Chapter 1
The Adult Central Nervous System: Anatomy and Physiology

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Overview

The human brain consists of three basic subdivisions – the cerebral hemispheres, the brain stem, and the cerebellum. Speech is represented by two main areas – Broca's area in the inferior frontal lobe (expressive speech) and the Wernicke's area in the temporoparietal cortex (interpretation of language) (Fig. 1.1). Language localization is found in 96% of population in the left. The basal ganglia are a collection of nuclei deep in the white matter of cerebral cortex and contain the substantia nigra. A decrease in function of the dopaminergic neurons located in the substantia nigra causes Parkinson's disease. The cerebral hemispheres are connected together medially by the corpus callosum. The limbic areas of the brain include the hypothalamus, amygdala, hippocampus, and limbic cortex. Optic nerve (second) leaves the retina of the eye and travels to the optic chiasm, located just below and in front of the pituitary gland. In the optic chiasm, the optic nerve fibers arising from the nasal half of each retina cross over to the other side; but the nerve fibers originating in the temporal retina do not cross over. The nerve fibers become the optic tract, the optic radiation and reach the visual cortex in the occipital lobe of the cerebrum.

The brainstem is located at the juncture of the cerebrum and the spinal column. It consists of the midbrain, pons, and the medulla oblongata. Two cranial nerves are associated with the midbrain, the oculomotor (3rd) which emerges from the interpeduncular fossa and the trochlear (4th) cranial nerves which emerges from the dorsal

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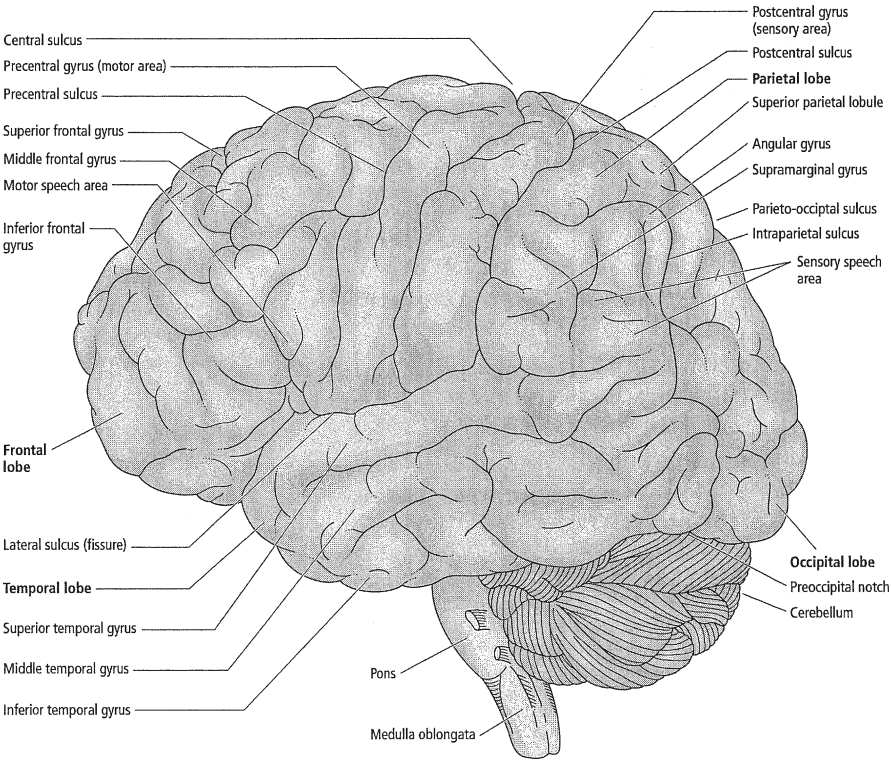

Fig. 1.1 Lateral surface of the brain

surface of the brain stem. The cranial nerves associated with the pons are the trigeminal (5th), abducens (6th), facial (7th), and the two components of the auditory (8th). The 5th cranial nerve passes through the rostral part of the middle cerebellar peduncle. The 6th lies on the floor of the fourth ventricle is partially encircled by the 7th cranial nerve and emerges from the ventral surface of the brain stem at the junction between the pons and medulla. The 7th and 8th cranial nerve emerge from the lateral surface of the pons at the cerebellopontine angle. The red nucleus is a structure in the rostral midbrain involved in motor coordination. It is less important in its motor functions for humans than in many other mammals because, in humans, the corticospinal tract is dominant. The reticular formation is composed of a number of diffuse nuclei in the medulla, pons, and midbrain. The ascending reticular formation is also called the reticular activating system (RAS) and is responsible for the sleep–wake cycle. The descending reticular formation is involved in posture, equilibrium, and motor movement. It is also responsible for autonomic nervous system activity (vasomotor center), and stimulation of different portions of this center causes either a rise in blood pressure and tachycardia (pressor area) or a fall in blood pressure and bradycardia (depressor area). Medulla is the most caudal part of the brain stem.

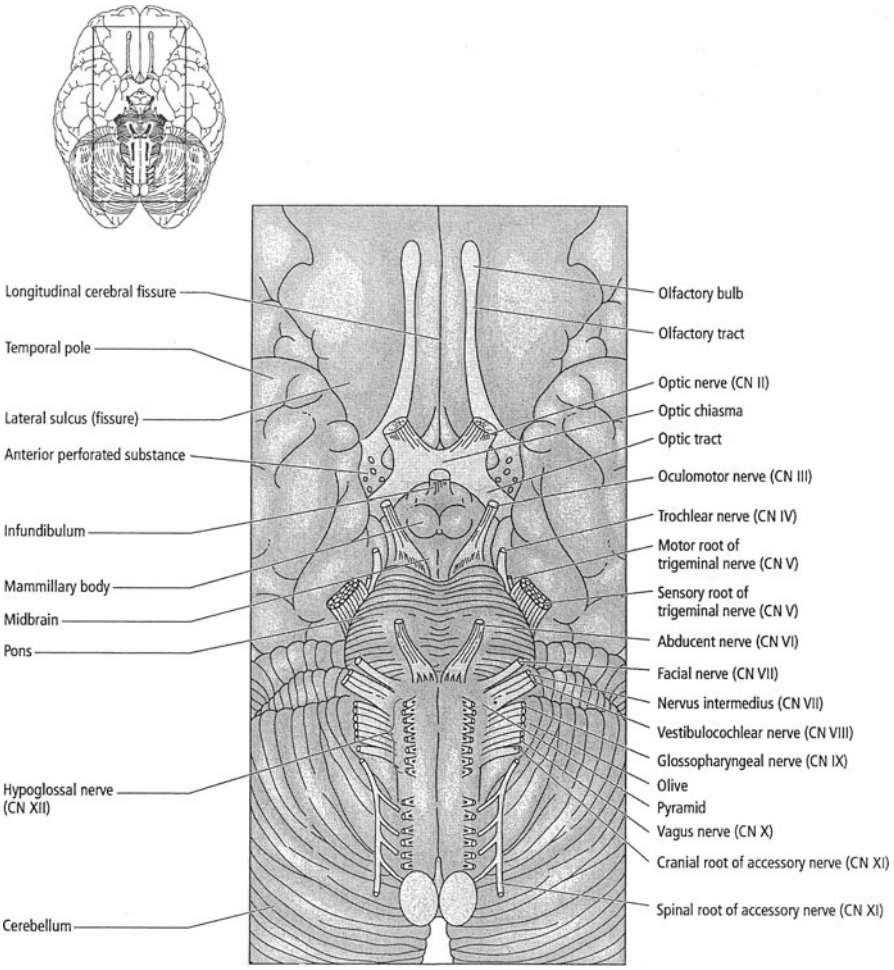

Fig. 1.2 Cranial nerves at the base of brain

The cranial nerves associated with the medulla are the glossopharyngeal nerve (9th), vagus (10th), accessory (11th), and the hypoglossal (12th) (Fig. 1.2).

The cerebellum is a trilobed structure, lying posterior to the pons and medulla oblongata and inferior to the occipital lobes of the cerebral hemispheres, that is responsible for the regulation and coordination of complex voluntary muscular movement as well as the maintenance of posture and balance. In contrast to the neocortex, cerebellar lesions produce ipsilateral disturbances.

The skull consists of the anterior, middle, and the posterior cranial fossa. Anterior cranial fossa accommodates the anterior lobe of brain. Middle cranial fossa contains the two temporal lobes, the parietal and part of the occipital lobe of brain. The posterior cranial fossa is part of the intracranial cavity located between the foramen magnum and

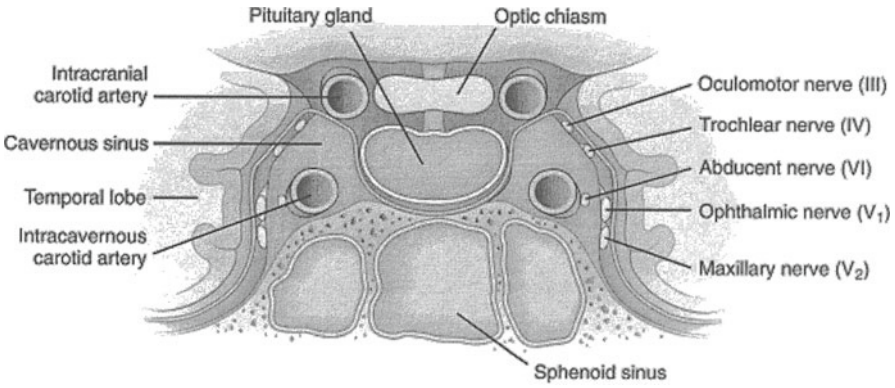

Fig. 1.3 Anatomical structure of cavernous sinus

tentorium cerebri. It contains part of the occipital lobe, brainstem, and cerebellum. The brain is covered by the dural membranes which enclose the venous sinuses.

The cavernous sinuses are paired, venous structures located on either side of the sella turcica (Fig. 1.3). They contain the carotid artery, its sympathetic plexus, and the third, fourth, and sixth cranial nerves. In addition, the ophthalmic branch and occasionally the maxillary branch of the fifth nerve traverse the cavernous sinus. The nerves pass through the wall of the sinus, while the carotid artery passes through the sinus itself. The pituitary gland is located inside the sella turcica, a round bony cavity that is separated from the sphenoid sinuses by a thin bone, the floor of the sella, which forms part of the roof of the sphenoid sinuses.

The Circle of Willis provides the blood supply to the cerebrum. It is formed by the two internal carotid arteries, which are responsible for 80% blood supply to the brain, and the vertebral artery, which is responsible for about 20% blood supply to the brain. The internal carotids and vertebral arteries anastomose together at the base of the brain. The Circle of Willis is associated with frequent anatomical variations, and a complete Circle of Willis is found only in 50% of the population. The anterior and middle cerebral arteries, which originate from the Circle of Willis, form the anterior circulation and supply the forebrain. Each gives rise to branches that supply the cortex and branches that penetrate the basal surface of the brain, supplying deep structures such as the basal ganglia, thalamus, and internal capsule. The lenticulostriate arteries arise from the middle cerebral artery and supply the basal ganglia and thalamus. The posterior circulation of the brain supplies the posterior cortex, the midbrain, and the brainstem and comprises arterial branches arising from the posterior cerebral, basilar, and vertebral arteries. Midline arteries supply medial structures; lateral arteries supply the lateral brainstem; and dorsal–lateral arteries supply dorsal–lateral brainstem structures and the cerebellum (Fig. 1.4).

Most of the blood in the brain can be found in its venous system. Blood is drained into superficial and deep cerebral veins and veins of the posterior fossa. The superficial veins drain the surface of the brain cortex and lie within the cortical sulci. The deep cerebral veins drain the white matter, basal ganglia, diencephalon, cerebellum,

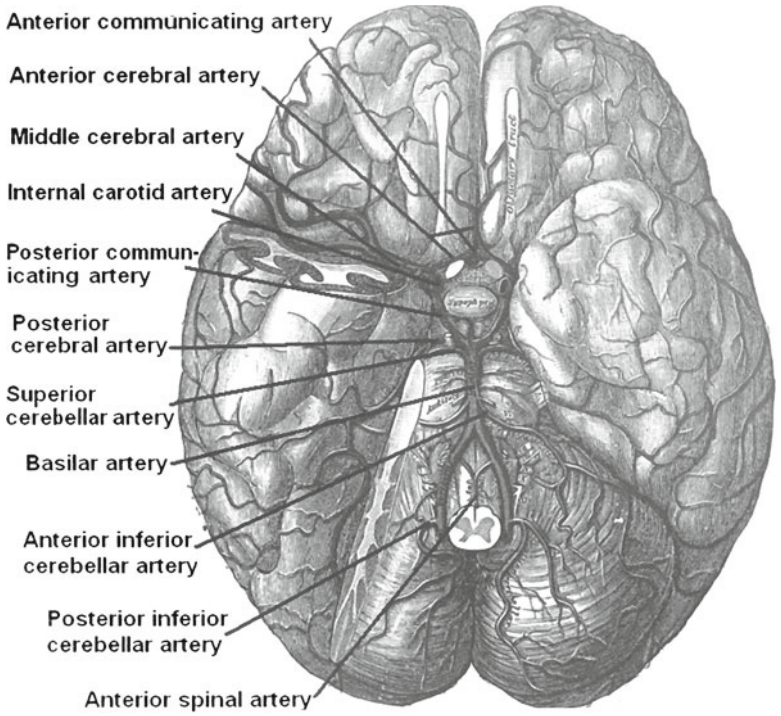

Fig. 1.4 Arterial supply of the brain

and brainstem. The deep veins join to form the great cerebral vein. The veins of posterior fossa drain blood from the cerebellar tonsils and the posteroinferior cerebellar hemispheres. In addition, the diploic veins drain the blood between layers of bone in the skull. Emissary veins connect the veins near the surface of the skull to the diploic veins and venous sinuses. All the blood is drained into the meningeal sinuses, which mainly drain into the internal jugular vein. Usually, the right jugular vein is the dominant one, receiving most of the blood from the brain. The veins and sinuses of the brain lack valves. Pressure of drainage vessels in the neck is directly transmitted to intracranial venous structures (Fig. 1.5).

The vertebral column is composed of 33 vertebrae. Each vertebra is composed of a vertebral body, neural arch, pedicle, and a lamina (Fig. 1.6). The two lamina join together posteriorly to form the spinous process. The ligaments stabilizing the vertebral column from exterior to interior are the supraspinous, interspinous, ligamentum flavum, posterior longitudinal, and anterior longitudinal ligaments (Fig. 1.7). The ligaments provide flexibility without allowing excessive movement which could damage the cord. The human spine is also affected by aging. The intervertebral disks become drier, more fibrous and less resilient.

The spinal cord is a long cylindrical structure covered by membranes which lies in the vertebral canal. The spinal cord is the continuation of the medulla. It extends

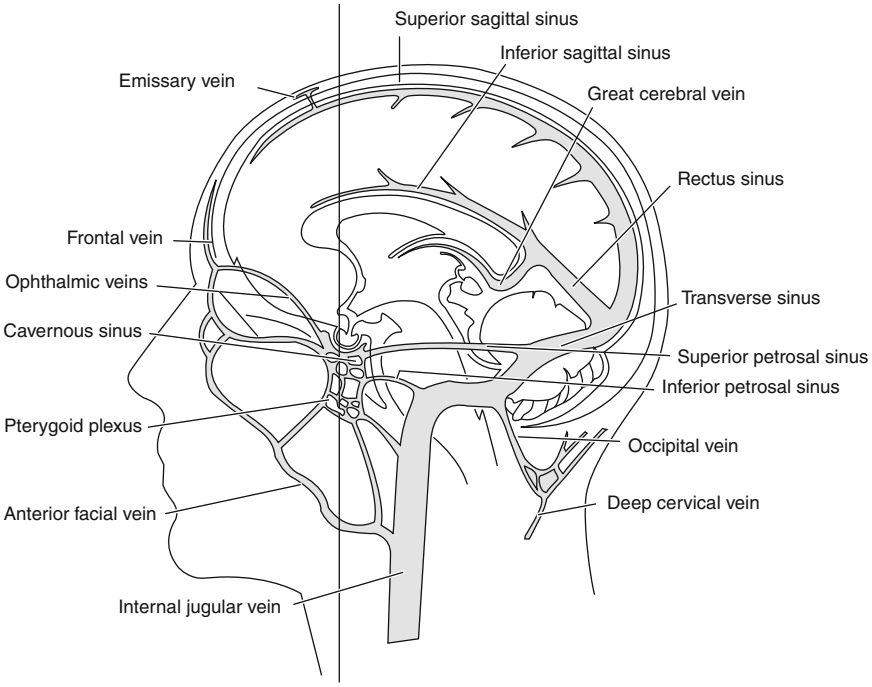

Fig. 1.5 Venous drainage of the brain

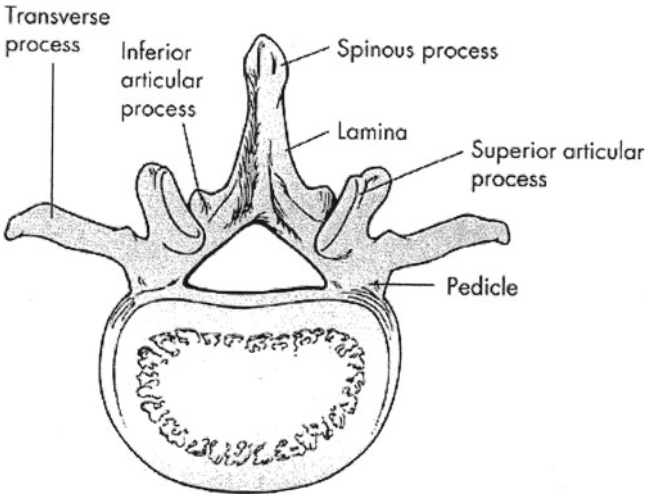

Fig. 1.6 Lumbar vertebra

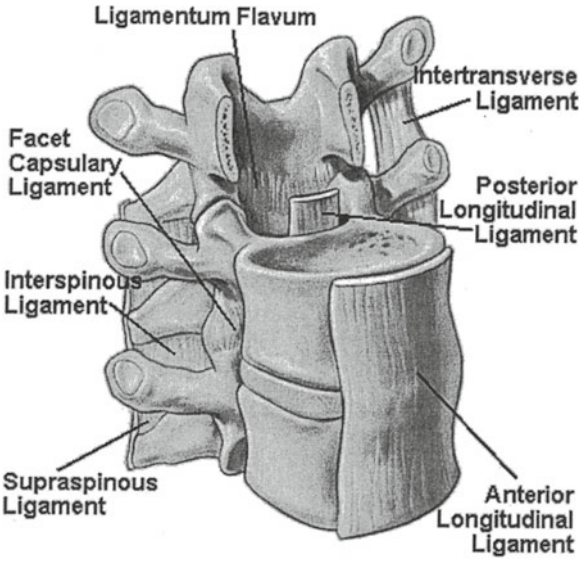

Fig. 1.7 Ligaments supporting the vertebrae

from the foramen magnum to the lower border of first lumbar vertebral. It has two enlargements, cervical and lumbosacral, corresponding to the innervation of the upper and lower extremities. It ends in the conus medullaris. The anterior portion of the spinal cord contains the motor tracts, while the posterior cord contains the sensory tracts. The mean spinal cord blood flow in the cervical and lumbar segments is 40% higher than the thoracic segment. The watershed area of the spinal cord blood flow is the mid-thoracic region. The spinal cord receives its blood supply principally from three longitudinal vessels. The single anterior spinal artery is formed by the two vertebral arteries which supply the anterior 75% of the cord and the two posterior spinal arteries formed by the posterior inferior cerebellar artery and supply the posterior 25% of the cord (Fig. 1.8). The anterior and posterior arteries alone can only supply enough blood to maintain the upper cervical segments of the spinal cord. The blood supply to the lower levels of the spinal cord is provided by the radicular arteries which anastamose with the anterior and posterior spinal arteries. The artery of Adamkiewicz, a major radicular artery located in lower thoracic or upper lumbar region, provides most of the blood supply to the lower cords. Autoregulation maintains spinal cord blood flow by altering vascular resistance in response to changes in the mean arterial blood pressure (MAP).

The ascending spinal tracts are contained in the posterior column of the spinal cord and terminate in the medulla. They are sensory in nature. After decussation in the medulla, the second-order neurons form an ascending bundle and terminate in the thalamus from where they reach the post-central gyrus via third-order neurons (Fig. 1.9). The descending tract, called the corticospinal tract, is motor in nature. Corticospinal tract fibers originate in the cerebral cortex in the precentral gyrus and 90% decussate at the level of the medulla. Lesions above the medullary decussation

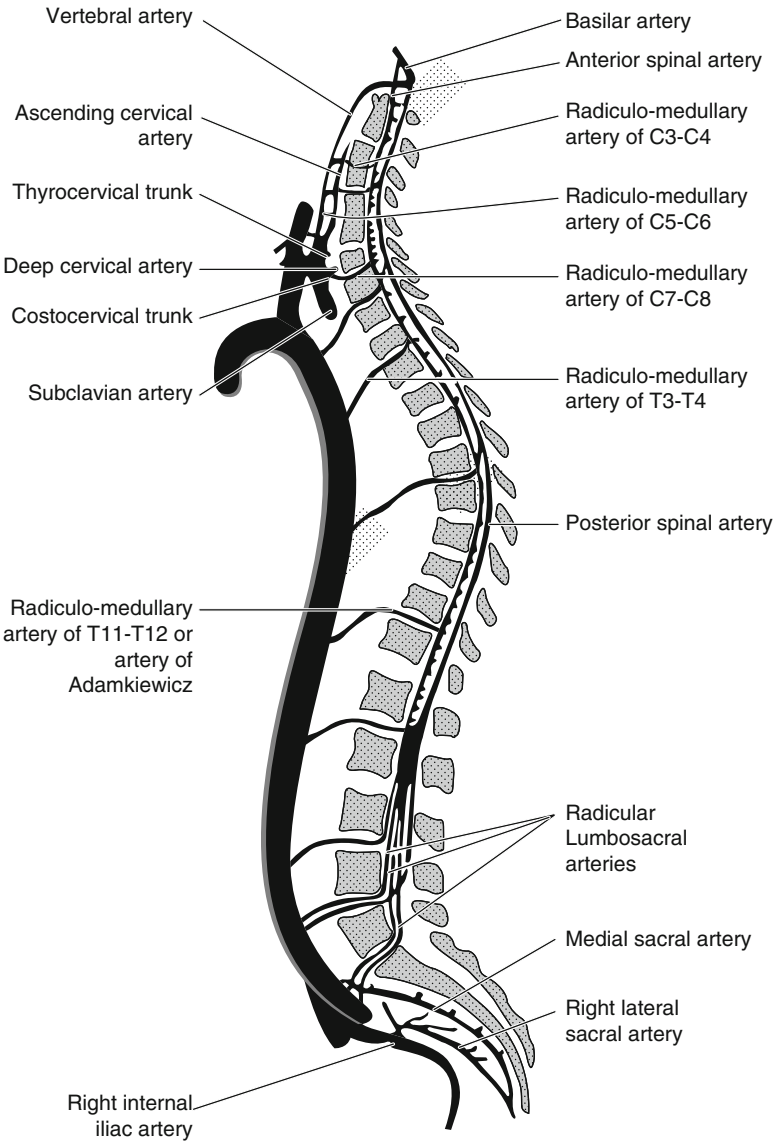

Fig. 1.8 Blood supply of the spinal cord

cause contralateral paralysis and those below medullary decussation cause ipsilateral paralysis (Fig. 1.10).

The ventricular system of the brain is composed of two lateral ventricles and two midline ventricles called the third and fourth ventricles which contain the cerebrospinal fluid (CSF). The chambers are connected to allow the flow of CSF between

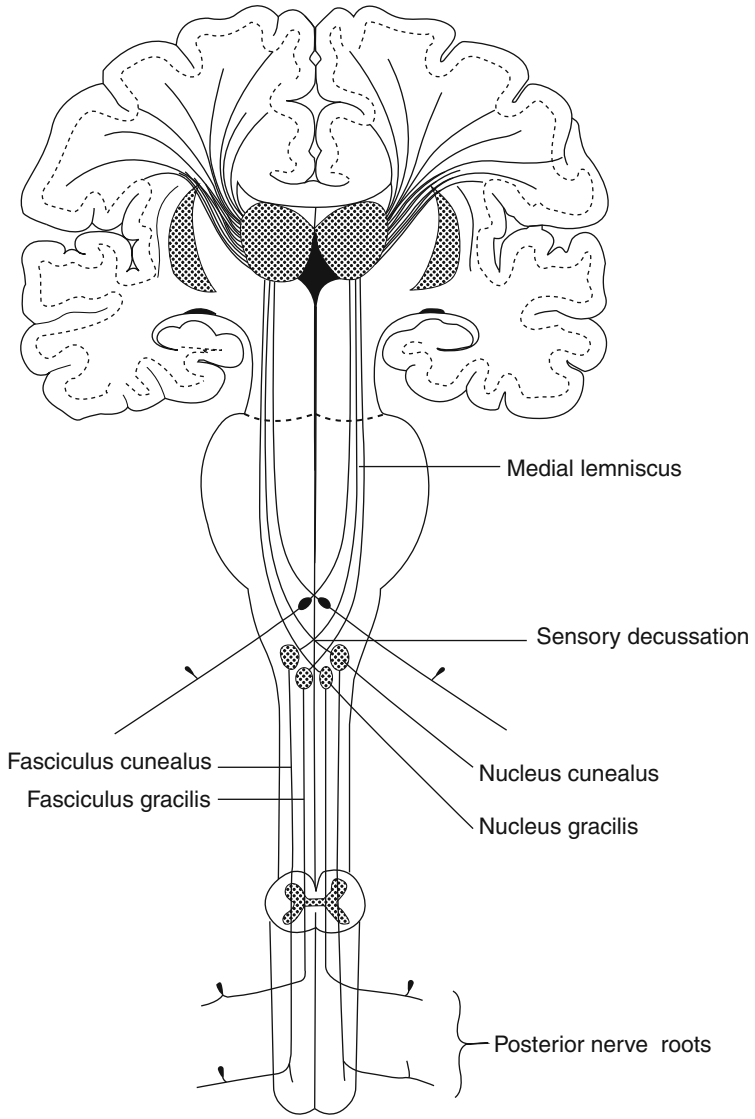

Fig. 1.9 Sensory pathway

two lateral ventricles to the third ventricles through the Foramen of Monroe. This then communicates through the aqueduct of Sylvius (Mesencephalic aqueduct) to the fourth ventricle (Fig. 1.11). The CSF flows out into the subarachnoid spaces of the brain and the spinal cord through the medial foramen of Magendie and lateral foramen of Luschka. The chambers of the ventricular system are lined with ependymal cells and are continuous with the central canal enclosed within the spinal cord. CSF is a dynamic medium, which is produced and absorbed constantly, and functions as

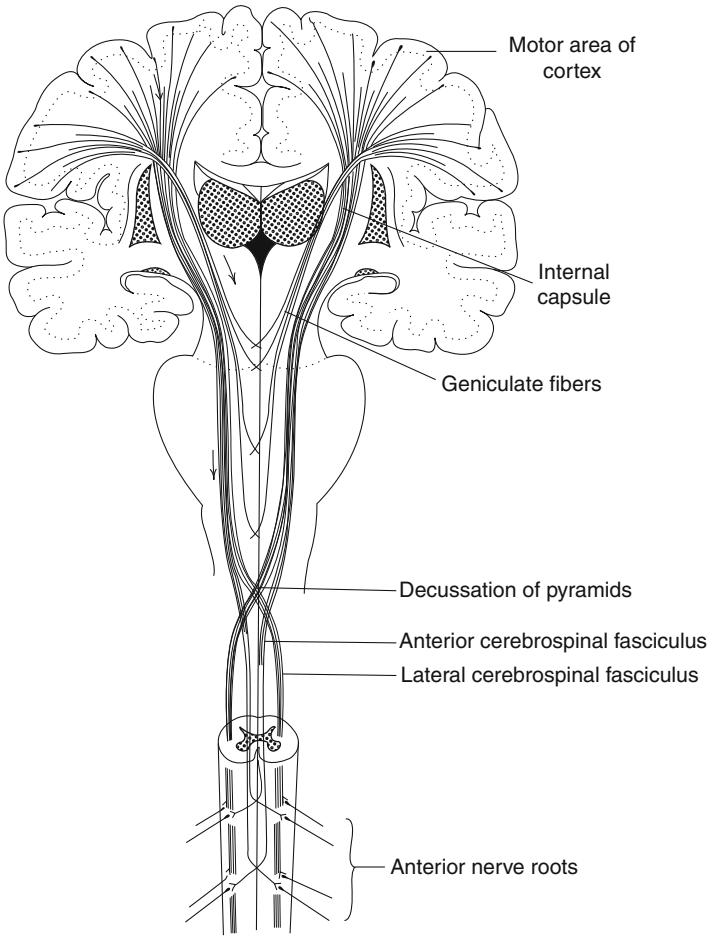

Fig. 1.10 Motor pathway (corticospinal tract)

the brain's drainage system. Under normal circumstances, a human being produces approximately 0.35 ml/min (500 ml/day) of CSF. The total volume of CSF at a given time is 150 ml, which means that the CSF is replaced approximately four times a day. The majority of CSF is formed in the choroid plexus of the lateral ventricles by filtration of plasma through fenestrated capillaries and also by the active transport of water and dissolved substances through the epithelial cells of the blood–CSF barrier. CSF may also be formed by the lymph-like drainage of the brain's extracellular fluid. Reabsorption of CSF takes place mostly in the arachnoid villi and granulations into the circulation. The mechanism behind the CSF reabsorption is the difference between the CSF pressure and venous pressure. CSF formation is reduced by decreased blood flow through the choroid, hypothermia, increased serum osmolarity, and increased ICP. The main compensatory mechanism for an increase

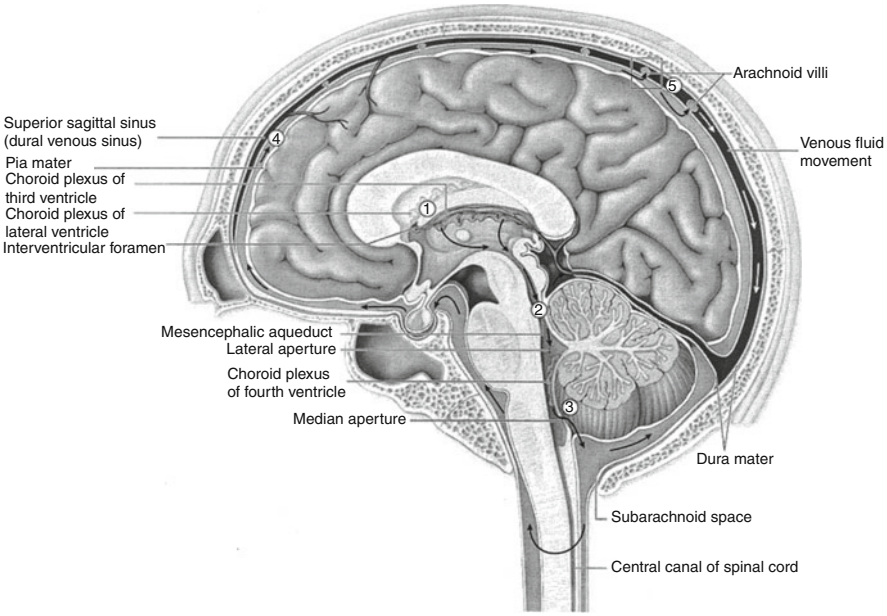

Fig. 1.11 Circulation of cerebrospinal fluid. (1) CSF formation, (2) circulation of CSF in the ventricle, (3) CSF flow into subarachnoid space, (4) CSF circulation around the brain, and (5) absorption into circulation

in CSF volume includes displacement of CSF from cranial to spinal compartment, increase in CSF absorption, decrease in CSF production, and a decrease in cerebral blood volume (mainly venous).

The blood–brain barrier (BBB) isolates the brain from the plasma and is formed by the interaction of capillary endothelial cells with astrocytes in the brain. Water, gases, glucose, and lipophilic substances are freely permeable through the BBB. Proteins and polar substances are poorly permeable through the BBB. The brain is protected from the circulating toxins by the BBB.

The brain tissue has a high energy requirement and is responsible for about 20% of total body oxygen consumption. The cerebral metabolic rate, expressed as oxygen consumption ($CMRO_2$), averages 3.5 ml/100 g/min in adults. $CMRO_2$ is greatest in the gray matter of the cerebral cortex. Brain oxygen consumption supports two major functions – basic cellular maintenance (45%) and nerve impulse generation and transmission (55%). Glucose is the main substrate for energy production in the brain.

Normal cerebral blood flow (CBF) varies with the metabolic activity. Blood flow in gray matter is about 80 ml/100 g/min and in white matter is 20 ml/100 g/min. The average CBF is about 50 ml/100 g/min. There is coupling between the CBF and $CMRO_2$. The precise mechanism responsible for this coupling has not been identified, but it has been suggested that local by-products of metabolism (K+, H+, lactate, adenosine) are responsible for this coupling. Nitric oxide, a potent vasodilator has also been suggested to play a role. Glial processes may serve as a conduit for the coupling.

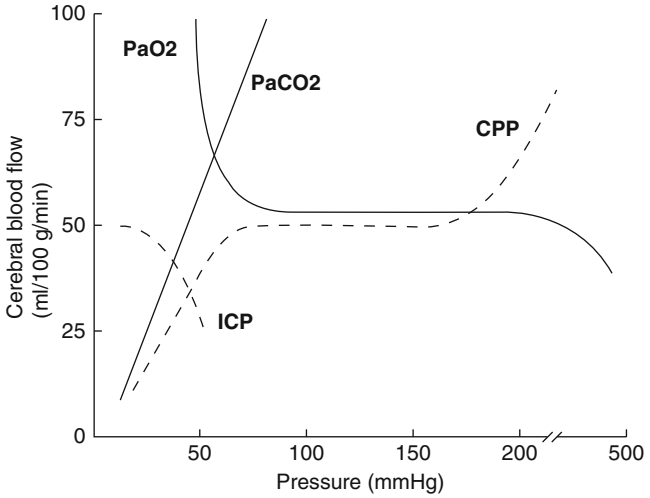

Fig. 1.12 Autoregulation of cerebral blood flow (CPP curve). Perfusion is increased in the setting of hypoxia or hypercarbia

Cerebral autoregulation maintains CBF relatively constant between cerebral perfusion pressures of 50 and 150 mmHg (Fig. 1.12). The mechanism of autoregulation is not understood but is probably due to myogenic and metabolic factors. Cerebral perfusion pressure (CPP) is calculated by subtraction of intracranial pressure (ICP) from mean arterial pressure, CPP = MAP − ICP.

The cranial vault is a rigid structure with fixed total volume, consisting of brain (80%), blood (12%), and CSF (8%). An increase in one component must be offset by an equivalent decrease in another to prevent a rise in the ICP. Intracranial compliance is measured by change in ICP in response to a change in intracranial volume ($\Delta V/\Delta P$). Intracranial elastance ($\Delta P/\Delta V$) is high because a small change in intracranial volume, ΔV, can cause a large change in ICP, ΔP. The pressure–volume relationship between ICP, volume of CSF, blood, and brain tissue, and CPP is known as the Monro-Kellie hypothesis (Fig. 1.13).

Anatomical changes that occur in the brain during normal aging are reduction in brain weight and volume with ventriculomegaly and sulcal expansion. The white matter volume of the cerebrum, cerebellum, corpus callosum, and pons remains fairly intact across all ages. Global CBF decreases about 10–20% because there is less brain mass to perfuse as we age.

Implications for the Neurosurgical Patient

Brain anatomy is important in identifying the different non-silent areas of the brain with respect to the surgical procedure being performed and helps to prevent devastating deficits. For instance, awake craniotomy or cortical mapping may be performed

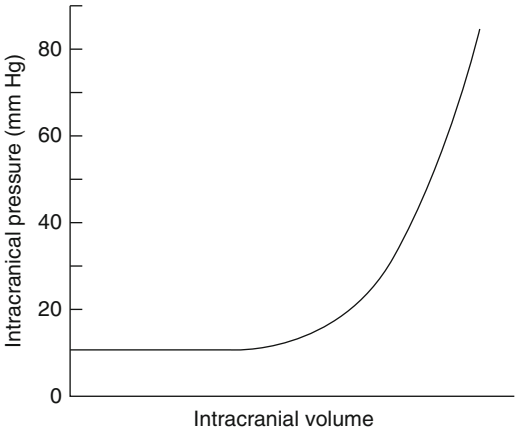

Fig. 1.13 Intracranial compliance curve – initial change in volume causes a slight rise in ICP; further increase causes a marked rise in ICP

when tumors or epileptic foci are close to cortical areas for speech or motor function or temporal structures critical to short-term memory. The Wada test determines the dominant lobe for suitability of temporal lobectomy. Deep brain stimulation of the globus pallidus interna or subthalamic nucleus improves many features of advanced Parkinson's disease. In addition, surgical approach and intraoperative events have anatomical implications. A frontotemporal craniotomy is used to approach anterior circulation aneurysms. Aneurysms originating from the posterior circulation require a subtemporal exposure, suboccipital exposure or a combined subtemporal, and suboccipital exposure. Brain stem stimulation can cause ventricular and supraventricular arrhythmias. Profound arterial hypertension can result from stimulation of the fifth cranial nerve. Significant bradycardia and escape rhythms can be caused from the stimulation of vagus nerve. Arterial hypotension can result from pontine or medullary compression. Brain stem lesions may result in abnormal breathing patterns.

Cranial nerve injury is a significant risk in surgery of the cerebellopontine angle and brain stem; therefore, intraoperative stimulation, monitoring and recording of electromyographic potential of cranial nerve with motor component is utilized to preserve the integrity of these nerves. Direct injury to portions of the RAS located within the pons, midbrain, or diencephalon (hypothalamus and thalamus) produces unconsciousness. Locked-in syndrome occurs with focal injury to the ventral pons and is often associated with basilar artery stroke. It results in patients who are quadriplegic, nonverbal, but awake and alert as the RAS is spared and volitional eye blinking is preserved.

The signs of corticospinal pathway dysfunction include initial weakness, followed by flaccid paralysis and then decorticate rigidity. Extensor (Babinski) toe response indicates acute or chronic injury of the corticospinal tracts in the brain and spinal cord. A Hoffman's response, which is the upper extremity equivalent of a Babinski response, can also provide additive lateralizing information. When damage involves subcortical structures (basal ganglia), decerebrate rigidity occurs.

Basal ganglia damage may also lead to athetoid or choreiform movements. The absence of vestibuloocular and oculocephalic reflex suggests brainstem damage in the pons between the vestibular nuclei (CN VIII) and oculomotor (CN III) and abducens (CN VI) nuclei, which control lateral eye movements. Flexor posturing (decorticate) indicates brain dysfunction above the level of the red nucleus in the midbrain (the area which mediates flexor response). Extensor posturing (decerebrate) indicates brainstem dysfunction below the level of the red nucleus. A hemispheric injury presents with contralateral hyporeflexia. Patients with acute spinal cord shock are hyporeflexive below the level of involvement (Table 1.1).

During spinal surgery, evoked potential monitoring may be used to evaluate spinal cord integrity. Sensory evoked potentials (SEP) evaluate the functional integrity of the ascending sensory pathways, mostly ending in the somatosensory cortex. Motor evoked potentials (MEP) test the functional integrity of the corticospinal tracts, which mainly originate in the motor cortex. The wake up test is also utilized to document the anterior motor component of the spinal cord (anterior column).

CBF is linearly related to $PaCO_2$ from 20 to 70 mmHg, when the autoregulation is intact. Hypocapnia causes cerebral vasoconstriction. Hypercapnea causes vasodilatation and increases the CBF. This change is mainly dependent on the pH alteration in the extracellular fluid of the brain. Change in PaO_2 from 60 to over 300 mmHg has little influence on CBF (Fig. 1.12). Hematocrit alters blood viscosity and affects blood flow; a low hematocrit increases blood flow by decreasing viscosity. Hypothermia decreases neuronal metabolism and reduces CBF, whereas hyperthermia has the opposite effect. $CMRO_2$ decreases by 6–7% for each reduction in 1°C.

Increased ICP may occur through one of several mechanisms: increase in CSF volume (due to blockage in CSF circulation or absorption), increase in brain tissue volume (tumor or edema), or increase in cerebral blood volume (intracranial bleed or vasodilatation). Communicating hydrocephalus occurs when the obstruction is at the point of CSF absorption (arachnoid granulation) and herniation is less likely to occur. In noncommunicating hydrocephalus, obstruction occurs within the ventricular system and herniation syndrome may be seen. Once there is a critical increase in ICP, brain herniation may occur. Ventriculostomy may be performed to decrease ICP in such cases. If drainage of CSF is performed via lumbar puncture in the presence of increased ICP, there is a risk of brain stem herniation through the foramen magnum. Aging increases intracranial CSF volume due to cerebral atrophy and creates nonpathological low-pressure hydrocephalus.

Concerns and Risks

Spinal cord lesions above C 3 lead to diaphragmatic paralysis, and the patient is totally ventilator dependent. This is because the phrenic nerve, which is the principal nerve supply of the diaphragm, is formed by C 3, 4, and 5 nerve roots. Acute spinal cord injury at the level of C 6 presents as spinal shock from a total loss of

Table 1.1 Diagnosis of cranial nerve damage in ICU

Cranial nerve	Test cranial nerve	Cerebral lobe affected	Presentation
Cranial nerve 1 (Olfactory nerve)	Not practical in ICU	Frontal lobe, pituitary tumor, anterior cranial fossa fracture	Loss of sense of smell (anosmia)
Cranial nerve 2 (Optic nerve)	Limited in unconscious patient	Distal to optic chiasm Lesion pressing on optic chiasm Lesion proximal to chiasm	Monocular blindness Bitemporal hemianopia Homonymous hemianopia
Cranial nerve 3 (Oculomotor nerve)	Pupillary exam	Uncal and temporal lobe herniation	Ptosis, divergent squint, pupillary dilatation, loss of accommodation, and light reflexes
Cranial nerve 4, 5, 6 (Trochlear, Trigeminal, Abducent)	Lightly poking the patient's cheeks with a sharp object or stimulating the nasal cavity with a cotton swab (CN V)	Cavernous sinus lesion, injury to base of skull	Loss of corneal reflex 5 (corneal damage), convergent squint 6
Cranial nerve 7 (Facial)	Facial movement	Large cerebellopontine tumors	Muscles of face, anterior two-thirds of tongue
Cranial nerve 8 (Auditory)		Cerebellopontine tumors, acoustic neuromas	Unilateral deafness
Cranial nerve 9, 10 (Glossopharyngeal, Vagus)	Gag reflex by tongue blade or suction catheter	Posterior third of tongue and pharynx	Absence of gag reflex (increased risk of aspiration), nasal speech, vocal cord paralysis 10
Cranial nerve 11 (Accessory)	Limited	Central branch arising from the medullary nuclei and spinal accessory branch arising in the first five to six cervical spinal segments from the lateral portion of the ventral horn	Central branch supplies the larynx and spinal accessory to trapezius and sternocleidomastoid – inability to shrug
Cranial nerve 12 (hypoglossal)	Limited in unconscious patient	Muscle of tongue, damaged during carotid surgery	Aspiration

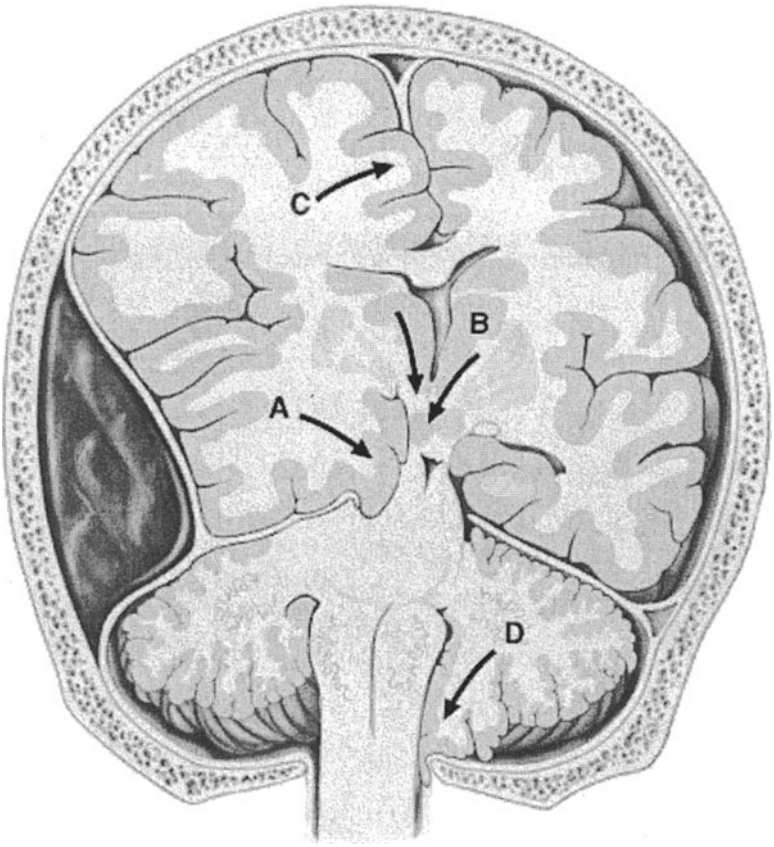

Fig. 1.14 Potential sites of brain herniation (A) uncal, (B) central, (C) subfalcine, (D) tonsillar. Transcalvarial is not shown in this figure

impulses from the higher centers. Epidural hematoma (EDH) is a traumatic accumulation of blood between the inner table of the skull and the stripped-off dural membrane. EDHs are usually arterial in origin with 70–80% located in the temporoparietal region where skull fractures cross the path of the middle meningeal artery or its dural branches. Expanding high-volume EDHs can produce a midline shift and subfalcine herniation of the brain resulting in compression of cerebral tissue and impingement on the third cranial nerve. This results in ipsilateral pupillary dilation and contralateral hemiparesis or extensor motor response. On the other hand, subdural hematomas are generally the result of venous bleeding of bridging veins (between the cortex and venous sinuses).

The brain is vulnerable to ischemic injury because of its high oxygen consumption and near-total dependence on aerobic glucose metabolism. Prolonged reduction

Table 1.2 Herniation syndromes and its clinical manifestations

Herniation of uncus of temporal lobe	Supratentorial	Pressure on ipsilateral occulomotor nerve, posterior cerebral arteries and cerebral peduncle-pupillary abnormalities, contralateral hemiplegia, possible ipsilateral medial occipital lobe infarction
Central herniation (midline structure)	Supratentorial	Downward movement of diencephalon through tentorial notch against immobile basilar artery may cause brain stem hemorrhage
Subfalcine (when the brain is pushed into the opposite half of the cranium under the falx cerebri)	Supratentorial	Cingulate gyrus injury, ischemia in anterior cerebral artery distribution
Tonsillar herniation	Supra or infratentorial	A rapid and fatal event unless it is recognized immediately and treated; the cerebral tonsil descends through the foramen magnum leading to the compression of brain stem and respiratory arrest; rarely posterior fossa mass pushing the posterior fossa contents through the tentorium cerebelli into the supratentorial compartment
Transcalvarial herniation	Supra or infratentorial	Any area beneath a defect in the skull

of CBF below 20–25 ml/100 g/min is associated with cerebral impairment (slowing of EEG), between 15 and 20 ml/100 g/min produces a flat EEG, while values below 10 ml/100 g/min are associated with irreversible structural brain damage. Autoregulation is impaired by hypoxia, ischemia, hypercapnia, trauma, and certain anesthetic agents. A rightward shift in the autoregulatory curve occurs with chronic hypertension and sympathetic activation (shock or stress). A leftward shift occurs in hypoxia, hypercarbia, and vasodialators. When CPP exceeds the upper limit of autoregulation, the BBB may be disrupted, leading to cerebral edema.

Any increase in ICP is initially well compensated; however, once the compliance is exhausted a small increase in volume causes an exponential rise in ICP leading to herniation (see Fig. 1.14). Herniation syndromes (Table 1.2) can be reversible if treated immediately and effectively and are encountered when the pressure in one compartment of the brain results in extrusion of the contents into an adjacent compartment with accompanying mechanical damage. Depressed level of consciousness with dilated pupils is the first clinical sign of a herniation syndrome and is usually accompanied by a Cushing response (elevation in the systolic blood pressure with accompanying bradycardia).

Key Points

- In the corticospinal tract, lesions above the medullary decussation cause contralateral paralysis and those below medullary decussation cause ipsilateral paralysis.
- Brain anatomy is important in identifying the different non-silent areas of the brain with respect to the surgical procedure being performed and helps to prevent devastating deficits.
- Cranial nerve injury is a significant risk in surgery of the cerebellopontine angle and brain stem.
- Lumbar puncture for drainage of CSF is performed below the L1 vertebra to prevent damage to the spinal cord. Most spinal cord injuries occur at the mid-cervical or thoracolumbar region. The watershed area of the spinal cord blood flow is the mid-thoracic region.
- CBF is linearly related to $PaCO_2$ from 20 to 70 mmHg when the autoregulation is intact. Hypocapnia causes cerebral vasoconstriction and can lead to brain ischemia. Hypercapnea causes vasodilatation and increases the CBF.
- Increased ICP may occur through one of several mechanisms: increase in CSF volume (due to blockage in CSF circulation or absorption), increase in brain tissue volume (tumor or edema), or increase in cerebral blood volume (intracranial bleed or vasodilatation).
- The brain is vulnerable to ischemic injury because of its high oxygen consumption and near-total dependence on aerobic glucose metabolism.
- Any increase in ICP is initially well compensated; however, once the compliance is exhausted, a small increase in volume causes an exponential rise in ICP leading to herniation.

Suggested Reading

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