The Nervous System

14 The Skull, the Brain, the Meninges, and the Blood Supply of the Brain Relative to Trauma and Intracranial Hemorrhage

Chapter Outline

THE SKULL

The Thinnest Part of the Lateral Wall of the Skull

The thinnest part of the lateral wall of the skull is where the anteroinferior corner of the parietal bone articulates with the greater wing of the sphenoid; this point is known as the **pterion.** This is a very important area since it overlies the anterior division of the middle meningeal artery and vein.

Fractures of the Skull

See Chapter 11 of the CD-ROM.

Fractures of the Facial Bones

See Chapter 11 of the CD-ROM.

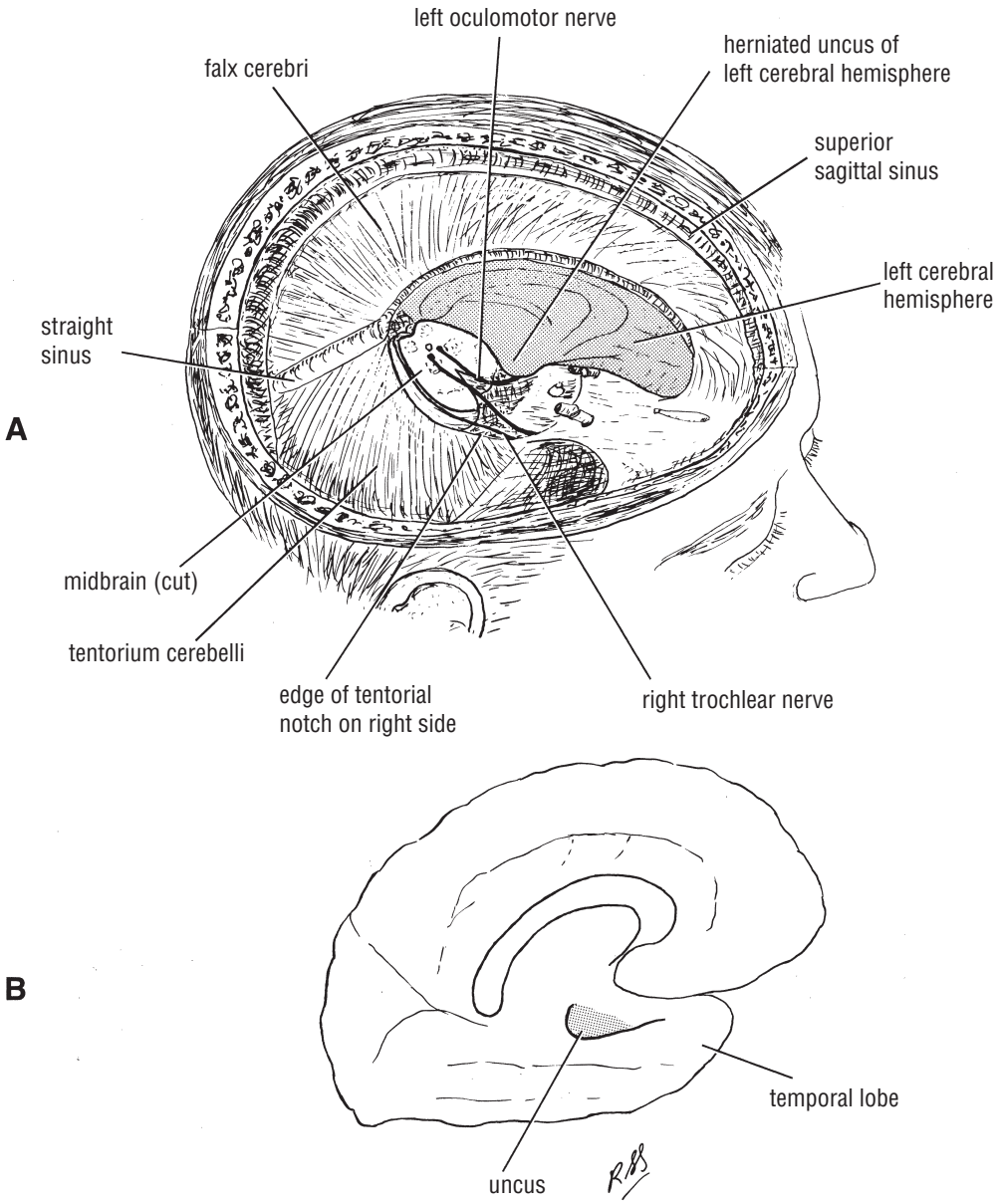
Rises in Supratentorial Pressure

The common causes of a rise in supratentorial pressure are intracerebral hemorrhage, subarachnoid hemorrhage, subdural hemorrhage, epidural hemorrhage, and cerebral edema. Two forms of caudal herniation of the brain through the tentorial notch of the tentorium cerebelli can occur as a result of the raised supratentorial pressure:

- **Central herniation syndrome:** In this syndrome the thalamus and midbrain are pushed caudally through the tentorial notch.
- **Uncal herniation syndrome:** In this syndrome the uncus of the temporal lobe is displaced medially and pushes the midbrain against the opposite sharp edge of the tentorial notch. At the same time, the displaced uncus presses on the ipsilateral oculomotor nerve at the notch, resulting in a sluggishly reactive and dilated pupil (CD Fig. 14-1).

Rises in Subtentorial Pressure

Posterior cranial fossa lesions such as those in cerebellar hemorrhage cause a rise in pressure that can directly com-

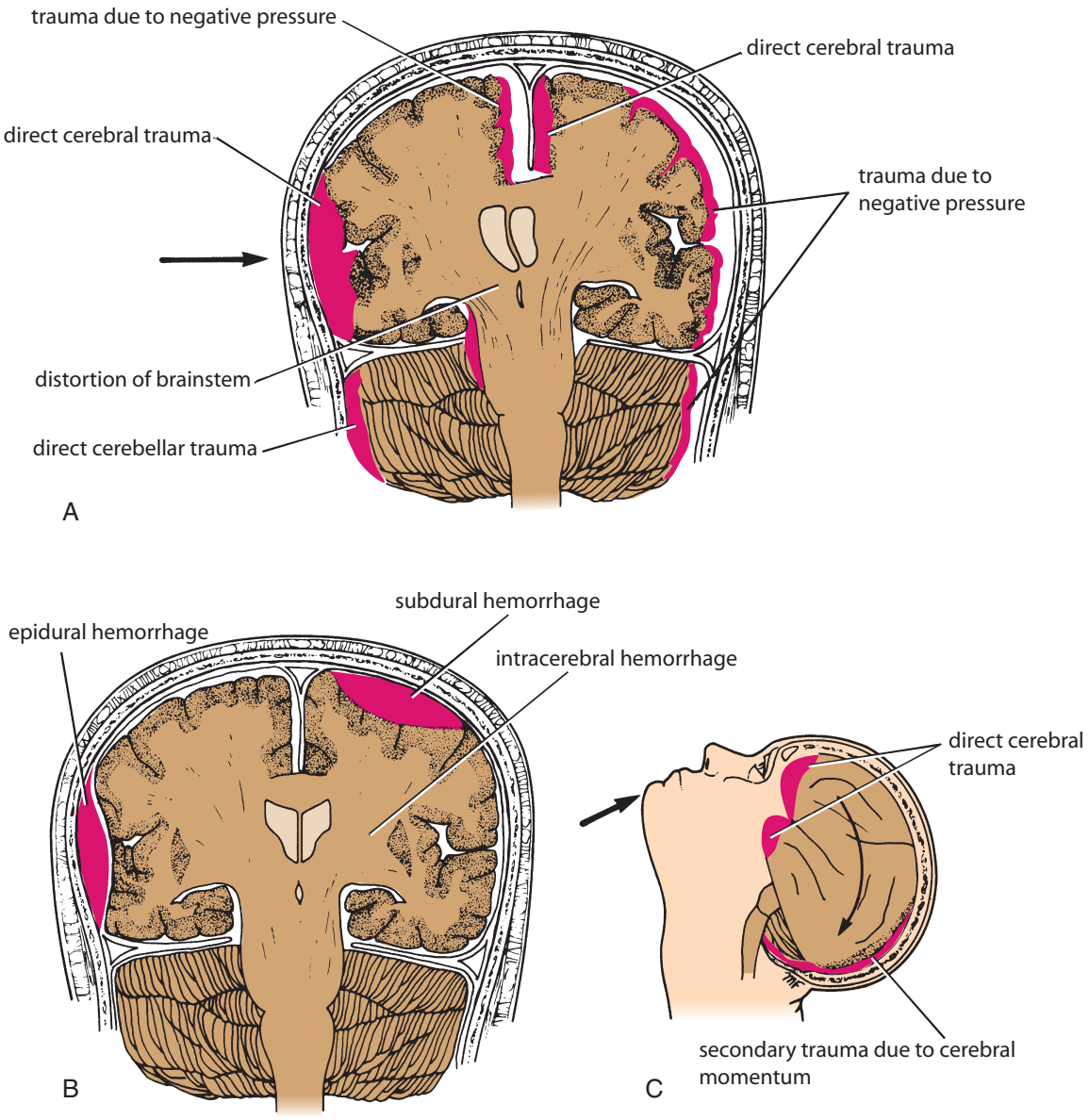


CD Figure 14-1 **A.** Lateral view of the interior of the skull showing the falx cerebri, tentorium cerebelli, and brainstem. As the result of abnormal supratentorial pressure, the uncus of the left cerebral hemisphere has herniated down through the tentorial notch of the tentorium cerebelli and is pressing on the left oculomotor nerve. **B.** The position of the uncus on the temporal lobe of the left cerebral hemisphere in a normal brain.

press the brainstem or its blood supply. Indirect compression can follow upward herniation of the cerebellum through the tentorial notch or downward herniation of the cerebellar tonsils through the foramen magnum. In the latter instance the medulla will also be displaced and pressed upon. The problem can be compounded by pressure on the cerebral aqueduct in the midbrain or the roof of the fourth ventricle, producing an acute obstructive hydrocephalus.

Movements of the Brain Relative to the Skull and Meninges in Head Injuries

Brain injuries are produced by displacement and distortion of the neuronal tissues at the moment of impact (CD Fig. 14-2).



CD Figure 14-2 **A.** Mechanisms of acute cerebral injury when a blow is applied to the lateral side of the head. **B.** Varieties of intracranial hemorrhage. **C.** Mechanism of cerebral trauma following a blow on the chin. The movement of the brain within the skull can also tear the cerebral veins.

The brain, which is incompressible, may be likened to a log soaked with water floating in water. The brain is floating in the cerebrospinal fluid in the subarachnoid space and is capable of a certain amount of anteroposterior and lateral gliding movement. The anteroposterior movement is limited by the attachment of the superior cerebral veins to the superior sagittal sinus. In lateral movements, the lateral surface of one hemisphere hits the side of the skull, and the medial surface of the opposite hemisphere hits the side of the falx cerebri (see CD Fig. 14-2). In superior movements, the superior surfaces of the cerebral hemispheres hit the vault of the skull, and the superior surface of the corpus callosum may hit the sharp free edge of the falx cerebri; the superior surface of the cerebellum presses against the inferior surface of the tentorium cerebelli.

It follows from these anatomic facts that blows on the front or back of the head lead to displacement of the brain, which may produce severe cerebral damage, stretching and distortion of the brainstem, and stretching and even tearing of the commissures of the brain. Blows to the side of the head produce less cerebral displacement, and the injuries to the brain consequently tend to be less severe. The falx cerebri, however, is a tough structure and may cause considerable damage to the softer brain tissue in cases where there has been a severe blow to the side of the head (see CD Fig.

14-2). Furthermore, glancing blows to the head may result in considerable rotation of the brain, causing shearing strains and distortion, particularly in areas where further rotation is prevented by bony prominences in the anterior and middle cranial fossae. Brain lacerations are likely to occur when the brain is forcibly thrown against the sharp edges of bone within the skull—the lesser wing of the sphenoid, for example.

When the brain is suddenly given momentum within the skull, the part of the brain that moves away from the skull wall is subjected to diminished pressure, because the cerebrospinal fluid has not had time to accommodate the brain movement (see CD Fig. 14-2). This results in a suction effect on the brain surface, with rupture of surface blood vessels.

A sudden severe blow to the head, as in an automobile accident, may result in damage to the brain at the following two sites: (1) at the point of impact and (2) at the pole of the brain opposite the point of impact, where the brain is thrown against the skull wall. This is referred to as **contrecoup injury**.

Movements of the brain relative to the skull and dural septa may seriously injure the cranial nerves that are tethered as they pass through the various foramina. This particularly applies to the long, slender nerves, such as the trochlear, abducent, and occulomotor nerves. Furthermore, the fragile cortical veins that tether the brain and drain into the dural sinuses may be torn, resulting in severe subdural or subarachnoid hemorrhage. The large arteries found at the base of the brain are tortuous, and this, coupled with their strong walls, explains why they are seldom damaged.

Intracranial Hemorrhage

Intracranial hemorrhage may result from trauma or cerebral vascular lesions. Four varieties are considered here: extradural, subdural, subarachnoid, and cerebral.

Extradural hemorrhage results from injuries to the meningeal arteries or veins. The most common artery to be damaged is the anterior division of the middle meningeal artery. A comparatively minor blow to the side of the head, resulting in fracture of the skull in the region of the anteroinferior portion of the parietal bone, may sever the artery. The arterial or venous injury is especially liable to occur if the artery and vein enter a bony canal in this region. Bleeding occurs and strips the meningeal layer of dura from the internal surface of the skull. The intracranial pressure rises, and the enlarging blood clot exerts local pressure on the underlying motor area in the precentral gyrus. Blood may also pass outward through the fracture line to form a soft swelling under the temporalis muscle.

To stop the hemorrhage, the torn artery or vein must be ligated or plugged. The burr hole through the skull wall should be placed about 1 to 1.5 in. (2.5 to 4 cm) above the midpoint of the zygomatic arch.

Subdural hemorrhage results from tearing of the superior cerebral veins at their point of entrance into the superior sagittal sinus. The cause is usually a blow on the front or the back of the head, causing excessive anteroposterior displacement of the brain within the skull.

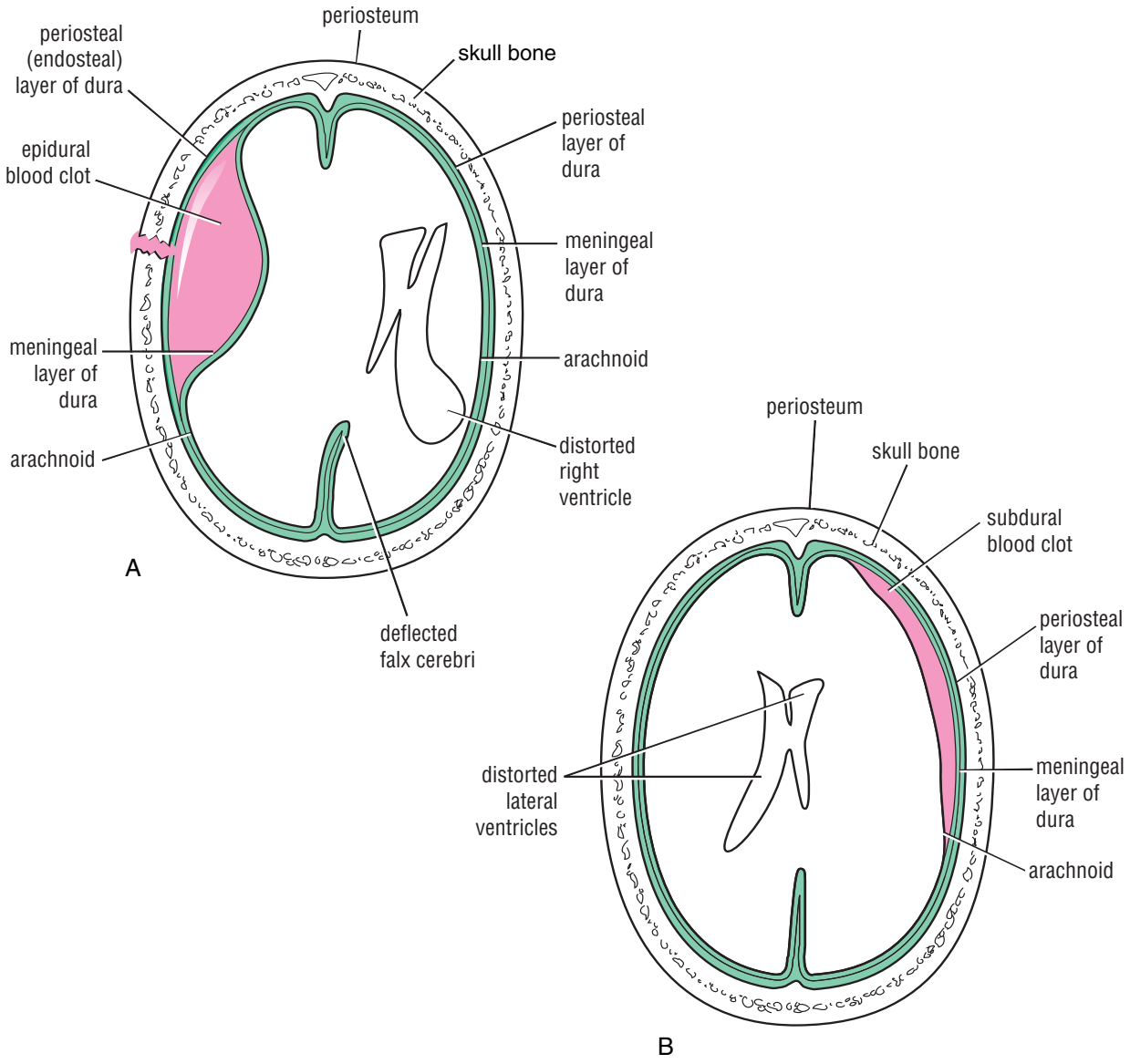
This condition, which is much more common than middle meningeal hemorrhage, can be produced by a sudden minor blow. Once the vein is torn, blood under low pressure begins to accumulate in the potential space between the dura and the arachnoid. In about half the cases the condition is bilateral.

Acute and chronic forms of the clinical condition occur, depending on the speed of accumulation of fluid in the subdural space. For example, if the patient starts to vomit, the venous pressure will rise as a result of a rise in the intrathoracic pressure. Under these circumstances, the subdural blood clot will increase rapidly in size and produce acute symptoms. In the chronic form, over a course of several months the small blood clot will attract fluid by osmosis so that a hemorrhagic cyst is formed, which gradually expands and produces pressure symptoms. In both forms the blood clot must be removed through burr holes in the skull.

COMPUTED TOMOGRAPHY SCANS OF EXTRADURAL AND SUBDURAL HEMATOMAS

The different appearances of blood clots in these two conditions as seen on computed tomography (CT) scans is related to the anatomy of the area (CD Fig. 14-3). In an extradural hemorrhage the blood strips the meningeal layer of the dura from the endosteal layer of dura (periosteum of the skull), producing a lens-shaped hyperdense collection of blood that compresses the brain and displaces the midline structures to the opposite side. The shape of the blood clot is determined by the adherence of the meningeal layer of dura to the periosteal layer of dura.

In patients with subdural hematoma the blood accumulates in the extensive potential space between the meningeal layer of dura and the arachnoid, producing a long, crescent-shaped, hyperdense rim of blood that extends from anterior to posterior along the inner surface of the skull. With a large hematoma, the brain sulci are obliterated, and the midline structures are displaced to the opposite side.

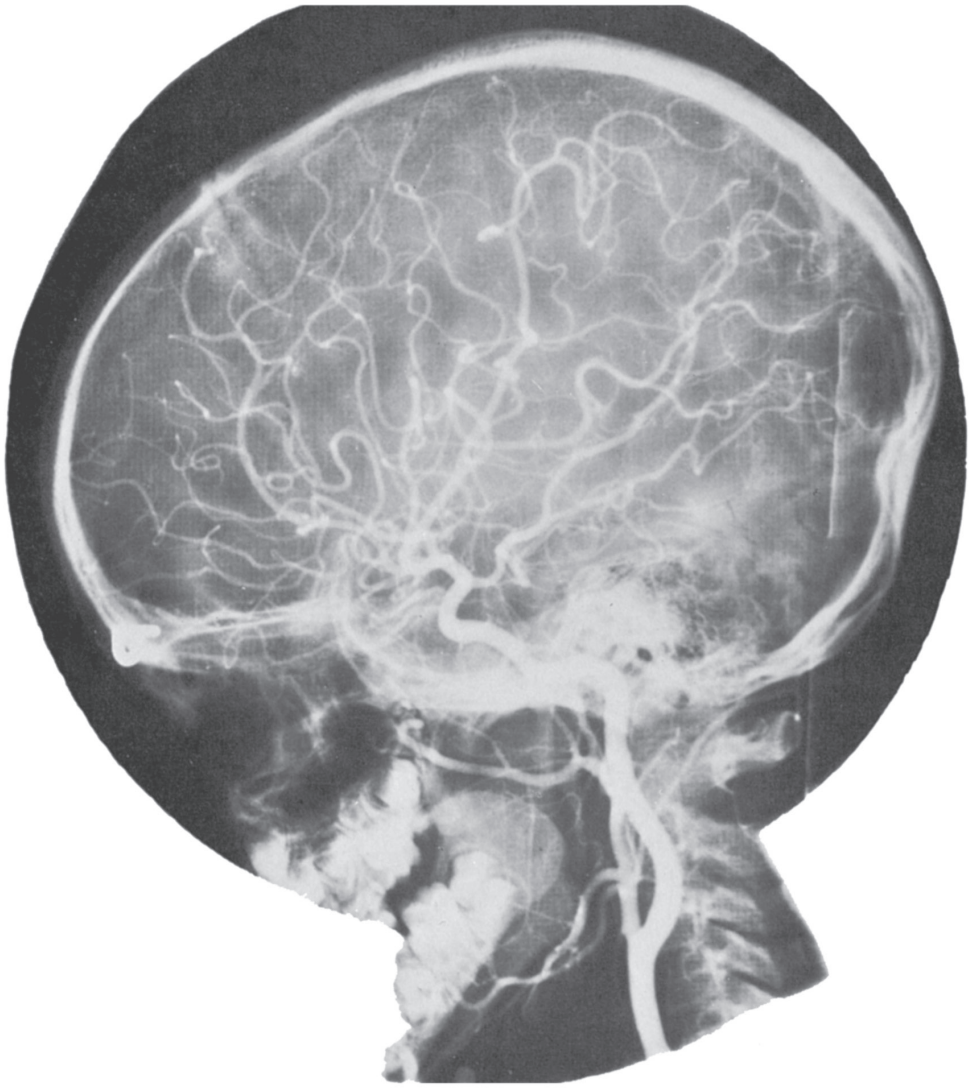


CD Figure 14-3 Diagrammatic representation of an extradural hemorrhage and a subdural hemorrhage. **A.** Extradural hemorrhage from the middle meningeal artery or vein on the left side. The hematoma is lens-shaped and occupies the space between the endosteal layer of dura (periosteum of the skull) and the meningeal layer of dura (true dura, hence the name extradural). **B.** Subdural hemorrhage from the cerebral veins at the site of entrance into the venous sinus on the right side. The hematoma is crescent-shaped and occupies the space between the meningeal layer of dura and the arachnoid, (i.e., beneath the dura).

CEREBRAL ARTERIOGRAPHY

The technique of cerebral arteriography is used to detect abnormalities of the cerebral arteries and localization of space-occupying lesions such as tumors, blood clots, or abscesses.

With the patient under general anesthesia and in the supine position, the head is centered on a radiographic apparatus that will take repeated radiographs at 2-second intervals. Both anteroposterior and lateral projections are obtained. A radiopaque medium is rapidly injected into the lumen of the common carotid or vertebral arteries. As the radiopaque material is introduced, a series of films are exposed. By this means the cerebral arteries can be demonstrated and their position and patency determined (CD Figs. 14-4 to 14-7).



CD Figure 14-4 Lateral internal carotid arteriogram.

This technique is not without risk because the insertion of a needle through the wall of an artery or the manipulation of a catheter within its lumen may dislodge an atheromatous plaque, leading to cerebral embolism.

Congenital Aneurysms

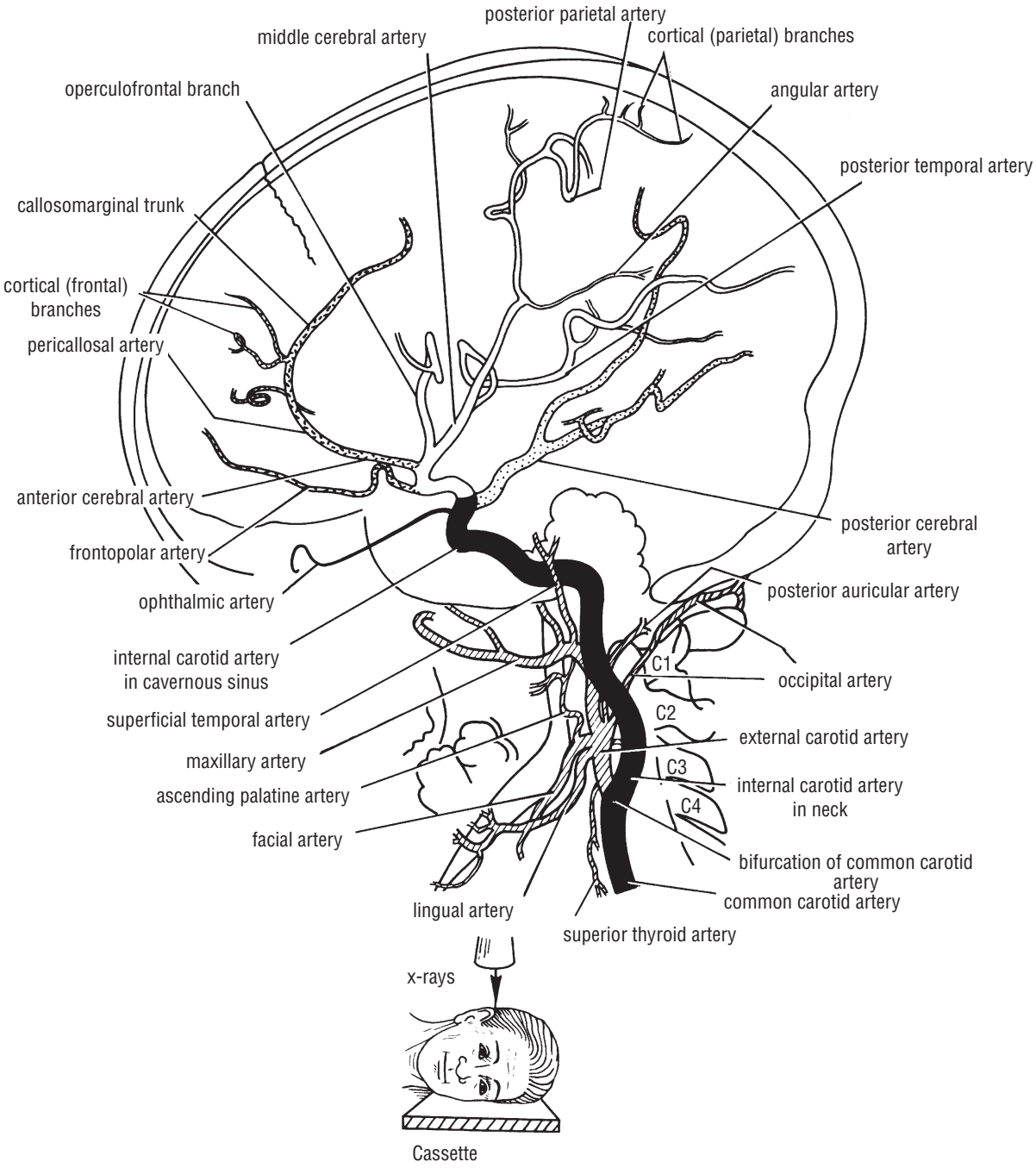
Congenital aneurysms occur most commonly at the site where two arteries join in the formation of the circle of Willis (CD Fig. 14-8). At this point, there is a deficiency in the tunica media that so weakens the arterial wall that an aneurysm develops. The enlarging aneurysm may press on neighboring structures, such as the optic, oculomotor, trochlear, and abducent nerves, and produce signs and symptoms or may suddenly rupture into the subarachnoid space.

Subarachnoid Hemorrhage

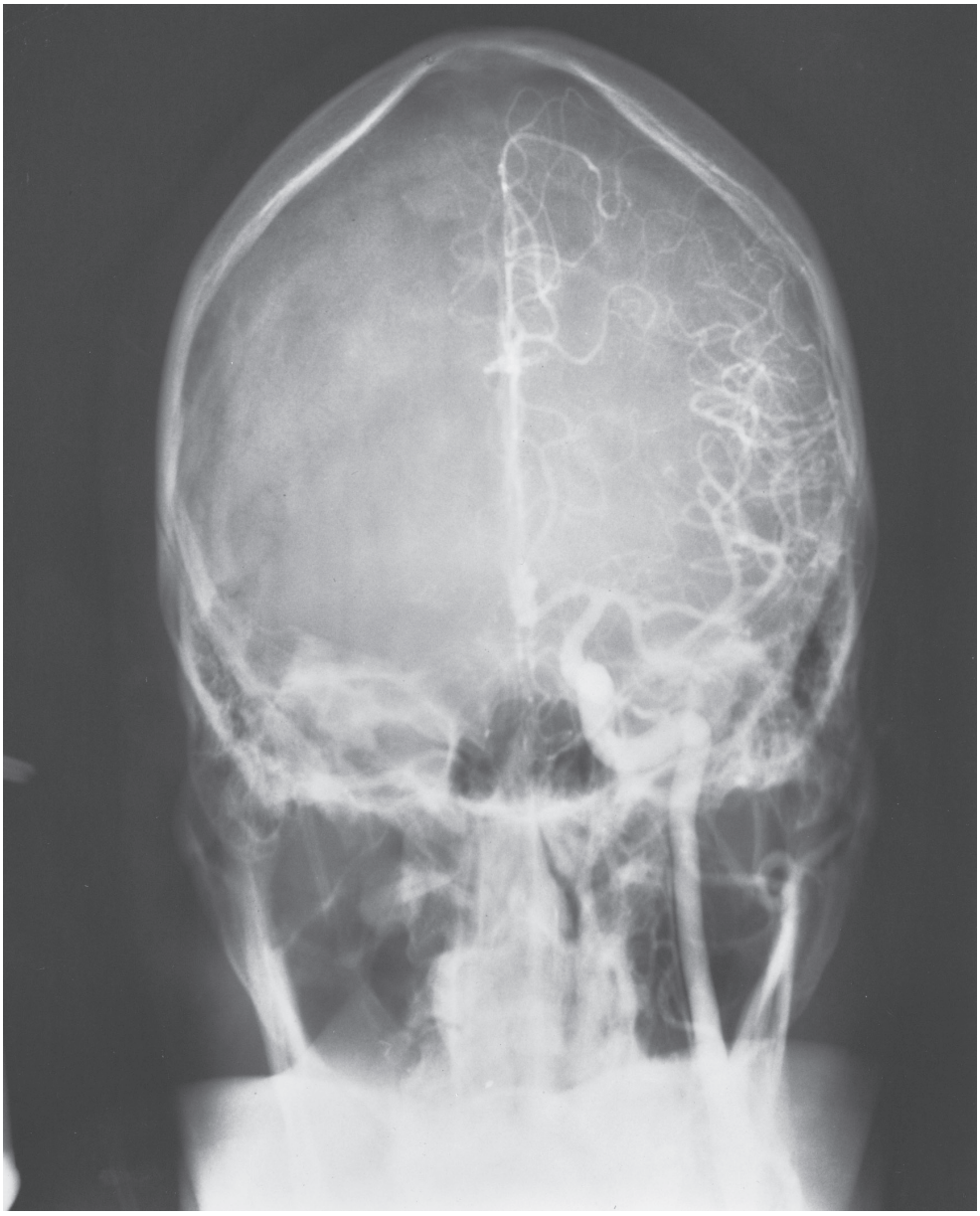
Subarachnoid hemorrhage results from leakage or rupture of a congenital aneurysm on the circle of Willis or, less commonly, from an angioma. The symptoms, which are sudden in onset, include severe headache, stiffness of the neck, and loss of consciousness. The diagnosis is established by withdrawing heavily blood-stained cerebrospinal fluid through a lumbar puncture (spinal tap).

Cerebral Hemorrhage

Cerebral hemorrhage is generally caused by rupture of the thin-walled lenticulostriate artery, a branch of the middle



CD Figure 14-5 Main features that can be seen in the arteriogram in CD Fig. 14-4.



CD Figure 14-6 Anteroposterior internal carotid arteriogram.

cerebral artery. The hemorrhage involves the vital corticobulbar and corticospinal fibers in the internal capsule and produces hemiplegia on the opposite side of the body. The patient immediately loses consciousness, and the paralysis is evident when consciousness is regained.

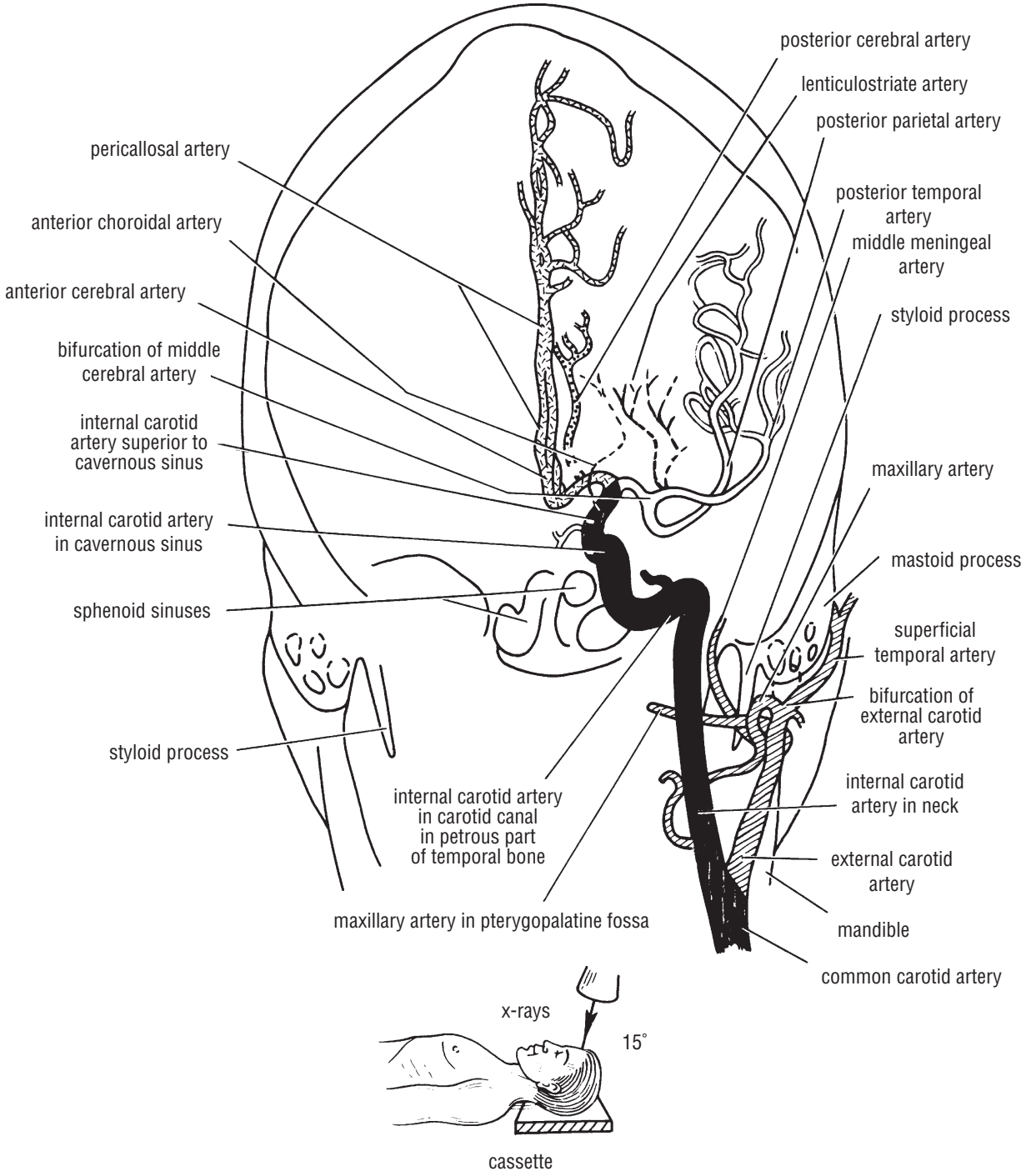
Cerebral Ischemia

It has been pointed out that there are two distinct yet interconnected vascular systems supplying the brain. The carotid arteries are the major suppliers of the cerebral hemispheres, and the basilar and vertebral arteries are the major suppliers of the brainstem and cerebellum. The neurologic deficit following blockage of one of the intracranial vessels will depend on the location of the blockage and the status of the collateral circulation. The blood supply to the functional areas of the cerebral cortex is shown in text Fig. 14-17.

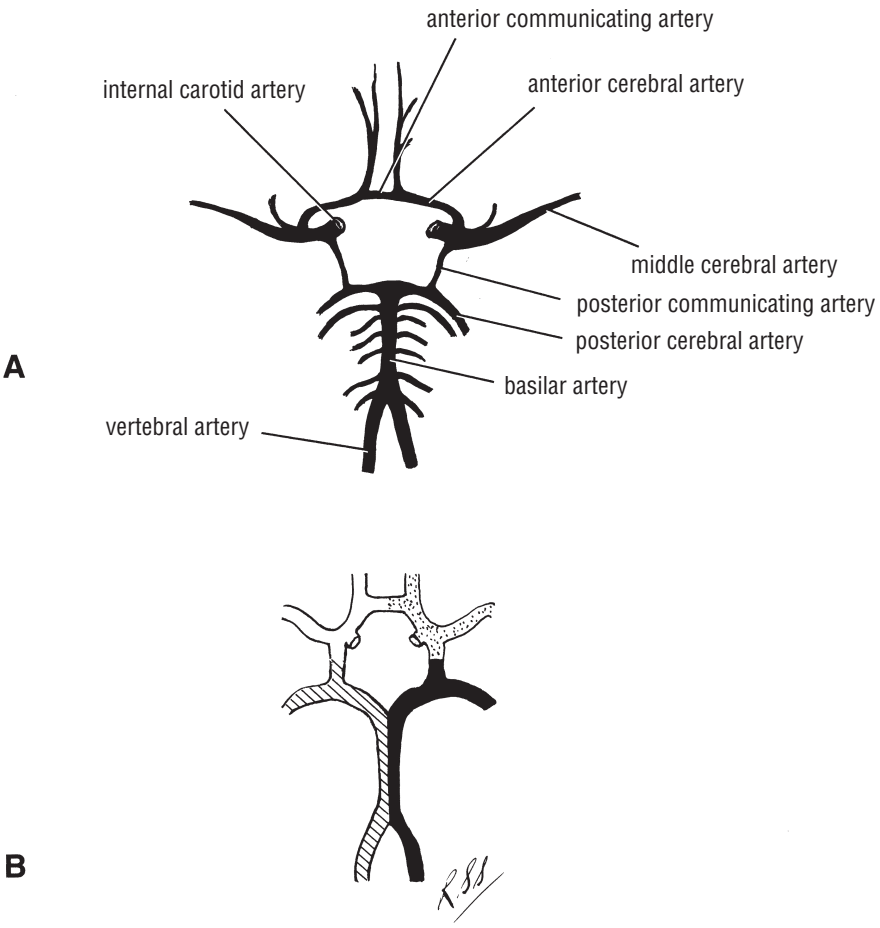
Cerebral Artery Occlusion

Anterior Cerebral Artery Occlusion

If the occlusion of the artery is proximal to the anterior communicating artery, the collateral circulation is usually adequate to preserve the circulation. Occlusion distal to the communicating artery may produce the following signs and



CD Figure 14-7 Main features that can be seen in the arteriogram in CD Fig. 14-6.



CD Figure 14-8 **A.** The formation of the circle of Willis from the two internal carotid and two vertebral arteries. **B.** The distribution of blood from the four main arteries.

symptoms:

- Contralateral hemiparesis and hemisensory loss involving mainly the leg and foot
- Inability to identify objects correctly, apathy, and personality changes

Middle Cerebral Artery Occlusion

Occlusion of the artery may produce the following signs and symptoms.

- Contralateral hemiparesis and hemisensory loss
- Aphasia if the left hemisphere is affected (rarely if the right hemisphere is affected)
- Homonymous hemianopia
- Anosognosia if the right hemisphere is affected (rarely if the left hemisphere is affected)

Internal Carotid Artery Occlusion

This may produce all the symptoms and signs of anterior and middle cerebral artery occlusion, depending on the degree of collateral circulation at the circle of Willis; in addition, the following may be seen:

- Loss of vision on the same side as the internal carotid artery occlusion due to blockage of the ophthalmic artery
- Decreased level of consciousnes

Vertebral Artery Occlusion

This produces a variable clinical picture and may include the following signs and symptoms:

- Ipsilateral pain and temperature sensory loss of the face and contralateral pain and temperature sensory loss of the body
- Ipsilateral loss of the gag reflex, dysphagia, and hoarseness as the result of lesions of the nuclei of the glossopharyngeal and vagus nerves
- Vertigo, nystagmus, nausea, and vomiting
- Ipsilateral Horner's syndrome
- Ipsilateral ataxia

If the lesion is more extensive, the corticospinal tracts may be involved, producing contralateral hemiparesis of the body. Contralateral loss of position and vibration sense may also be lost due to damage to the medial lemniscus.

Basilar Artery Occlusion

Since this artery gives off numerous branches to the pons, cerebellar peduncles, and cerebellum, total blockage of this artery can produce lesions of the trigeminal, abducent, and facial nerve nuclei, quadriplegia, and coma (reticular formation). If occlusion is restricted to branches of the basilar artery, there may be contralateral hemiparesis, contralateral sensory loss, or evidence of cerebellar dysfunction.

Central Branch Artery Occlusion

Small artery occlusion will cause discrete areas of brain necrosis. The signs and symptoms produced will obviously depend on the area involved. For example, a lesion of the internal capsule may result in contralateral hemiplegia.

Transient Ischemic Attacks

These are brief, self-limited focal neurologic deficits caused by embolic or thrombotic occlusion of arteries supplying the brain. The signs and symptoms will depend on the area of brain involved.

For further information on the clinical neuroanatomy of this area, please consult *Clinical Neuroanatomy*, 6th ed., by R.S. Snell, Lippincott Williams & Wilkins.

Hydrocephalus

Hydrocephalus is an abnormal increase in the volume of the cerebrospinal fluid within the skull. If hydrocephalus is accompanied by raised cerebrospinal fluid pressure, it is caused by either (1) an abnormal increase in fluid formation, (2) blockage of the fluid circulation, or (3) diminished absorption of the fluid. Rarely, hydrocephalus occurs with normal cerebrospinal fluid pressure, and in these patients compensatory hypoplasia or atrophy of the brain substance exists.

When the block of the movement of cerebrospinal fluid lies within the brain, the hydrocephalus is the **noncommunicating** type (i.e., the cerebrospinal fluid inside the brain does not communicate with that on the outside). If the fluid is able to pass through the roof of the fourth ventricle into the subarachnoid space and cannot be absorbed by the arachnoid villi, the hydrocephalus is the **communicating** type (i.e., the cerebrospinal fluid inside the brain communicates with that on the outside).

Hydrocephalus Resulting from Excessive Formation of Cerebrospinal Fluid

This condition is rare and may occur when there is a tumor of the choroid plexuses.

Hydrocephalus Resulting from Blockage of Cerebrospinal Fluid Circulation

An obstruction of the interventricular foramen by a tumor will block the drainage of the lateral ventricle on that side. The continued production of cerebrospinal fluid by the choroid plexus of that ventricle will cause distention of that ventricle and atrophy of the surrounding neural tissue.

An obstruction in the cerebral aqueduct in the midbrain may be congenital or result from inflammation or pressure from a tumor. This causes a symmetrical distension of both lateral ventricles and distension of the third ventricle.

Obstruction of the foramina in the roof of the fourth ventricle by inflammatory exudate, or by tumor growth, will produce symmetrical dilatation of both lateral ventricles and the third and fourth ventricle.

Sometimes inflammatory exudate secondary to meningitis will block the subarachnoid space and obstruct the flow of cerebrospinal fluid over the outer surface of the cerebral hemispheres. Here, again, the entire ventricular system of the brain will become distended.

Hydrocephalus Resulting from Diminished Absorption of Cerebrospinal Fluid

Interference with the absorption of cerebrospinal fluid at the arachnoid granulations can be caused by inflammatory exudate, venous thrombosis or pressure on the venous sinuses, or obstruction of the internal jugular vein.

Clinical Problem Solving Questions

Read the following case histories/questions and give the best answer for each.

1. A 36-year-old man was admitted to the emergency department unconscious. He had been hit on the side of the head by a taxi while crossing the road. On examination, he was found to have a dough-like swelling over the right temporalis muscle. He also had the signs of right-sided hemiplegia. Later, a right-sided, fixed, dilated pupil developed. A lateral radiograph of the skull showed a linear fracture running downward and forward across the right pterion. His coma deepened, and he died 6 hours

after the accident. Using your knowledge of anatomy, make the diagnosis. Explain the clinical findings. How would you explain the homolateral hemiplegia?

2. Severe injury to the soft structures within the skull may follow head trauma. What structures exist within the skull to limit damage to the cerebral hemispheres and other parts of the brain? Which blood vessels are damaged more commonly, the cerebral arteries or the cerebral veins? Which cranial nerves are likely to be damaged in head injuries? What is the reason for their susceptibility?

3. A 51-year-old woman was examined in the emergency department complaining of a severe headache. She said that the headache had started about 1 hour after she had hit her head on the mantle piece of a fireplace after bending down to poke the fire. Four hours later it was noticed that she was becoming mentally confused and was developing a left-sided hemiplegia on the side opposite the head injury. Her deep reflexes were exaggerated, and she had a positive Babinski response on the left side. A CT scan demonstrated a right subdural hematoma. Explain in anatomic terms the development of a subdural hematoma.

4. Which part of the base of the skull is most prone to fracture? Can you give an anatomic reason for this answer?

5. It is not uncommon to read in newspapers of the survival of a baby that has fallen from a great height, such as a third-floor window, and yet it is known that if an adult falls from a similar height, it would be fatal. Can you give an anatomic explanation, based on age, for this difference in survival?

6. Using your knowledge of the anatomic pathways along which the cerebrospinal fluid flows, name the sites at which pathologic blockage may occur.

7. There are no anastomoses of clinical importance between the terminal end arteries within the brain substance, but there are many important anastomoses between the large arteries, both within and outside the skull, and these may play a major role in determining the extent of brain damage in cerebral vascular disease. Name the sites at which important arterial anastomoses take place.

8. A 35-year-old man was seen in the emergency department with a history of sudden excruciating, generalized headache while gardening. Ten minutes later the patient collapsed to the ground in a state of unconsciousness. After being carried indoors and placed on a settee, he regained consciousness but appeared confused. He complained of a severe headache and a stiff neck. Physical examination revealed some rigidity of the neck but nothing further. A careful neurologic examination 4 days later revealed some loss of tone of the muscles of the left leg. Using your knowledge of anatomy, make a diagnosis. What caused the neck rigidity?

Answers and Explanations

1. The initial loss of consciousness was due to cerebral trauma. The swelling over the right temporalis muscle and the radiographic finding of a linear fracture over the anterior inferior angle of the right parietal bone (pterion) would suggest that the right middle meningeal artery had been damaged and an extradural hemorrhage had occurred. Blood had extravasated through the fracture line into the overlying temporalis muscle and soft tissue. The right homolateral hemiplegia was due to the compression of the left cerebral peduncle against the edge of the tentorial notch of the tentorium cerebelli. This is unusual. A left hemiplegia due to pressure on the right precentral gyrus is more common.

   The right-sided, fixed, dilated pupil was due to the pressure on the right oculomotor nerve by the hippocampal gyrus, which had herniated through the tentorial notch.

2. The meninges and the cerebrospinal fluid afford a remarkable degree of protection to the brain tissue. The dural partitions limit the extent of side-to-side, forward and backward, and rotation movements of the brain within the skull.

   The thin-walled cerebral veins are liable to be damaged during excessive movements of the brain relative to the skull, especially at the point where the veins join the dural venous sinuses. The thick-walled cerebral arteries are rarely damaged.

   The small-diameter cranial nerves of long length are particularly prone to damage during head injuries. The trochlear, abducent, and oculomotor nerves are commonly injured.

3. A subdural hematoma is an accumulation of blood clot in the interval between the meningeal layer of dura and the arachnoid mater. It results from tearing of the supe-

rior cerebral veins at their point of entrance into the superior sagittal sinus. The cause is usually a blow to the front or the back of the head (which may be minor), resulting in excessive anteroposterior displacement of the brain within the skull.

4. The middle cranial fossa is the part of the skull most prone to fracture, since it possesses numerous foramina and canals and has air spaces, namely, the sphenoid air sinus and the tympanic cavity. The foramen magnum in the posterior cranial fossa is very large but its boundaries are extremely thick.

5. In infants the skull bones are more resilient than in adults, and they are separated by fibrous sutural ligaments. In adults the inner table of the skull is particularly brittle and the sutural ligaments begin to ossify during middle age.

6. The common sites for blockage of the flow of cerebrospinal fluid are where the passages are narrowest, namely, the interventricular foramina (between the lateral and third ventricles), the cerebral aqueduct of the midbrain, the median aperture, and the lateral apertures in the roof of the fourth ventricle. It is possible for inflammatory exudate secondary to meningitis or a cerebral tumor to narrow down or even obliterate the opening in the tentorial notch so that the passage of the cerebrospinal fluid to the outer surface of the cerebral hemisphere is impeded or stopped. Inflammatory exudate may also block the drainage of the fluid into the superior sagittal sinus at the arachnoid villi.

7. Once the terminal branches of the cerebral arteries enter the brain substance, no further anastomoses occur. Blockage of such end arteries by disease is quickly followed by neuronal death and necrosis. The following important anastomoses exist between the cerebral arteries: (1) the circle of Willis, (2) anastomoses between the branches of the cerebral arteries on the surface of the cerebral hemispheres and the cerebellar hemispheres, and (3) anastomoses between the branches of the internal and external carotid arteries at their origin at the common carotid artery, at the anastomosis between the branches of the ophthalmic artery within the orbit and the facial and maxillary arteries, and between the meningeal branches of the internal carotid artery and the middle meningeal artery.

8. This patient had a congenital aneurysm of the anterior communicating artery. The sudden onset of a severe headache, which is often so dramatic that the patient feels as though he has been hit on the head, is characteristic of rupture of a congenital aneurysm into the subarachnoid space. The stiff or rigid neck is due to meningeal irritation caused by the presence of blood in the subarachnoid space. This patient had no evidence of previous pressure on the optic nerve leading to unilateral visual defect, which sometimes occurs when the aneurysm is situated on the anterior part of the circle of Willis. The loss of tone in the left leg muscles is difficult to explain, although it may be due to penetration of the hemorrhage into the right cerebral hemisphere.