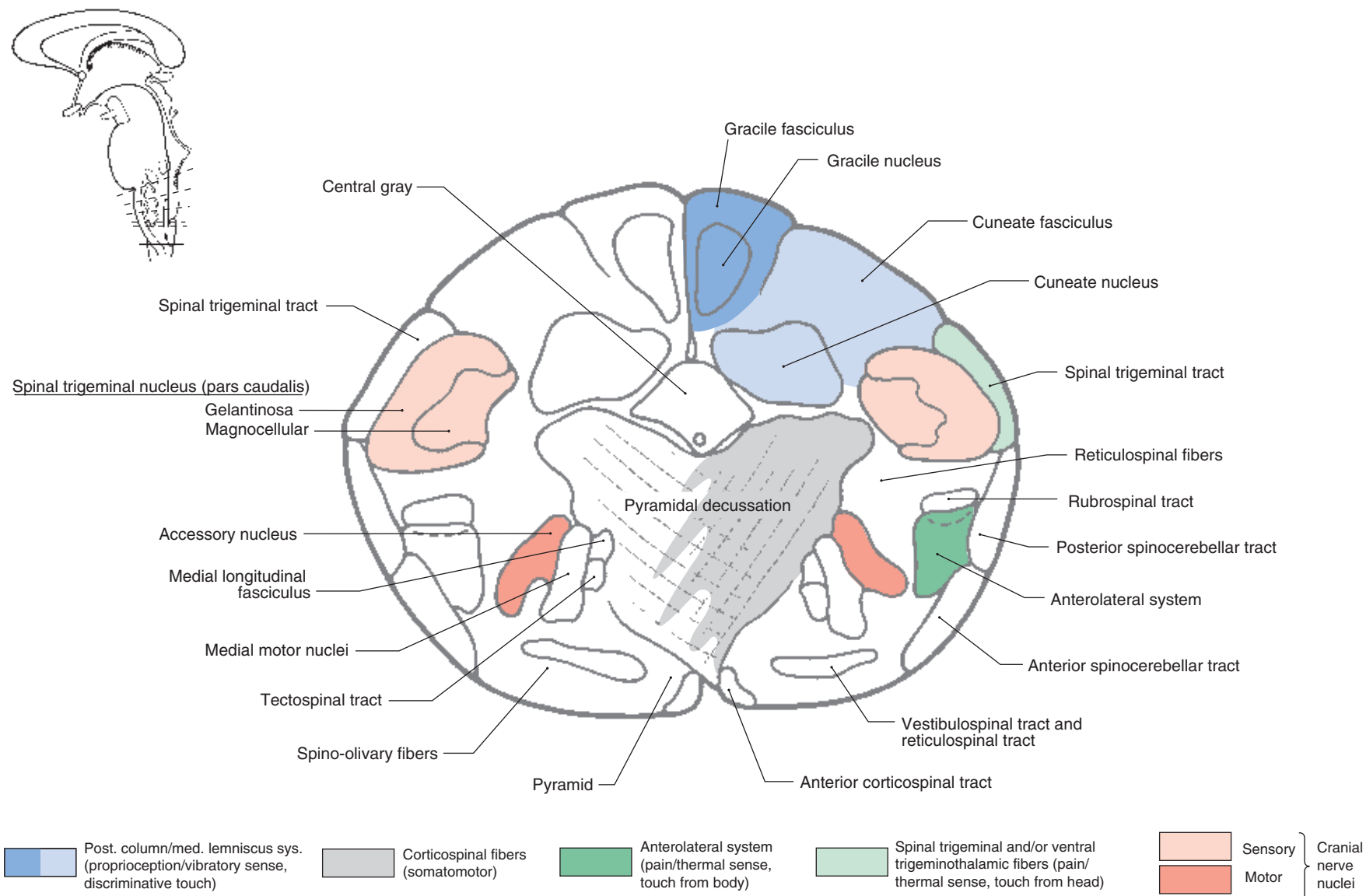
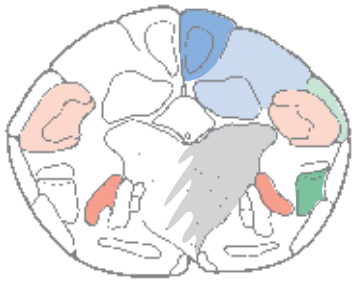
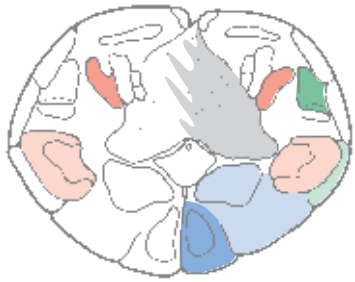


5-8 Transverse section of the medulla through the *decussation of the pyramids* (motor decussation, pyramidal decussation, crossing of corticospinal fibers). This is the level of the spinal cord–medulla transition. The corticospinal fibers have moved from their location in the lateral funiculus to the motor decussation and will cross to form the pyramid on the opposite side.

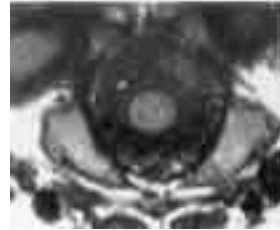




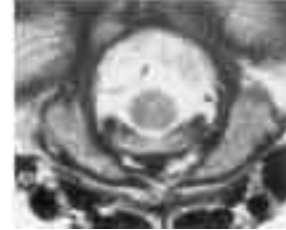
Anatomical orientation



Clinical orientation



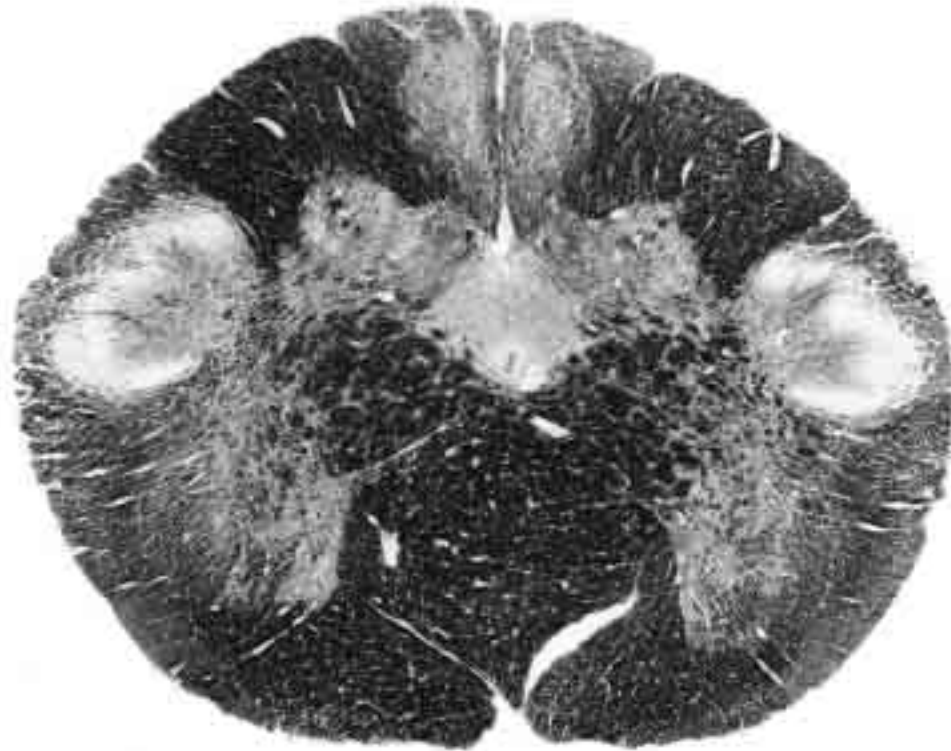
MRI, T1-weighted image



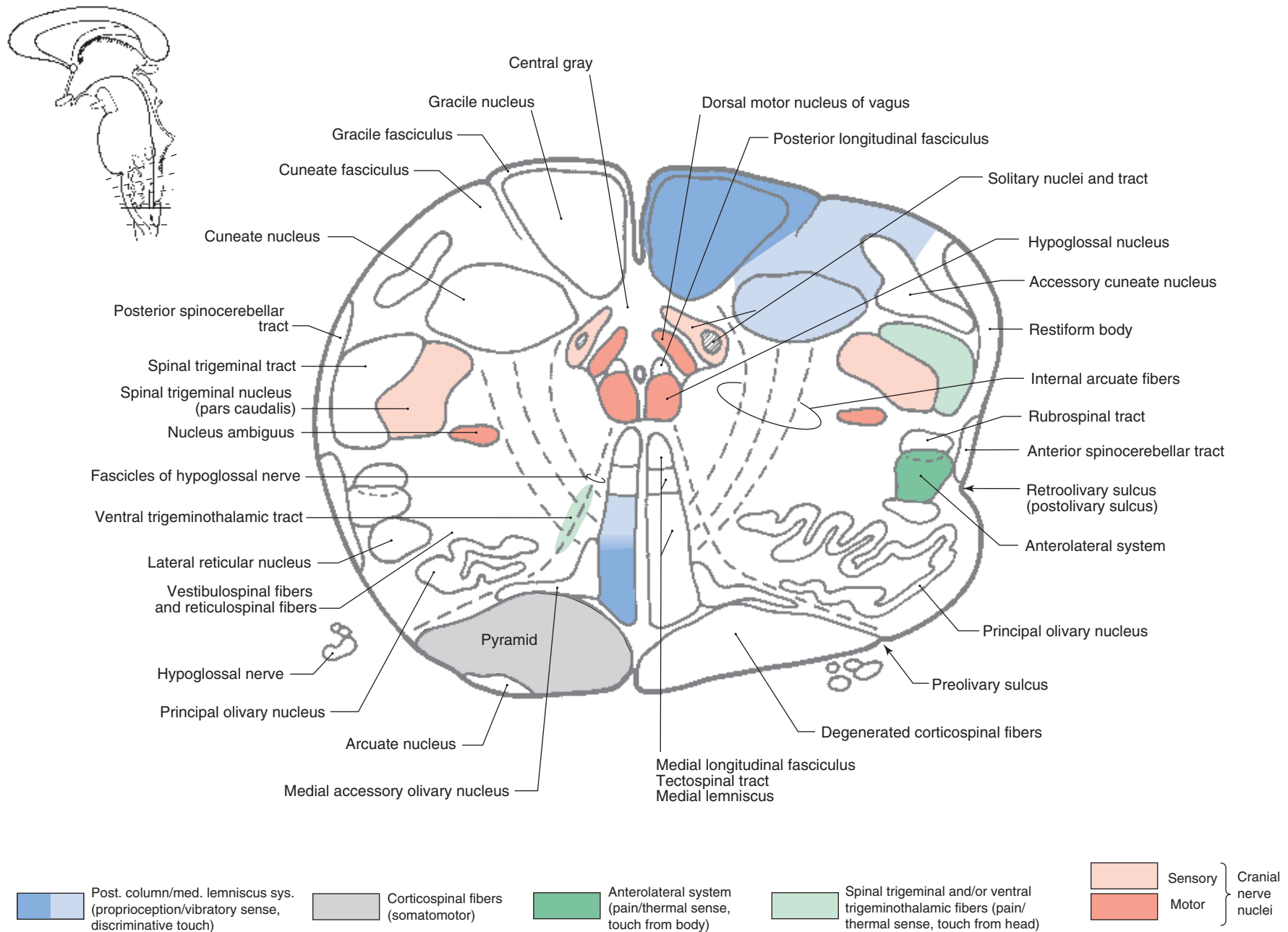
MRI, T2-weighted image

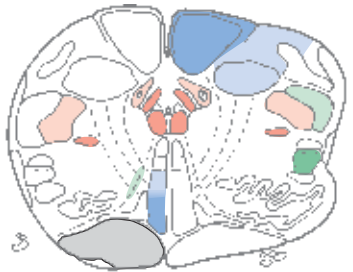


CT cisternogram

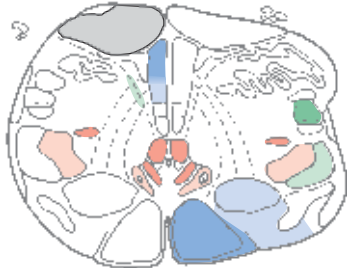


5-9 Transverse section of the medulla through the *dorsal column nuclei* (nucleus gracilis and nucleus cuneatus), caudal portions of the *hypoglossal nucleus*, caudal end of the *principal olivary nucleus*, and middle portions of the *sensory decussation* (crossing of internal arcuate fibers).





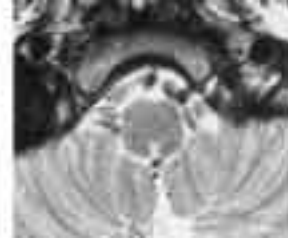
Anatomical orientation



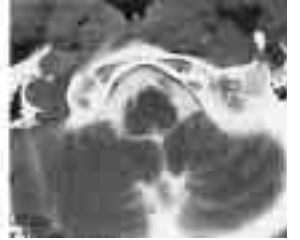
Clinical orientation



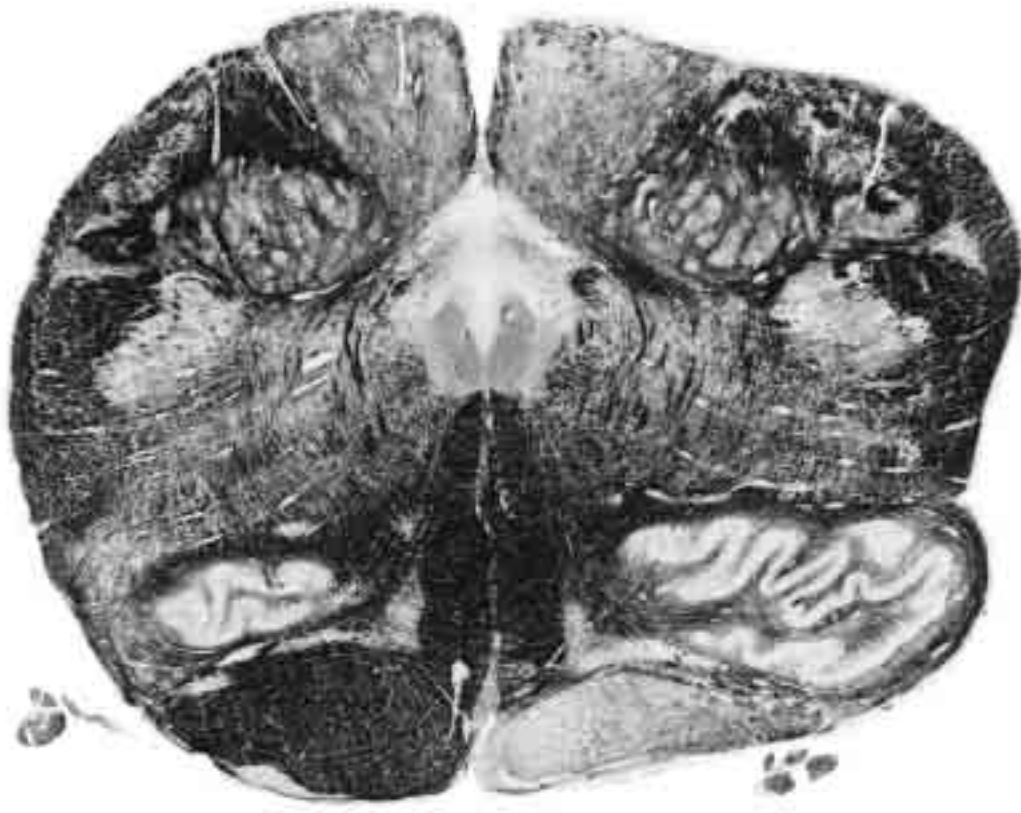
MRI, T1-weighted image



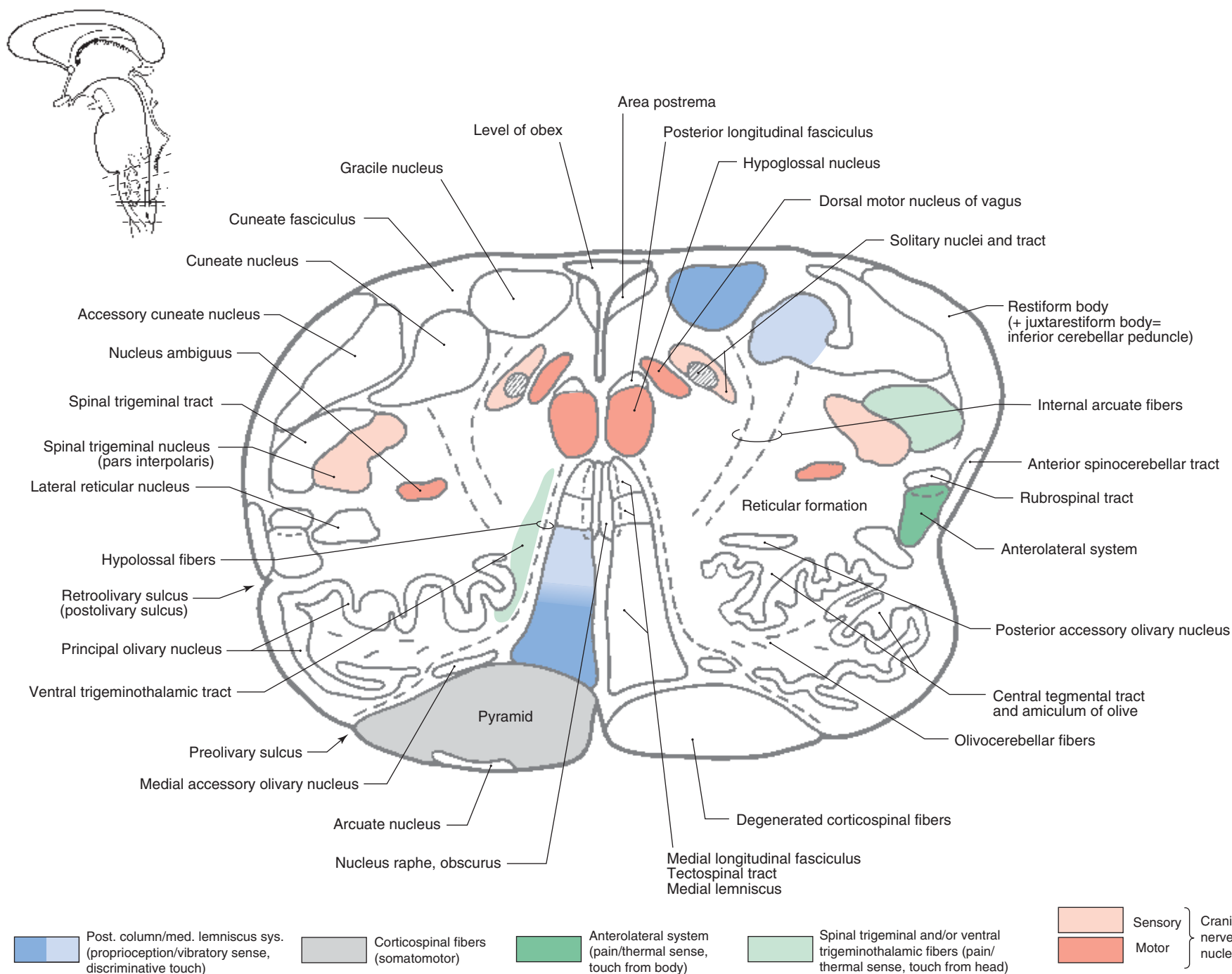
MRI, T2-weighted image

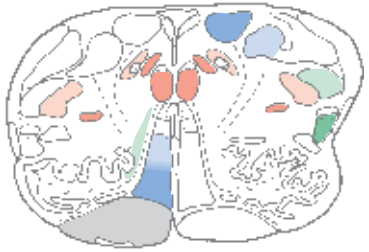


CT cisternogram

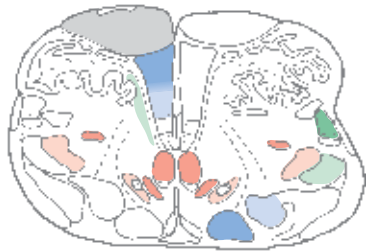


5-10 Transverse section of the medulla through rostral portions of the sensory decussation (*crossing of internal arcuate fibers*), obex, and the caudal one-third of the *hypoglossal* and *principal olivary* nuclei.

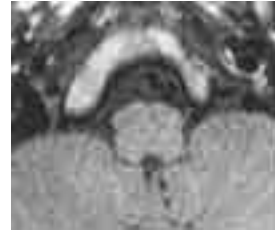




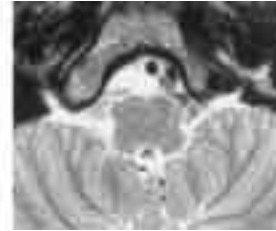
Anatomical orientation



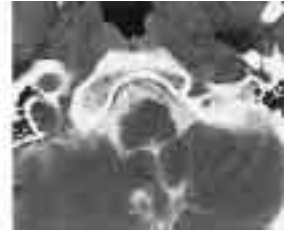
Clinical orientation



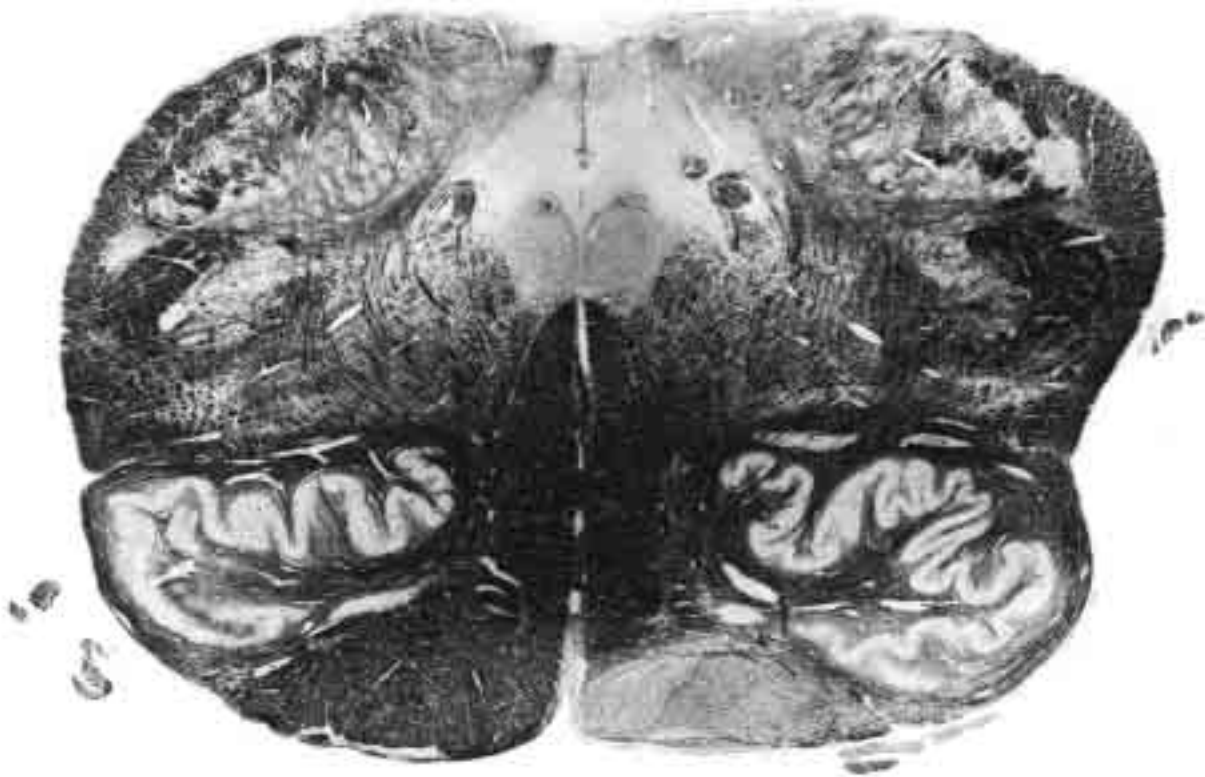
MRI, T1-weighted image



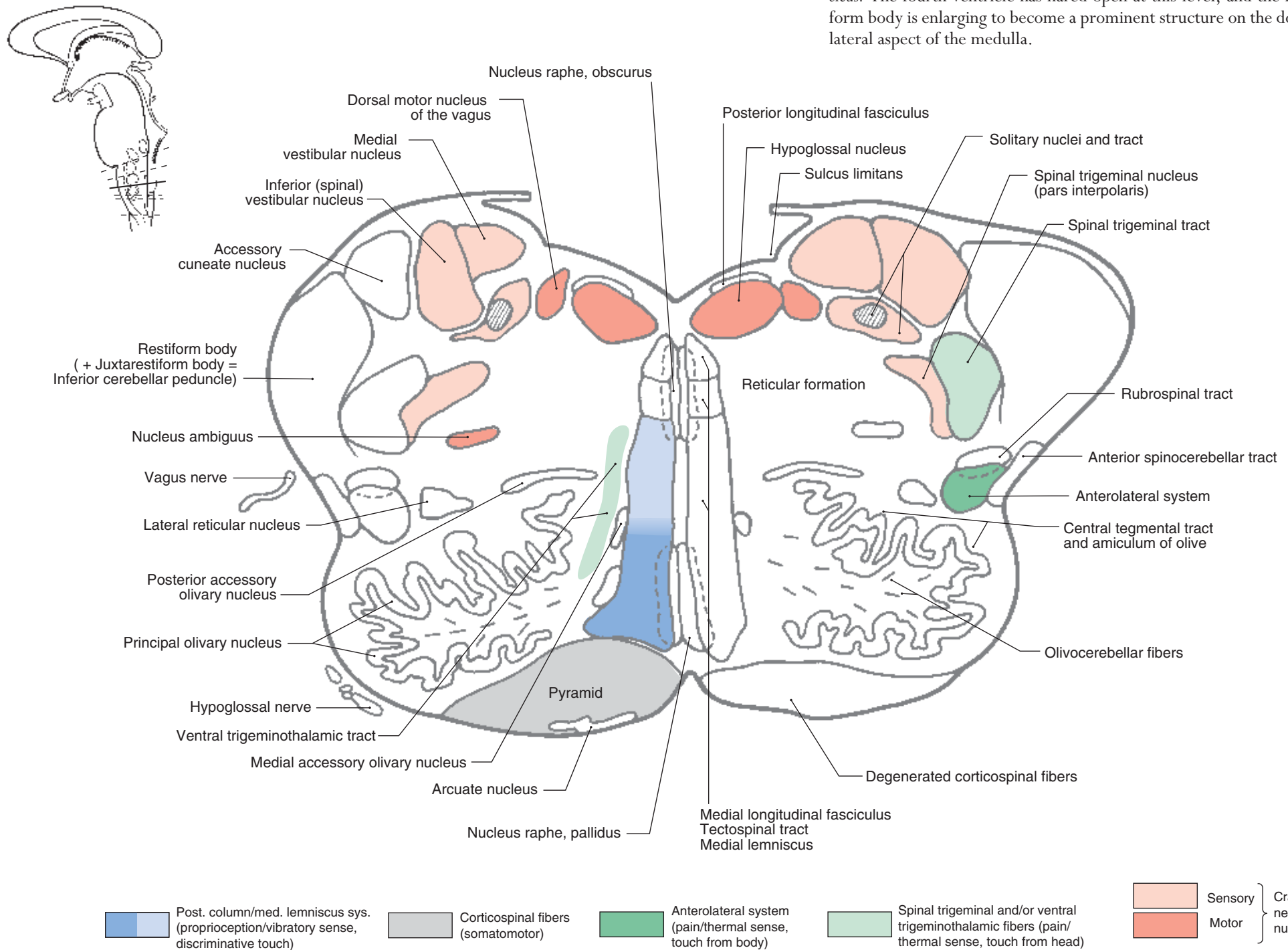
MRI, T2-weighted image

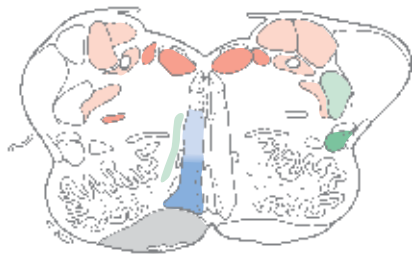


CT cisternogram

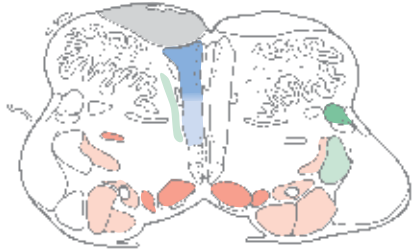


5-11 Transverse section of the medulla through rostral portions of the *hypoglossal nucleus* and the middle portions of the *principal olivary nucleus*. The fourth ventricle has flared open at this level, and the restiform body is enlarging to become a prominent structure on the dorso-lateral aspect of the medulla.





Anatomical orientation



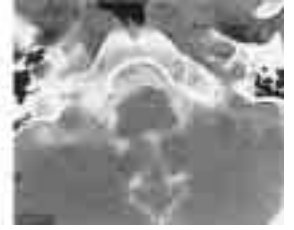
Clinical orientation



MRI, T1-weighted image



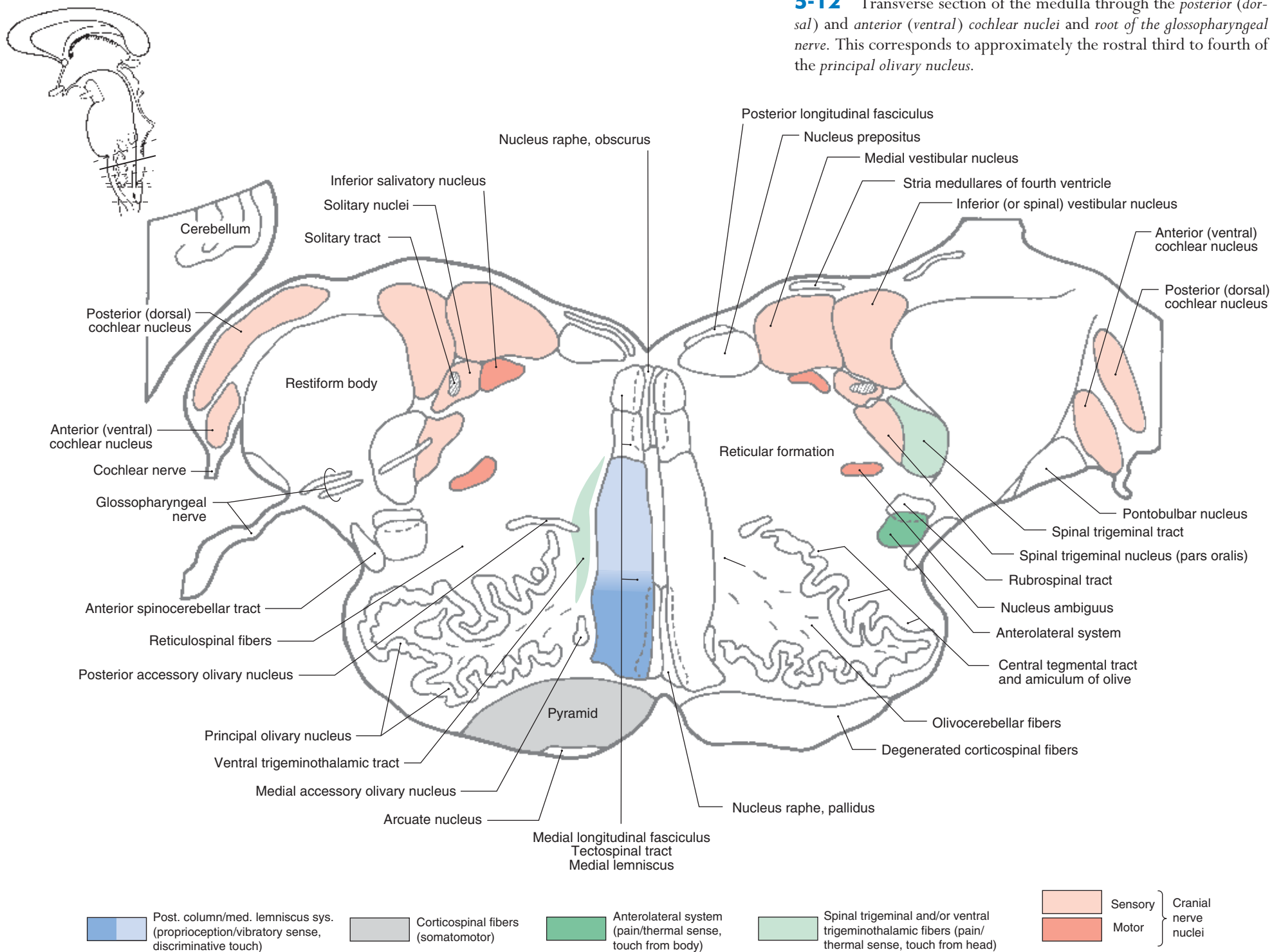
MRI, T2-weighted image

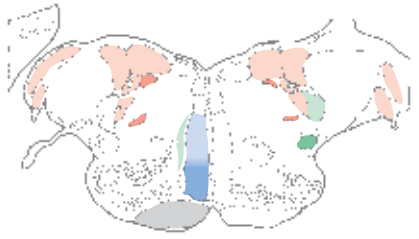


CT cisternogram

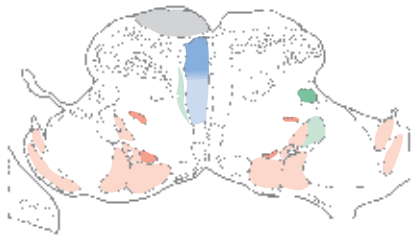


5-12 Transverse section of the medulla through the *posterior (dorsal) and anterior (ventral) cochlear nuclei and root of the glossopharyngeal nerve*. This corresponds to approximately the rostral third to fourth of the *principal olivary nucleus*.





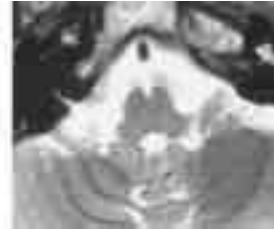
Anatomical orientation



Clinical orientation



MRI, T1-weighted image



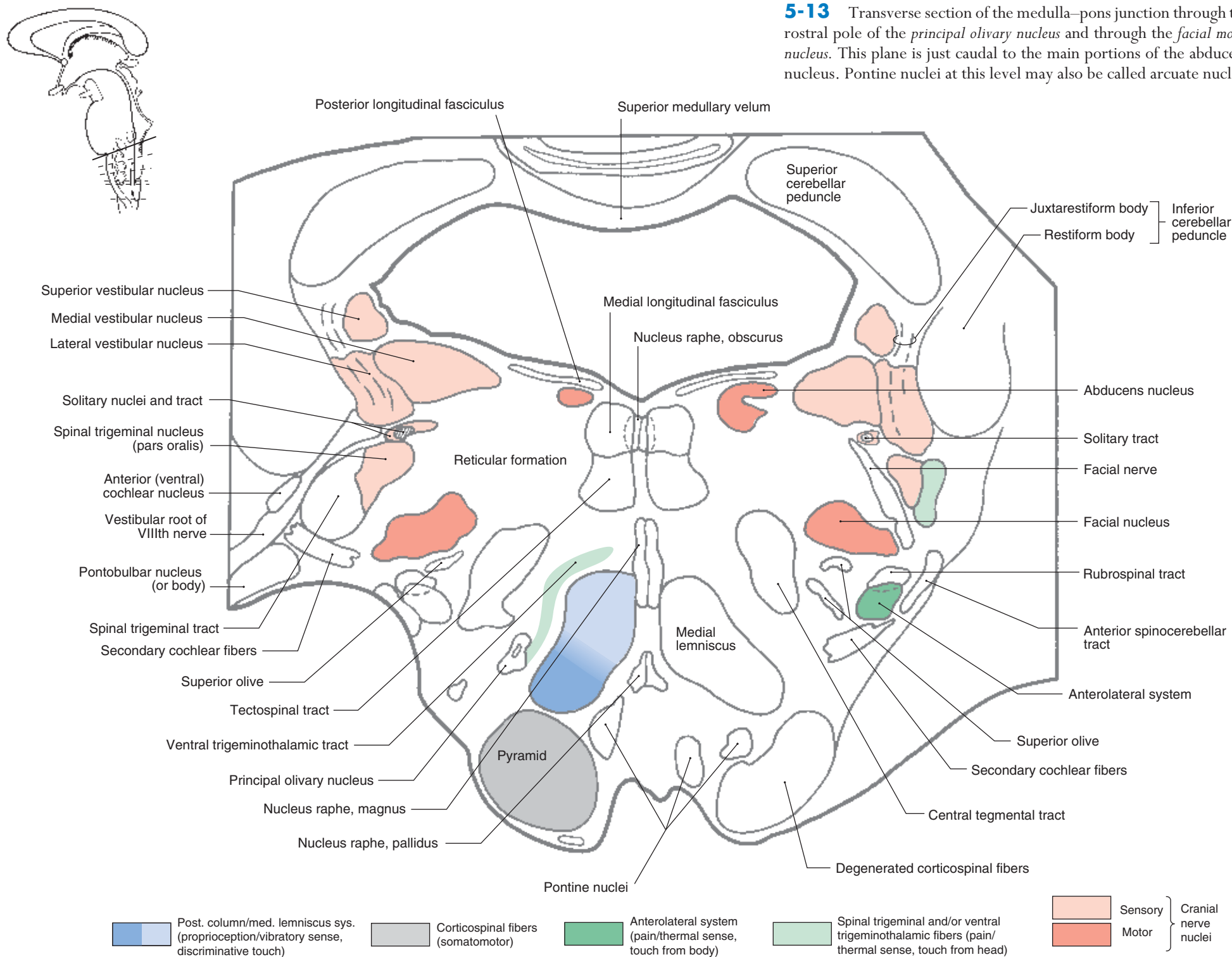
MRI, T2-weighted image



CT cisternogram

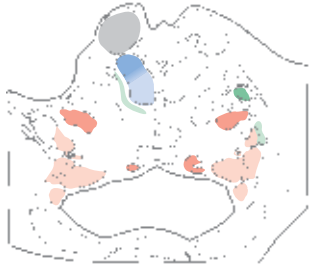


5-13 Transverse section of the medulla–pons junction through the rostral pole of the *principal olivary nucleus* and through the *facial motor nucleus*. This plane is just caudal to the main portions of the abducens nucleus. Pontine nuclei at this level may also be called arcuate nuclei.





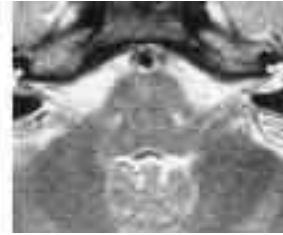
Anatomical orientation



Clinical orientation



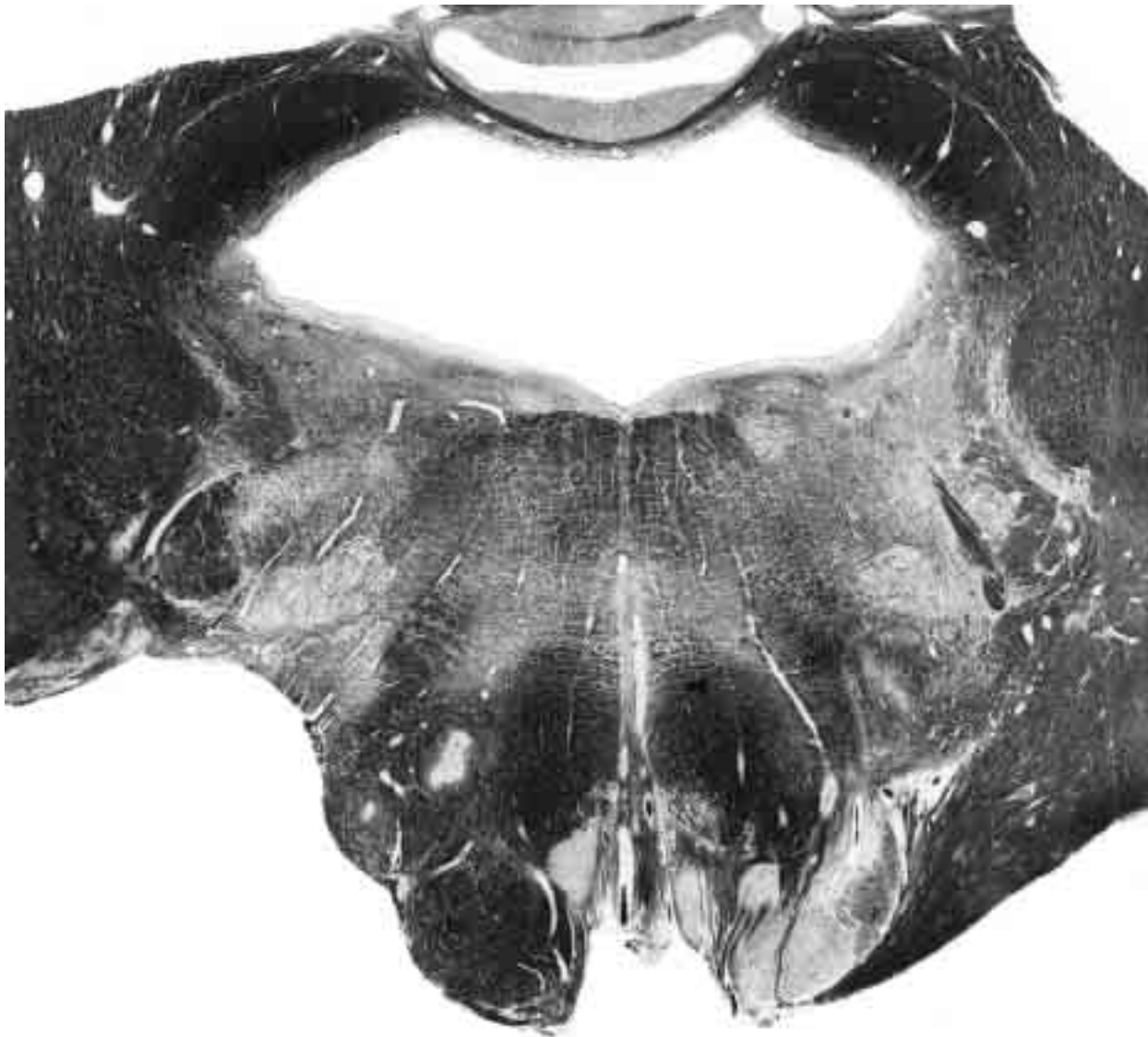
MRI, T1-weighted image



MRI, T2-weighted image



CT cisternogram



Vascular Syndromes or Lesions of the Medulla Oblongata

Medial Medullary Syndrome: Results from occlusion of branches of anterior spinal artery.

Deficits	Structure Damaged
<ul style="list-style-type: none"> • Contralateral hemiplegia of arm and leg • Contralateral loss of position sense, vibratory sense and discriminative touch • Deviation of tongue to ipsilateral side when protruded; muscle atrophy and fasciculations 	<ul style="list-style-type: none"> • Pyramid (cortico-spinal fibers) • Medial lemniscus • Hypoglossal nerve in medulla or hypoglossal nucleus

Comment: The medial medullary syndrome is rare compared to the more common occurrence of the lateral medullary syndrome. *Nystagmus* may result if the lesion involves the medial longitudinal fasciculus or the nucleus prepositus hypoglossi. The lesion may involve ventral trigeminothalamic fibers, but diminished pain and thermal sense from the contralateral side of the face is rarely seen. The combination of a contralateral hemiplegia and ipsilateral deviation of the tongue is called an *inferior alternating hemiplegia* when the lesion is at this level.

Lateral Medullary Syndrome: Results from occlusion of posterior inferior cerebellar artery or branches of PICA to dorsolateral medulla (PICA syndrome, Wallenberg syndrome). In many cases the lateral medullary syndrome frequently results from occlusion of the vertebral artery with consequent loss of flow into PICA.

Deficits	Structure Damaged
<ul style="list-style-type: none"> • Contralateral loss of pain and thermal sense on body • Ipsilateral loss of pain and thermal sense on face • Dysphagia, soft palate paralysis, hoarseness, diminished gag reflex 	<ul style="list-style-type: none"> • Anterolateral system fibers • Spinal trigeminal tract and nucleus • Nucleus ambiguus, roots of 9th and 10th nerves

- Ipsilateral Horner syndrome (miosis, ptosis, anhidrosis, flushing of face)
- Nausea, diplopia, tendency to fall to ipsilateral side, nystagmus, vertigo
- Ataxia to the ipsilateral side
- Descending hypothalamospinal fibers
- Vestibular nuclei (mainly inferior and medial)
- Restiform body and spinocerebellar fibers

Comment: In addition to the above, involvement of the solitary tract and nucleus may (rarely) cause *dysageusia*. *Dyspnea* and *tachycardia* may be seen in patients with damage to the dorsal motor nucleus of the vagus. It is also possible that damage to respiratory centers in the reticular formation or to the vagal motor nucleus may result in hiccup (*singultus*). Bilateral medullary damage may cause the syndrome of “Ondine’s curse,” an inability to breathe without willing it or “thinking about it.”

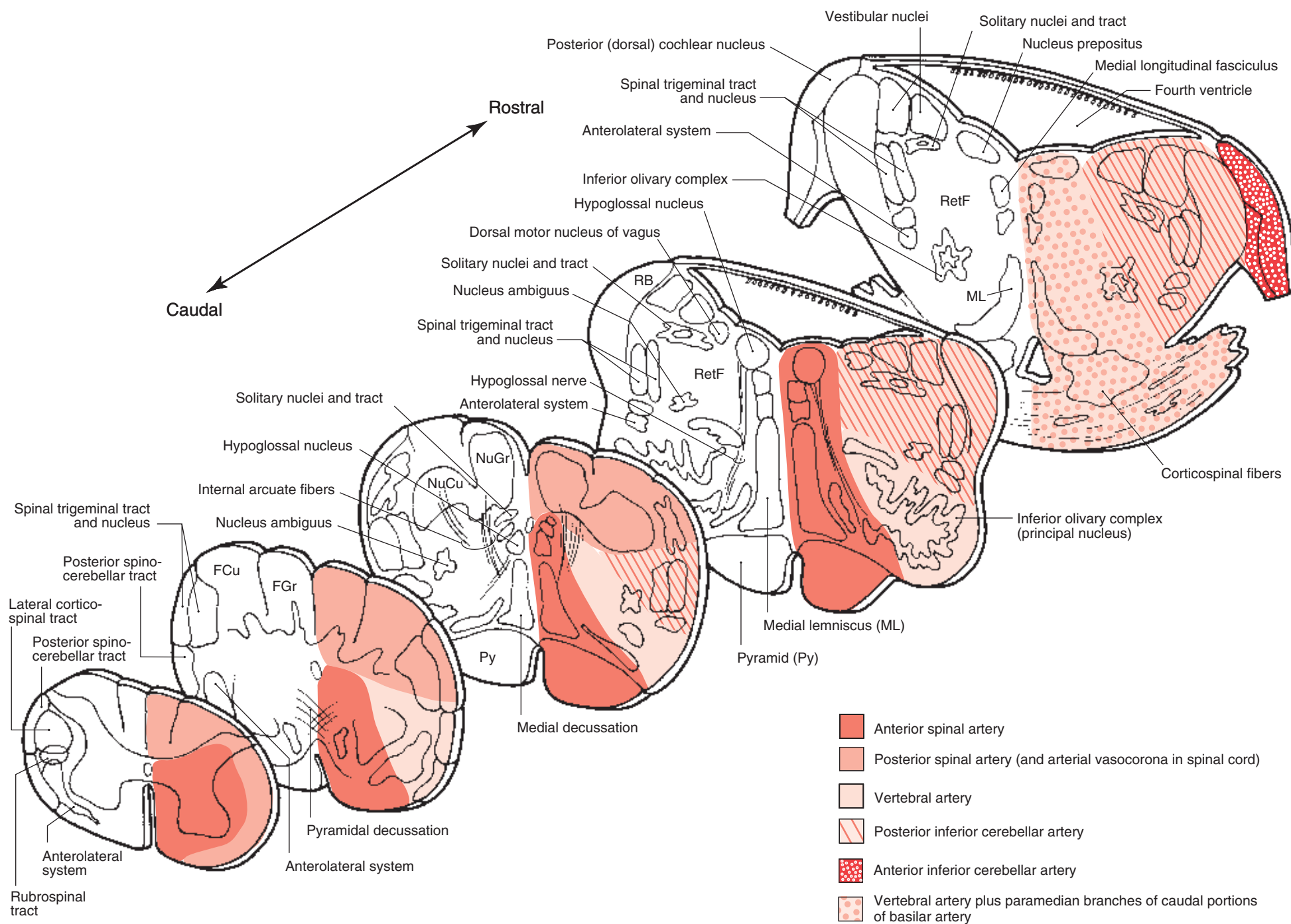
Tonsillar Herniation: Although the cerebellar tonsil is not part of the medulla, the herniation of this structure (*tonsillar herniation*) down through the foramen magnum has serious consequences for function of the medulla. The coneing of the cerebellar tonsils into, and through, the foramen magnum may compress the medulla resulting in *cardiac and respiratory arrest*. This is due to a combination of pressure on the medulla and the occlusion of small vessels serving cardiac and respiratory centers in the lateral area of the medulla. Patients experiencing a sudden herniation of the cerebellar tonsils may lose consciousness rapidly and die.

Syringobulbia: A cavitation within the brainstem (*syringobulbia*) may exist with syringomyelia, be independent of syringomyelia, or in some cases both may exist and communicate with each other. The cavity in syringobulbia is usually on one side of the midline of the medulla. Signs and symptoms of syringobulbia may include *weakness of tongue muscles* (hypoglossal nucleus or nerve), *weakness of pharyngeal, palatal, and vocal musculature* (ambiguus nucleus), *nystagmus* (vestibular nuclei), and *loss of pain and thermal sensation on the ipsilateral side of the face* (spinal trigeminal tract and nucleus or crossing of trigeminothalamic fibers).

5-14 Semidiagrammatic representation of the internal distribution of arteries in the medulla oblongata. Selected main structures are labeled primarily on the left side of each section and the general pattern of arterial distribution overlies these structures on the right side. The general distribution patterns of arteries in the medulla as illustrated here may vary from patient to patient. For example, the territories served by adjacent vessels may overlap to differing degrees at their margins or the territory of a particular vessel may be smaller or larger than seen in the typical pattern.

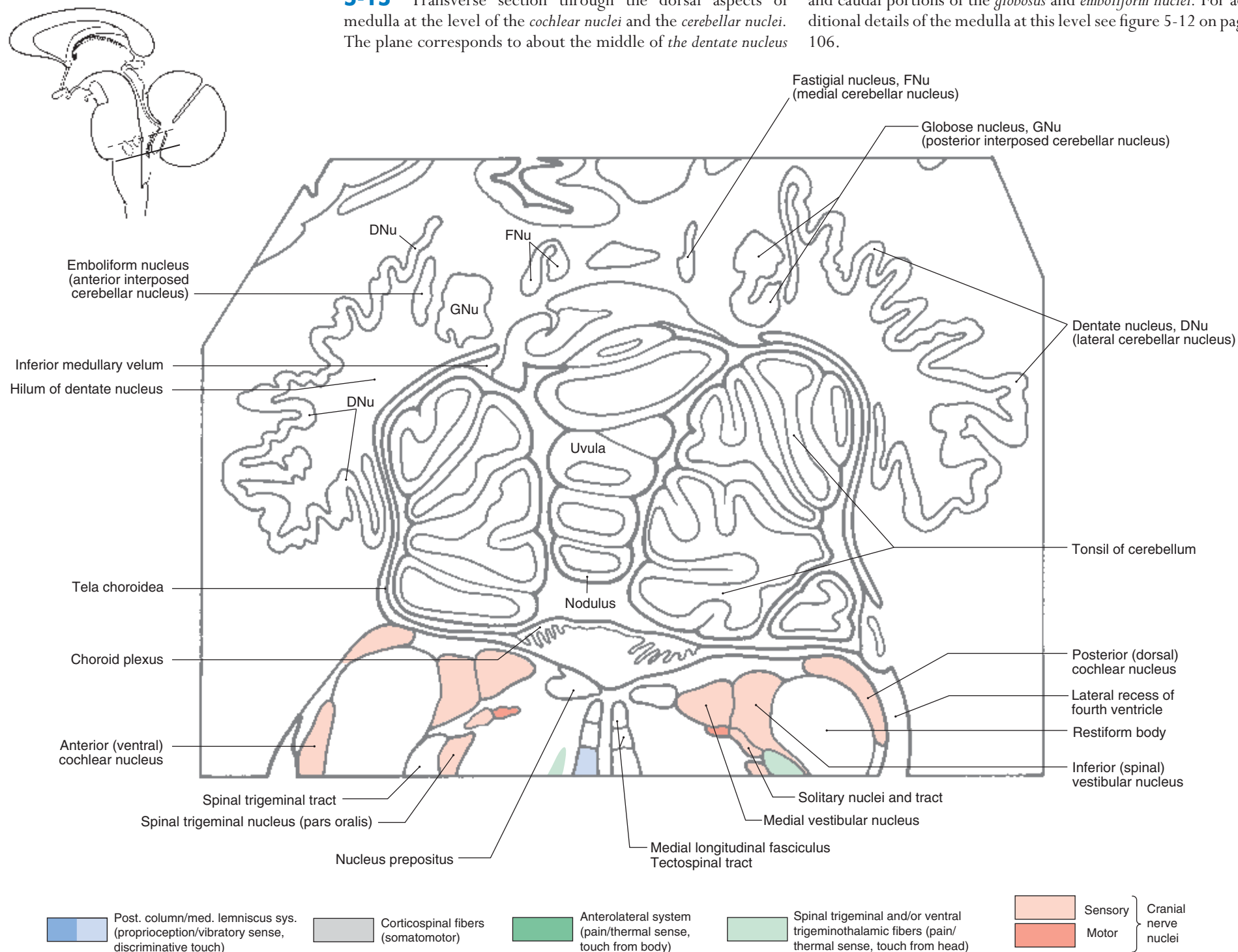
Abbreviations

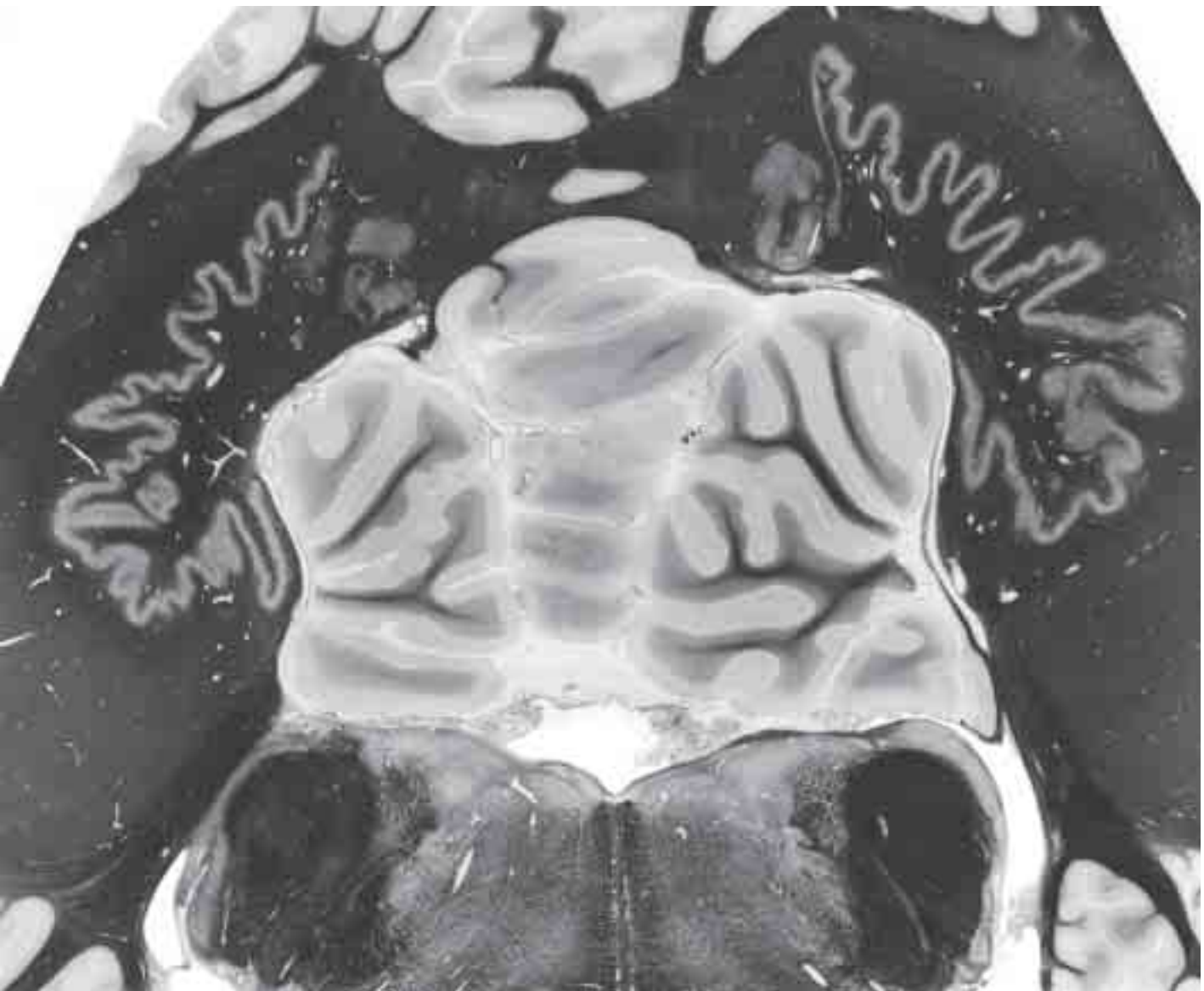
FCu	Cuneate fasciculus
FGr	Gracile fasciculus
ML	Medial lemniscus
NuCu	Cuneate nucleus
NuGr	Gracile nucleus
Py	Pyramid
RB	Restiform body (+ juxtarestiform body = inferior cerebellar peduncle)
RetF	Reticular formation



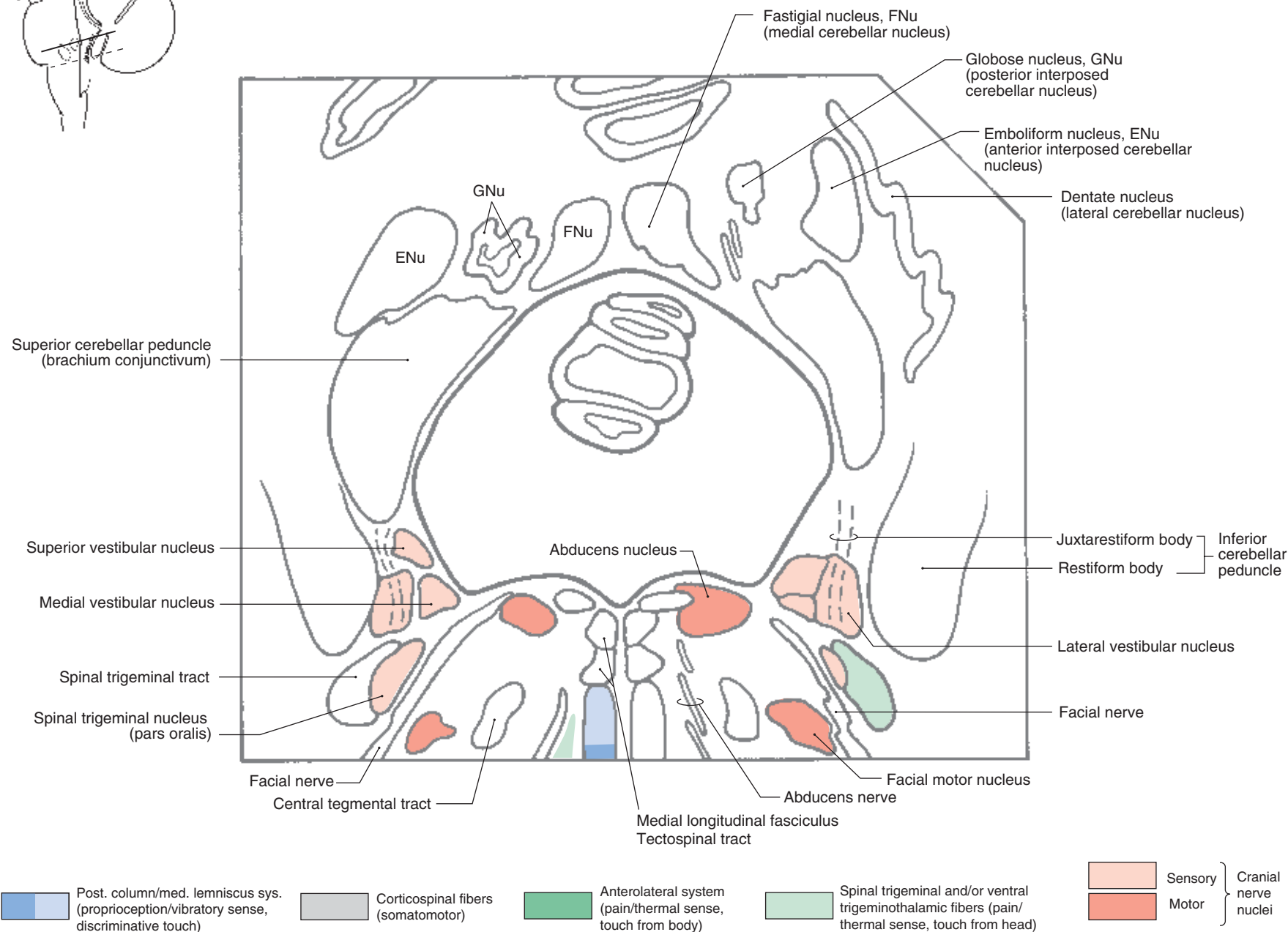
5-15 Transverse section through the dorsal aspects of medulla at the level of the *cochlear nuclei* and the *cerebellar nuclei*. The plane corresponds to about the middle of the *dentate nucleus*

and caudal portions of the *globosus* and *emboliform nuclei*. For additional details of the medulla at this level see figure 5-12 on page 106.



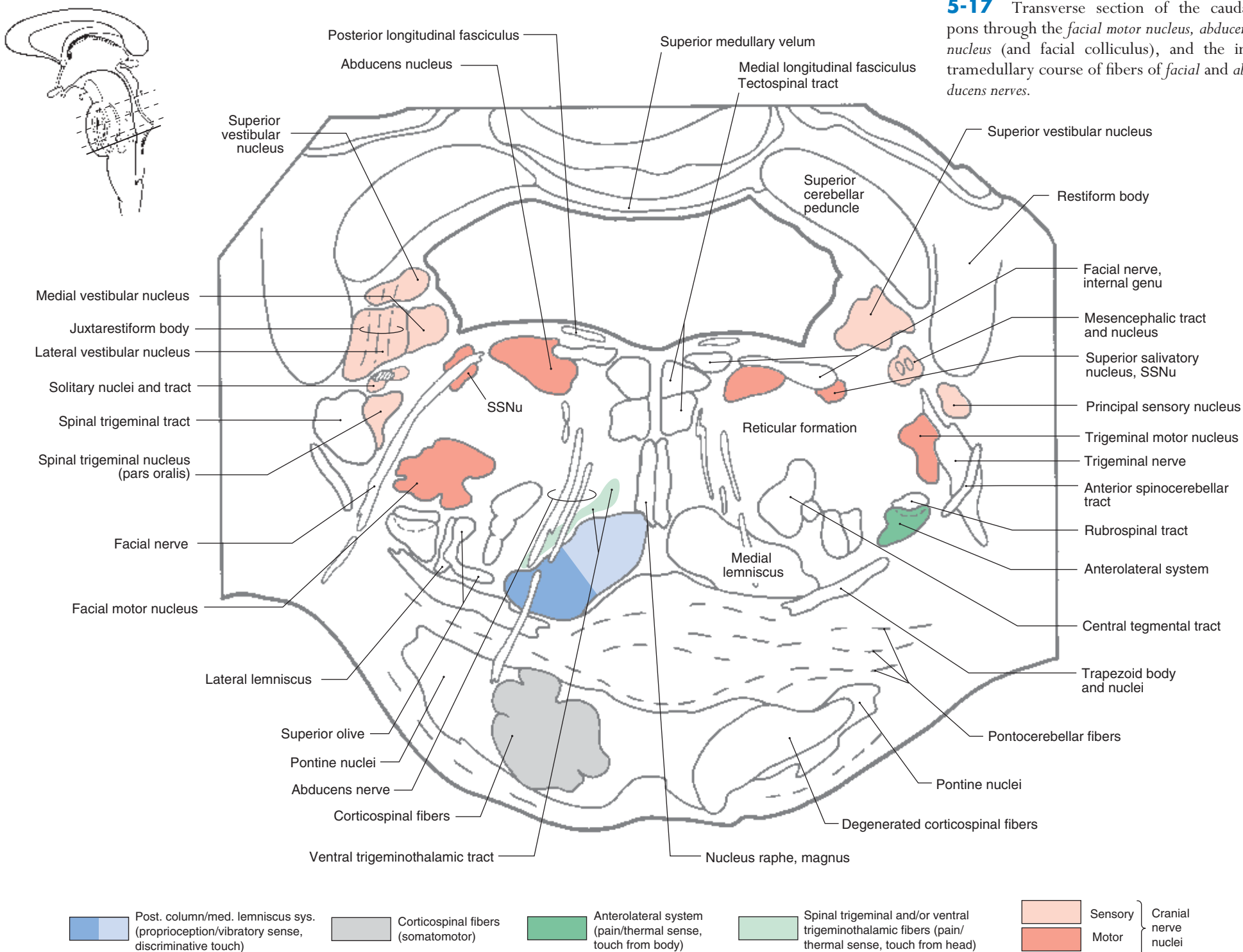


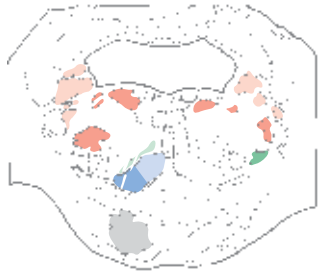
5-16 Transverse section through dorsal portions of pons at the level of the *abducens nucleus* (and *facial colliculus*) and through rostral portions of the *cerebellar nuclei*. For additional details of the pons at this level see Figure 5-17 on page 116.



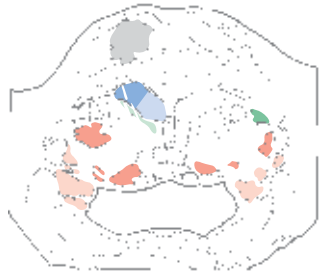


5-17 Transverse section of the caudal pons through the *facial motor nucleus*, *abducens nucleus* (and facial colliculus), and the intramedullary course of fibers of *facial* and *abducens* nerves.





Anatomical orientation



Clinical orientation



MRI, T1-weighted image



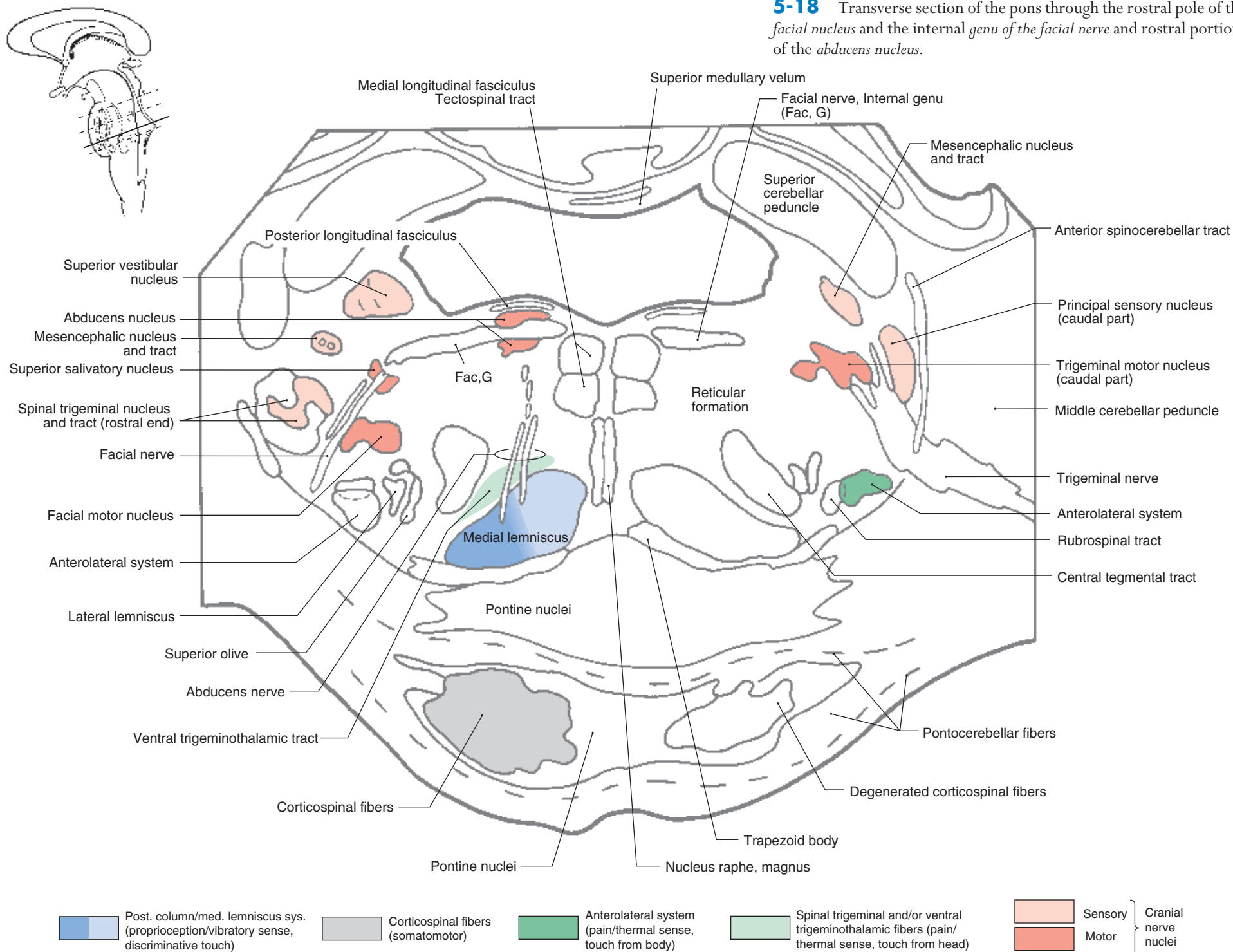
MRI, T2-weighted image

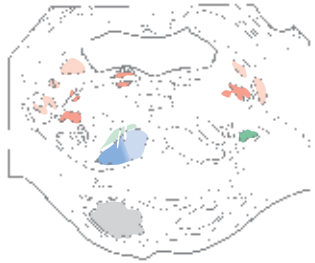


CT cisternogram

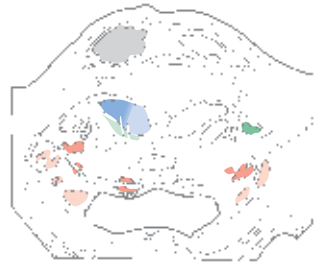


5-18 Transverse section of the pons through the rostral pole of the *facial nucleus* and the *internal genu of the facial nerve* and rostral portions of the *abducens nucleus*.





Anatomical orientation



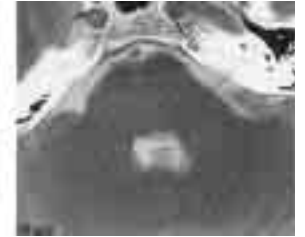
Clinical orientation



MRI, T1-weighted image



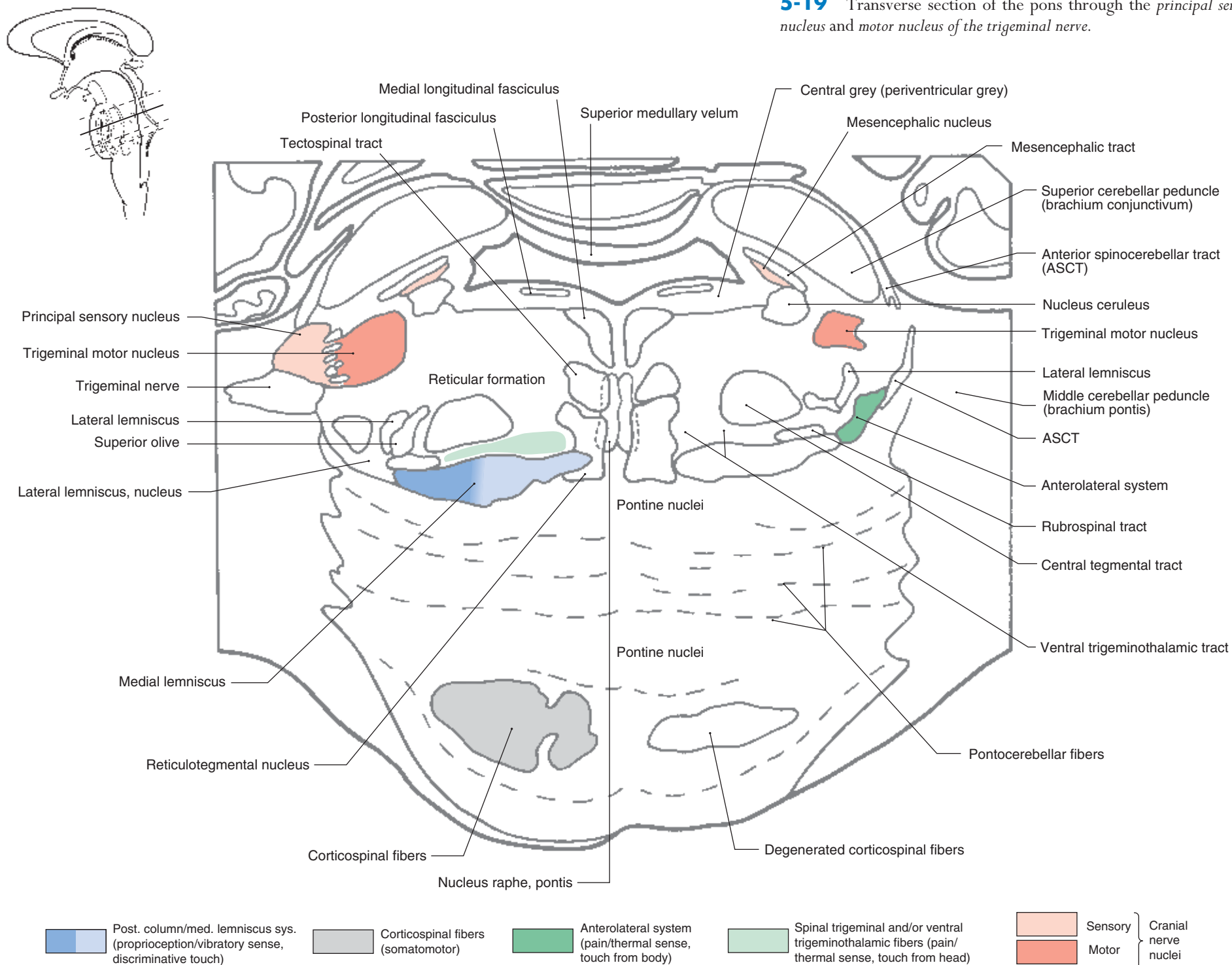
MRI, T2-weighted image

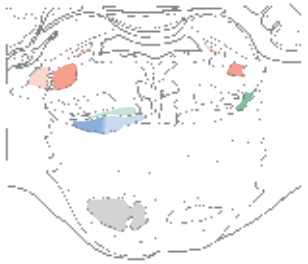


CT cisternogram

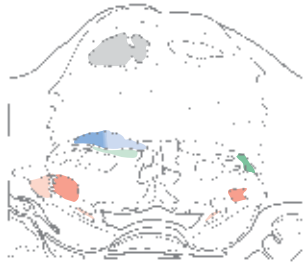


5-19 Transverse section of the pons through the *principal sensory nucleus* and *motor nucleus of the trigeminal nerve*.

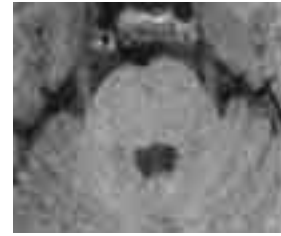




Anatomical orientation



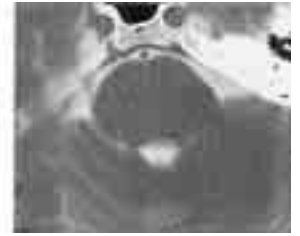
Clinical orientation



MRI, T1-weighted image



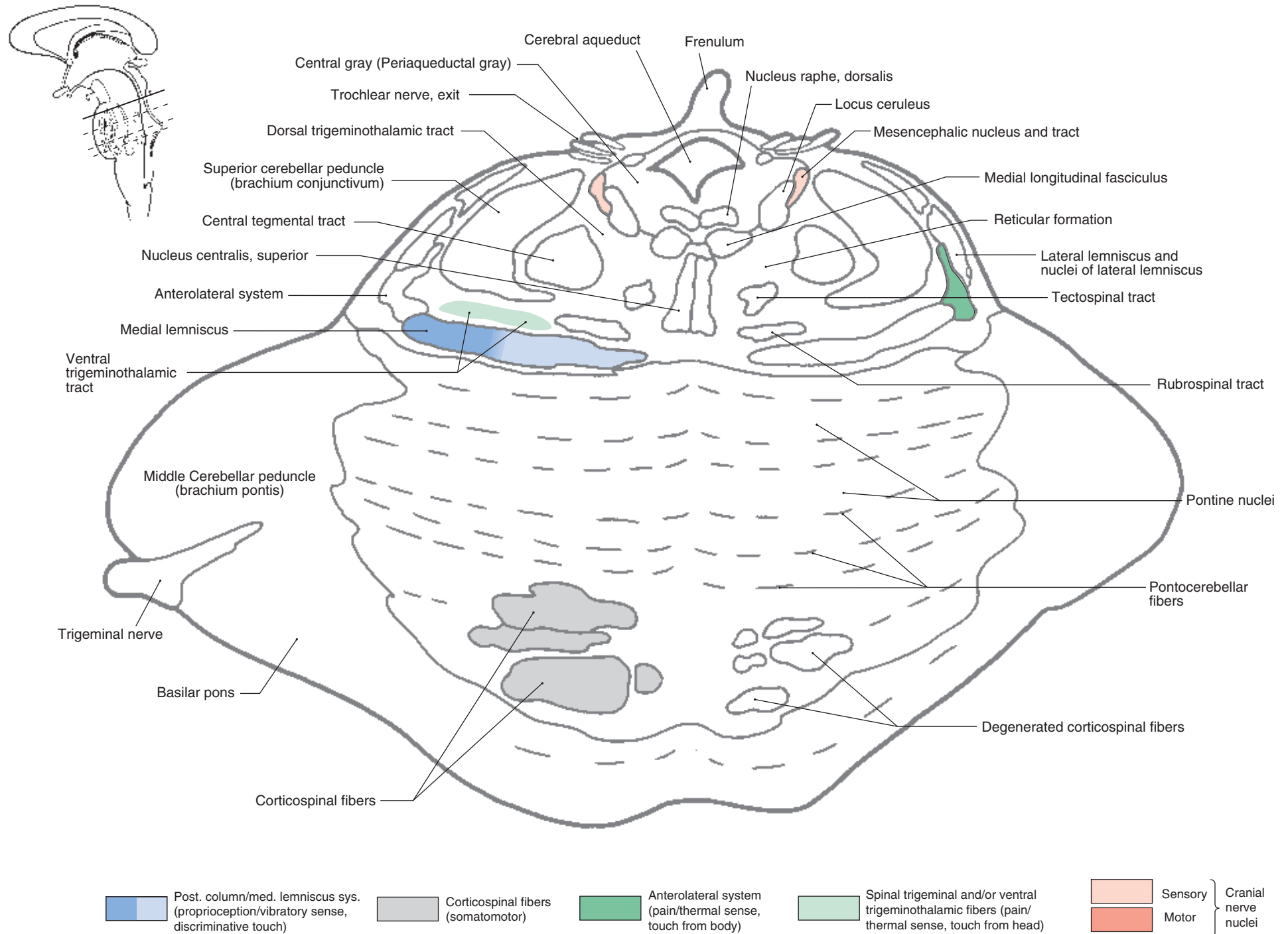
MRI, T2-weighted image

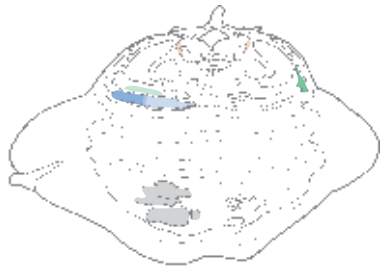


CT cisternogram



5-20 Transverse section of the rostral pons through the *exit of the trochlear nerve* and rostral portions of the *exit of the trigeminal nerve*.





Anatomical orientation



Clinical orientation



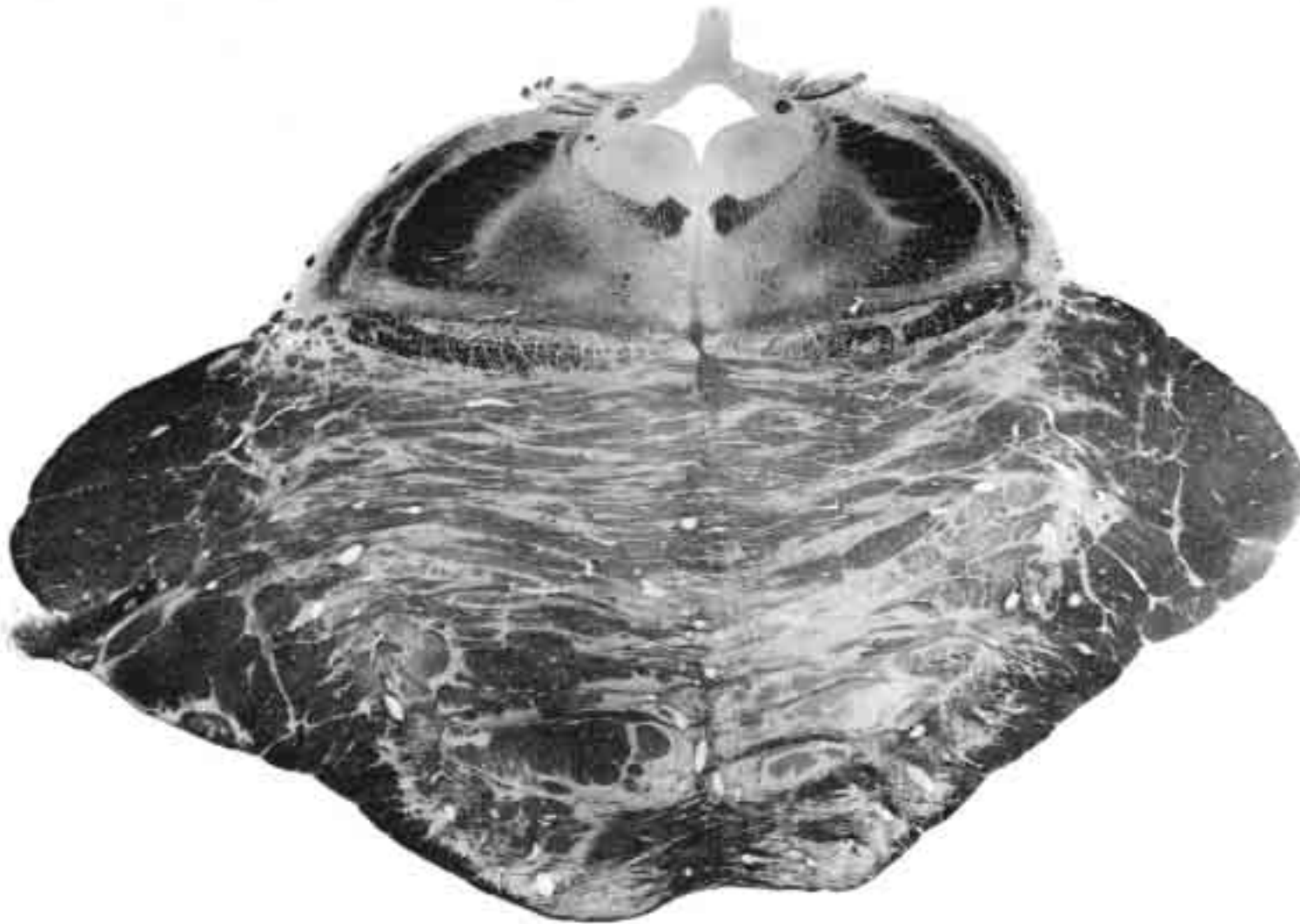
MRI, T1-weighted image



MRI, T2-weighted image



CT cisternogram



Vascular Syndromes or Lesions of the Pons

Medial Pontine Syndrome: Results from occlusion of paramedian branches of basilar artery.

Deficits	Structure Damaged
• Contralateral hemiplegia of arm and leg	• Corticospinal fibers in basilar pons
• Contralateral loss or decrease of position and vibratory sense and discriminative touch (arm and leg)	• Medial lemniscus
• Ipsilateral lateral rectus muscle paralysis	• Abducens nerve fibers or nucleus
• Paralysis of conjugate gaze toward side of lesion	• Paramedian pontine reticular formation (pontine gaze center)

Comment: The combination of corticospinal deficits on one side of the body coupled with a cranial nerve motor deficit on the opposite is called a *middle alternating hemiplegia* when the lesion is at this level. *Diplopia* will result (abducens nerve lesion) on gaze toward the side of the lesion. Involvement of the abducens nucleus may also result in an inability to adduct the contralateral medial rectus muscle (damage to abducens internuclear neurons).

At caudal levels the lesion may extend lateral to involve the lateral lemniscus (*hypacusis*), parts of the middle cerebellar peduncle (some *ataxia*), the facial motor nucleus (*ipsilateral facial paralysis*), the spinal trigeminal tract and nucleus (*ipsilateral loss of pain and thermal sensation from the face*), and the anterolateral system (*contralateral loss of pain and thermal sensation from the body*). At rostral pontine levels the lesion may extend into the medial lemniscus or may involve only the arm fibers within this structure (*contralateral loss of vibratory sense, proprioception, and discriminative touch*), the motor nucleus of the trigeminal nerve (*ipsilateral paralysis of masticatory muscles*), or may damage the anterolateral system and rostral portions of the spinal trigeminal tract and nucleus (*loss of pain and thermal sensation from the body [contralateral] and from the face [ipsilateral]*).

Lateral Pontine Syndrome: Results from occlusion of long circumferential branches of basilar artery.

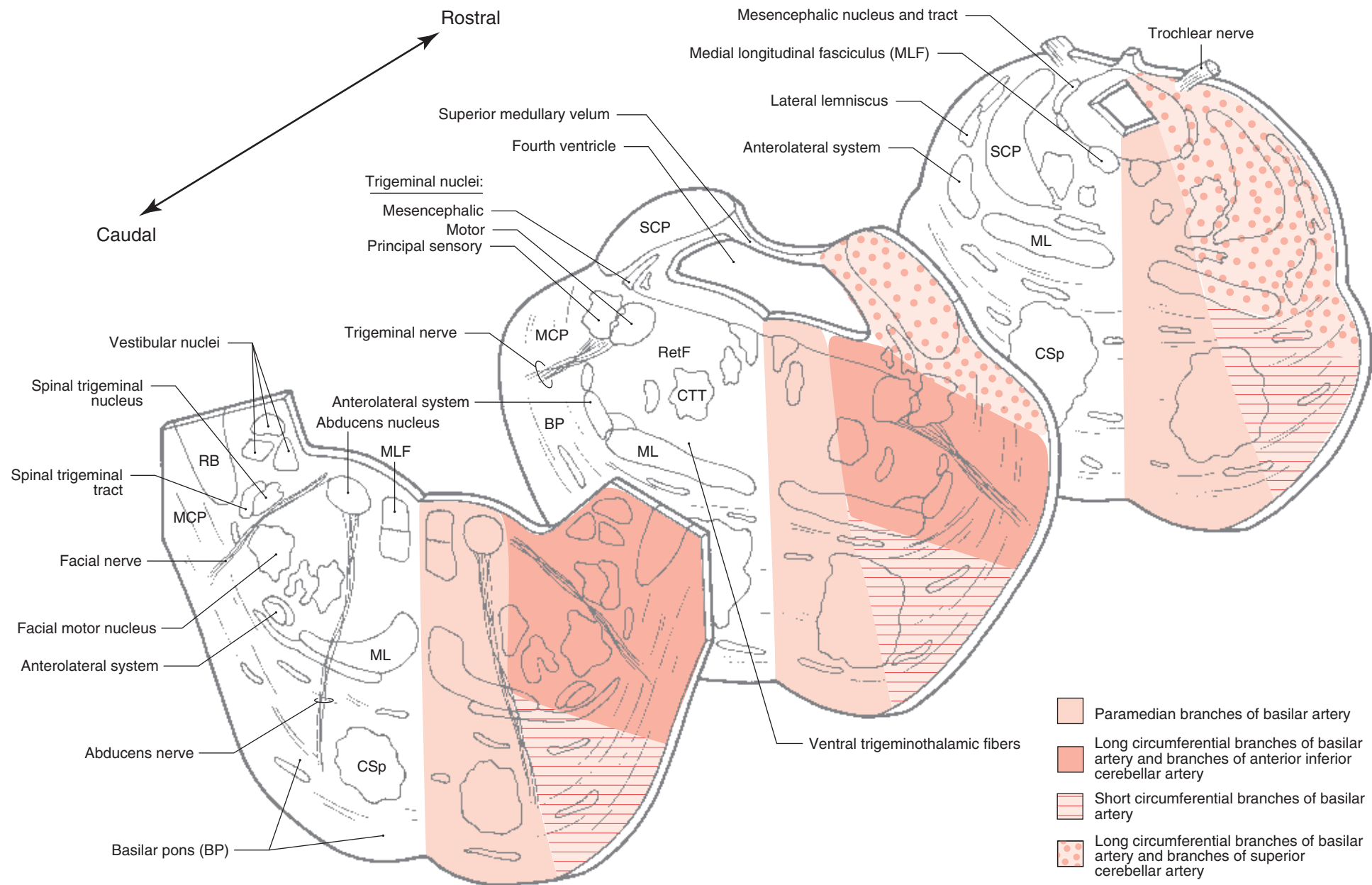
Deficit	Structure Damaged
• Ataxia, unsteady gait, fall toward side of lesion	• Middle and superior cerebellar peduncles (caudal and rostral levels)
• Vertigo, nausea, nystagmus, deafness, tinnitus, vomiting (at caudal levels)	• Vestibular and cochlear nerves and nuclei
• Ipsilateral paralysis of facial muscles	• Facial motor nucleus (caudal levels)
• Ipsilateral paralysis of masticatory muscles	• Trigeminal motor nucleus (midpontine levels)
• Ipsilateral Horner syndrome	• Descending hypothalamospinal fibers
• Ipsilateral loss of pain and thermal sense from face	• Spinal trigeminal tract and nucleus
• Contralateral loss of pain and thermal sense from body	• Anterolateral system
• Paralysis of conjugate horizontal gaze	• Paramedian pontine reticular formation (at mid to caudal levels)

Comment: The various combinations of these deficits may vary depending on whether the lesion is located in lateral pontine areas at caudal levels versus lateral pontine areas at rostral levels. As noted above lesions located in lateral portions of the pontine tegmentum may also extend medial at either caudal or rostral levels and give rise to some of the deficits discussed above in the section on medial pontine syndrome.

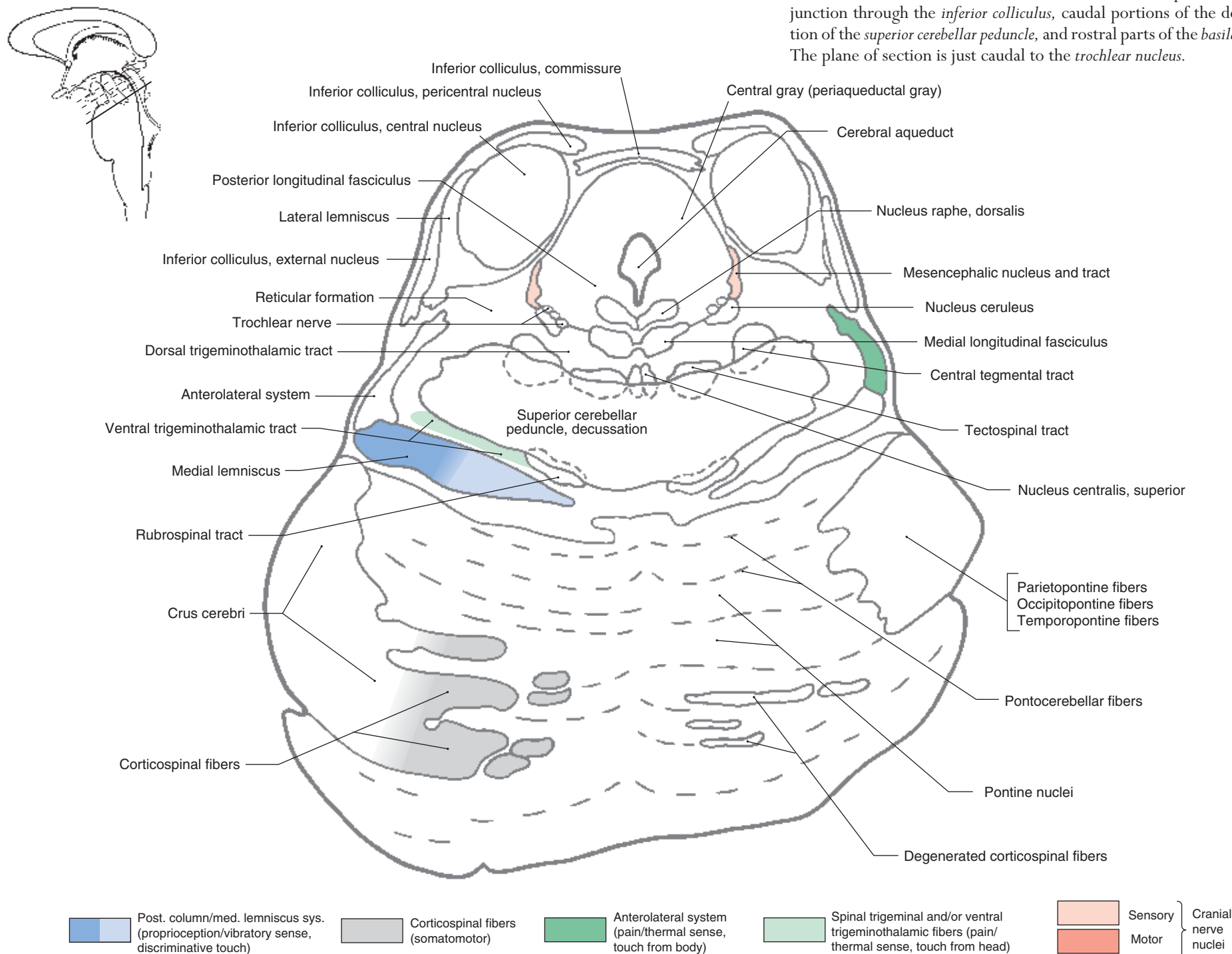
5-21 Semidiagrammatic representation of the internal distribution of arteries in the pons. Selected main structures are labeled on the left side of each section; the general pattern of arterial distribution overlies these structures on the right side. Some patients may have variations of the general distribution patterns of arteries to the pons as shown here. For example, the adjacent territories served by vessels may overlap to differing degrees at their margins or the territory of a particular vessel may be smaller or larger than seen in the general pattern.

Abbreviations

BP	Basilar pons
CSp	Corticospinal fibers
CTT	Central tegmental tract
MCP	Middle cerebellar peduncle (brachium pontis)
ML	Medial lemniscus
MLF	Medial longitudinal fasciculus
RB	Restiform body (+ juxtarestiform body = inferior cerebellar)
RetF	Reticular formation
SCP	Superior cerebellar peduncle (brachium conjunctivum)

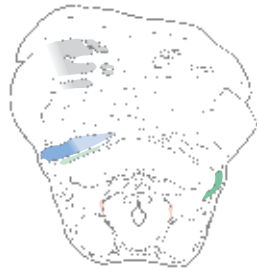


5-22 Transverse section of the brainstem at the pons–midbrain junction through the *inferior colliculus*, caudal portions of the decussation of the *superior cerebellar peduncle*, and rostral parts of the *basilar pons*. The plane of section is just caudal to the *trochlear nucleus*.





Anatomical orientation



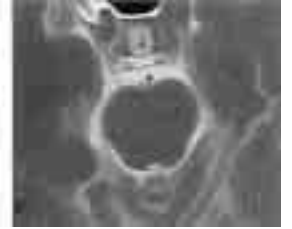
Clinical orientation



MRI, T1-weighted image



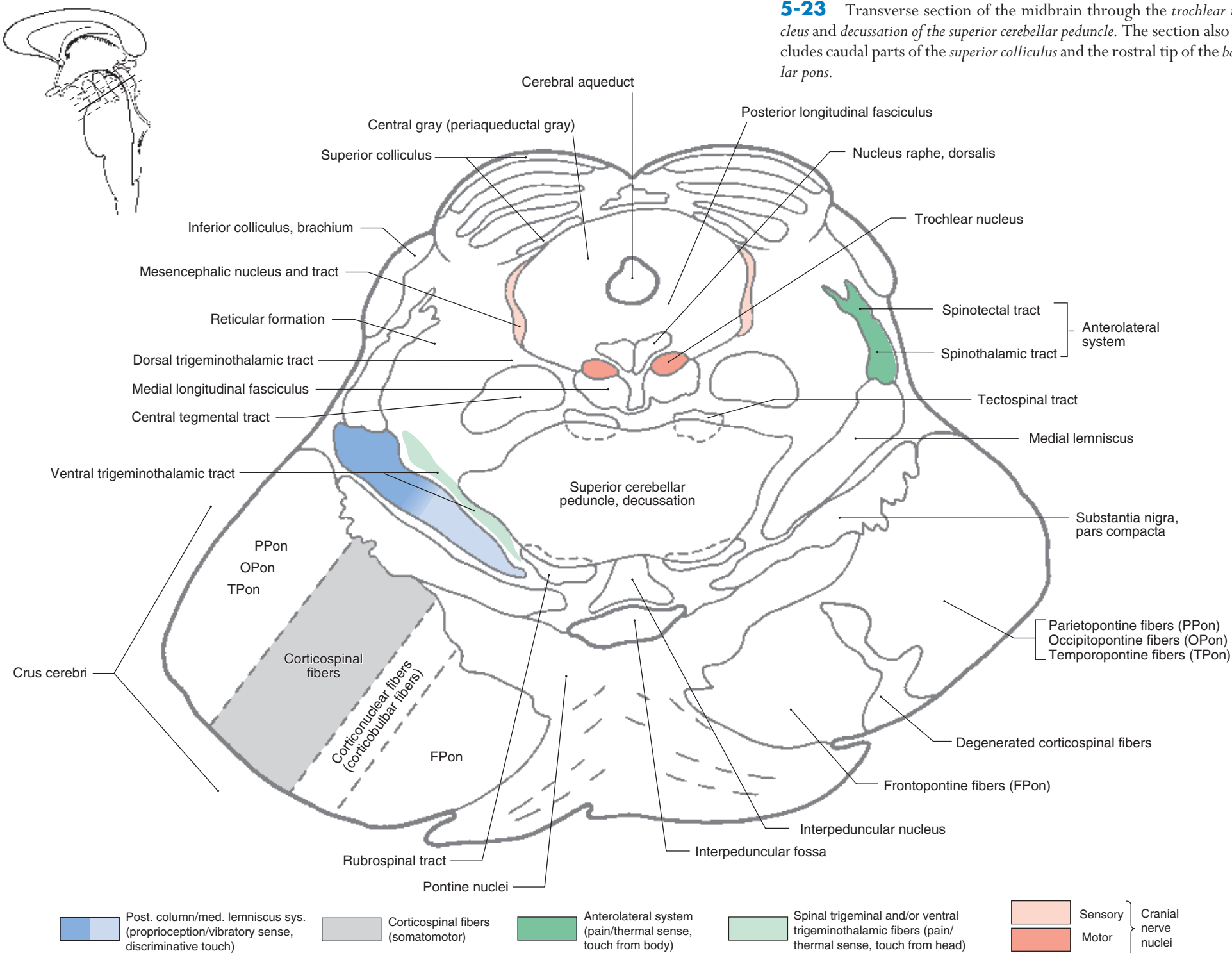
MRI, T2-weighted image



CT cisternogram

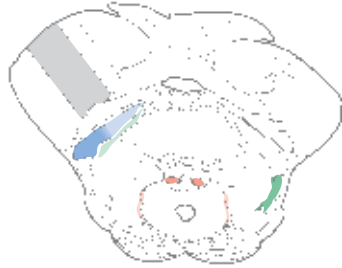


5-23 Transverse section of the midbrain through the *trochlear nucleus* and *decussation of the superior cerebellar peduncle*. The section also includes caudal parts of the *superior colliculus* and the rostral tip of the *basilar pons*.





Anatomical orientation



Clinical orientation



MRI, T1-weighted image



MRI, T2-weighted image

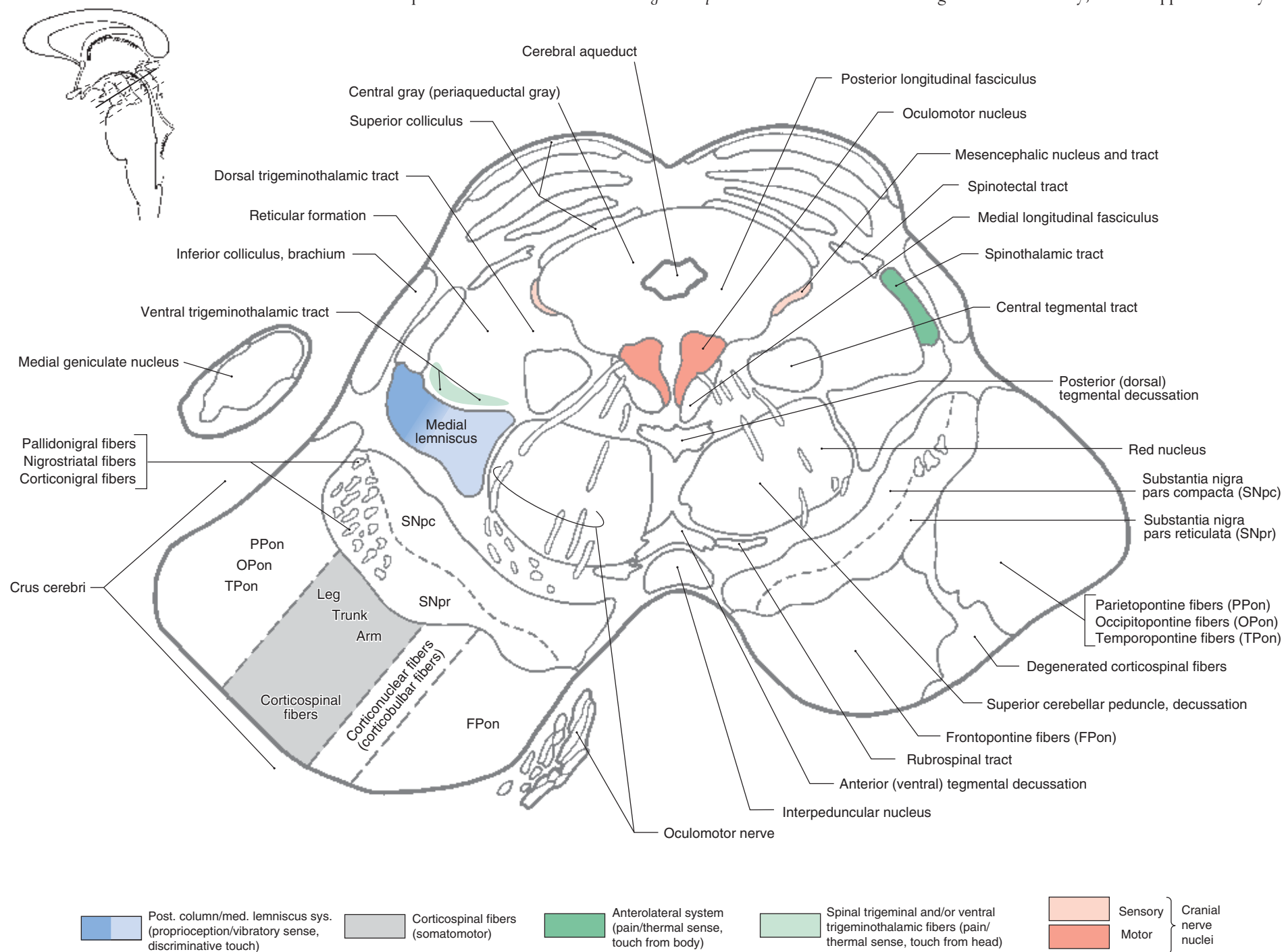


CT cisternogram



5-24 Transverse section of the midbrain through the *superior colliculus*, caudal parts of the *oculomotor nucleus*, and caudal parts of the *red nucleus*. The plane of section is caudal to the *Edinger-Westphal nucleus* but

includes rostral portions of the *decussation of the superior cerebellar peduncle*, which, at this level, are intermingled with the caudal part of the *red nucleus*. Leg = lower extremity; Arm = upper extremity.





Anatomical orientation



Clinical orientation



MRI, T1-weighted image



MRI, T2-weighted image

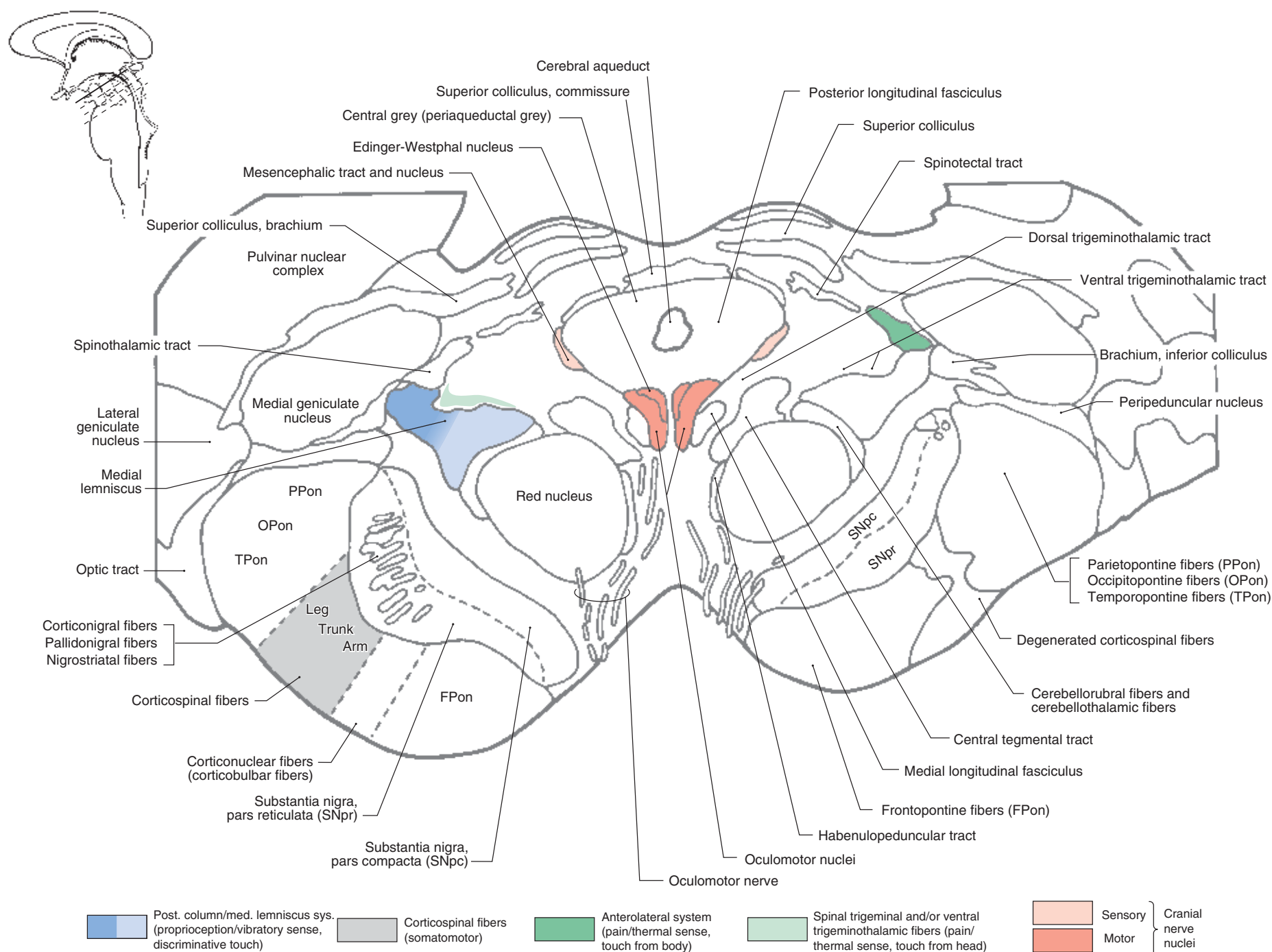


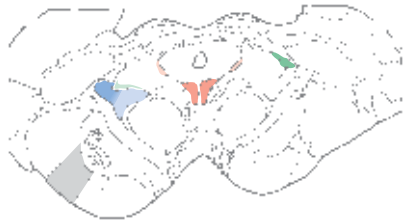
CT cisternogram



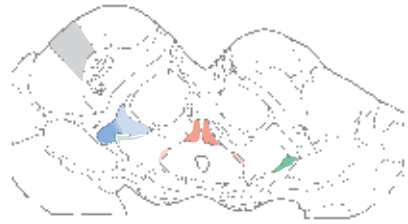
5-25 Transverse section of the midbrain through the *superior colliculus*, rostral portions of the *oculomotor nucleus*, including the *Etinger-Westphal nucleus*, and the exiting fibers of the *oculomotor nerve*. The

plane of this section is also through caudal portions of the diencephalon including the *pulvinar nuclear complex* and the *medial* and *lateral geniculate nuclei*. Leg = lower extremity; Arm = upper extremity.





Anatomical orientation



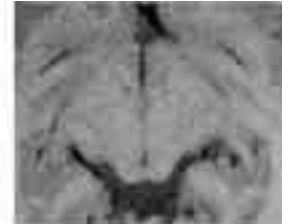
Clinical orientation



MRI, T1-weighted image



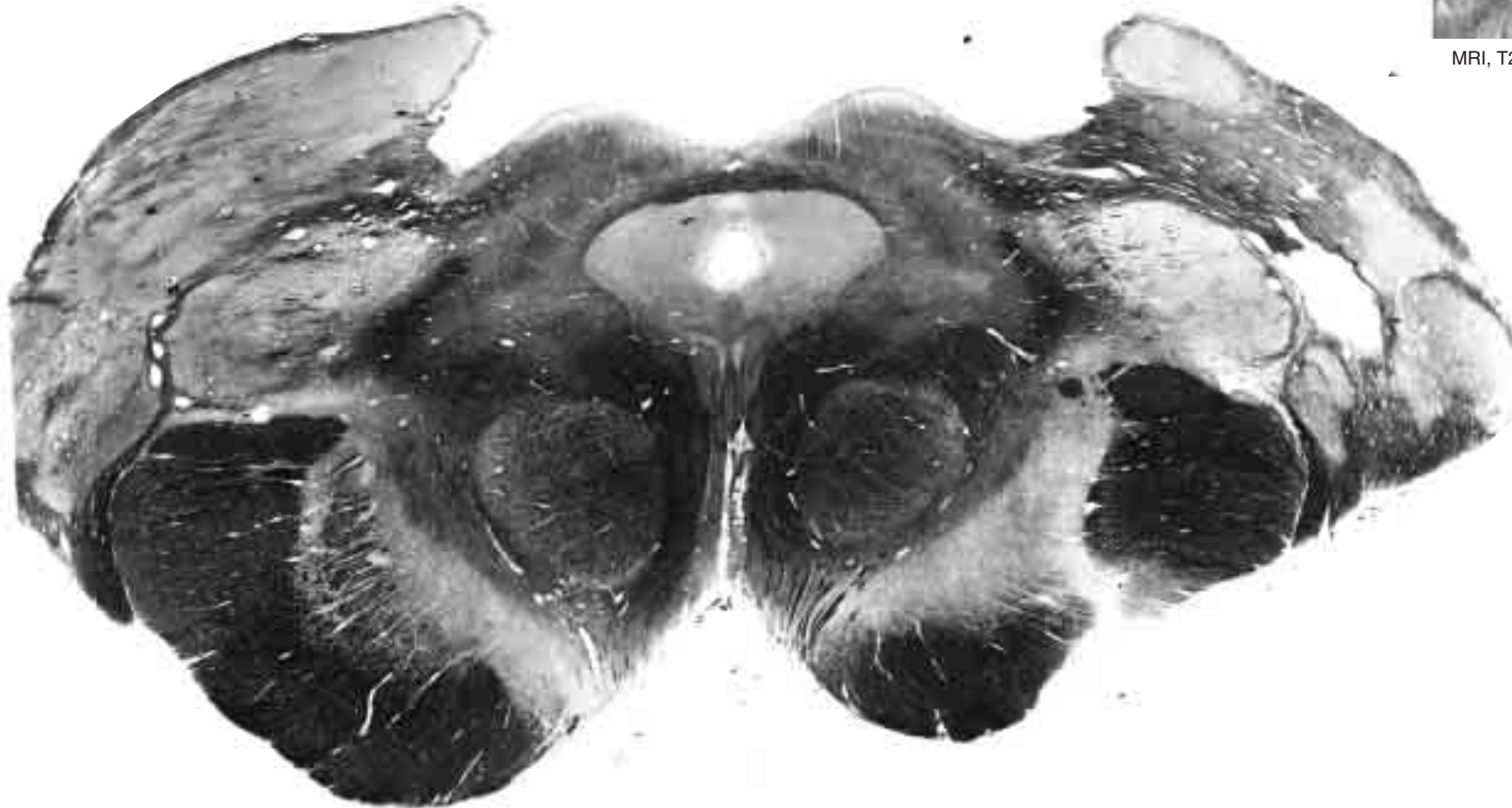
MRI, T2-weighted image



MRI, T1-weighted image

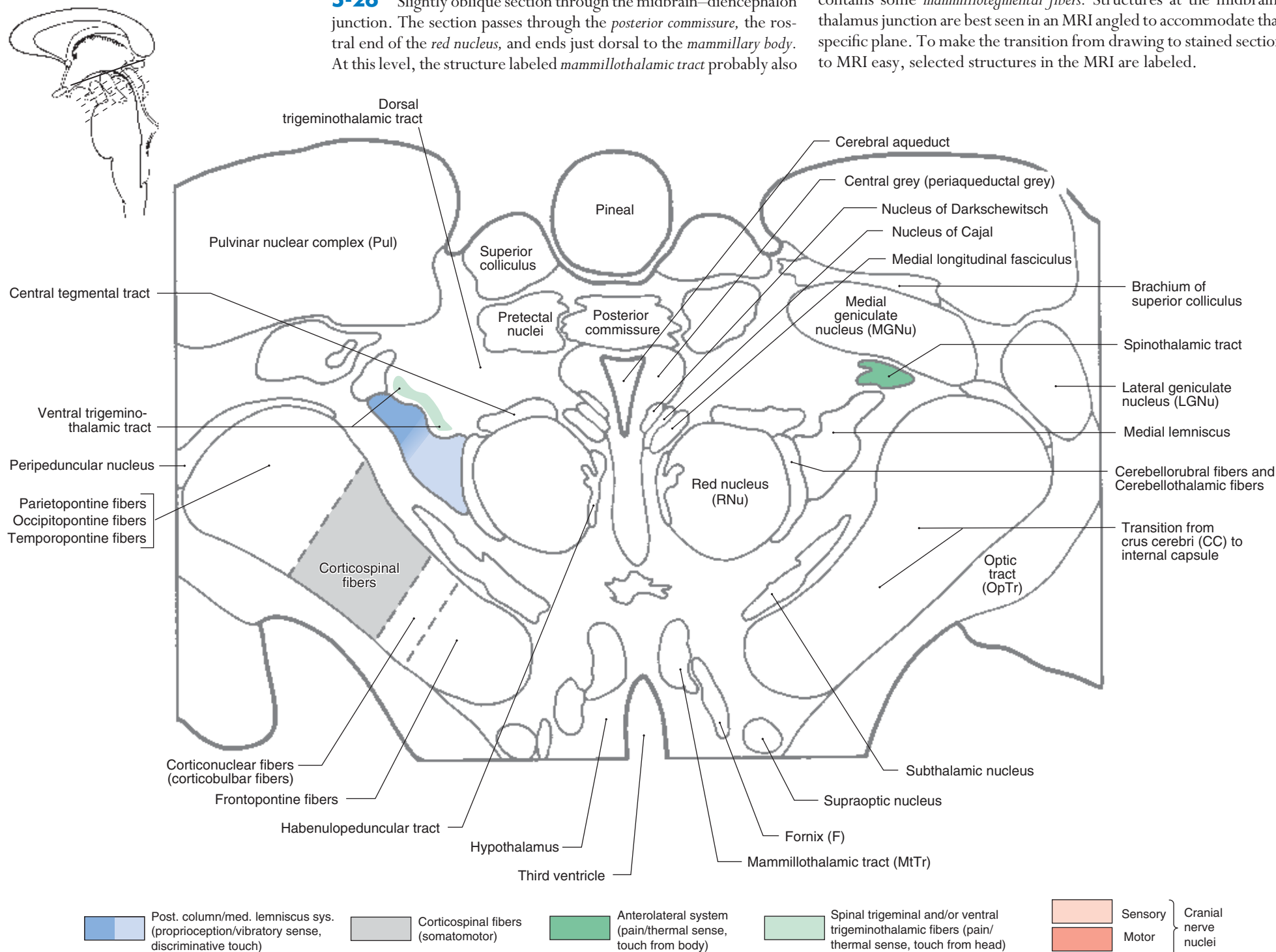


MRI, T2-weighted image



5-26 Slightly oblique section through the midbrain–diencephalon junction. The section passes through the *posterior commissure*, the rostral end of the *red nucleus*, and ends just dorsal to the *mammillary body*. At this level, the structure labeled *mammillothalamic tract* probably also

contains some *mammillotegmental fibers*. Structures at the midbrain–thalamus junction are best seen in an MRI angled to accommodate that specific plane. To make the transition from drawing to stained section to MRI easy, selected structures in the MRI are labeled.

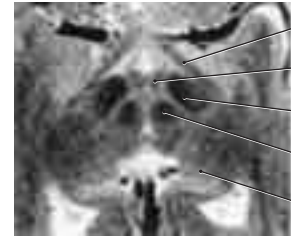




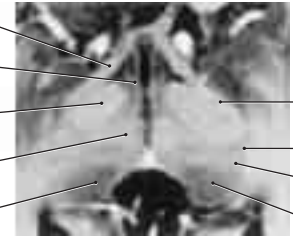
Anatomical orientation



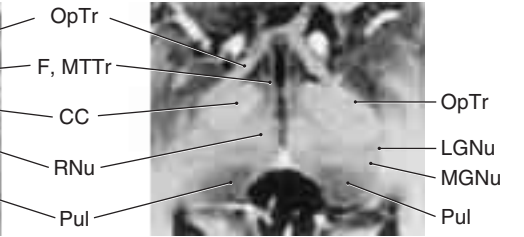
Clinical orientation



MRI, T2-weighted image



MRI, inversion recovery



Vascular Syndromes or Lesions of the Midbrain

Medial Midbrain (Weber) Syndrome: May result from occlusion of paramedian branches of P_1 segment of posterior cerebral artery.

Deficit	Structure Damaged
• Contralateral hemiplegia of arm and leg	• Corticospinal fibers in crus cerebri
• Ipsilateral paralysis of eye movement: eye oriented down and out and pupil dilated and fixed	• Oculomotor nerve

Comment: This combination of motor deficits at this level of the brainstem is called a *superior alternating hemiplegia*. This pattern consists of *ipsilateral paralysis of eye movement* and *contralateral hemiplegia* of the upper and lower extremities. Damage to the corticonuclear (corticobulbar) fibers in the crus cerebri may result in a partial deficit in tongue and facial movement on the contralateral side. These cranial nerve deficits are seen as a *deviation of the tongue* to the side opposite the lesion on attempted protrusion and a *paralysis of the lower half of the facial muscles* on the contralateral side. Although parts of the substantia nigra are frequently involved, *akinesia* or *dyskinesia* are not frequently seen.

Central Midbrain Lesion (Claude syndrome)

Deficit	Structure Damaged
• Ipsilateral paralysis of eye movement: eye oriented down and out and pupil dilated and fixed	• Oculomotor nerve
• Contralateral ataxia and tremor of cerebellar origin	• Red nucleus and cerebellothalamic fibers

Comment: The lesion in this syndrome may extend laterally into the medial lemniscus and the dorsally adjacent ventral trigeminothalamic fibers. If this was the case, there could conceivably be a loss or diminution of

position and vibratory sense and of discriminative touch from the contralateral arm and partial loss of pain and thermal sensation from the contralateral face.

Benedikt syndrome: This results from a larger lesion of the midbrain that essentially involves both of the separate areas of Weber and Claude. The main deficits are contralateral hemiplegia of arm and leg (corticospinal fibers), ipsilateral paralysis of eye movement with dilated pupil (oculomotor nerve), and cerebellar tremor and ataxia (red nucleus and cerebellothalamic fibers). Slight variations may be present based on the extent of the lesion.

Parinaud syndrome: This syndrome is usually caused by a tumor in the pineal region, such as germinoma, astrocytoma, pineocytoma/pineoblastoma, or any of a variety of other tumors that impinge on the superior colliculi. The potential for occlusion at the cerebral aqueduct in these cases also indicates that hydrocephalus may be a component of this syndrome. The deficits in these patients consist of a *paralysis of upward gaze* (superior colliculi), *hydrocephalus* (occlusion of the cerebral aqueduct), and eventually a *failure of eye movement* due to pressure on the oculomotor and trochlear nuclei. These patients may also exhibit *nystagmus* due to involvement of the medial longitudinal fasciculus.

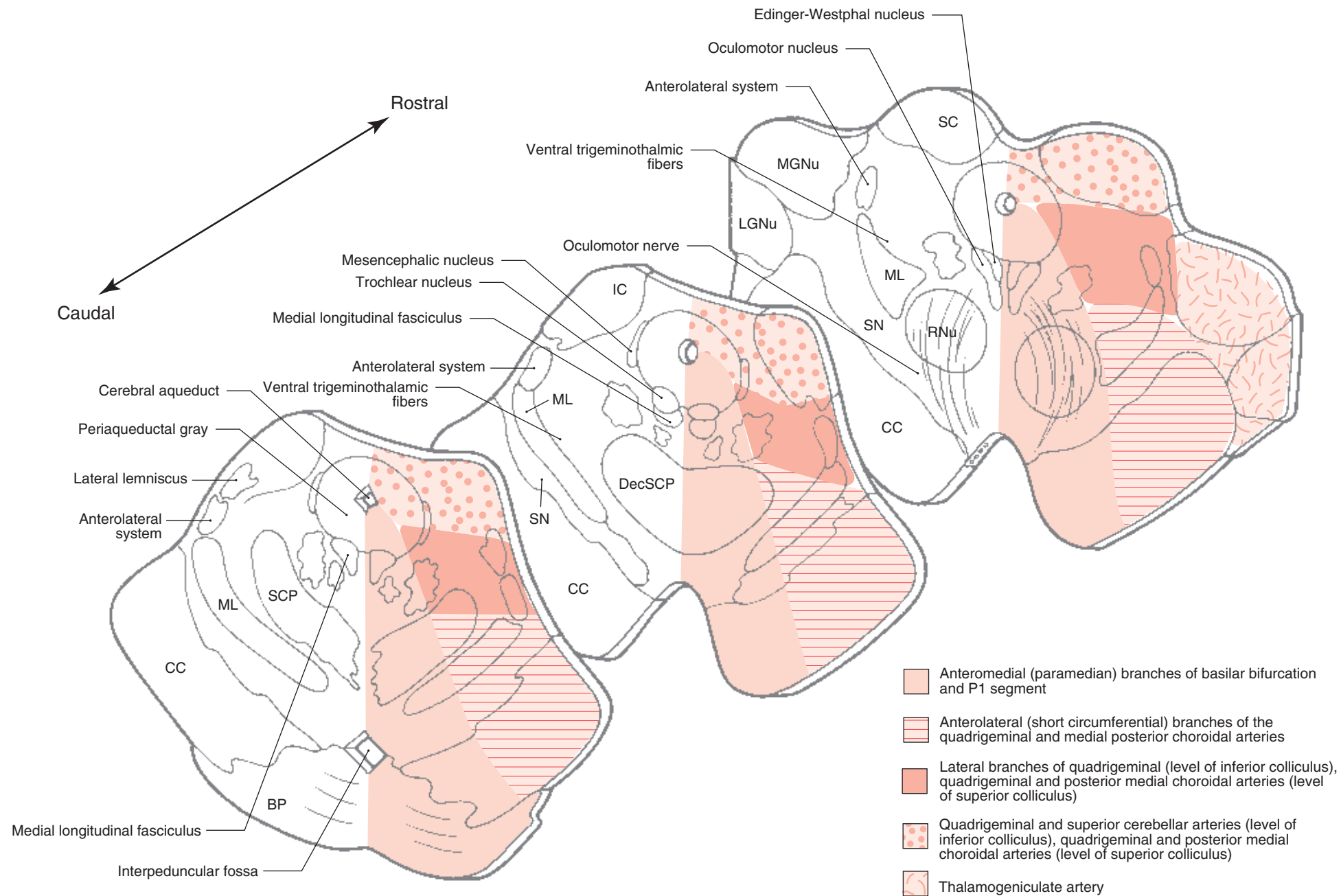
Uncal Herniation: Herniation of the uncus occurs in response to large and rapidly expanding lesions in the cerebral hemisphere, this being a supratentorial location. *Uncal herniation* is an extrusion of the uncus through the tentorial notch (tentorial incisura) with resultant pressure on the oculomotor nerve and the crus cerebri of the midbrain. Initially the pupils, unilaterally or bilaterally, may dilate or respond slowly to light, followed by weakness of oculomotor movement. As herniation progresses the pupils will be fully dilated, eye movements regulated by the oculomotor nerve may be slow or absent, and the eyes will deviate slightly laterally due to the unopposed actions of the abducens nerves. There is usually weakness on the contralateral side of the body due to compression of corticospinal fibers in the crus cerebri. However, if pressure is sufficient the entire midbrain may shift so that there can be contralateral as well as ipsilateral weakness due to pressure on the same side and pressure on the opposite side of the crus cerebri. This hemiplegia ipsilateral to the

herniation and ipsilateral to the oculomotor deficits is called the *Kernohan phenomenon*. As damage from the pressure on the midbrain extends down and into the upper pons the pupils are dilated and fixed, eye movement is largely absent, respiration is decreased, and the patient will become decerebrate (upper and lower extremities extended, toes pointed inward, fingers flexed, forearm pronated, head and neck extended).

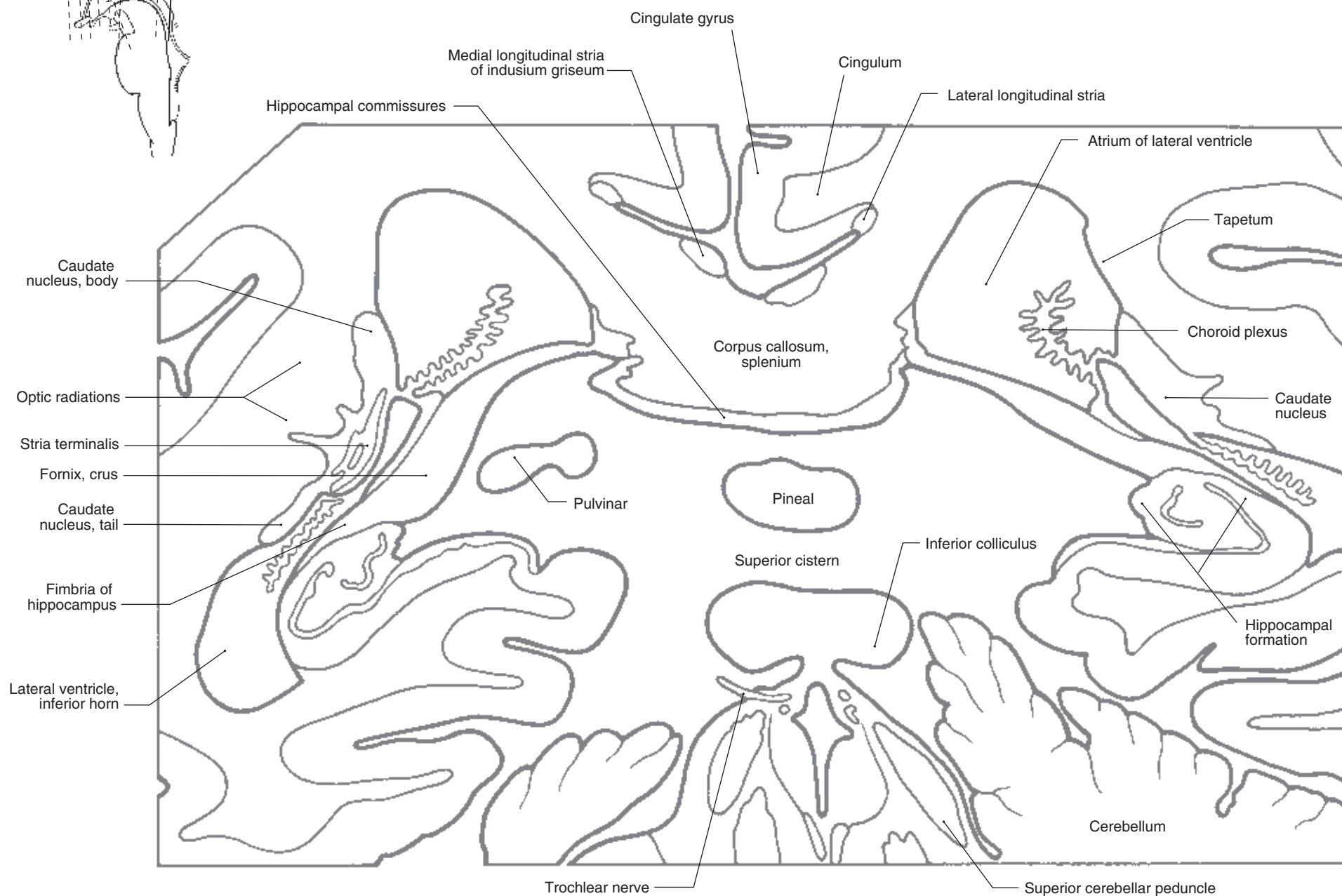
5-27 Semidiagrammatic representation of the internal distribution of arteries in the midbrain. Selected main structures are labeled on the left side of each section; the typical pattern of arterial distribution overlies these structures on the right side. The general distribution patterns of the vessels to the midbrain as shown here may vary somewhat from patient to patient. For example, the adjacent territories served by neighboring vessels may overlap to differing degrees at their margins or the territory of a particular vessel may be larger or smaller than seen in the general pattern.

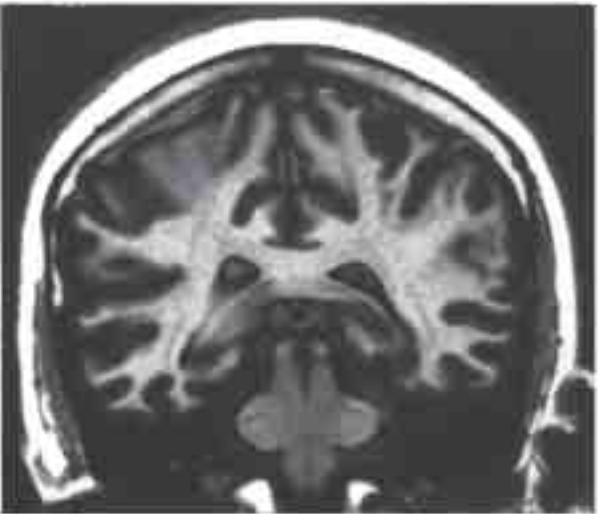
Abbreviations

BP	Basilar pons
CC	Crus cerebri
DecSCP	Decussation of the superior cerebellar peduncle
IC	Inferior colliculus
LGNu	Lateral geniculate nucleus
MGNu	Medial geniculate nucleus
ML	Medial lemniscus
RNu	Red nucleus
SC	Superior colliculus
SCP	Superior cerebellar peduncle
SN	Substantia nigra



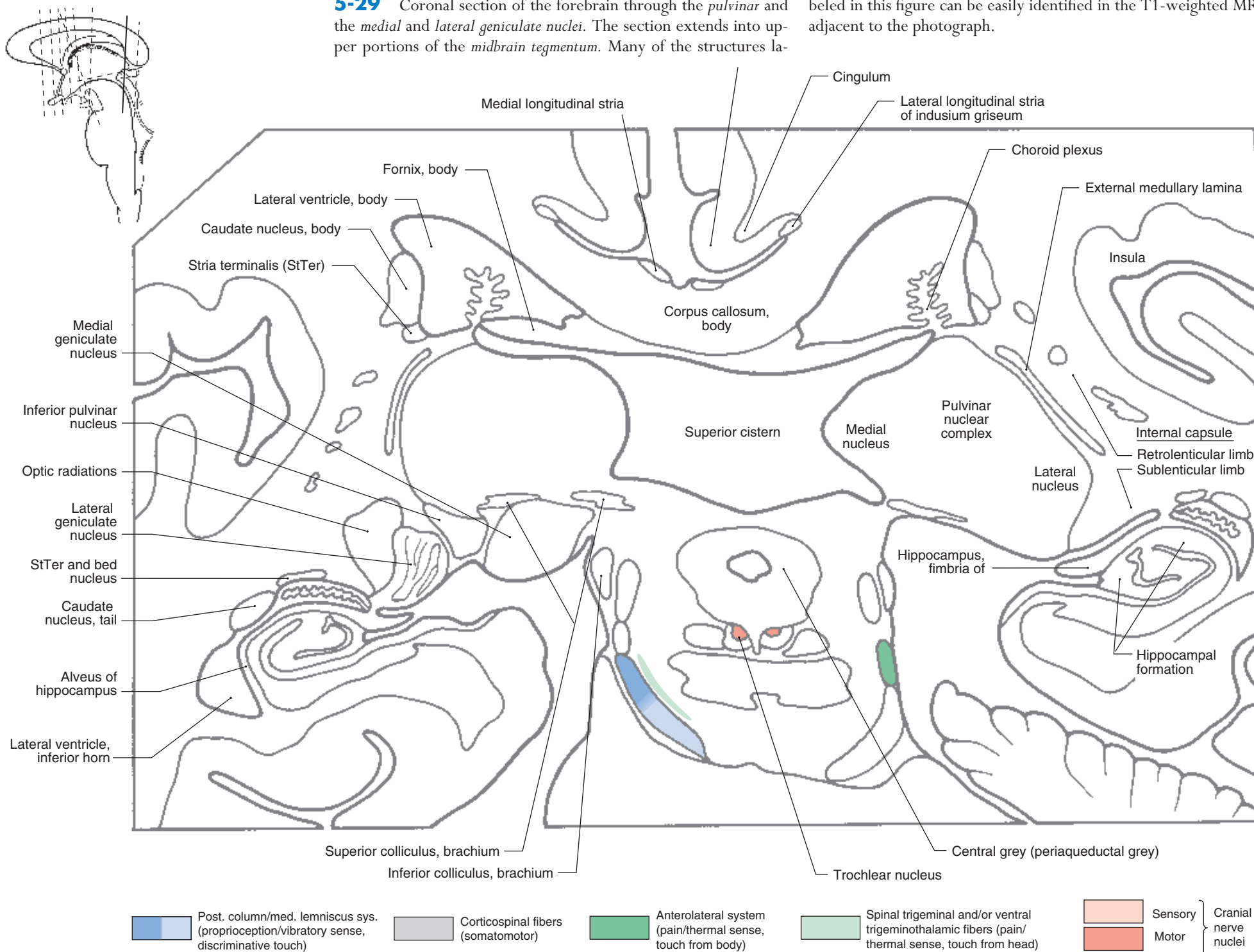
5-28 Coronal section of forebrain through the *splenium of the corpus callosum* and *crus of fornix*, and extending into the *inferior colliculus* and exit of the *trochlear nerve*. Many of the structures labeled in this figure can be easily identified in the T1-weighted MRI adjacent to the photograph.

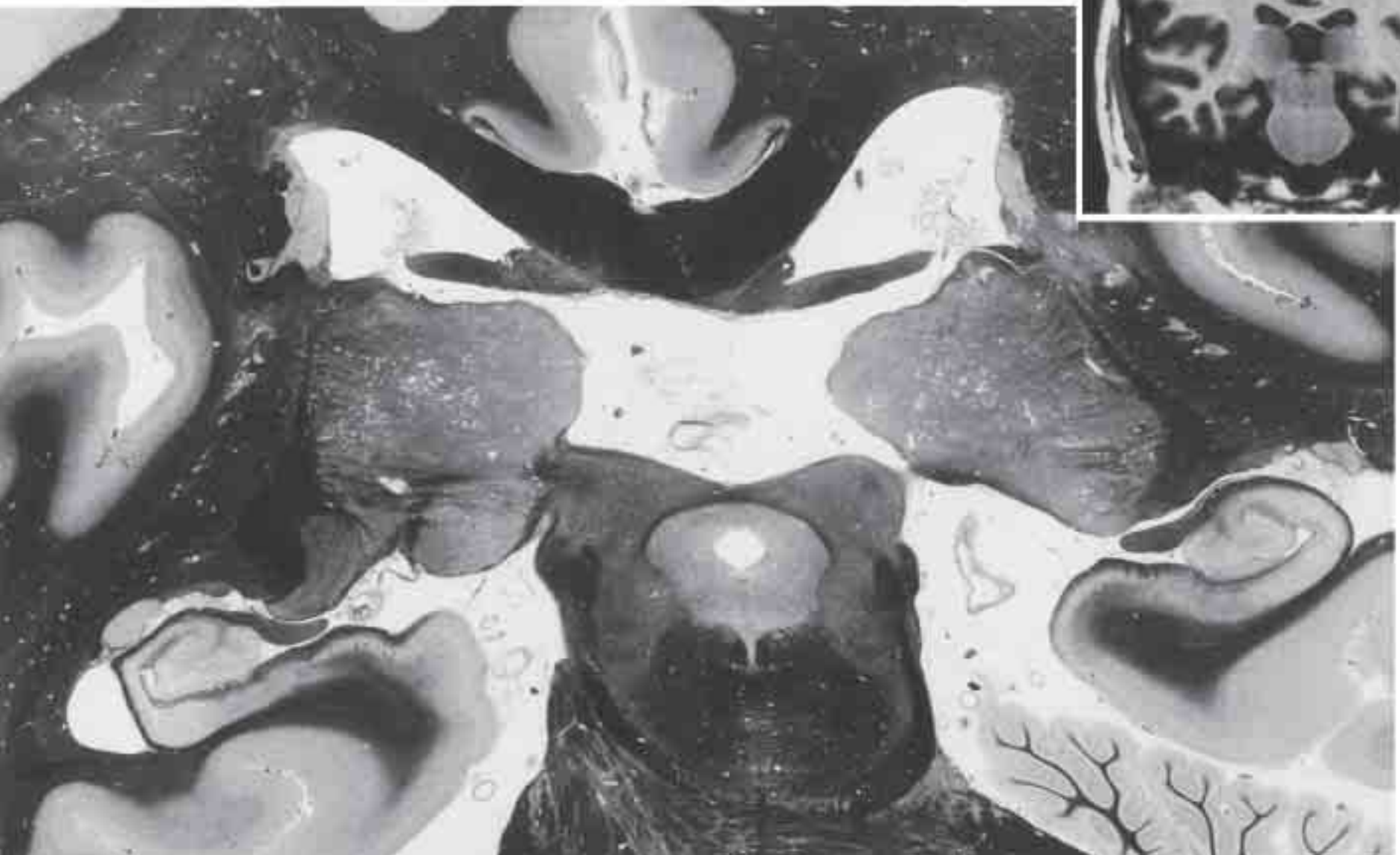
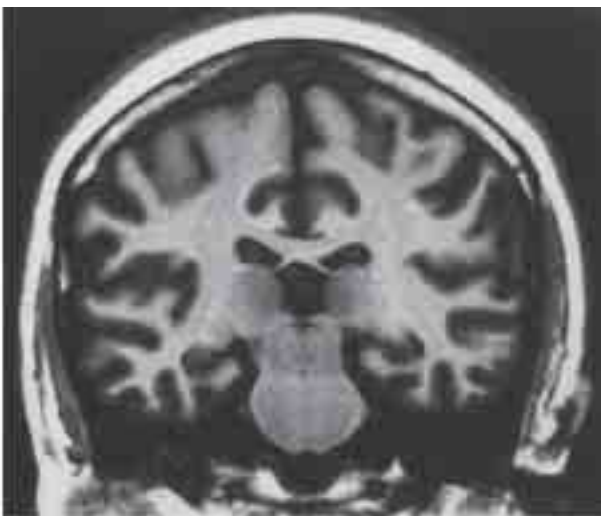




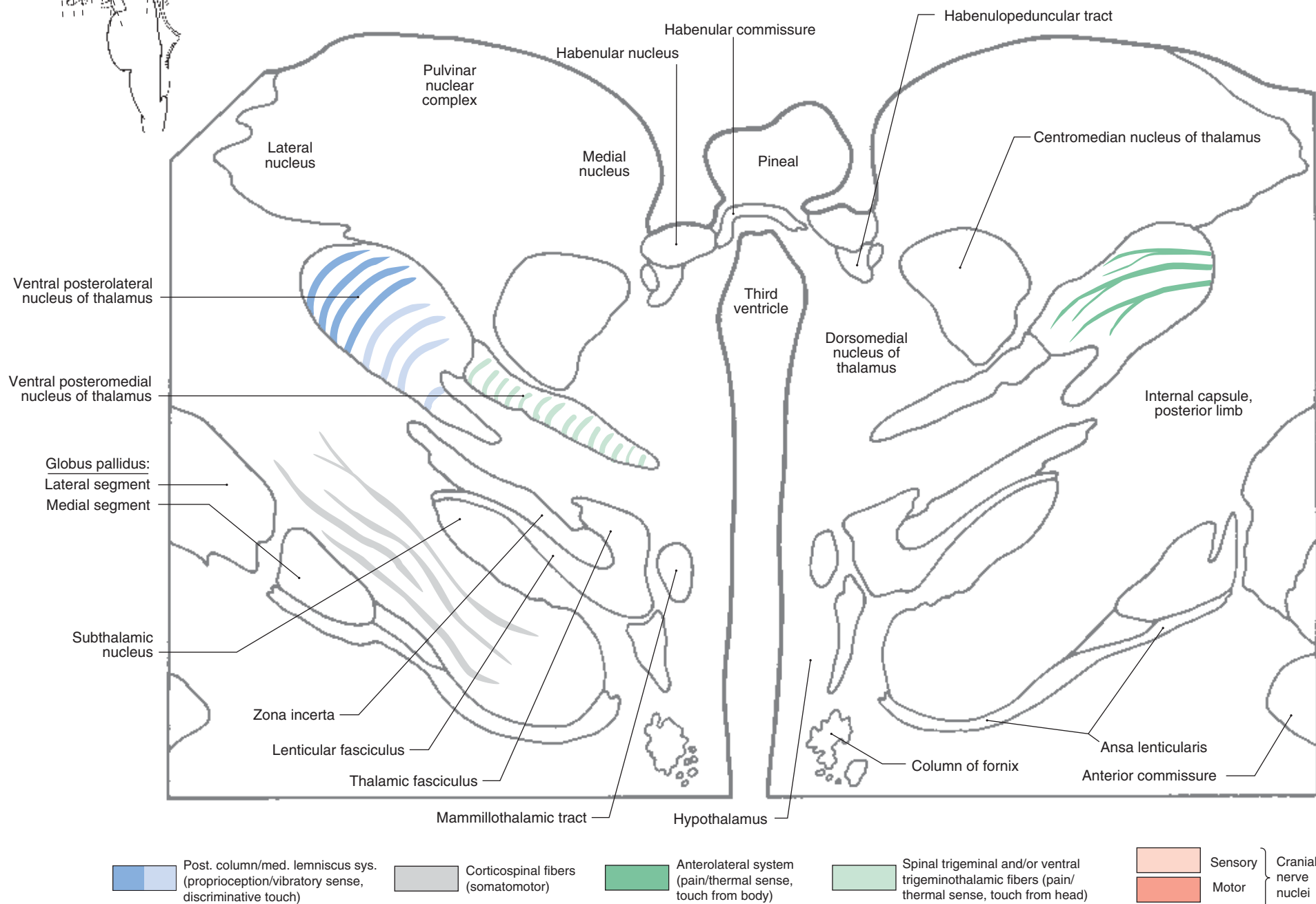
5-29 Coronal section of the forebrain through the *pulvinar* and the *medial* and *lateral geniculate nuclei*. The section extends into upper portions of the *midbrain tegmentum*. Many of the structures la-

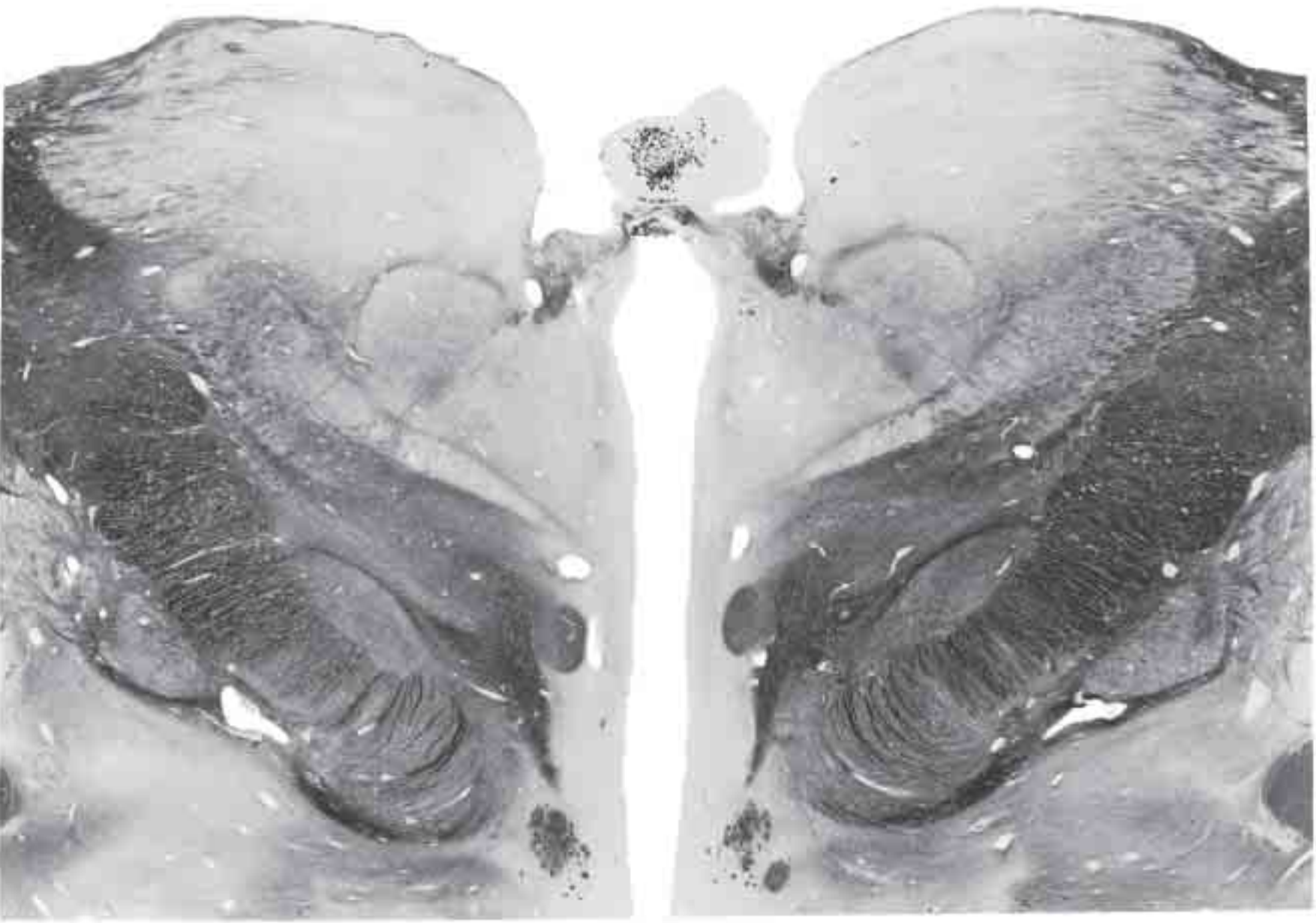
beled in this figure can be easily identified in the T1-weighted MRI adjacent to the photograph.





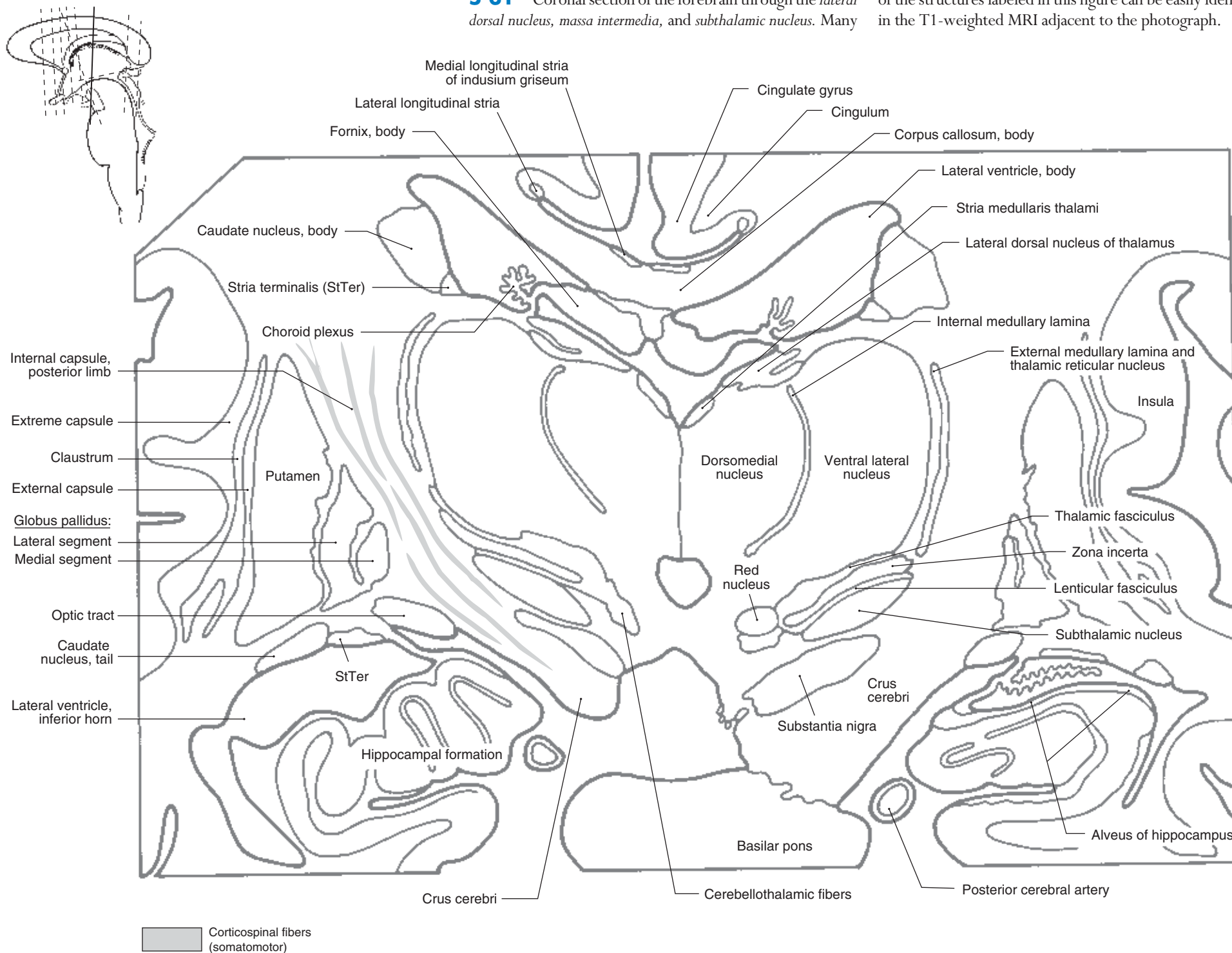
5-30 Slightly oblique section of the forebrain through the *pulvinar*, *ventral posteromedial*, and *ventral posterolateral nuclei*. The section extends rostrally through the *subthalamic nucleus* and ends in the *caudal hypothalamus* just dorsal to the *mammillary bodies* as seen by the position of the (postcommissural) fornix.

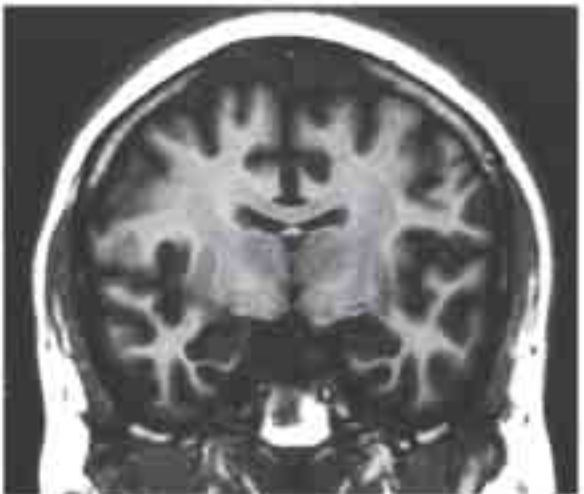
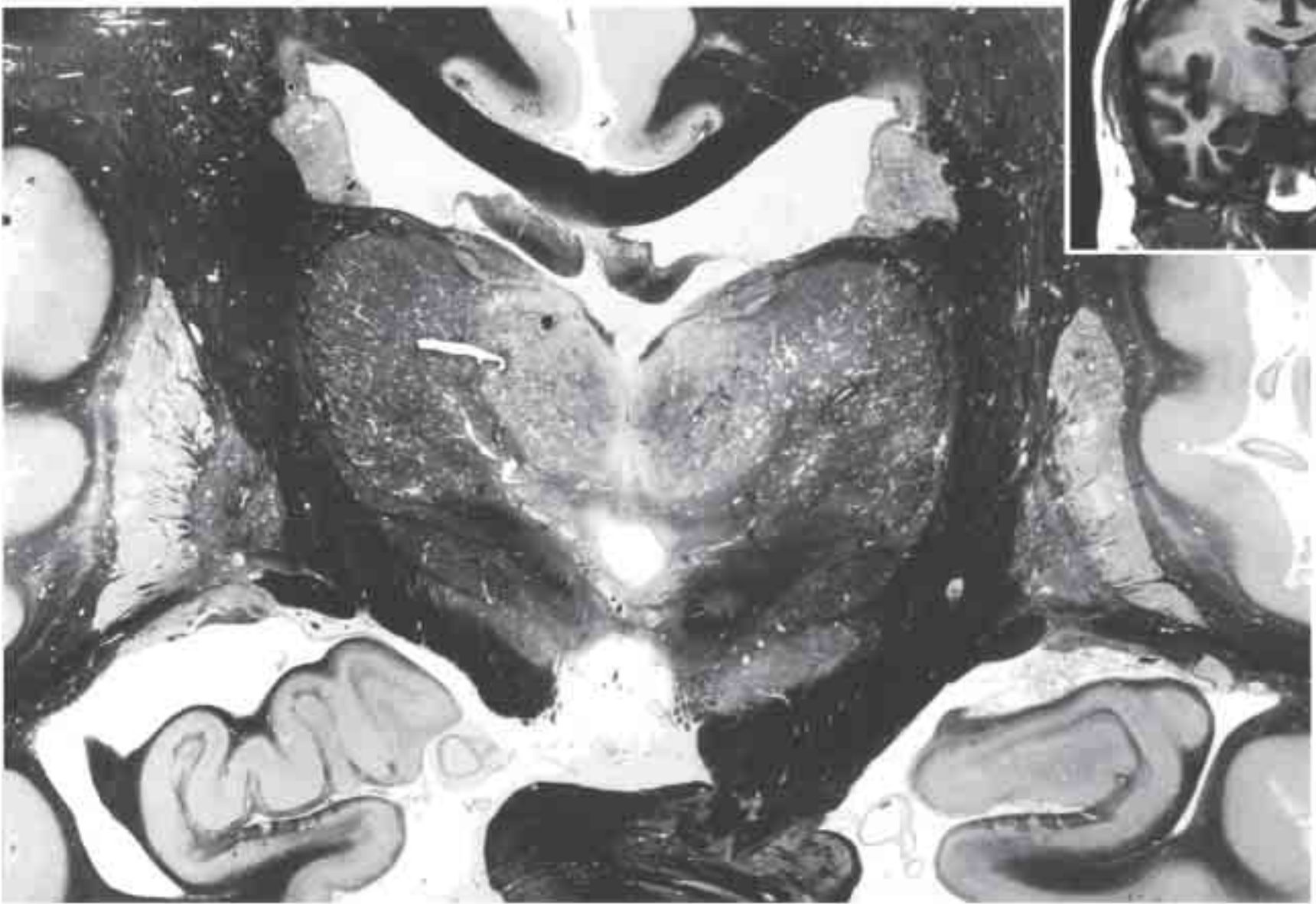




5-31 Coronal section of the forebrain through the *lateral dorsal nucleus, massa intermedia, and subthalamic nucleus*. Many

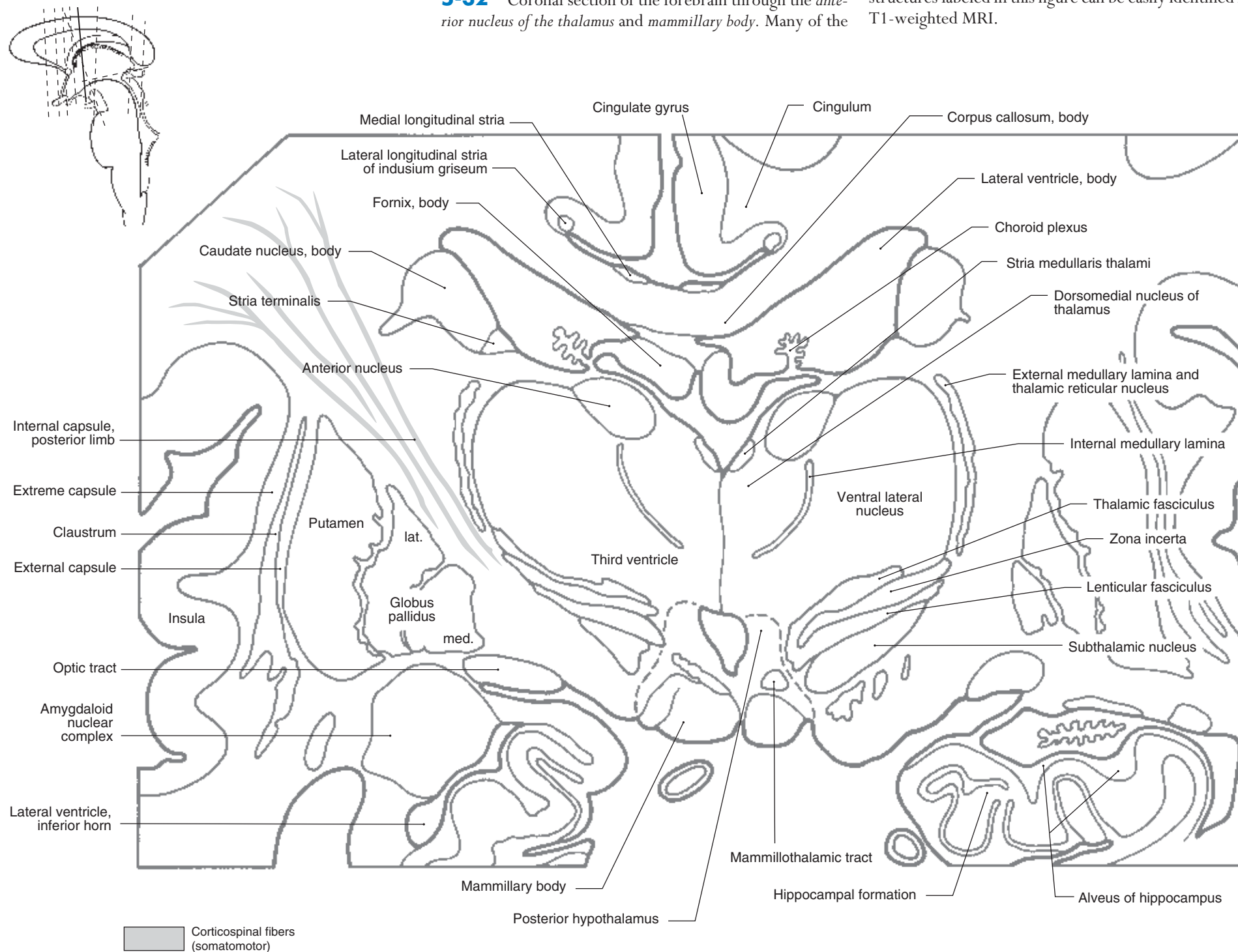
of the structures labeled in this figure can be easily identified in the T1-weighted MRI adjacent to the photograph.

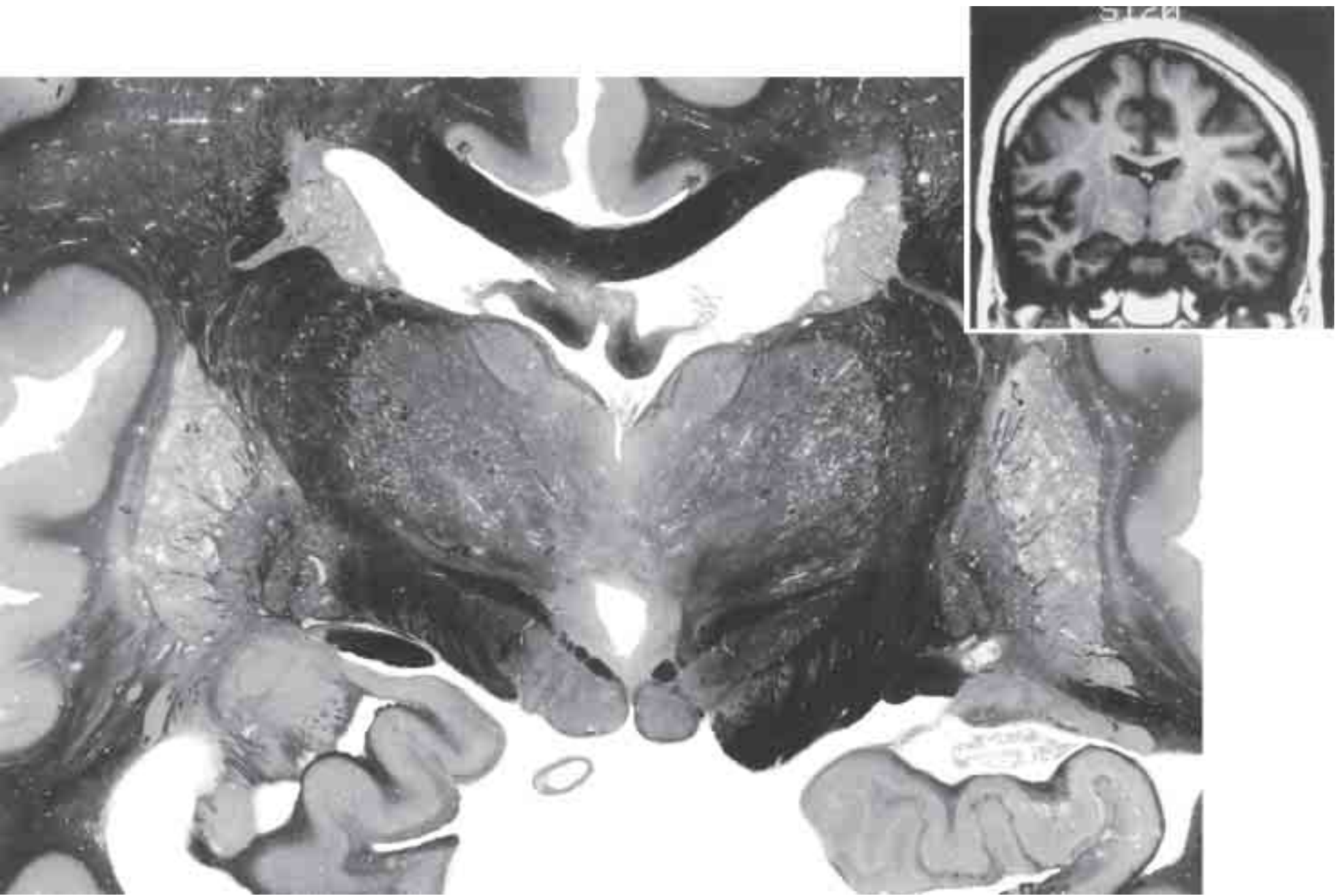




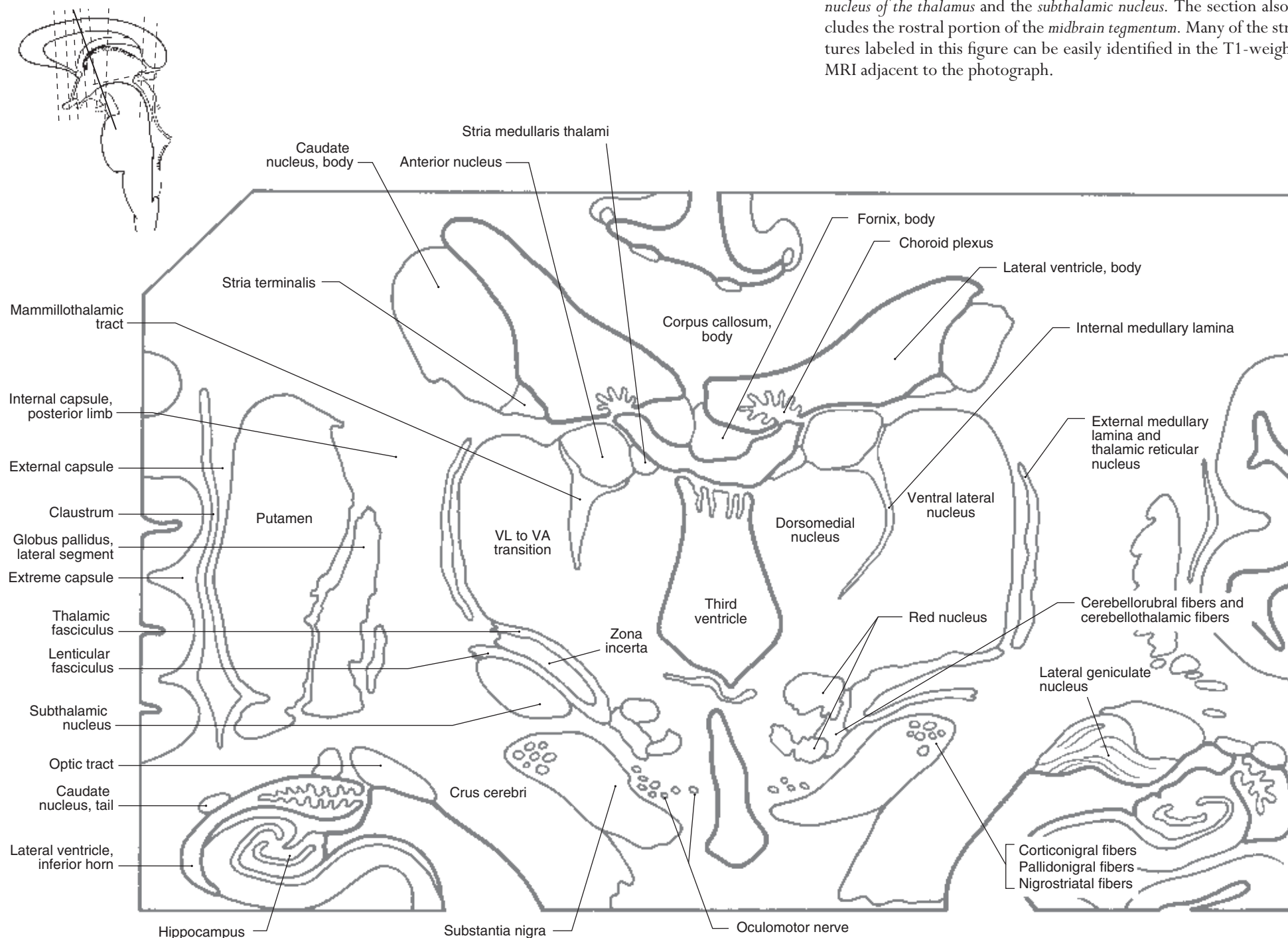
5-32 Coronal section of the forebrain through the *anterior nucleus of the thalamus* and *mammillary body*. Many of the

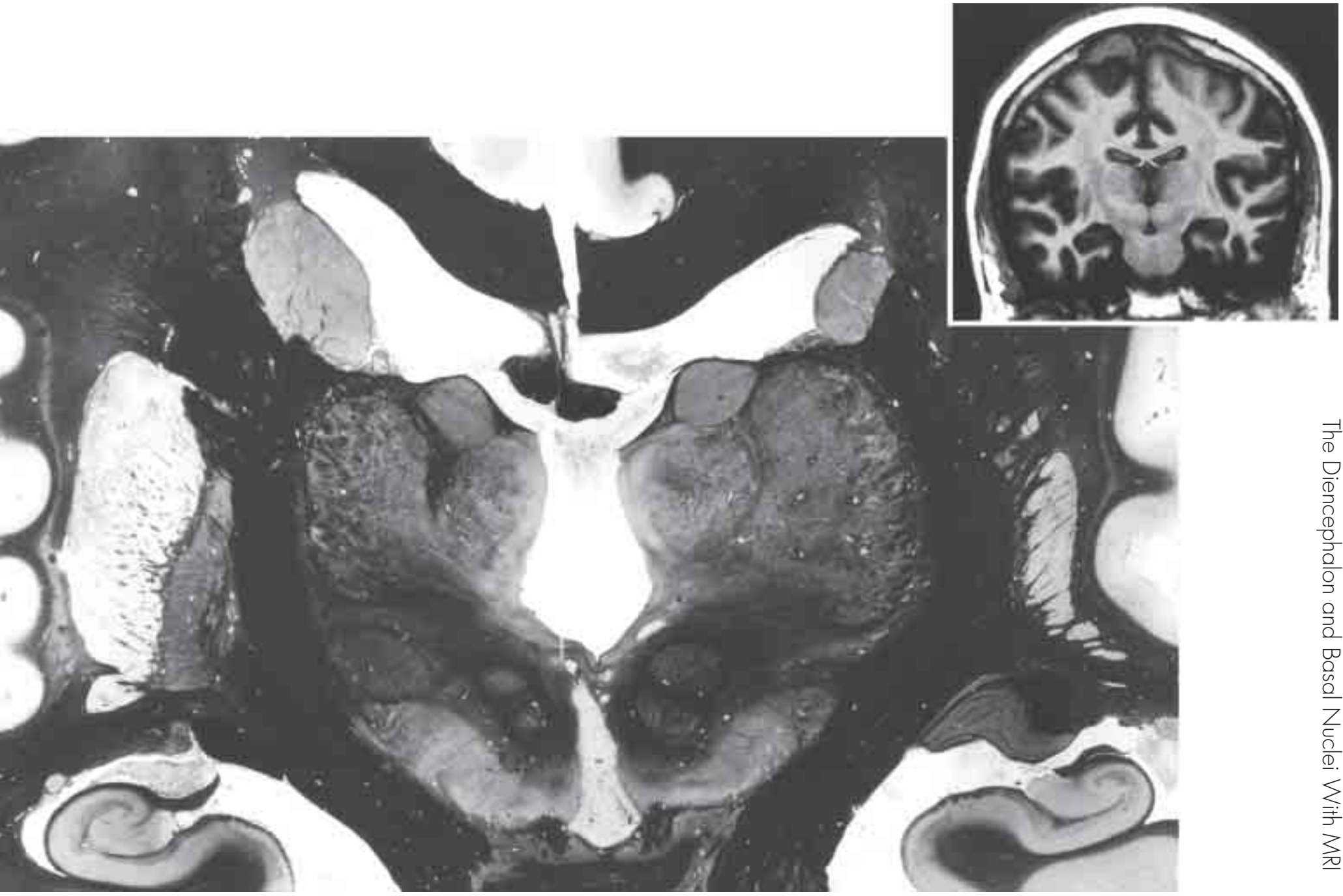
structures labeled in this figure can be easily identified in the T1-weighted MRI.





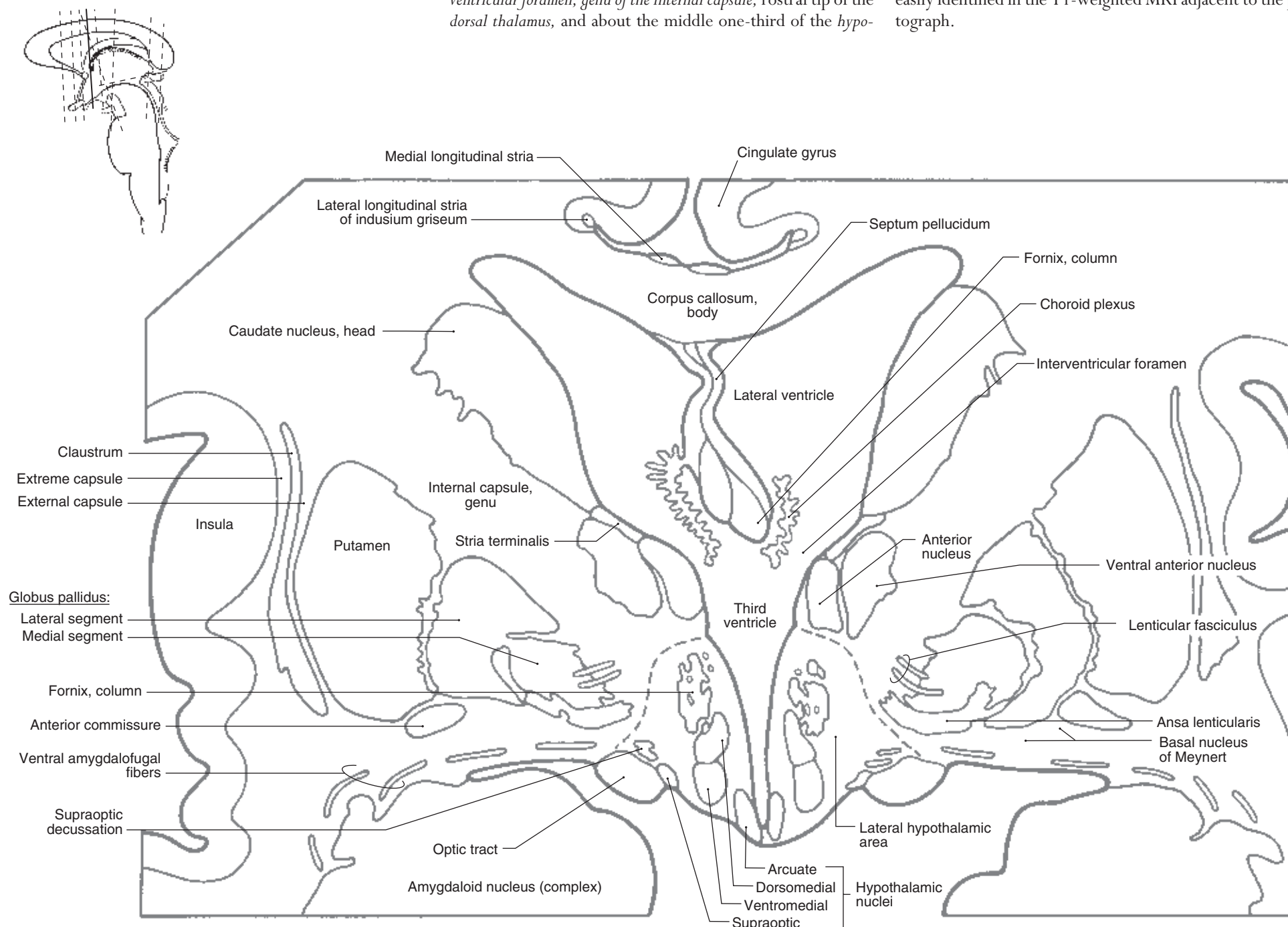
5-33 Slightly oblique section of the forebrain through the *anterior nucleus of the thalamus* and the *subthalamic nucleus*. The section also includes the rostral portion of the *midbrain tegmentum*. Many of the structures labeled in this figure can be easily identified in the T1-weighted MRI adjacent to the photograph.

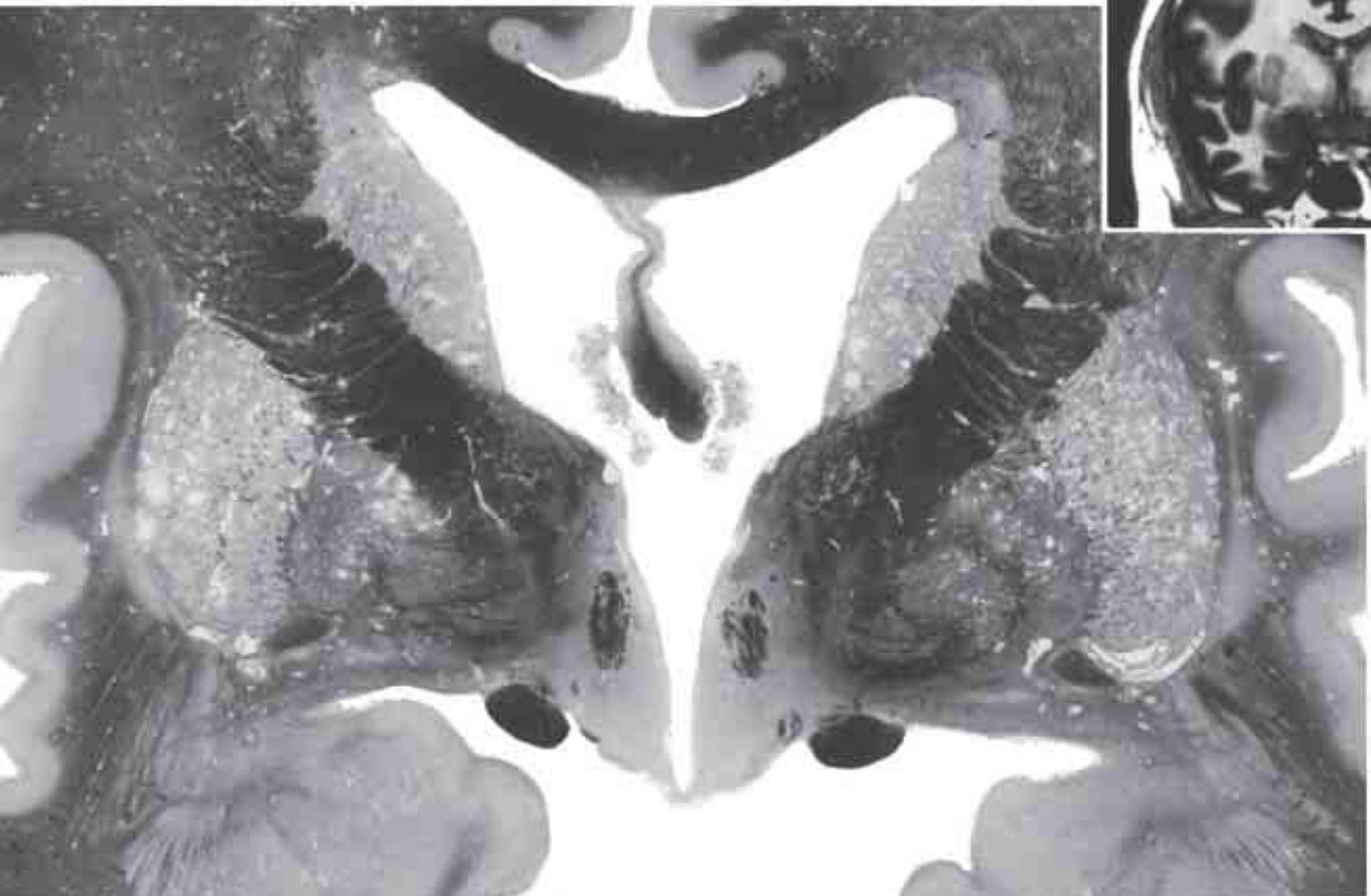
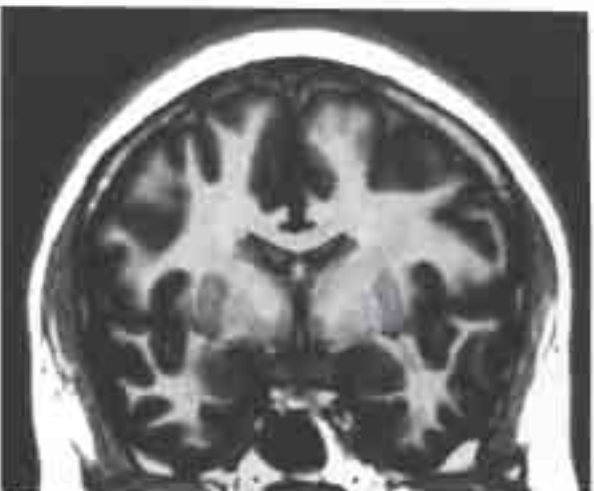




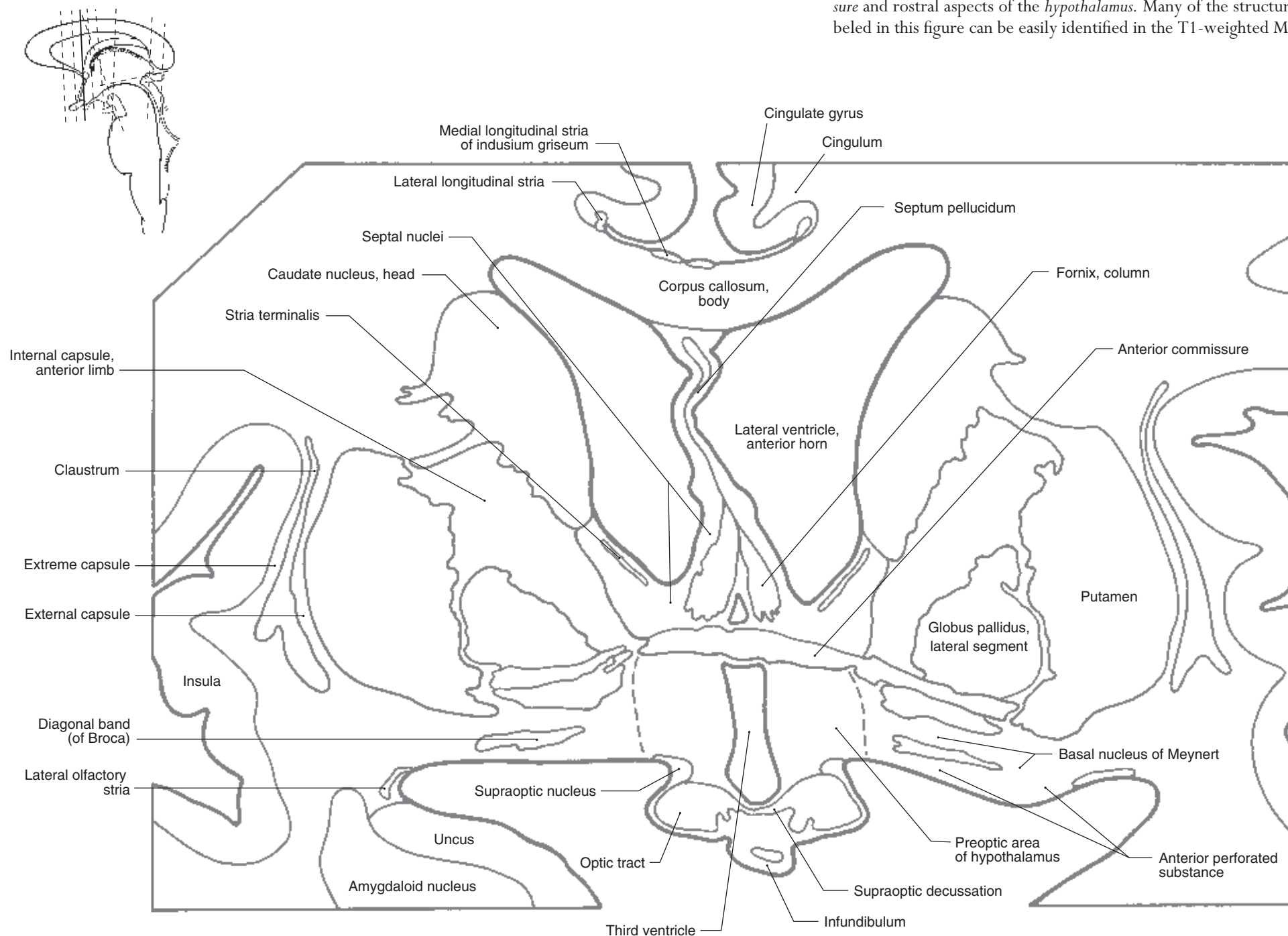
5-34 Coronal section of the forebrain through the *inter-ventricular foramen*, *genu of the internal capsule*, rostral tip of the *dorsal thalamus*, and about the middle one-third of the *hypo-*

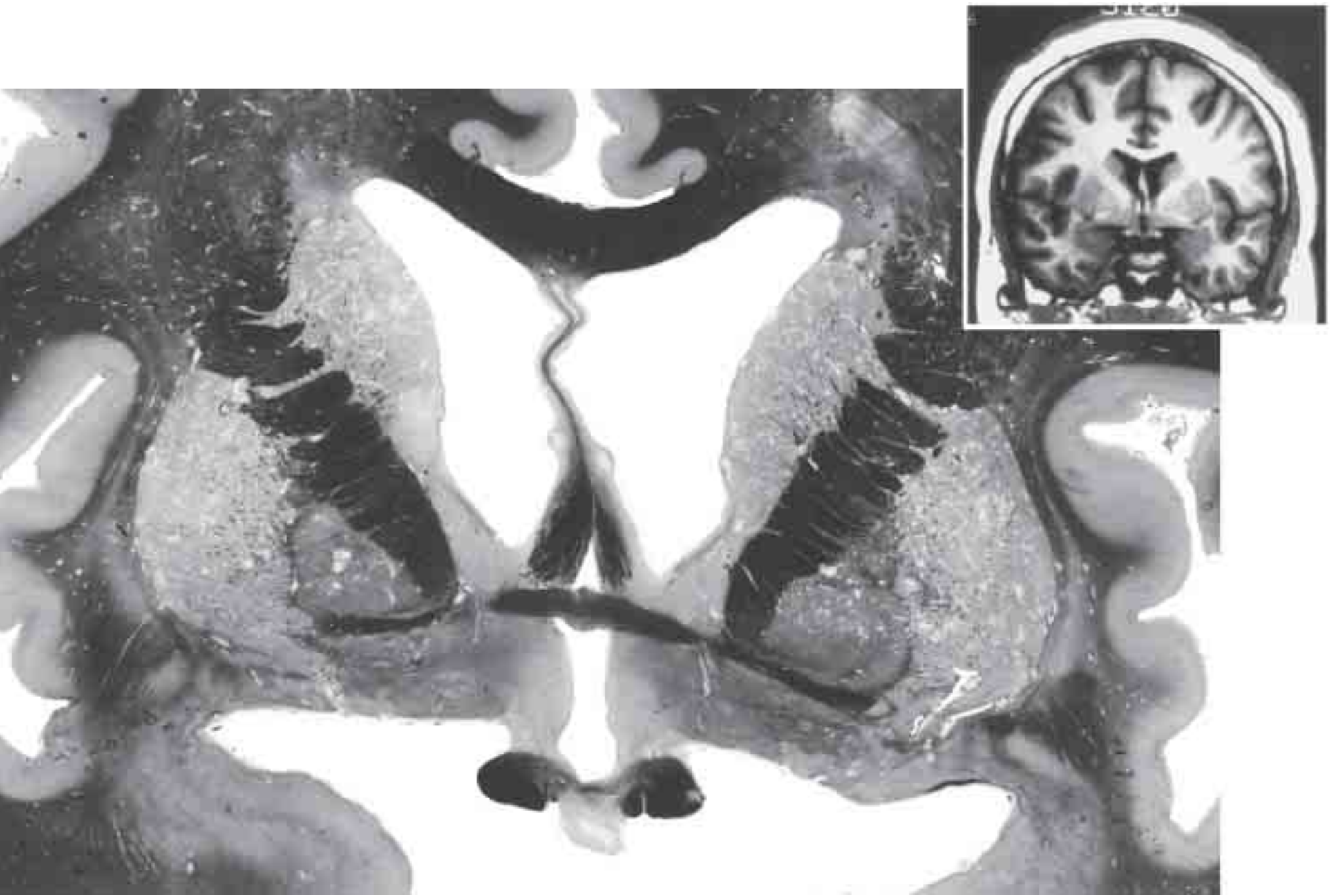
thalamus. Many of the structures labeled in this figure can be easily identified in the T1-weighted MRI adjacent to the photograph.



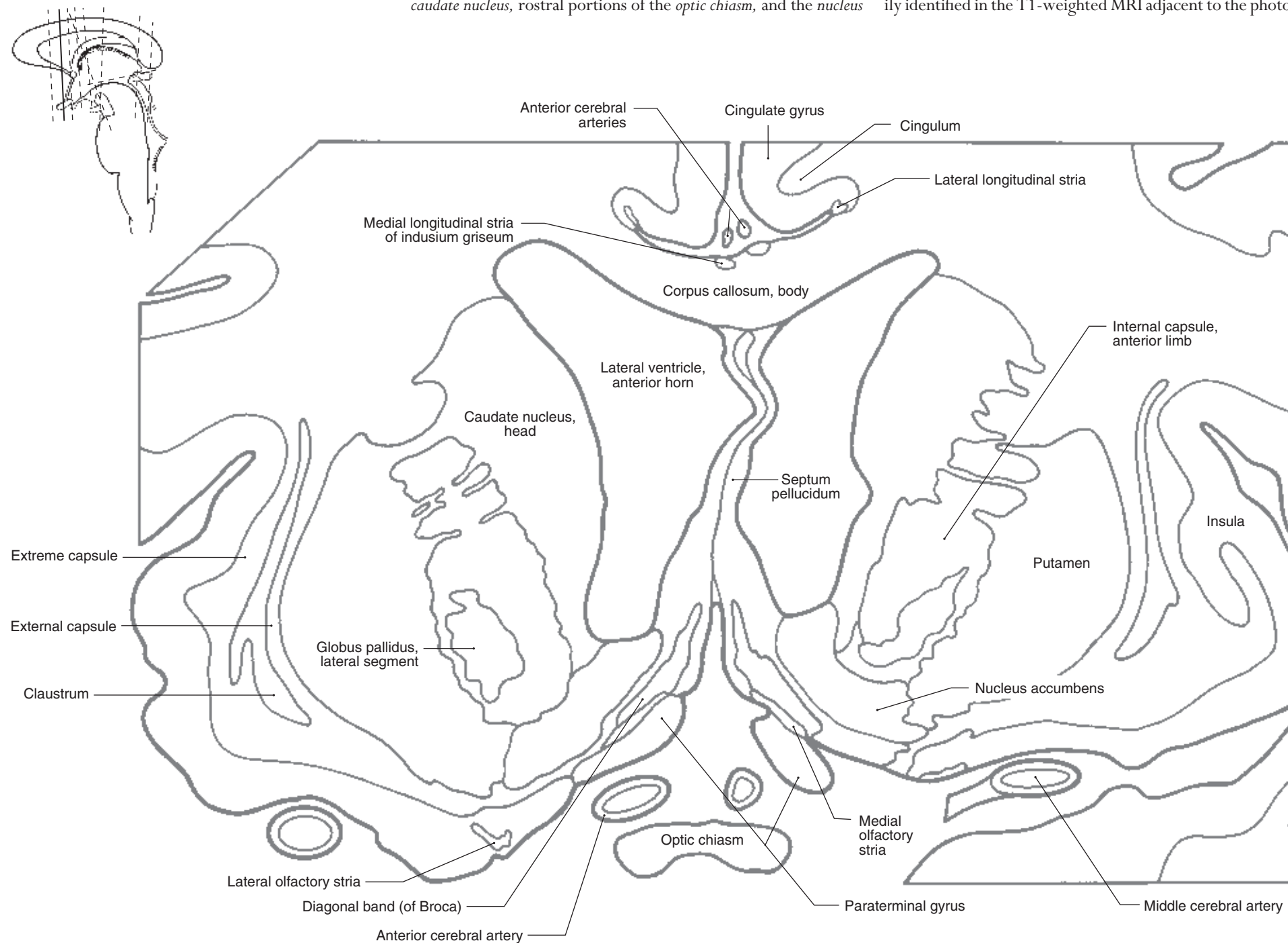


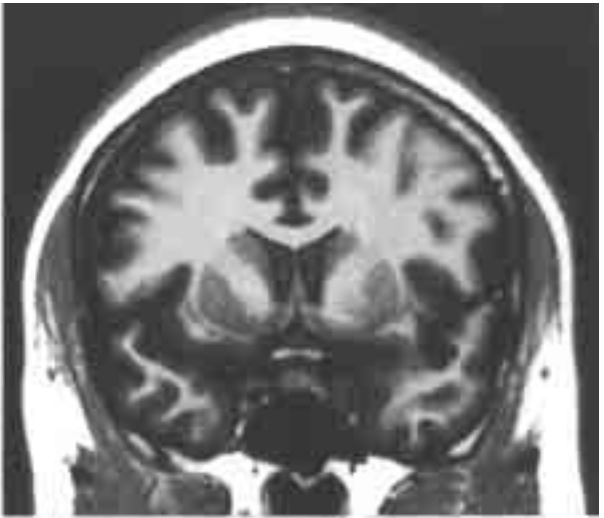
5-35 Coronal section of the forebrain through the *anterior commissure* and rostral aspects of the *hypothalamus*. Many of the structures labeled in this figure can be easily identified in the T1-weighted MRI.



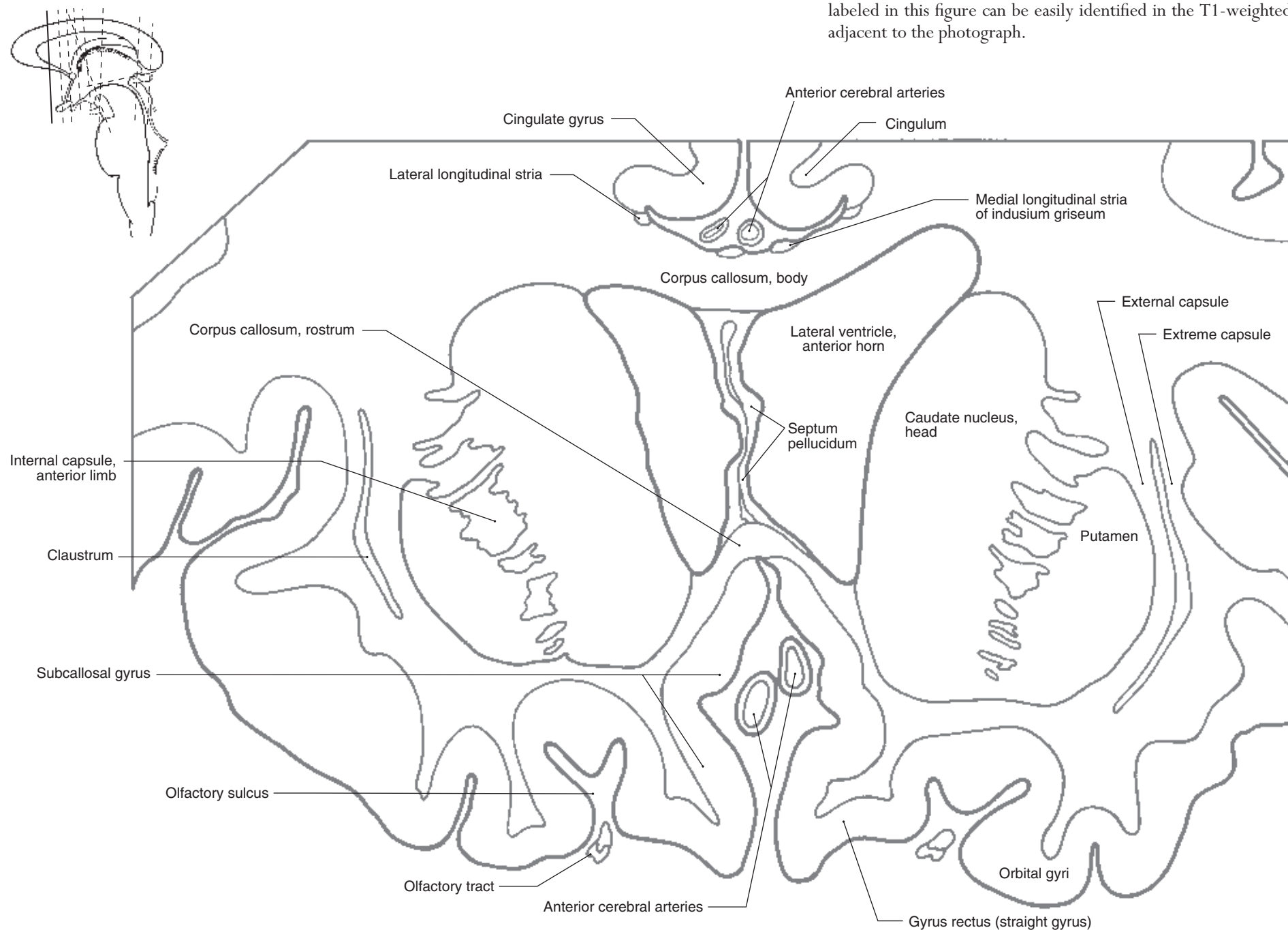


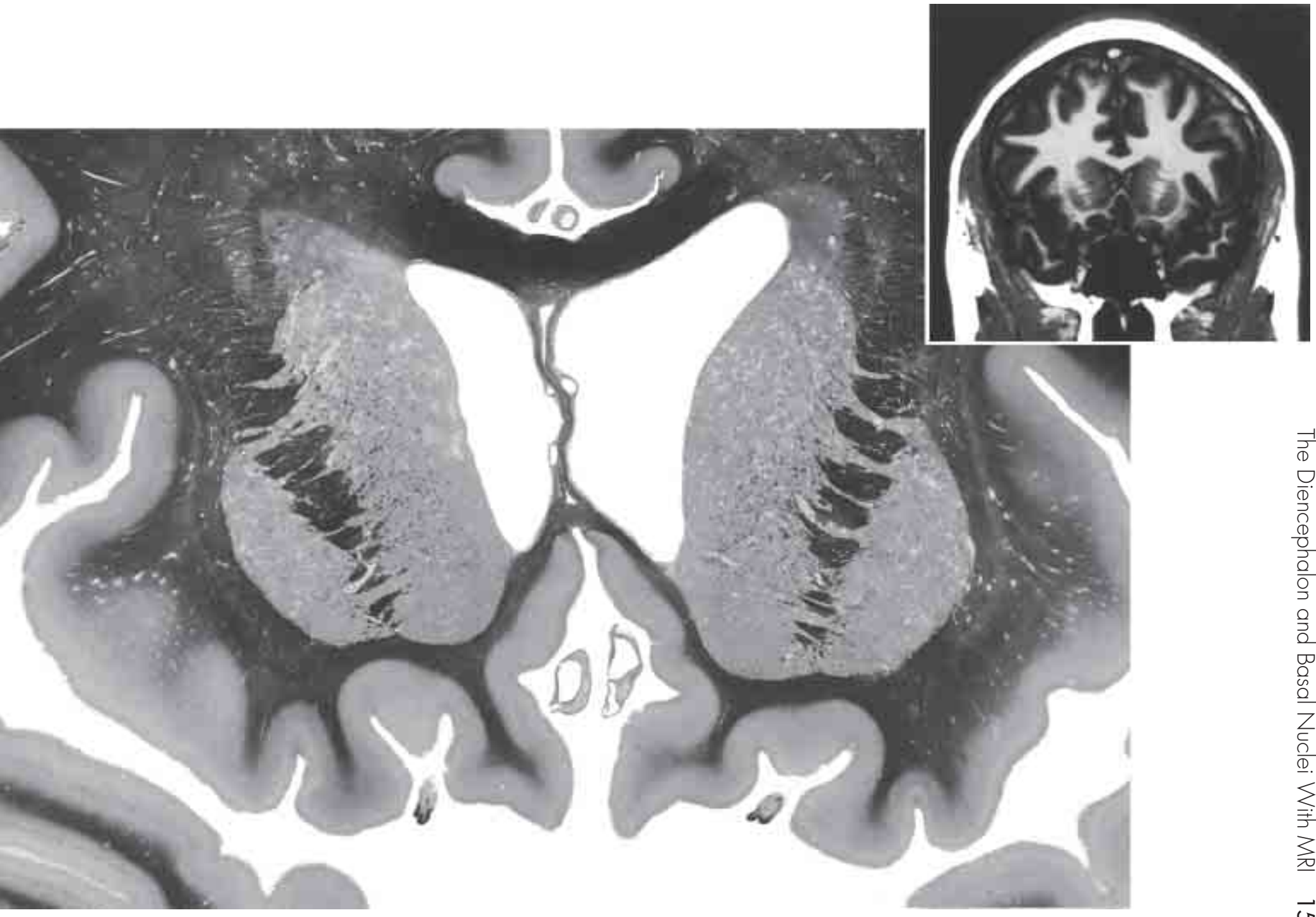
5-36 Coronal section of the forebrain through the head of the caudate nucleus, rostral portions of the optic chiasm, and the nucleus accumbens. Many of the structures labeled in this figure can be easily identified in the T1-weighted MRI adjacent to the photograph.





5-37 Coronal section of forebrain through the *head of the caudate nucleus* and the *anterior horn of the lateral ventricle*. Many of the structures labeled in this figure can be easily identified in the T1-weighted MRI adjacent to the photograph.





Vascular Syndromes or Lesions of the Forebrain

Forebrain vascular lesions result in a wide range of deficits that include motor and sensory losses and a variety of cognitive disorders. Forebrain vessels may be occluded by a *thrombus*. This is a structure (usually a clot) formed by blood products and frequently attached to the vessel wall. Deficits may appear slowly, or wax and wane, as the blood flow is progressively restricted.

Vessels may also be occluded by *embolization*. A foreign body, or *embolus* (fat, air, piece of thrombus, piece of sclerotic plaque, clump of bacteria, etc.), is delivered from some distant site into the cerebral circulation where it lodges in a vessel. Since this is a sudden event deficits usually appear quickly and progress rapidly. Interruption of blood supply to a part of the forebrain will result in an *infarct* of the area served by the occluded vessel.

Lesion in the Subthalamic Nucleus: Small vascular lesions occur in the subthalamic nucleus, resulting in rapid and unpredictable flailing movements of the contralateral extremities (*hemiballismus*). Movements are more obvious in the arm than in the leg. The clinical expression of this lesion is through corticospinal fibers, therefore it is on the contralateral side of the body.

Occlusion of Lenticulostriate Branches to Internal Capsule: Damage to the internal capsule may result in contralateral *hemiplegia* (corticospinal fibers) and a loss, or diminution, of sensory perception (pain, thermal sense, proprioception) caused by damage to thalamocortical fibers traversing the posterior limb to the overlying sensory cortex. If the lesion extends into the genu of the capsule, a partial paralysis of facial muscles and tongue movement may also occur contralaterally.

Infarction of Posterior Thalamic Nuclei: Occlusion of vessels to posterior thalamic regions results in either a *complete sensory loss* (pain/thermal sense, touch, vibratory and position sense) on the contralateral side of the body, or a *dissociated sensory loss*. In the latter case the patient may experience pain/thermal sensory losses but not position/vibratory losses, or vice versa. As the lesion resolves the patient may experience intense persistent pain, *thalamic pain*, or *anesthesia dolorosa*.

Occlusion of Distal Branches of the Anterior (ACA) or Middle (MCA) Cerebral Arteries: Occlusion of distal branches of the ACA results in motor

and sensory losses in the contralateral foot, leg, and thigh owing to damage to the anterior and posterior paracentral gyri (primary motor and sensory cortices for lower extremity). Occlusion of distal branches of MCA results in contralateral motor and sensory losses of the upper extremity, trunk, and face with sparing of the leg and foot, and a consensual deviation of the eyes to the ipsilateral side. This represents damage to the precentral and postcentral gyri and to the frontal eye fields.

Watershed Infarct: Sudden systemic hypotension, hypoperfusion, or embolic showers may result in infarcts at border zones between the territories served by the ACA, MCA, and posterior cerebral artery (PCA). *Anterior watershed infarcts* (at the ACA–MCA junction) result in a contralateral hemiparesis (mainly leg) and expressive language or behavioral changes. *Posterior watershed infarcts* (MCA–PCA interface) result in visual deficits and language problems.

Anterior Choroidal Artery Syndrome: Occlusion of this vessel may result from small emboli or small vessel disease. This syndrome may also occur as a complication of temporal lobectomy (removal of the temporal lobe to treat intractable epilepsy). The infarcted area usually includes the optic tract, lower portions of the basal nuclei, and lower aspects of the internal capsule. The patient experiences a contralateral *hemiplegia*, *hemihypesthesia*, and *homonymous hemianopsia*. These deficits are due to, respectively, involvement of corticospinal fibers in the posterior limb of the internal capsule or possibly in the crus cerebri, involvement of thalamocortical fibers in the posterior limb of the internal capsule, and involvement of the fibers of the optic tract.

Parkinson Disease: Parkinson disease (paralysis agitans) results from a loss of the dopamine-containing cells in the substantia nigra. Although this part of the brain is located in the midbrain, the terminals of these nigrostriatal fibers are in the putamen and the caudate nucleus. The classic signs and symptoms of this disease are a *stooped posture*, *resting tremor*, *rigidity*, *shuffling or festinating gait*, and difficulty initiating or maintaining movement (*akinesia*, *hypokinesia*, or *bradykinesia*). Initially, the tremor and walking difficulty may appear on

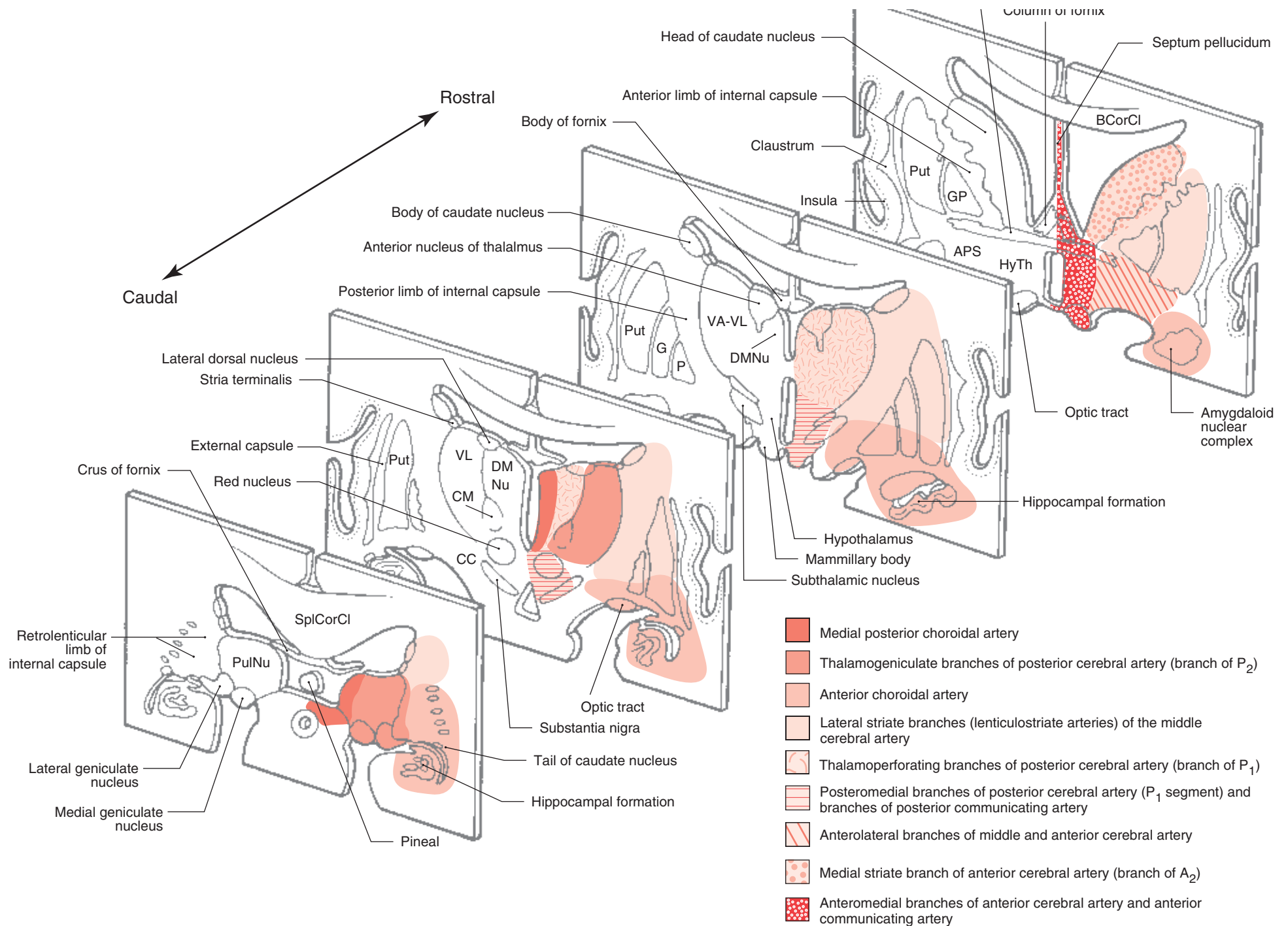
one side of the body, but these signs usually spread to both sides with time. This is a neurodegenerative disease that has, in its later stages, a dementia component.

Transient Ischemic Attack: A transient ischemic attack, commonly called TIA, is a temporary (and frequently focal) neurologic deficit that usually resolves within 10 to 30 minutes from the onset of symptoms. The cause is temporary occlusion of a vessel or inadequate perfusion of a restricted vascular territory. TIA that last 60+ minutes may result in some permanent deficits. This vascular event may take place anywhere in the central nervous system but is more common in the cerebral hemisphere.

5-38 Semidiagrammatic representation of the internal distribution of arteries to the diencephalon, basal ganglia, and internal capsule. Selected structures are labeled on the left side of each section; the general pattern of arterial distribution overlies these structures on the right side. The general distribution patterns of arteries in the forebrain as shown here may vary from patient to patient. For example, the adjacent territories served by neighboring vessels may overlap to varying degrees at their margins or the territory of a particular vessel may be larger or smaller than seen in the general pattern.

Abbreviations

APS	Anterior perforated substance
BCorCl	Body of corpus callosum
CC	Crus cerebri
CM	Centromedian nucleus of thalamus
DMNu	Dorsomedial nucleus of thalamus
GP	Globus pallidus
HyTh	Hypothalamus
PulNu	Pulvinar nuclear complex
Put	Putamen
SplCorCl	Splenium of the corpus callosum
VA	Ventral anterior nucleus of thalamus
VL	Ventral lateral nucleus of thalamus



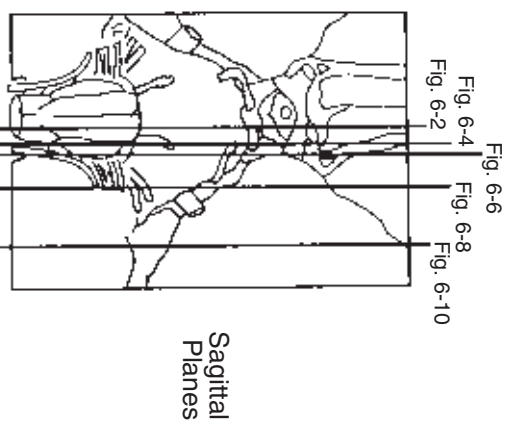
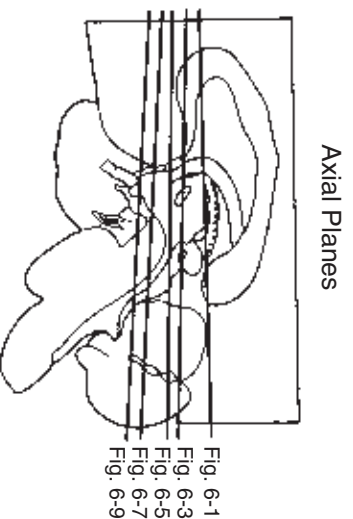
Internal Morphology of the Brain in Stained Sections: Axial–Sagittal Correlations with MRI

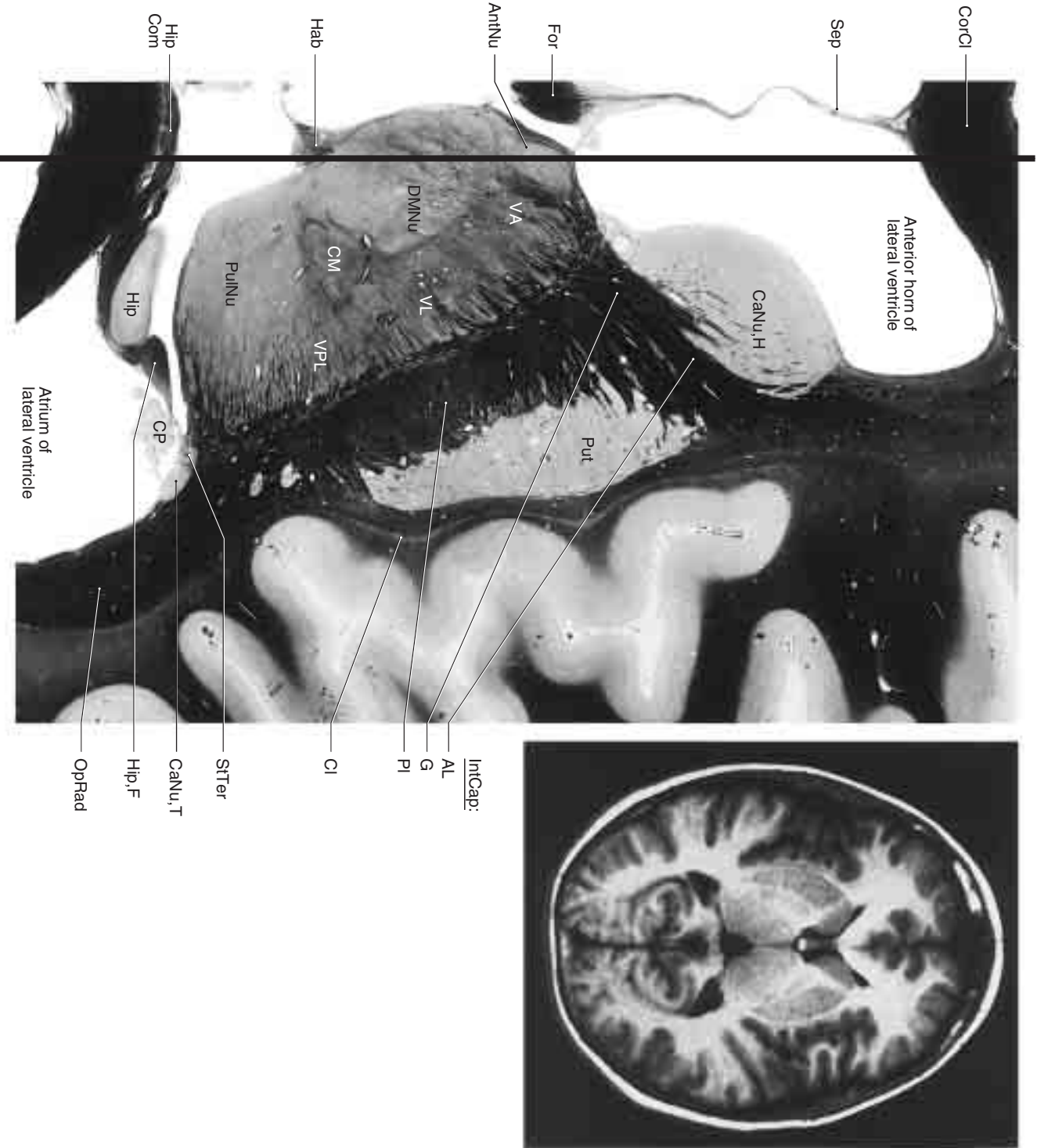
Although the general organization of Chapter 6 has been described in Chapter 1 (the reader may wish to refer back to this section), it is appropriate to reiterate its unique features at this point. Each set of facing pages has photographs of an axial stained section (left-hand page) and a sagittal stained section (right-hand page). In addition to individually labeled structures, a heavy line appears on each photograph. This prominent line on the axial section represents the approximate plane of the sagittal section located on the facing page. On the sagittal section this line signifies the approximate plane of the corresponding axial section. The reader can identify features in each photograph and then, using this line as a reference point, visualize structures that are located either above or below that plane (axial to sagittal comparison) or medial or lateral to that plane (sagittal to axial comparison). This method of presentation provides a format for reconstructing and understanding three-dimensional relationships within the central nervous system.

The magnetic resonance image (MRI) placed on every page

in this chapter gives the reader an opportunity to compare internal brain anatomy, as seen in stained sections, with those structures as visualized in clinical images generated in the same plane. Even a general comparison reveals that many features, as seen in the stained section, can be readily identified in the adjacent MRI.

This chapter is also organized so that one can view structures in either the axial or the sagittal plane only. Axial photographs appear on left-hand pages and are sequenced from dorsal to ventral (odd-numbered Figures 6-1 through 6-9), while sagittal photographs are on the right-hand pages and progress from medial to lateral (even-numbered Figures 6-2 through 6-10). Consequently, the user can identify and follow structures through an axial series by simply flipping through the left-hand pages or through a sagittal series by flipping through the right-hand pages. The inherent flexibility in this chapter should prove useful in a wide variety of instructional/learning situations. The drawings shown in the following illustrate the axial and sagittal planes of the photographs in this chapter.

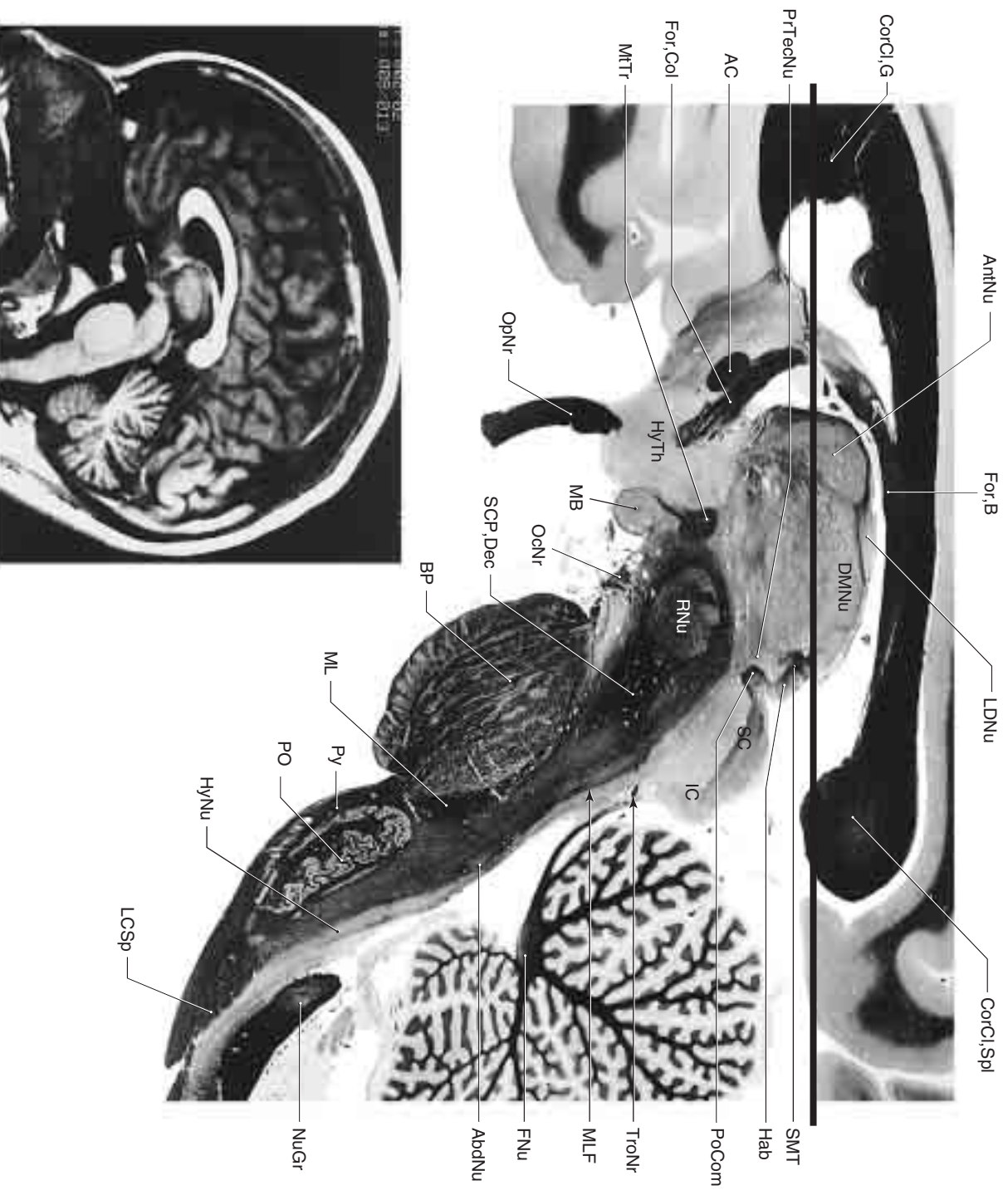




6-1 Axial section through the head of the caudate nucleus and several key thalamic nuclei (*anterior, centromedian, pulvinar, habenular*). The heavy line represents the approximate plane of the sagittal section shown in Figure 6-2 (facing page). Many of the structures labeled in this photograph can be clearly identified in the adjacent T1-weighted MRI.

Abbreviations

AntNu	Anterior nucleus of thalamus	HipCom	Hippocampal commissure
CaNu,H	Caudate nucleus, head	IntCap,AL	Internal capsule, anterior limb
CaNu,T	Caudate nucleus, tail	IntCap,G	Internal capsule, genu
CI	Clastrum	IntCap,PL	Internal capsule, posterior limb
CM	Centromedial nucleus of thalamus	OpRad	Optic radiations
CorCI	Corpus callosum	PuNu	Pulvinar nuclear complex
CP	Choroid plexus	Put	Putamen
DMNu	Dorsomedial nucleus of thalamus	Sep	Septum pellucidum
For	Fornix, column	StTer	Stria terminalis
Hab	Habenular nucleus	VA	Ventral anterior nucleus of thalamus
Hip	Hippocampal formation	VL	Ventral lateral nucleus of thalamus
Hip,F	Hippocampus, fimbria	VPL	Ventral posterolateral nucleus



6-2 Sagittal section through the *column of the fornix*, *anterior thalamic nucleus*, *red nucleus*, and *medial portions of the pons* (*abducens nucleus*), *cerebellum* (*fastigial nucleus*), and *medulla* (*nucleus gracilis*). The heavy line

represents the approximate plane of the axial section shown in Figure 6-1 (facing page). Many of the structures labeled in this photograph can be clearly identified in the adjacent T1-weighted MRI.

Abbreviations

AbdNu	Abducens nucleus	MB	Mammillary body
AC	Anterior commissure	ML	Medial lemniscus
AntNu	Anterior nucleus of thalamus	MLF	Medial longitudinal fasciculus
BP	Basilar pons	MTr	Mammillothalamic tract
CorCl,G	Corpus callosum, genu	NuGr	Nucleus gracilis
CorCl,Spl	Corpus callosum, splenium	OoNr	Oculomotor nerve
DMNu	Dorsomedial nucleus of thalamus	OpNr	Optic nerve
FNu	Fastigial nucleus (medial cerebellar nucleus)	PO	Principal olivary nucleus
For,B	Fornix, body	PoCom	Posterior commissure
For,Col	Fornix, column	PrTCeNu	Pretectal nuclei
Hab	Habenular nuclei	Py	Pyramid
HyNu	Hypoglossal nucleus	RNu	Red nucleus
HyTh	Hypothalamus	SC	Superior colliculus
IC	Inferior colliculus	SCP,Dec	Superior cerebellar peduncle, decussation
LCSp	Lateral corticospinal tract	SMT	Stria medullaris thalami
LDNu	Lateral dorsal nucleus	TroNr	Trochlear nerve