. Occlusive vascular disease, which results from a blockage (*infarction)* in a particular vessel, interrupts blood flow to the brain. Occlusive lesions may involve either arterial vessels or the venous system, although the former is much more common and will be the focus of the discussion. Three etiological subtypes of ischemic arterial occlusive disorders generally identified are: **thrombotic**, **thromboembolic**, and **embolic**. **Thrombotic disease** produces a gradual, progressive narrowing of the arterial lumen or *arterial stenosis* that inhibits or restricts the flow of blood. This condition most commonly results from atherosclerosis, which in turn results from a buildup of fibrous tissue within the interior walls of the vessel. This atherosclerotic condition is compounded by the adherence of fatty plaques and blood platelets to these fibrous plaques. Although atherosclerosis occurs with aging, certain conditions appear to accelerate this process. These include hypertension, diabetes, elevated serum lipoproteins, smoking, and genetic predisposition.

The large vessels of the neck (e.g., the internal carotid system) and the areas of bifurcation or branching in the proximal portions of the cerebral arteries are sites that offer particularly high risks as sources of thrombotic strokes. Not all thrombotic occlusions result in a stroke (i.e., persistent, focal neurological deficit associated with a disturbance of the circulatory system). If the arterial stenosis occurs very gradually, particularly if the site is proximal

to the circle of Willis, compensatory collateral circulation may develop via other arterial systems (e.g., the opposite internal carotid or posterior vessels). The circle of Willis is not the only potential source of collateral blood supply. In certain circumstances, branches from the external carotid artery may shunt blood via anastomoses with the ophthalmic artery into the carotid system. While the areas in which the distal distributions of the ACA, MCA, and PCA overlap (watershed territories) also may form anastomoses providing potential sources of collateral interchanges between two arterial distributions, if present these generally have limited clinical significance.

In addition to thrombosis, another cause for occlusive vascular disease is emboli. An **embolus** is a bit of organic material or foreign matter that travels from one part of the body (or vascular system) through the arterial system where it eventually lodges, occluding the vessel. Common sources of emboli are bits of thrombotic plaques from the heart or cardiocephalic arteries, bacterial endocarditis, air or nitrogen bubbles, and fat. The heart is the most common source of embolic stroke (i.e., cardioembolic stroke). Cardioembolic stroke is associated with several cardiac conditions, including arrhythmias, acute myocardial infarction (MI), cardiac wall motion abnormalities due to an old MI or cardiomyopathy, valvular disease, and some congenital cardiac diseases. Cardiac arrhythmias (particularly atrial fibrillation) greatly increase the risk for the development of cardiac emboli in susceptible individuals, for example, those with history of rheumatic fever, myocardial infarction, or other conditions that lead to scarring or plaque development with the walls of the heart or its valves.

Fat emboli most commonly results following fractures of the long bones of the leg. Fat globules are carried by the venous system through the heart to the lungs where most are filtered out of the circulating blood. However, it is possible for some of the smaller fat cells to enter into the arterial system where they can be transported to the brain (or other organs) causing embolic infarctions. Because the lungs already have filtered out most of the larger fat emboli, the resulting cerebral infractions tend to be small, but may be multiple.

Distant emboli, again with the heart being the most common source, account for about 20% of ischemic strokes. Emboli that originate from thrombotic plaques in the neck or travel up the carotid system from the heart are considerably more likely to enter the middle as opposed to the anterior cerebral circulation. Emboli that originate from extracranial or large cerebral vessels that are stenosed often are referred to as thromboembolic strokes, as these occlusive strokes are produced by an embolus breaking off of a thrombosed vessel.

While there are no hard and fast rules, there are several factors that may differentiate thrombotic infarction from embolic infarctions. First, in general, embolic infarctions are somewhat more likely to occur during periods of activity rather than at rest (as is slightly more typical of thrombotic infarctions). Thrombotic infarctions are more likely to have been preceded by transient ischemic attacks (TIAs) than are embolic strokes. Embolic infarctions are more likely to evidence secondary hemorrhage (hemorrhagic infarctions) than are those caused by thrombosis. Thrombotic infarctions more often than not are likely to occur while sleeping or shortly after awakening in the morning. This is probably related to changes in hemodynamics and/or blood chemistries that can occur during periods of prolonged quiescence.

Although there are many ways to conceptualize stroke subtypes, one method is based on the vascular anatomy and pathophysiology. In addition to the MCA, ACA, and PCA distributions, another common dichotomy is *large* versus *small* vessels. Large cerebral vessels generally include the main surface vessels of the ACA, MCA, and PCA that supply the lateral, medial, and inferior aspects of the cerebral cortex. Small cerebral vessels, on the other hand, generally are defined as those central perforating vessels (such as the lenticulostriate arteries) that, as we saw earlier, tend to supply internal cortical structures (basal ganglia, internal capsule, and thalamus). Therefore, stroke syndromes often are subtyped

as large vessel or small vessel strokes. The mechanisms and deficits associated with each of these subtypes differ. Large vessel strokes usually are caused by a localized thrombus to a large vessel or a branch of a large vessel (like a branch of the MCA) or by an embolus propagated from an area of atherosclerosis to a more distal branch. An example might be an atherosclerotic plaque at the internal carotid artery bifurcation throwing off an embolus to the MCA).

Deficits corresponding to the vascular territory that is occluded often are discernable with a comprehensive neurological and mental status examination, although neuroimaging generally will better define its anatomical boundaries. The more proximal the occlusion in the vascular tree, the larger the area of infarction, and hence, the greater the deficits that are likely to occur. By contrast, more distally located occlusions will produce smaller areas of infarction and, as a general rule, more restricted deficits (see Figure 10–7). The nature and prominence of the symptoms also will depend on the locus of the lesion. For example, a stroke produced by a more proximal occlusion of the internal carotid or MCA of the dominant hemisphere typically would result in massive motor, sensory, language (global aphasia), and other cognitive deficits, while an infarction that is limited to a particular branch of the MCA would result in an incomplete MCA syndrome with the exact symptoms dependent upon the vessel(s) involved.[3] Although less common, large-vessel strokes can be limited to the ACA distribution or the PCA (the latter being derived from the vertebrobasilar system). Again, specific complaints from the patient's history and signs and symptoms obtained from an examination of the patient often can identify the vascular territory most likely involved. Many of the specific findings and complaints commonly associated with particular arterial territories will be reviewed later in this chapter.

While hypertension frequently is a factor in the development of generalized, large-vessel atherosclerotic disease, a history of chronic hypertension almost universally is present in

(a)

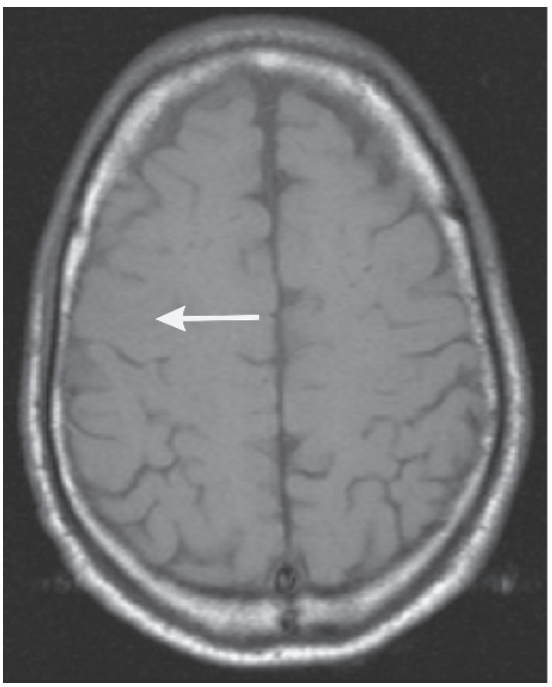
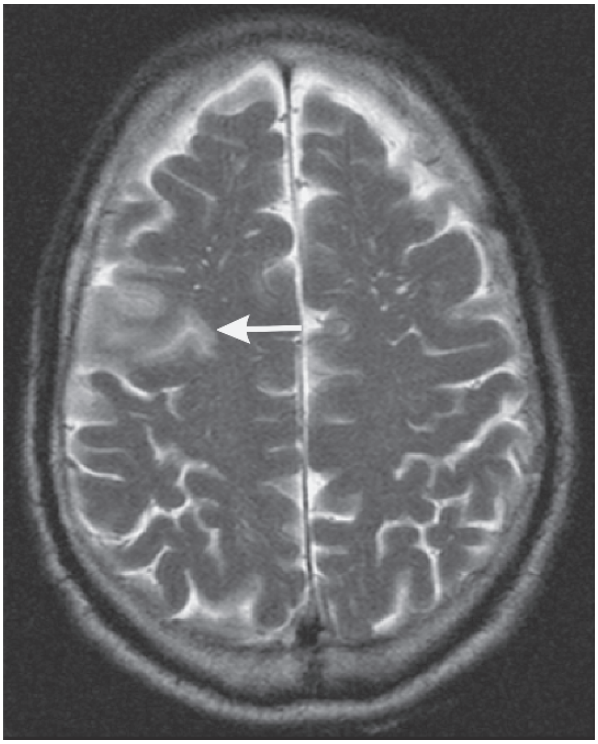
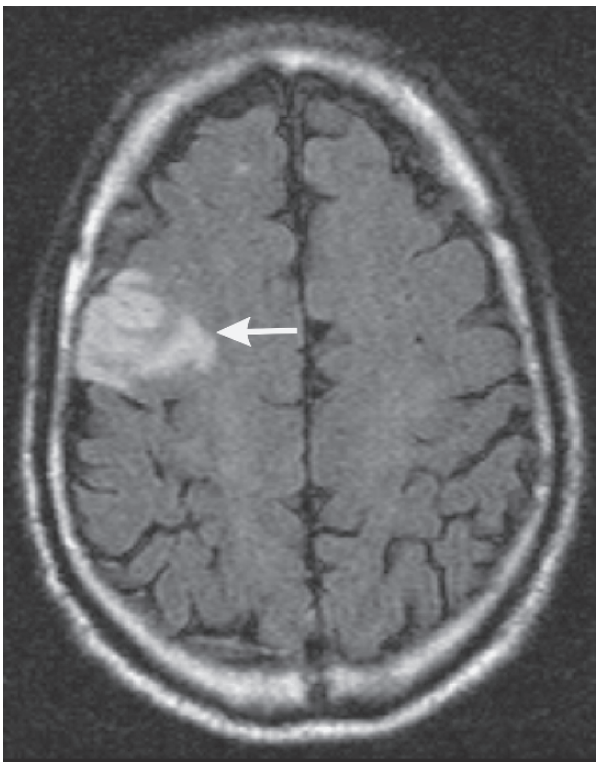


Figure 10–7. (*Continued*)

(b)



(c)



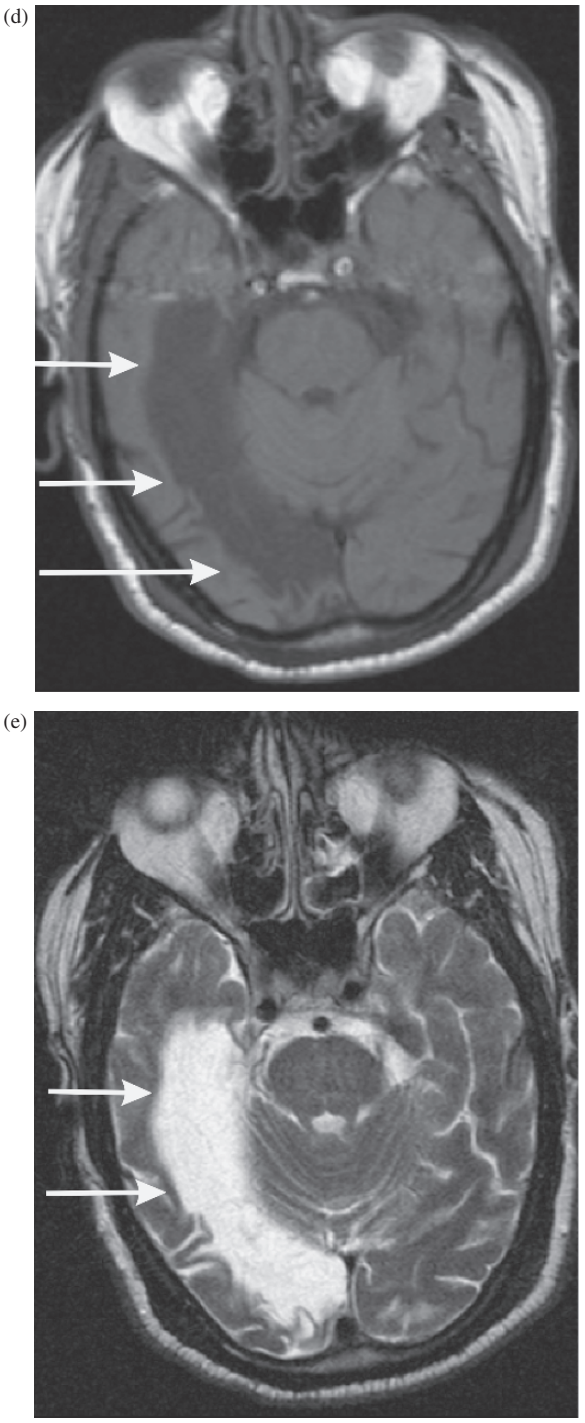
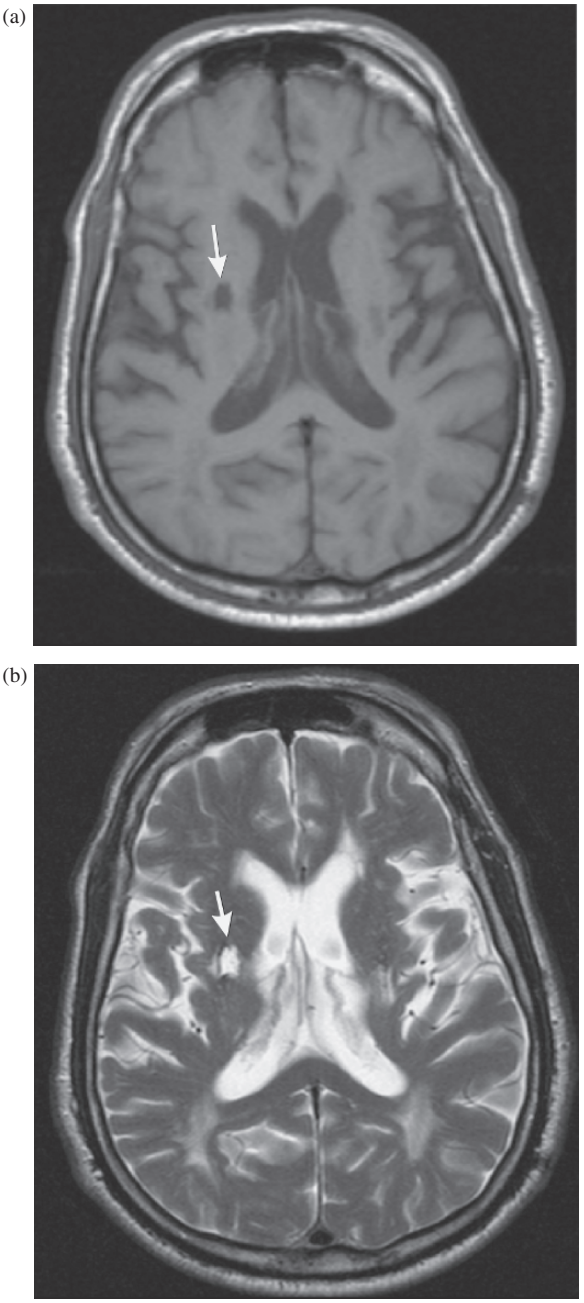


Figure 10–7. Acute infarction of distal branch of the right MCA as seen on (a), T-1, (b), T-2, and (c) FLAIR images. Compare the acute lesion as seen on (a) T-1 image with an old infarction of more proximal portions of the right PCA as seen on (d) T-1 and (e) T-2 weighted images. **Note:** Infarctions are less conspicuous in T-1 weighted images in their acute stage, but show up more prominently in latter stages as the infarcted tissue is displaced by water molecules. Nonetheless, careful observation of (a) reveals slight sulcal effacement in the affected area, a subtle or indirect sign of edema following acute infarction.

small-vessel disease, whether of occlusive or hemorrhagic origin. The mechanism behind hypertensive infarcts involves localized thrombosis of the small, penetrating vessels due to lipohyalinoid degeneration that leads to occlusion of the vessel.[4] These types of small-vessel strokes produce **lacunar infarcts**, which by definition are lesions of 1.0 to 1.5 centimeters in size (Figure 10–8). Because of the vascular anatomy and vulnerability of the deep penetrating small vessels that supply subcortical and brainstem areas, there are specific stroke syndromes



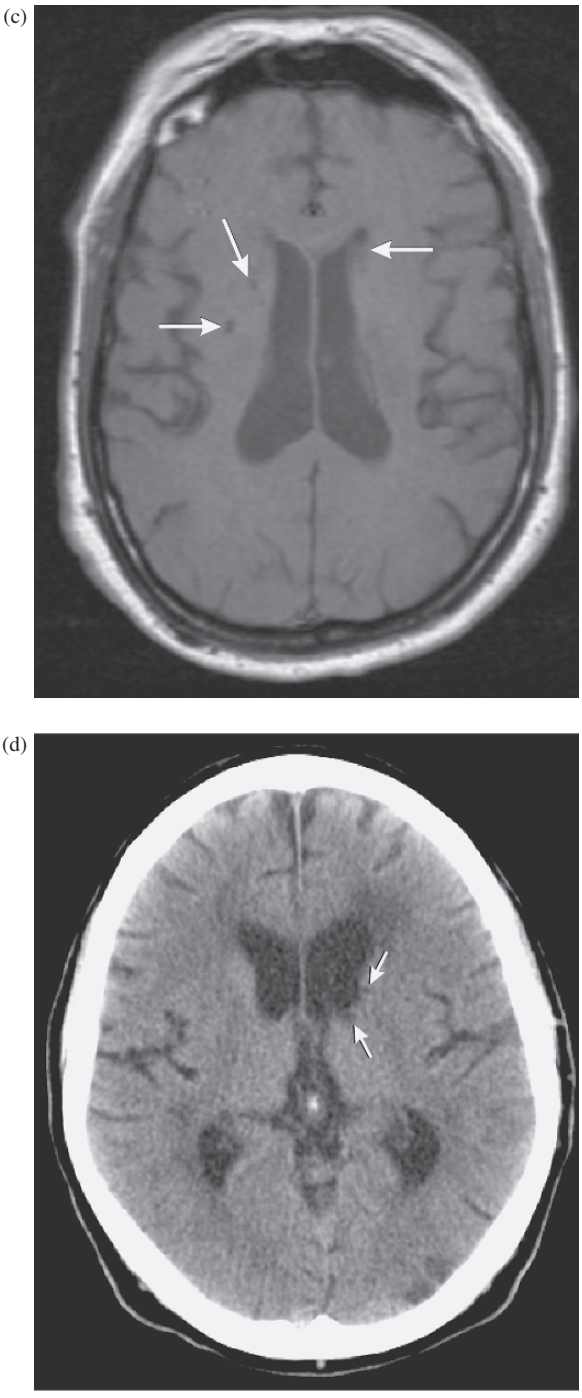


Figure 10–8. Lacunar infarctions involving smaller penetrating vessels. Figures (a) and (b) are the same lesions as seen on T-1 and T-2 MRI imaging, respectively. Multiple slightly smaller infarcts can be seen on (c) (T-1 weighted image), while (d) shows comparable small infarcts on a CT scan.

due to lacunar infarcts that most commonly involve the basal ganglia, thalamus, and internal capsule. These lacunar syndromes include:

1. Pure motor strokes
2. Pure sensory strokes
3. Mixed sensory-motor strokes
4. Ataxic hemiparesis
5. Clumsy hand, with dysarthria

Before proceeding to the hypoxic or insufficiency syndromes, it should be noted that while atherosclerotic disease and cardiac emboli are the most common causes of occlusive cerebrovascular disease (stroke), they are not the only causative factors. Other causes of strokes include trauma to the vessels themselves (especially in the neck resulting in carotid artery dissection), inflammatory or infectious processes directly involving the proximal or cerebral vessels (vasculitis), amyloid angiopathy, right-to-left shunt in the heart due to patent foramen ovale producing a paradoxical emboli, hematological disorders (e.g., sickle cell disease, thrombocytopenia), intravenous drug abuse, and pregnancy.

In addition to occlusive vascular disease that results in a total and extended shutting off of the blood supply of blood to a cerebral vessel and necrosis in its area of distribution (infarction), there are other conditions that produce more temporary or incomplete circulatory restrictions. The etiologies are similar to those for other forms of occlusive disorders: diseases affecting the vessels themselves (e.g., atherosclerosis), blood factors including emboli that contribute to the restriction of blood flow, or conditions that reduce cerebral perfusion (e.g., steal syndromes or reduced arterial pressure, especially in the presence of preexisting vascular disease). In the first situation, there may be a more or less complete but temporary restriction of blood flow through an artery as a result of either a thrombotic or embolic process. Commonly referred to as a **transient ischemic attack** (TIA) (see below), the individual may experience a temporary loss or impairment of functioning, particularly if a primary sensory, motor, or language region of the cortex is affected. Again, the specific symptoms will depend on the artery and branches affected and the hemisphere involved.

Hypoxic Lesions

Ischemic strokes result from lack of sufficient oxygenation of brain tissue. In addition to an inadequate profusion of the brain secondary to an occlusion of vessels (either as a result of a thrombus or embolus), there are other conditions in which the blood vessels themselves may remain patent but the brain is still oxygen-deprived. Commonly referred to as **hypoxic syndromes**, these conditions may result from either a lack (or displacement) of oxygen in the blood (*primary hypoxia*) or a systemic circulatory failure (*secondary hypoxia*), both of which result in failures to adequately replenish oxygen to the brain.

Less common than occlusive vascular disease, hypoxia or lack of sufficient oxygenation of the brain can produce either temporary or permanent neurological deficits. The nature, pattern, or severity of these deficits in part will depend on the severity, length, and etiology of the deprivation. Hypoxia may result from either chronic or acute conditions. Causes of chronic hypoxia include chronic obstructive pulmonary disease (COPD), congestive heart failure, or severe anemia. Subacute changes can result from unaccustomed exposure to high altitudes ("mountain sickness"). Chronic or subacute conditions are not usually associated with "strokes" in the usual sense of the word or with loss of consciousness. Chronic hypoxic syndromes frequently will present with a picture of more subtle cognitive or behavioral changes, although more dramatic effects, such as delirium, might occur particularly in the elderly who may have less cognitive reserve. Acute oxygen deprivation is more likely to be associated with loss of consciousness and more serious and permanent consequences. The latter can result from such events as airway obstructions (e.g., choking), cardiac arrest,

carbon monoxide poisoning, near drowning, "partially successful" suicide attempts by hanging, respiratory paralysis (e.g., Guillain–Barré), or severe hypotension.

In more acute cases of anoxia, it is not uncommon to find more permanent, focal-type deficits, particularly if consciousness has been lost. In these cases, selective deficits may be related to the specific oxygen needs of the brain tissue itself (Adams & Graham, 1988). Certain brain regions are believed to be more vulnerable to oxygen depletion than others. The areas of greatest vulnerability are CA1 of the hippocampus, the basal ganglia, the cerebellum, and laminar necrosis to cortical layers 3 and 5. It is hypothesized that the brain tissue in these areas (i.e., the metabolism that is necessary to sustain function) is more sensitive to oxygen deprivation, and hence, more readily affected by oxygen deprivation. Thus, changes in memory and motor functions are fairly common in such cases. Certainly the brain as a whole has a very high rate of metabolism and uses vast amounts of oxygen relative to its size. Under normal circumstances, oxygen deprivation for more than 5 to 6 minutes can be expected to result in neuronal damage or death. Comas lasting more than 48 hours following an acute anoxic episode, especially if attended by absence of brainstem responses, generally are associated with severe, permanent brain damage. Conditions that tend to slow down brain metabolism and as a result its need for oxygen consumption, such as being immersed in frigid water, can lengthen the time of oxygen deprivation before permanent damage may result. Using this principle, it is common practice to lower the body temperature of patients undergoing certain types of heart surgery.

Conditions that result in reduced arterial pressure also may create states of hypoperfusion leading to either temporary or permanent changes in brain function. Two general types of conditions likely are to be responsible for brain lesions secondary to hypoprofusion. One is a systemic lowering of blood pressure. Some common causes include postural (orthostatic) hypotension, cardiac insufficiency or myocardial infarction, or severe systemic hemorrhaging. Postural hypotension (a sudden drop in blood pressure upon rising to a vertical position) can result in dizziness or a syncopal episode, without any permanent sequelae. If the hypotension is severe and long enough, all cerebral vessels can be affected and permanent deficits may ensue. However, arteries that are nearly completely occluded from atherosclerotic disease might be differentially affected, resulting in more focal deficits. In instances of decreased blood pressure or markedly reduced cardiac output, the areas maximally affected may be those where the distribution of the MCA, PCA, and ACA overlap, producing what is known as a *watershed* or *borderzone* syndrome (Figure 10–9).

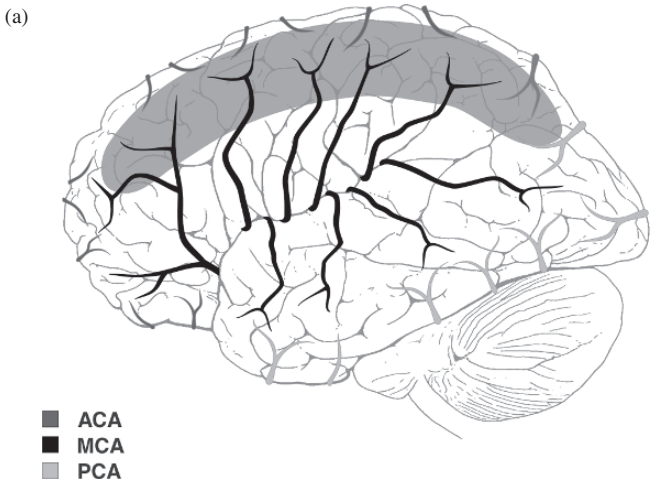


Figure 10–9. (*Continued*)

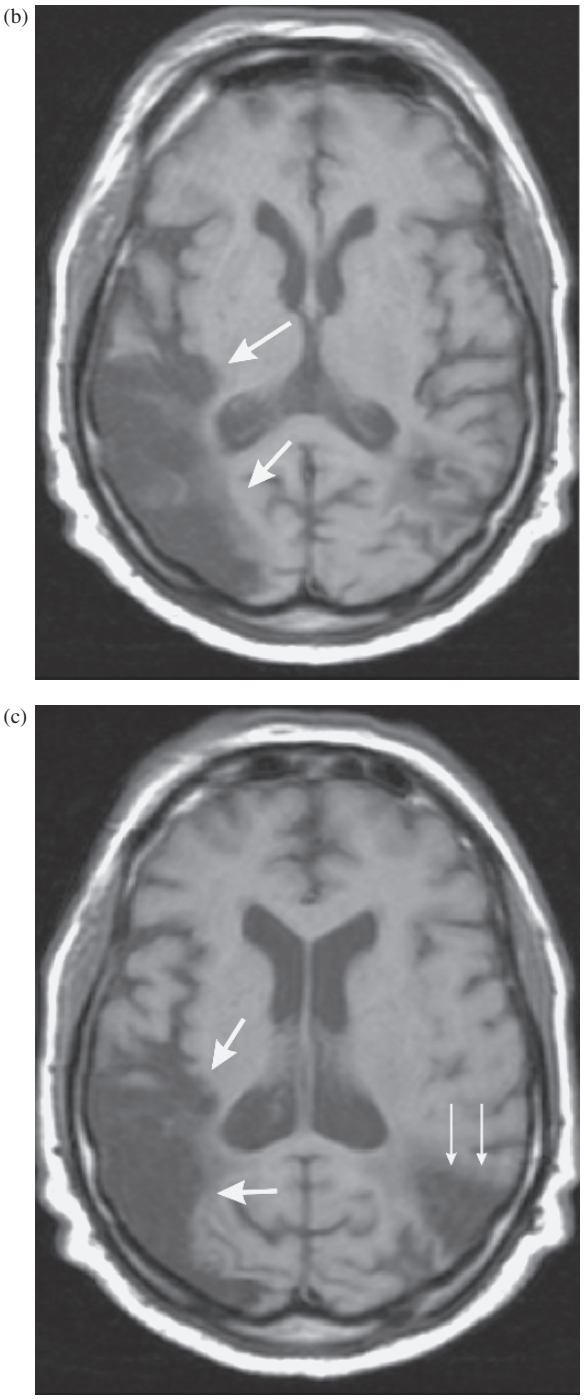


Figure 10–9. (a) Schematic representation of "borderzone" infarction involving the terminal branches of the anterior, middle, and posterior cerebral artery distributions. Figures (b) and (c) show right-hemisphere lesion (large arrows) thought to represent possible borderzone infarction involving the area of the MCA and PCA distributions, although direct occlusion of one or more of these vessels cannot be ruled out. (c) Also seen is an older, smaller infarction in the left posterior parietal area likely involving the MCA.

This syndrome will vary depending on the particular hemisphere and arterial distributions affected; however, the symptoms often will result in complex cognitive, behavioral disturbances (e.g., agnosias, aphasias, apraxias, frontal lobe-type syndromes).

A second situation that may produce secondary hypoxia is where arterial hypotension is limited to the arterial distribution distal to an area of infarction. A relatively common example might involve the occlusion of an internal carotid artery. While collateral circulation via the opposite carotid artery or via the vertebral system may allow for some circulation through the affected system, the pressure will be reduced. In this situation, the more proximal portions of the arterial distribution may be adequately perfused, but the more distal portions might suffer.

Transient Ischemic Attacks

By definition, if a deficit resulting from an ischemic infarction resolves (at least for most clinical purposes) within an hour, the episode is referred to as a *transient ischemic attack* (TIA). If it lasts longer but seems to completely clear within a relatively short time (e.g., within a few days), it may be referred to as a *reversible ischemic neurological deficit* (RIND).[5] The symptoms of a completed stroke generally come on rather suddenly, although they may evolve over time (typically a matter of minutes, but occasionally over hours or even days). An individual may have a TIA without ever experiencing a completed stroke, but the presence of multiple TIAs affecting the same vascular distribution normally is associated with increased risk for a permanent infarction. A single such episode may be either embolic or thrombotic in origin, while multiple episodes (again, within the same distribution) suggest arteriosclerotic disease. It should be noted that while by definition a TIA or RIND is completely clinically resolved within a short period of time, in what initially might appear to be a TIA the possibility of more subtle, residual deficits on occasion, can be detected if sufficiently sensitive measures are employed.[6]

Transient Global Amnesia

Transient global amnesia (TGA) is a syndrome in which the patient experiences a sudden loss of recent memory as well as an inability to lay down new memories (anterograde amnesia). Like TIAs, TGA is characterized by an acute onset, is generally limited in scope (the patient may evidence mild confusion secondary to the memory loss but no deterioration of global cognitive functions), and typically resolves within a few hours. While the capacity to lay down new memories returns, the patient typically remains amnestic for events surrounding the episode itself. Also like TIAs, transient global amnesia is thought to most likely represent a vascular type of event; however, unlike a TIA, it is not associated with increased risk of a subsequent stroke. If indeed this syndrome is of vascular origin (the exact etiology is not clear, but some type of vascular spasm is possible), the temporal branches of the PCA and/or some of the posterior penetrating arteries are most likely involved.

Recovery from Ischemic Strokes

Following occlusive or hypoxic syndromes, the patient often recovers significant function over time. What are the mechanisms that allow for this? Certainly one mechanism is that other areas of the brain "learn" to compensate or take over for the damaged area. The patient also may "relearn" how to do certain things using alternative pathways or behavioral strategies. However, a large amount of recovery probably takes place independent of what the patient learns or experiences poststroke and may be explained at a more mechanical level. When an infarct occurs, several things disrupt neuronal function, some of which appear to be reversible. First, it is commonly accepted that if neurons supplied by a vessel are

deprived of oxygen for a sufficient period, they die, and once dead they do not regenerate. However, as one goes out from the center of the lesion (the *umbra*), there may be brain tissue that is not completely shut off from the supply of blood (perhaps as a result of partial collateral or overlapping circulation or vasospasms), which becomes hypoprofused. In this area (the *penumbra*), the cells become dysfunctional as a result of lack of sufficient oxygen, but still are viable and may recover over time as additional collateral circulation develops and/or the vasospasms diminish—the *idling neuron* hypothesis. Additionally, within the area of the infarct, some cytotoxic edema is invariably present.[7] As the edema subsides, those neurons that may have been adversely affected by the edema may recover. Some minor hemorrhaging around the site of an infarction into the surrounding brain tissue is not uncommon following an ischemic stroke. The direct imposition of red blood cells on neural cells is not compatible with their normal functioning. (i.e., the blood appears to have a toxic effect on the nerve cells). Once this blood breaks down and is absorbed, some of these affected cells may return to normal or near normal functioning. While there are likely additional explanations for partial recovery of function on a cellular level, the mechanisms discussed above are among the most commonly suggested.

Hemorrhagic Vascular Disease

Following occlusive (ischemic) disorders, hemorrhages are the next most common cause of stroke. Hemorrhages involve the rupture of a blood vessel, most commonly an artery. The major differences in the types of hemorrhagic stroke to be discussed below simply reflect the site of the bleed. Certainly a second major consideration is the size or extent of the resulting hemorrhage, which is largely a function of the size and site of the ruptured vessel. Intracranial hemorrhages tend to have increased mortality in the acute phase compared to occlusive strokes. However, if the patient survives the acute stage, the prognosis for improvement typically is somewhat better than that for occlusive infarction. While this may be an oversimplification of the situation, at one level this readily makes sense. As noted above, in a completed occlusive stroke or infarction, the blood supply is shut off from the cells in the arterial distribution distal to the occlusion. Without a supply of blood (oxygen), the cells die, and once dead there is no chance of regeneration.

Bleeds, by contrast, potentially are very destructive for other reasons. First, like occlusive infarcts they can disrupt the blood supply to adjacent nervous tissue or distal to the site of the hemorrhage. In addition, secondary vasospasms (particularly in subarachnoid bleeding) may cause additional expansion of the ischemic infarction. The extravasation (leakage) of free blood into the extracellular spaces will disrupt the normal functioning of the cells due to its direct toxic effect on neural tissue. These effects obviously will be maximal where the free or extravascular blood directly interfaces with the nervous tissue and the clinical signs and symptoms will depend on the location and size of the hemorrhage. Hemorrhagic strokes also can be associated with significant increases in intracranial pressure. As with infarcts, cytotoxic edema results from damage to the surrounding cells, with the degree of edema associated with the size of the bleed. However, in the case of hemorrhages, there potentially is another source of increased intracranial pressure, compounding the cytotoxic edema. The blood itself can act like a space-occupying lesion, causing compression of the brain tissue and possibly leading to herniation and death if the bleed is sufficiently large. Small-vessel or venous bleeds generally pose considerably less risk than a rupture of larger arterial vessels as might be found with aneurysms or epidural hematomas (see below). While these pressure effects may be disruptive to normal functioning of the cells, in many cases these effects are limited and transient. In the case of intraparenchymal hemorrhages, once the blood is reabsorbed, many cells might return to normal functioning. If the bleed is outside the brain tissue itself (e.g., subdural or epidural hematomas), the major destructive

damage will be from pressure effects, which again might be overcome in the long run if the patient survives.

As suggested above, the classification of the sites of intracranial hemorrhages falls into four general categories:

1. Within the brain tissue itself (**intracerebral** or **intraparenchymal** hemorrhages).
2. In the subarachnoid space (**subarachnoid** hemorrhage).
3. In the potential space between the dura and the arachnoid (**subdural** hematoma).
4. Outside the dura (**epidural** hematoma).

As we shall see, the typical cause of such bleeds tends to vary depending on their sites.

Epidural hematomas most commonly result from skull fractures where the meningeal arteries are ruptured. Because the arterial blood is under greater pressure than venous blood, the bleeding is frequently profuse and can result in a rapid increase in intracranial pressure (Figure 10–10). Consciousness usually is impaired as the pressure increases, and unless the blood is surgically evacuated and the pressure relieved, death may ensue as a result of mass effect and brainstem herniation. Because the dura is attached to the skull along its suture lines, the lateral extent of the bleeding usually is somewhat restricted, resulting in a "lens"-shaped hematoma on imaging studies.

Subdural hematomas are produced when bleeding occurs into the "potential space" between the dura and the arachnoid (Figure 10–11). Subdural hematomas are also associated with head trauma but, unlike an epidural bleed, are more often associated with a closed head injury (i.e., without skull fractures). Subdural hematomas most frequently are due to rupture of the bridging veins into the dural sinuses as a result of the shearing forces, as in a deceleration-type injury. If the formation of the hematoma is acute and

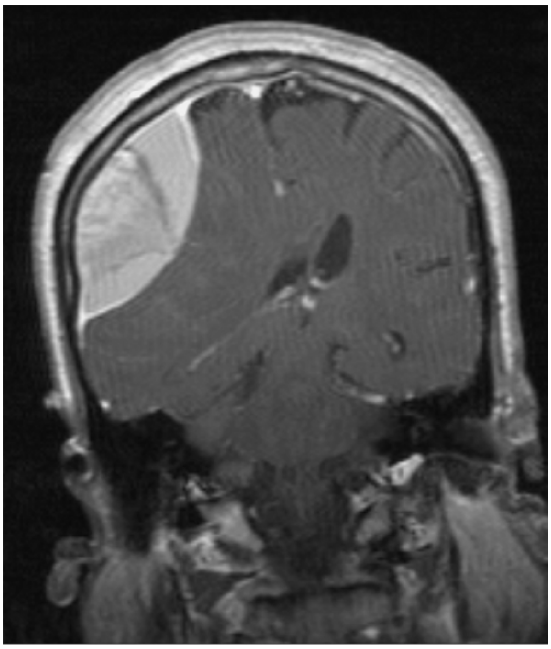
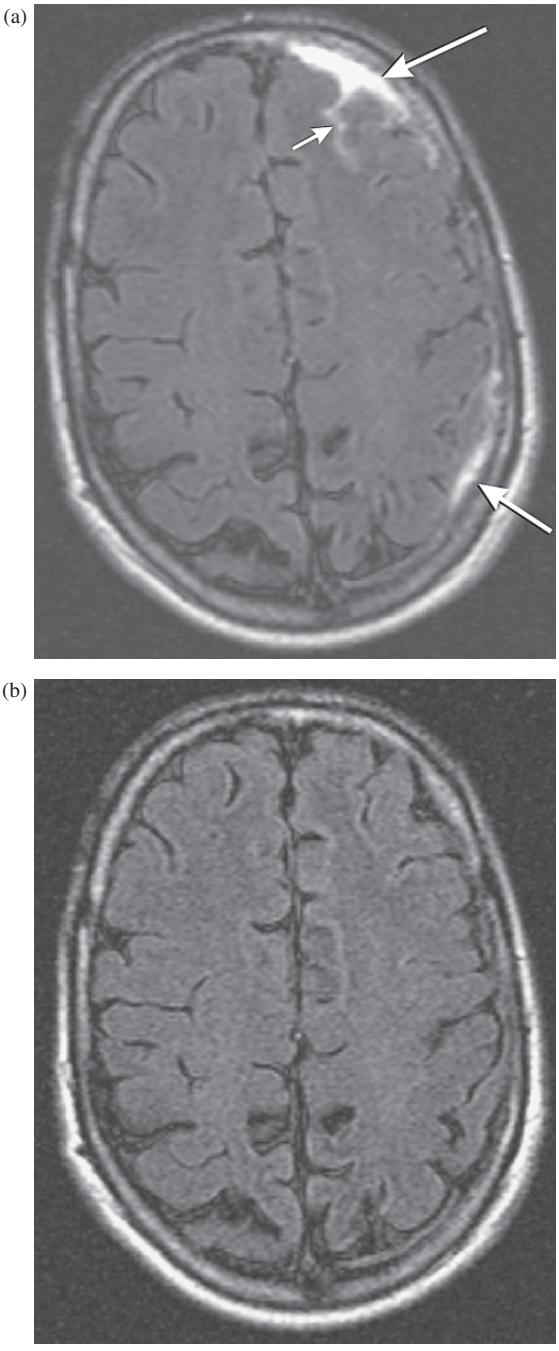


Figure 10–10. Example of an epidural hematoma. Due to the rapid increase in intracranial pressure, significant mass effect is seen. Unless evacuated soon after the injury, such lesions often prove fatal.

extensive, it also can produce increased intracranial pressure and brainstem compression. In such cases, surgery generally is indicated, especially if there are signs of rapidly increasing intracranial pressure. In many cases, the subdural bleeding is much more controlled or protracted (i.e., subacute). While signs of focal pressure may be present, the danger of herniation is much less. Particularly in chronic alcoholics, it is not uncommon



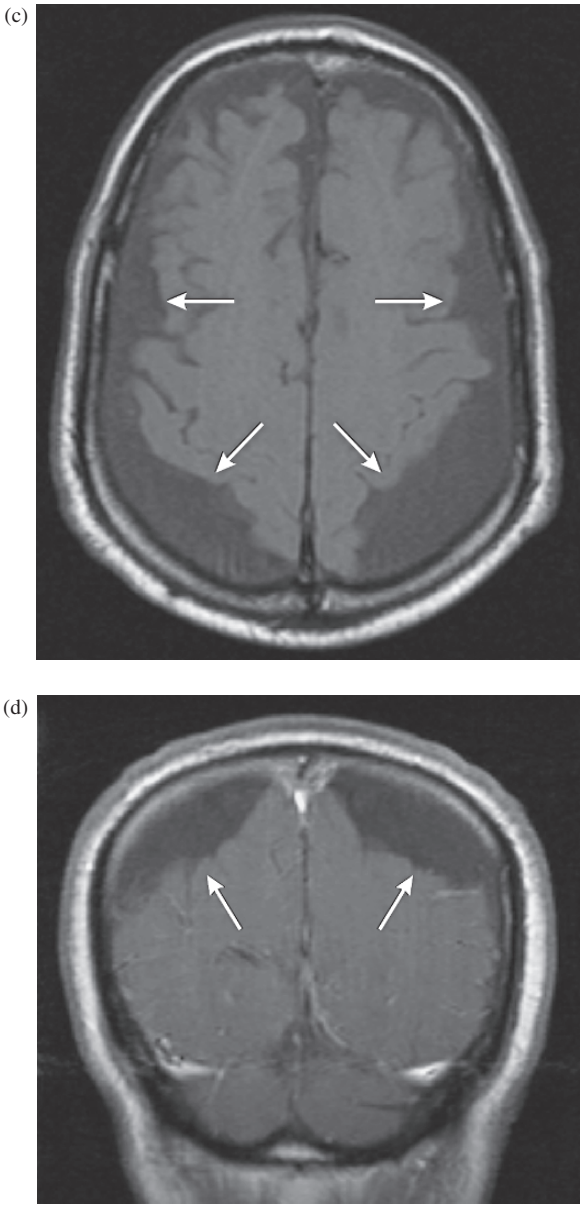


Figure 10–11. Because they typically involve venous bleeding, subdurals often develop more slowly and may be diffused over the surface of the hemisphere, and thus often associated with more minimal clinical effects. Figure 10–11a shows two more limited subdurals (large arrows), combined with area of subarachnoid bleeding (small arrow) as seen on FLAIR images (a CT scan on this same patient was read as "normal"). A second FLAIR image 2 months later (b) shows the same patient after the blood had been reabsorbed. Images (c) and (d) show residuals of large chronic bilateral subdural hematomas that have become filled with fluid. At this stage these fluid-filled pockets are often referred to as "hygromas."

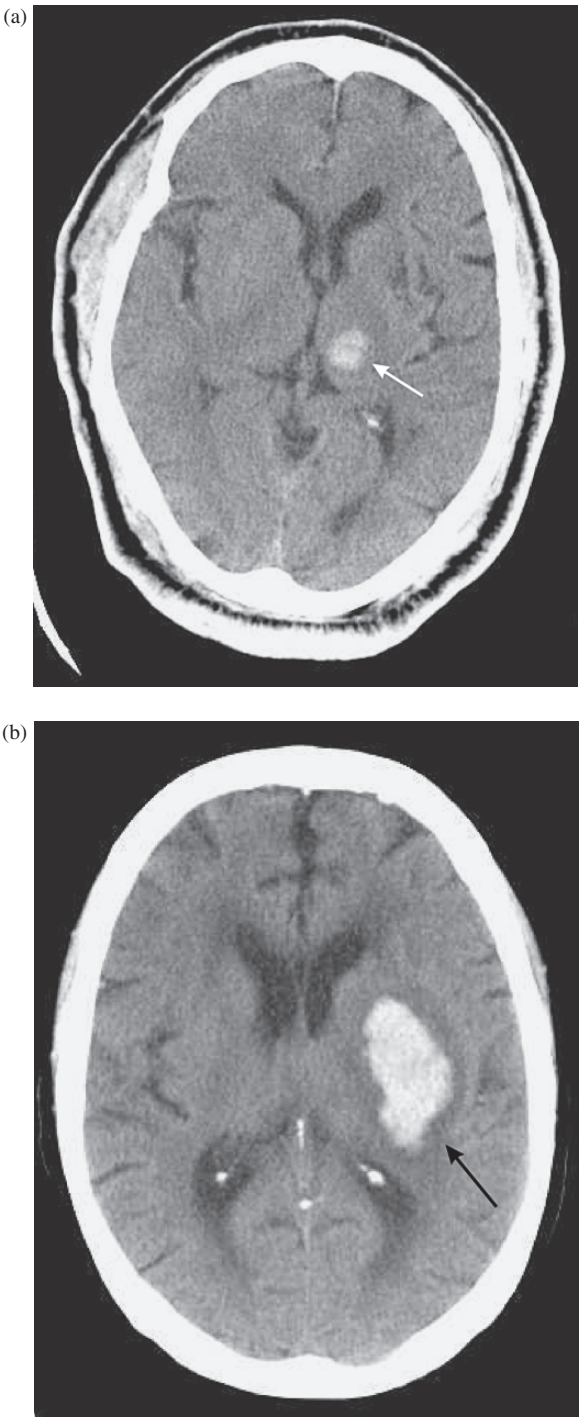
to find evidence of chronic or old subdural hematomas on autopsy or subsequent MRI imaging that clinically had gone undetected at the time.

Subarachnoid hemorrhages, along with intraparenchymal bleeds, represent one of the most common causes of hemorrhagic strokes. Bleeding into the subarachnoid space may result from various events, but the most common are rupture of an aneurysm, leakage from an arteriovenous malformation (see below), extension of a primary intracerebral hemorrhage into the subarachnoid space, and trauma. At times the bleeding may be slow or subacute and very limited, and the symptoms, which usually include a headache, may go unheeded by the patient at the time. More commonly, the onset is quite acute and rather dramatic. Frequently occurring during strenuous physical activity, patients often experience the sudden onset of headache, which is typically described as the "worst of their life." There also may be some disturbance or loss of consciousness, confusion, nausea, dizziness, or vomiting. Seizures are not uncommon. Pain or stiffness of the neck commonly is present, and when present is strongly suggestive of a subarachnoid hemorrhage. Except in severe cases in which there may be a rapid rise in intracranial pressure, focal neurological signs, such as sensorimotor disturbances, often either are absent or transitory. Imaging studies and/or a spinal tap typically will confirm the diagnosis. In rare cases of small bleeds that may not show up on imaging, a tap will reveal grossly bloody or xanthochromic cerebral spinal fluid.

Additional complications of subarachnoid hemorrhage include the possibility of rebleeding (especially if a ruptured aneurysm was the initial cause of the bleed), bleeding into the parenchyma (brain tissue) itself, vasospasms with secondary ischemic infarctions, edema, and increased intracranial pressure (that may be exacerbated by the vasospasms). An acute hydrocephalus may result as the flow of CSF is obstructed. A communicating-type hydrocephalus (e.g., "normal pressure hydrocephalus") may develop at a later date due to the reduced absorption capacity of the villi in the subarachnoid space as a result of the original bleed. This latter syndrome typically presents with disturbances of gait, urinary urgency or incontinence, and changes in mental status.

Intracerebral or **intraparenchymal hemorrhages** are those that invade the brain tissue itself (Figure 10–12). The most common cause of intracerebral hemorrhage is hypertension, which produces changes in the walls of the small, central penetrating vessels. Chronic hypertension weakens the vessel wall and can result in a ballooning of the vessel wall, producing what is termed **pseudoaneurysms**.[8] These pseudoaneurysms may rupture with extravasation of blood into adjacent brain regions. The regions most vulnerable to small-vessel, hypertensive hemorrhagic strokes are the putamen, thalamus, cerebellum, and pons, but may occur in any subcortical structure supplied by these small, penetrating vessels (e.g., subcortical grey and white matter and brainstem). Although hypertension and its related arteriolosclerotic changes in the small cerebral vessels are the most common causes of spontaneous intracerebral hemorrhages, a variety of other conditions also are associated with increased risk for intracerebral hemorrhage. Among the more common are AVMs, aneurysms, amyloid angiopathy, blood dyscrasias, drugs (particularly anticoagulant therapy, amphetamines, and cocaine), primary brain tumors, and embolic infarctions. Trauma, especially any type of penetrating head wound, is another common cause of hemorrhaging within the brain.

The signs and symptoms of intracerebral hemorrhage, as well as its prognosis or morbidity, depend on a variety of factors including the size of the bleed, its progression, and its location. With regard to size and progression, two general scenarios may be identified. In the first, the bleeding may be slow and/or relatively circumscribed. Since the brain tissue



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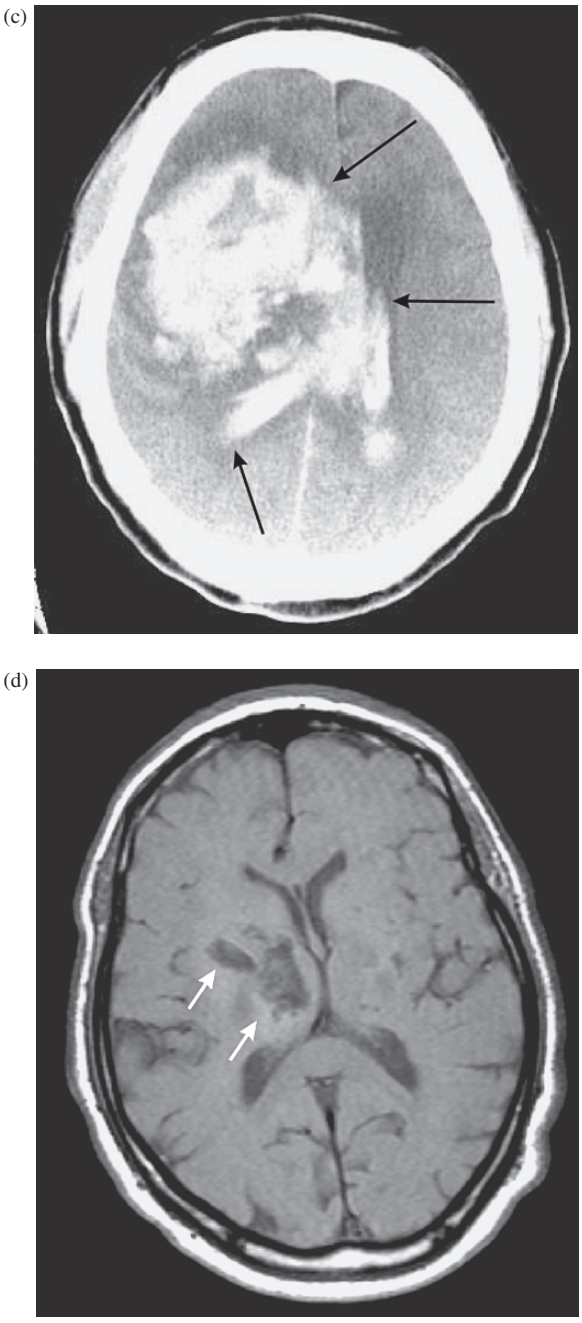


Figure 10–12. (a) Small thalamic, (b) larger putamenal, and (c) massive intraparenchymal hemorrhages. (d) Smaller hemorrhages may eventually (c) resolve with minimal to moderate clinical effects, although here signs of mass effect and gliosis are still present. Larger hemorrhages, as seen in (c) with intraventricular extension are typically incompatible with survival due to a rapid and massive increase in intracranial pressure and herniation.

itself as opposed to the meninges has no pain receptors, the patient may experience little or no headache. If headache does occur, it is most likely related to increased intracranial pressure. In addition to focal findings that are dependent on the site of the bleed, some patients may experience general confusion, nausea and vomiting, seizures, and/or changes in the level of consciousness. If the hemorrhage is relatively small, as in many small-vessel, hypertensive hemorrhages, the patient may present with focal findings and it may be difficult to differentiate clinically from an occlusive ischemic stroke, except on CT scan. In the second scenario, if the bleeding is more rapid and/or continues to expand to a critical size, the initial onset may be heralded by focal symptoms, as well as by headaches and nausea and vomiting (signs of increased intracranial pressure). Very quickly the patient will generally lapse into coma as a result of this rapidly increasing pressure and brain herniation. The latter picture is much more likely to be associated with mortality or increased morbidity.

Both the symptoms and the rate of survival from intracerebral hemorrhages also depend on the locus of the lesion. As will be discussed in greater detail in the next section, the clinical syndrome in large part will reflect the areas of the brain and/or pathways most directly impacted by the hemorrhage. Unlike occlusive (ischemic) infarcts, which deprive a large area of the brain cells of life-supporting oxygen, bleeding into the parenchyma affects the brain in a different fashion. While occlusive or ischemic lesions have their main effects in the regions of their distribution distal to the site of the blockage, hemorrhagic lesions primarily impact the area surrounding the bleed itself. First, intracerebral hemorrhage interrupts normal brain function by its direct mass effect. The hemorrhagic lesion puts increased pressure on the cells, axonal pathways, and capillary vessels, particularly those that are at the boundaries or in the immediate vicinity of the clot. In addition to this focal pressure, there also are the adverse effects due to a more generalized increase in intracranial pressure. These effects are enhanced by the secondary edema that occurs in addition to the space-occupying presence of the clot. Free blood in the extracellular spaces also seems to have a toxic or detrimental effect on the functioning of the cells that it directly impacts. Secondary ischemia may result from the various changes in blood flow and the effects on local vessels. Nonetheless, as noted earlier, if the organism survives the initial shock of an intracerebral hemorrhage, the possibility of recovery of function seems to be greater than if a comparable area was totally infarcted by occlusive cerebrovascular disease.

As might be expected, lesions that are confined to the area of the basal ganglia are likely to produce contralateral motor deficits (dyskinesias). However, somatosensory symptoms also may result. Thalamic bleeds may evoke similar symptoms except that, in the earlier stages especially, somatosensory deficits might predominate over motor deficits. The internal capsule commonly is involved following both thalamic and basal ganglia hemorrhages, frequently resulting in hemiplegia or hemiparesis. In capsular lesions, the upper face typically is spared because of its bilateral innervation, but the lower face, along with the leg and the arm, equally may be affected. In either site, but particularly with primary thalamic bleeds, a dysphasic syndrome (subcortical aphasia) may accompany left hemispheric hemorrhages. Other cognitive deficits, such as hemispatial neglect, also may follow thalamic lesions. Disturbances of oculomotor function, as well as visual field defects (hemianopia), are not uncommon with either basal ganglia or thalamic bleeds as a result of encroachment onto various pathways (e.g., from frontal eye fields, subthalamic connections, or optic tracts or radiations).

Intracerebral hemorrhages that occur in one of the lobes of the brain (lobar hemorrhage) will produce symptoms normally associated with that area of the brain. For example, contralateral motor findings are common with frontal lesions and contralateral sensory losses or neglect with parietal damage. Visual field loss may occur following occipital hemorrhages, although partial visual field cuts also can result from temporal (*superior quadrantanopia*) or parietal (*inferior quadrantanopia)* lesions, as a function of the interruption of the underlying

optic radiations. Disturbances of higher cortical functions also would expect to be affected, such as language, visual spatial constructions, or selective memory. Often these cortical syndromes will be both subtler but at the same time more inclusive than those caused by occlusive disease. With lobar hemorrhages, the patient may experience focal headaches, (again due to pressure effects), especially with frontal or occipital involvement, and focal or generalized seizures are not uncommon.

Another relatively common site of hemorrhage is in the brainstem, particularly in the pons. Rapid loss of consciousness is typical and the mortality rate is high. Neurologically, the patient frequently is quadriplegic and may show decerebrate rigidity. If the signs of corticospinal involvement are unilateral, opposite-sided cranial nerve findings are likely. Oculomotor deviations and loss of pupillary responses are common in brainstem lesions involving the pons or midbrain. Breathing also is commonly affected.

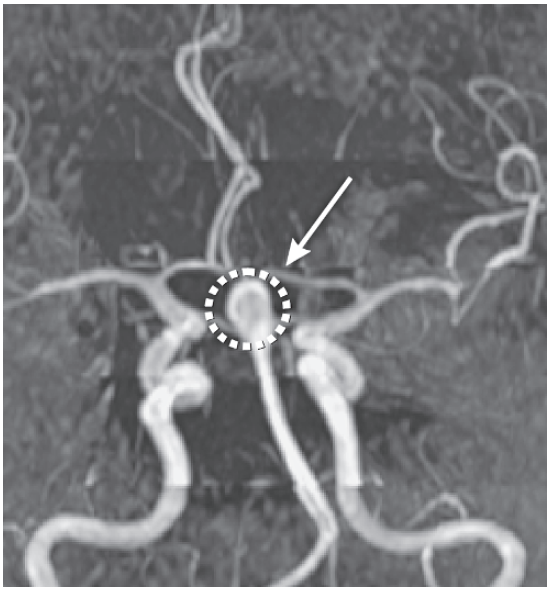
Cerebellar hemorrhages typically are associated with nausea, vertigo, vomiting, and posterior headaches. Neurological examination typically reveals marked disturbances of gait and truncal ataxia, upper limb dysmetria, and dysarthric speech. Hemiparesis or visual field defects are not present as part of the cerebellar picture, but additional signs of brainstem compression may develop, including oculomotor findings, extensor plantar responses, and loss of consciousness, or more commonly obtundation if the hemorrhage reaches sufficient magnitude.

Vascular Anomalies

Aneurysms

Aneurysms represent the ballooning of the wall of a vessel, typically an artery (Figure 10–13. They can be extremely small, as in the case of the *microaneurysms* that may develop on the smaller penetrating arteries as a result of chronic hypertension. Microaneurysms likely represent one cause of lacunar infarctions or hemorrhages deep within the brain. In contrast, other aneurysms may enlarge to greater than 1 centimeter in diameter (*giant aneurysms*). Giant aneurysms usually are derived from the more proximal vessels, and if of sufficient size can act like a space-occupying lesion, exerting pressure on adjacent neurons or fiber

(a)



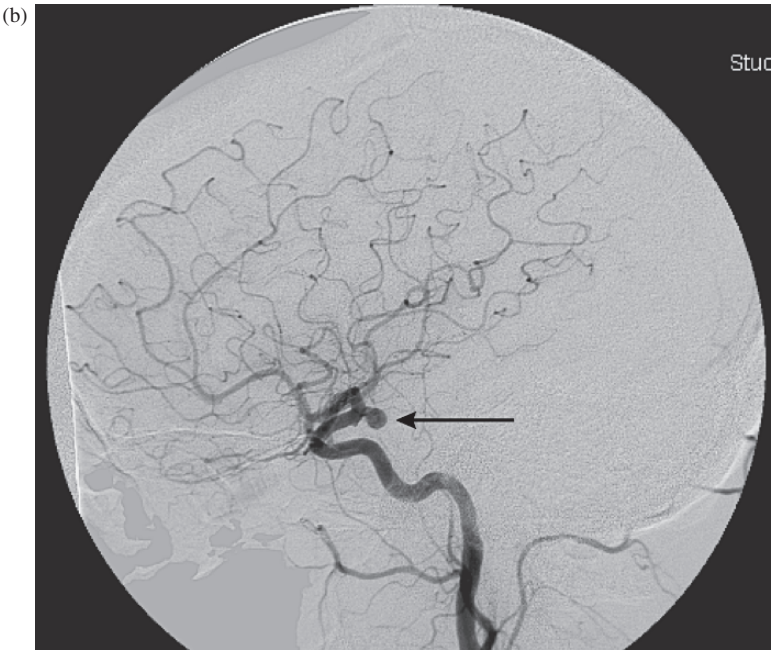


Figure 10–13. (a) Aneurysms (circled) at the tip of the basilar artery and (b) at the point of origin of the posterior communicating artery. The former is a standard MRA, while the latter represents a DSA (digital subtraction or conventional angiogram.

tracts. The majority of aneurysms appear to develop from a congenital weakness in the wall of the vessel, typically at the site of the bifurcation or branching of the artery or possibly at the site of a vestigial artery. These are known as *saccular* or *berry* aneurysms, the former representing a broad-based bulge in the artery, while the latter has a clearly defined stalk or "neck." Both of these are most commonly found in the carotid system, in or around the circle of Willis. Aneurysms also may be formed as a result of bacterial infection (*mycotic aneurysms*) or arteriosclerotic disease (*fusiform aneurysms*). The mycotic aneurysms often resemble berry-type aneurysms and more commonly are found in the carotid distribution (e.g., ACA or MCA). Fusiform aneurysms are more equally distributed in the carotid and vertebral systems and tend to be of the saccular type.

Many if not most aneurysms go undetected until they leak or rupture. Except for microaneurysms that may present as small infarcts, most ruptured aneurysms result in subarachnoid hemorrhages. The presenting symptoms are similar to that of other forms of subarachnoid hemorrhage discussed previously. Occasionally, a ruptured aneurysm may force blood directly into the parenchyma itself.

Special attention should be given to the rupture of aneurysms involving the area of the anterior communicating artery, a frequent site of cerebral aneurysms. Because the rupture of anterior communicating artery (ACoA) aneurysms directly affects the hypothalamus (including the mammillary bodies), the basal frontal regions, and medial temporal structures, significant behavioral symptoms typically are associated with this syndrome. Although ACoA aneurysms initially may present like most subarachnoid hemorrhages with severe headache and loss of consciousness, if and when the patient recovers, various personality, behavioral, and memory changes frequently are noted (Alexander & Freedman, 1984; Damasio et al., 1985; De Luca & Diamond, 1995; Fasanaro et al., 1989: Gade, 1982; Greene

et al., 1995; Okawa et al., 1980; Talland et. al., 1967; Vilkki, 1985). These patients commonly demonstrate marked disorientation and major amnestic deficits, involving both retrograde and particularly severe anterograde losses. Elaborate confabulations are relatively frequent, although they typically will subside over time (as do most confabulatory tendencies). The memory disturbances also generally will improve with time, but the extent of final recovery will vary among patients. In addition to memory, behavioral and affective changes also are quite common. While some variation is found, these patients typically demonstrate a number of symptoms associated with frontal pathology, including apathy, disinhibition, poor judgment, euphoria, dysphoria, or increased irritability. Cognitive or intellectual impairment may occur, but the memory and behavioral changes usually are more dramatic. As with memory loss, many patients will evidence an improvement in behavioral disturbances over time.

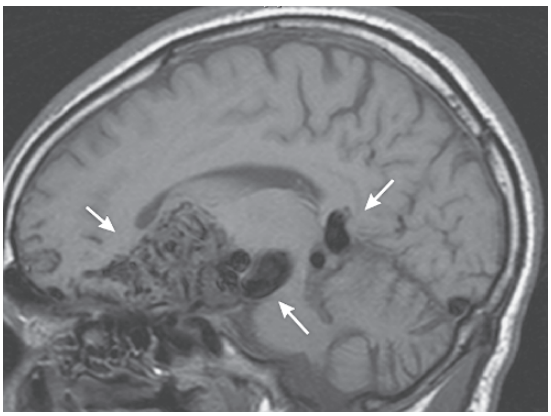
Even without rupturing, aneurysms still may present symptomatically because of their mass effect. This is particularly true around the circle of Willis and brainstem where they may impinge on sensitive structures, such as cranial nerves. Thus, unilateral anosmia, visual field cuts, and oculomotor disturbances (diplopia, ptosis, gaze palsy, unequal pupillary responses) are not uncommon.

Arteriovenous Malformations

Arteriovenous malformations (AVMs) represent another type of developmental vascular malformation. There are multiple types of AVMs, but perhaps most typically they represent a lack of development of the normal capillary network between a group of arteries and veins. As a result, there is a more or less direct shunting of blood from the arterial to venous system in the affected vessels. Over time these vessels tend to enlarge into a massive network of engorged artery–venous anastomoses. The AVMs can be composed primarily of surface vessels or can be embedded deep within the hemisphere. Size of AVMs may vary between relatively small or focal vascular malformations to quite massive anomalies, sometimes encompassing huge portions of a hemisphere, as seen in Figure 10–14.

The effects of vascular malformations can be multiple. First and foremost, AVMs and other vascular anomalies are subject to leakage or rupture, producing either a subarachnoid or an intracerebral hemorrhage. Because there is a direct shunting of blood from the arteries to the veins, lowering of arterial blood pressure and focal ischemia may occur. Because of their size, which increases over time, AVMs also may create a mass effect; however, since their growth is extremely slow, this effect usually is minimal. It is not uncommon for patients to go into their third or fourth decade or beyond before the AVM is discovered, and even

(a)



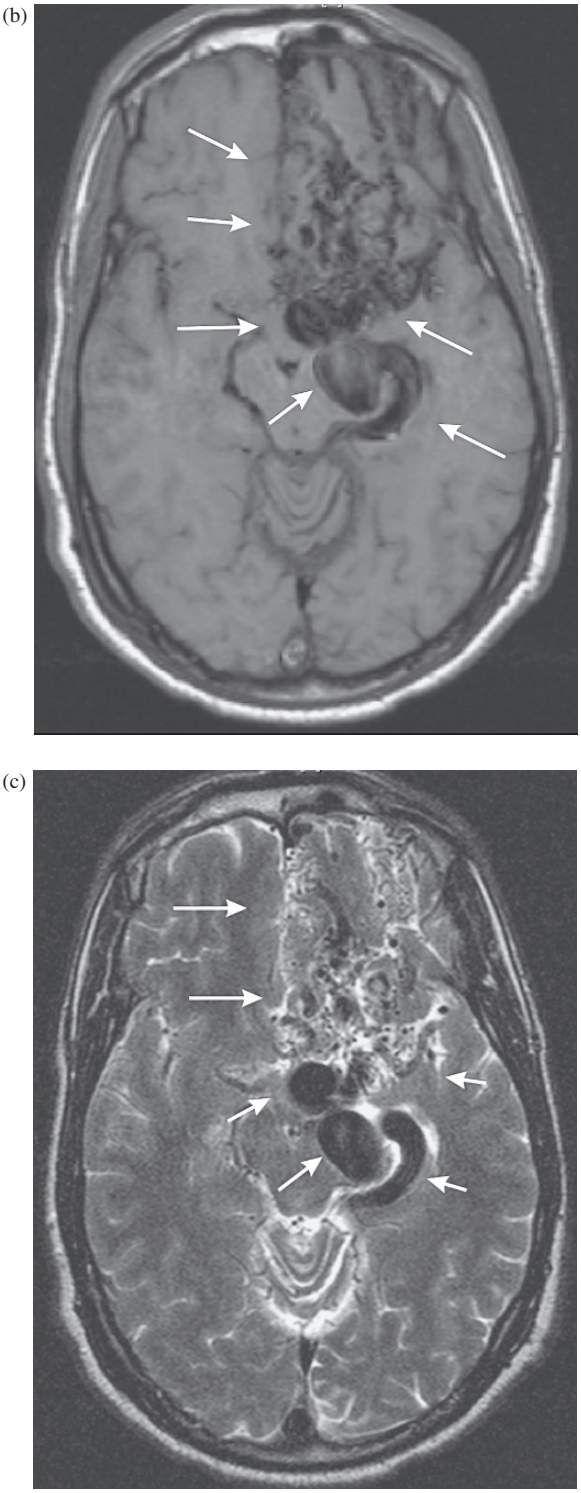
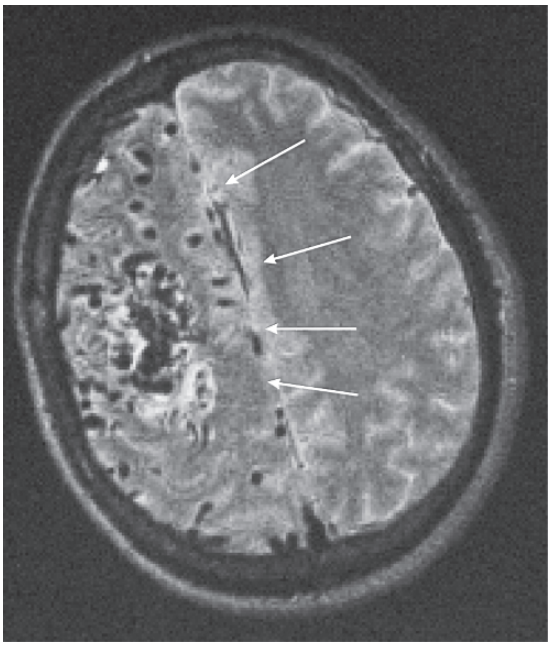
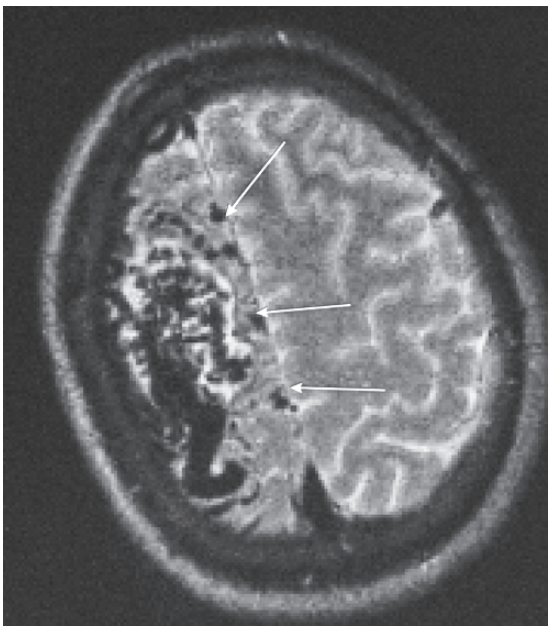


Figure 10–14. (*Continued*)

(d)



(e)



then usually only after some hemorrhaging occurs. Late-onset seizures perhaps are the most common presenting symptom of non-hemorrhagic AVMs, especially when accompanied by persistent focal vascular headaches. Over time, more neurological or behavioral symptoms may develop, as the AVM continues to enlarge and greater ischemic effects occur.

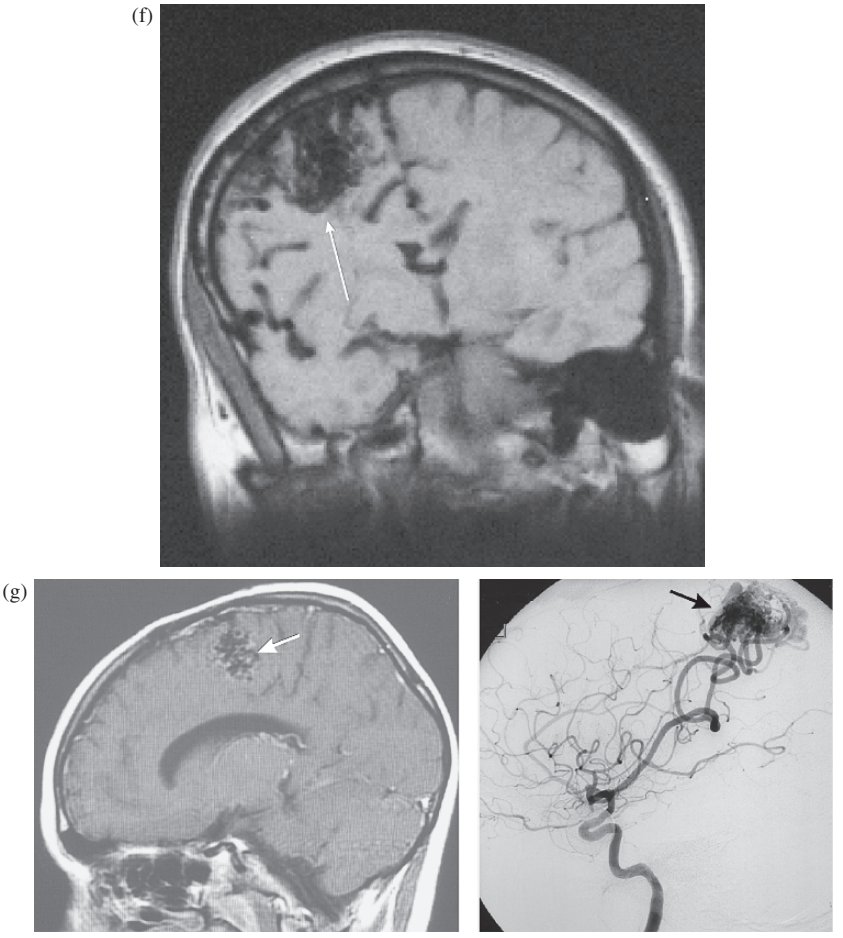


Figure 10–14. Large AVMs of (a–c) left and (d–f) right cerebral hemispheres. Because of slow growth, such lesions often are asymptomatic until they bleed and/or result in seizures. First patient (a–c), in addition to seizures, presented only with a mild right-sided weakness. The second patient (d–f) also had a history of seizures, but no other clear neurobehavioral or cognitive symptoms. Last image (g) compares the results of a MRI and DSA in the same patient. (MRIs shown in a, b, f, and g are T-1 weighted images; c, d, and e are T-2 weighted). (g) Courtesy of Dr. Jose Suros.

SPECIFIC VASCULAR SYNDROMES

As previously discussed, a number of variables play a role in determining the exact effects of vascular lesions. Hemorrhagic lesions produce very different effects from occlusive, ischemic lesions. The more proximal the lesion in a particular arterial distribution, the more devastating the impact and different patterns of deficits will emerge depending on the particular distal branches affected. Occlusive disease secondary to atherosclerotic processes, which develop slowly, allow for the possibility of greater collateral circulation development, thus possibly mitigating the effects of the stroke. Multiple strokes have more than a simple "additive" effect. While the overall vascular patterns generally are similar from one individual to the next, important variations may be common. The bottom line is that it is difficult to completely recreate or compare the particular stroke suffered by one

patient with the stroke of another. However, it is possible to discuss, at least in general terms, the syndromes produced by the blockage of certain arteries or arterial systems.[9]

Internal Carotid Artery Syndrome

As noted, a major factor in internal carotid disease symptomatology is the period of time over which the stenosis takes place. If it happens slowly enough, sufficient collateral circulation may develop so that the patient may experience relatively mild, if any, overt neurological deficits. Often, however, transient ischemic attacks (TIAs) may signal the presence of internal carotid artery stenosis. Amaurosis fugax (temporary blindness or dimming of vision in one eye as a result of the involvement of the ophthalmic artery) is a relatively common warning sign of carotid disease. Other indications might include transient episodes of weakness, somatosensory disturbances, or impairments of speech or language. The presence of a bruit (abnormal swishing sound) in auscultation of the neck is further suggestive of carotid artery stenosis and might indicate either Doppler studies or angiography for a more definitive diagnosis. Then, depending on the nature and severity of the disease process, differential treatment options might be considered. The possibility of complete internal carotid artery occlusion potentially is catastrophic, especially in the absence of adequate collateral circulation. Not only might both the ACA and MCA distributions be compromised (resulting in massive functional deficits), but also the resulting edema from such a large area of infarction can be life-threatening. If the patient survives such a stroke, among the impairments that might be expected are:

1. Contralateral hemiplegia and hemisomatosensory disturbances.
2. Homonymous hemianopia (secondary to involvement of the optic radiations).
3. Marked or global aphasia (if dominant hemisphere is involved).
4. Severe visual–spatial and other "higher-cognitive" deficits.
5. Emotional–behavioral changes.

More commonly, the loss of circulation will be somewhat more restricted, especially in embolic disease, typically affecting the MCA more than the ACA. Some variants of the typical vascular pattern may occur in which, for example, both ACAs as well as one of the PCAs may be supplied by one internal carotid. In these cases it is possible, although not common, to develop a bilateral frontal syndrome or a PCA syndrome with or without additional findings from occlusions of this single carotid system. A third possible outcome of stenosis of the internal carotid artery is the development of a borderzone or watershed-type infarction. As previously noted, this syndrome also can result from systemic hypotension, rapid blood loss, or hypoxia. In this situation the patient typically will manifest various cognitive or behavioral deficits consistent with the hemisphere involved that usually will not be restricted to the symptom pattern seen with focal cerebral infarctions. Sensorimotor losses may be minimal compared to the cognitive disturbances following watershed infarctions involving the lateral cortices.

Anterior Cerebral Artery Syndrome

Focal infarctions of the ACA are considerably less common than infarctions of the MCA, especially those of embolic origin. Some of the expected findings following infarction of the ACA are contralateral weakness, greater in the lower extremity and possibly proximal portions of the upper extremity. Some cortical sensory disturbances (e.g., two-point discrimination) may be present in the same distribution due to involvement of the medial–dorsal parietal cortex. Involvement of the cingulate gyrus may lead to urinary urgency or incontinence, at least temporarily. This latter problem is likely to be more severe and more

permanent if previous comparable lesions in the opposite hemisphere also are present. Since the ACA supplies the anterior portions of the corpus callosum, a partial disconnection syndrome may be detected, particularly ideomotor apraxia involving the nondominant hand. Again, while the effect is likely to be much more dramatic if bilateral lesions are present, frontal lobe symptomatology (e.g., decreased initiative or spontaneity, emotional lability or apathy, disinhibition, perseveration, frontal release signs) may be observed. In addition to contralateral leg weakness, gait ataxia may be present, along with an initial gaze preference toward the side of the lesion. If the dominant hemisphere is involved, elements of transcortical motor aphasia also may be seen.

Anterior Choroidal Artery Syndrome

Because this artery supplies blood to the posterior limb of the internal capsule, as well as to the lateral geniculates, infarction may lead to the combination of contralateral hemiparesis (or hemiplegia) and a contralateral homonymous hemianopia or quadrantanopia. Mild somatosensory deficits also may be found on the involved side.

Middle Cerebral Artery Syndrome

The anatomy of the MCA makes this vessel a prime candidate for embolic infarction from the carotid arteries, the heart, or lungs. Since the MCA is the largest cerebral artery, a proximal occlusion is quite devastating, producing a syndrome similar to that described for the internal carotid artery. Complete occlusion can result in:

1. Total contralateral hemiplegia (sparing the upper portion of the face), hemisensory impairment and hemianopia.
2. Unilateral neglect, global aprosodia, and/or marked visual spatial deficits (more common with right hemisphere lesions).
3. Global aphasia and ideomotor apraxia (left hemisphere lesions).
4. Memory and perceptual deficits (the exact nature of which will depend on the hemisphere involved).
5. Frontal executive deficits.[10]

Since the ACA is spared, initial lower extremity weakness is likely to improve faster than that of the upper extremity. It is not uncommon for more distal, individual branches of the MCA to be selectively occluded. In these cases the symptoms will depend on the specific territories and hemisphere involved, as well as the efficiency of collateral circulation.

Occlusion of one or more anterior branches of the MCA (e.g., the prefrontal or orbitofrontal branch) may produce frontal lobe symptoms (these often can be subtle with unilateral disease: see Chapter 9). Motor weakness or paralysis and/or cortical somatosensory symptoms with the upper limb generally being more affected than the lower can occur following an occlusion of the middle branches of the MCA (e.g., precentral, central branches, or postcentral branches). Syndromes of nonfluent aphasia (dominant hemisphere) or expressive aprosodia (nondominant hemisphere) also may occur following infarction of the anterior branches of the MCA. Occlusion of the more posterior parietal branches of the MCA (e.g., anterior, posterior, and/or angular parietal branches) may produce visual spatial problems (right or left hemisphere) or language problems (particularly visual-based processes such as reading and writing) if the left hemisphere is affected. An inferior quadrantanopia may result from an extension of the lesion to the underlying optic radiations. If the angular gyrus in the dominant hemisphere is affected, most if not all of the symptoms of Gerstmann's syndrome may be present. Contralateral neglect or anosognosia is common, especially in the acute stages of nondominant (right) hemispheric lesions.

If the posterior temporal branches of the MCA are involved, fluent aphasia (left hemisphere) or receptive aprosodia (right hemisphere) may be observed, with little or no motor weakness. Interruption of the optic radiations in the temporal region (Meyer's loop) may produce a superior quadrantanopia. Selective memory deficits may occur, probably secondary to perceptual or encoding difficulties. With selective involvement of the lenticulostriate arteries that supply the basal ganglia and parts of the anterior and posterior limbs of the internal capsule, dyskinesias or a contralateral hemiparesis or hemiplegia with dysarthria may result, in the relative absence of "higher-cortical" deficits.

Posterior Cerebral Artery Syndrome

The symptom most commonly associated with posterior cerebral artery disease is contralateral hemianopia due to lesions affecting the primary visual (striate) cortex (calcarine artery). If the calcarine branches of both PCAs are simultaneously affected, not only will the patient be cortically blind, but he or she may develop anosognosia for visual input (*Anton's syndrome*), characterized by lack of awareness or denial of his or her blindness) (Redlich & Dorsey, 1945). If both the left calcarine cortex, as well as the splenium of the corpus callosum are affected, the patient may manifest the syndrome of *alexia without agraphia* (Damasio & Damasio, 1983). However, depending on the particular branch of the PCA that is involved, visual field cuts may or may not be present. Despite the fact that there may not be a demonstrable field cut, other visual disturbances may be present, such as *apperceptive agnosias, color recognition* or *color-naming deficits, simultanagnosia* or a more complete *Balint's syndrome*, and *prosopagnosia* (difficulty recognizing faces). These syndromes usually occur following parietal–occipital or temporal–occipital lesions that may result from borderzone lesions affecting the PCA and MCA distributions (for review, see: Bauer, 1993; Damasio, 1985; DeRenzi, 1997; Farah, 1997; Tranel, 1997). Occasionally, visual hallucinations may result from occlusions of the PCA, but these are more commonly found with irritative or toxic lesions.

In addition to supplying the area around the calcarine fissure, the anterior and posterior temporal branches of the PCA also supply the inferior and medial temporal regions. Infarction of this area can produce severe impairment in the ability to encode new information (anterograde memory), especially if both hemispheres are involved. An acute state of confusion or delirium also may result. As noted above, the syndrome of transient global amnesia is thought to perhaps reflect a type of transient compromise of the temporal branches of the posterior cerebral arteries.

The posterior cerebral artery system also is responsible for supplying blood to parts of the upper portions of the brainstem and represents the main source of blood to the thalamus via the posteromedial and posterolateral penetrating or central arteries. Occlusion of these vessels can produce a variety of midbrain or thalamic syndromes. The resulting brainstem syndromes may include ipsilateral oculomotor (third nerve) findings, contralateral motor deficits (cerebral peduncle), tremor, hemiballismus or other extrapyramidal symptoms (red nucleus), and ataxia (superior cerebellar fibers). The combination of ipsilateral third nerve palsy and contralateral hemiplegia as a result of unilateral infarction of the midbrain is known as **Weber's syndrome**. Lesions of the thalamus can produce a combination of hemiparesis and hemisomatosensory loss, combined with hyperesthesia or thalamic pain syndrome. Disturbances of higher cortical functions, for example, aphasia or neglect, also may be observed.

Infarctions involving branches of the vertebral or basilar arteries will result in either brainstem and/or cerebellar lesions. In addition to cerebellar symptomatology (dyskinesias), infarctions of the pons (most frequent) or other areas of the brainstem can produce any of a large variety of symptoms associated with a disruption of the corticospinal and/or

spinothalamic tracts, combined with cranial nerve findings. See Chapter 4 for a review of several common brainstem syndromes.

NEUROPSYCHIATRIC SYNDROMES ASSOCIATED WITH VASCULAR LESIONS

When examining patients for the sequelae of cerebral vascular accidents, there is a natural tendency to focus primarily on signs and symptoms of motor, sensory, language, and other cognitive dysfunctions. However, as has been previously noted, particularly in the preceding chapter, lesions in various parts of the brain often are associated with significant "behavioral disturbances." Recall, for example, problems of disinhibition and agitation that can be associated with orbital–frontal lesions, the paranoia that occasionally may accompany fluent aphasic syndromes, the emotional flatness (*expressive aprosodia*) that can result from frontal–temporal lesions in the right hemispheric, or conversely the emotional incontinence (pathological laughter or crying) that is most commonly associated with deep bilateral frontal lesions. Another important concept to keep in mind is the possibility of neurologically based depression and the differentiation between depression versus apathy. If an individual suffers a stroke (with associated physical or cognitive deficits), it is easy to attribute his or her sadness to this subjective sense of "loss." While such "reactive" depressions indeed may occur, the increased frequency of clinically depressive syndromes following left anterior CVAs strongly suggests the possibility of a neurological substrate in many of these cases. Finally, it also is important to distinguish between depressive syndromes, which are more likely to be associated with strokes affecting the MCA territories (especially the left frontal dorsolateral cortex) versus apathy that is more likely to result from strokes affecting the midline frontal cortices (ACA distribution).[11]

Finally, diffuse cerebrovascular disease that results in multiple large- or small-vessel strokes may present as a progressive dementia. There is some controversy regarding the subtypes of vascular dementias (for a brief review, see Cummings & Benson, 1992). Although the majority of dementias may be the result of degenerative rather than vascular changes, vascular disease also is recognized as a common cause of dementia. In fact, it now is suspected that in many cases both degenerative and vascular components are contributory factors. What types of vascular diseases or changes are most likely to produce a dementia? Certainly multiple, large cortical infarcts can result in significant, generalized deterioration of cognitive and behavioral function consistent with a dementia. Multiple and clearly demonstrable lacunar infarctions also may produce a comparable condition. Bilateral borderzone infarctions, although not common, will produce a clinical picture of dementia. Infarctions that appear predominately in the periventricular white matter, the basal ganglia, and thalamus (*Binswanger's disease*) also have been associated with dementias.[12] Etiological factors that may facilitate these conditions include chronic hypertension, amyloid angiopathy, and atherosclerosis.

Endnotes

1. The major parts of the thoracic and lower portions of the cord are supplied by the radicular arteries, which enter the cord at the various segmental levels.
2. For a more complete discussion of this topic, the following resources are suggested: Biller (1990); Bornstein and Brown (1991); Caplan and Bogousslavsky (2001); Millikan,

McDowell, and Easton (1987); Toole (1990); for exceptionally comprehensive treatment of this subject, see Welch et al. (1997) and Mohr et al. (2004).

3. While neuroimaging techniques (e.g., MRIs and CT scans) provide more definitive evidence of the locus and extent of an infarction, false-negative results may be obtained, especially in the early stages of a stroke. Regardless of the presence or absence of such radiographic evidence, knowledge of functional neuroanatomy is still essential when attempting to map out the functional deficits resulting from the stroke.
4. These changes also can lead to the formation of microaneurysms in walls of the small vessels that may subsequently hemorrhage.
5. The meaning and use of these terms are subject to change. TIAs used to be defined as any deficit that appeared to resolve within 24 hours and an RIND as a deficit that cleared after 24 hours. Now TIAs commonly are defined as deficits that last from minutes to an hour (more or less), while the term "RIND" is rarely used.
6. The author (JEM) found that both finger tapping and a simple bedside coin rotation task often revealed mild residual impairments days after what was reported to be a TIA supposedly had cleared. (Mendoza, et al, 2007).
7. Edema is simply a collection of fluid within or surrounding certain tissues. Three types of edema generally are identified with regard to the brain. **Cytotoxic** or cellular edema results from the disruption of the normal metabolic processes within the cell, particularly the disruption of the "sodium–potassium pump" (see Chapter 11). As a result of this disruption, sodium accumulates within the cell, which in turn attracts water molecules, thus producing the edema. This type of edema is most commonly associated with vascular disease. **Vasogenic** edema, on the other hand, results from a breakdown of the blood–brain barrier through a disruption of the walls of the capillary vessels allowing plasma fluids to leak into the interstitial spaces. This type of edema, which tends to be more commonly associated with tumors, is often characterized by its frondlike projections within the white matter surrounding the neoplasm. **Hydrocephalic** edema typically is associated with obstructive hydrocephalus. As a result of the increased pressure within the ventricles from the buildup of cerebrospinal fluid, there is leakage through the ependymal cell walls of the ventricle into the surrounding white matter.
8. These are also commonly referred to as "micro-aneurysms" or Charcot-Bouchard aneurysms, but the term pseudoaneurysms may be preferable, as they do not appear to be true aneurysms, as classically defined. Like true aneurysms, however, they do represent a weakness in the walls of the vessel.
9. The following will focus primarily on arterial syndromes affecting the cerebral hemispheres. See respective chapters for lesions affecting other regions of the CNS.
10. See Chapter 9 for a more detailed listing of functions associated with right and left hemispheric lesions, as well as descriptions of "frontal executive functions."
11. For a more detailed review of the neuropsychiatric sequelae of strokes, the reader is referred to Robinson (1997, 1998).
12. Not infrequently, CT scan or MRI reports will note the presence of periventricular changes in the absence of any evidence of mental status change. Hence, caution is advised against making a diagnosis of subcortical leukoencephalopathy or Binswanger's disease based on neuroimaging techniques alone.

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