

Posture & Movement

In veterinary neurology, abnormalities of posture & movement are more important than sensory disorders because animals readily express motor behavior but hardly at all report their feelings.

Preview: Posture/Movement Hierarchy

Spinal Cord and Cranial Nerve Motor Nuclei

Local reflex—useful response to a stimulus (determined by local interneuron circuits).

Hindbrain

Standing posture—excitation of alpha & gamma motor units of extensor muscles (driven by spontaneous activity of reticular formation & vestibular neurons).

Equilibrium—maintaining normal position of eyes, head, & body (vestibular system).

Midbrain

Orientation—orienting head/eyes/ears toward abrupt visual/auditory stimuli (tectum).

Specific movements—moving individual joints (via red nucleus and rubrospinal tract).

Forebrain

Inherent movement sequences—species-specific patterns of posture/movement/gait (basal nuclei interacting with thalamus & motor areas of cerebral cortex).

Learned movements—including learned movement sequences performed too rapidly for sensory feedback (involves pattern generation in premotor cerebral cortex).

Brain Structures Concerned with Posture & Movement

-----*Hindbrain*

Reticular Formation

Anatomy: network (mixture) of gray & white matter, found throughout the brainstem
— gets synaptic input from collateral branches of ascending tracts (e.g., spinothalamic tract)

Physiology: spontaneously active neuronal circuits; perform three major functions:

1. Ascending system to alert cerebral cortex (via non-specific thalamic nuclei) vs. coma
2. Vegetative centers: regulate heart rate, respiration, digestion, micturition, etc.
3. Standing posture and muscle tone via two pathways to alpha & gamma neurons:
 - pontine reticulospinal tract — arises from neurons located laterally in pons & medulla
 - dominant and spontaneously active; runs ipsilaterally in the ventral funiculus
 - activates alpha & gamma neurons to extensor muscles of proximal joints
 - medullary reticulospinal tract — arises from neurons located medially in medulla
 - inhibits neurons to extensor mm. & excites neurons to flexor mm.;
 - not spontaneously active — driven by cerebral cortex to preset movement posture
 - descends bilaterally in the lateral funiculus

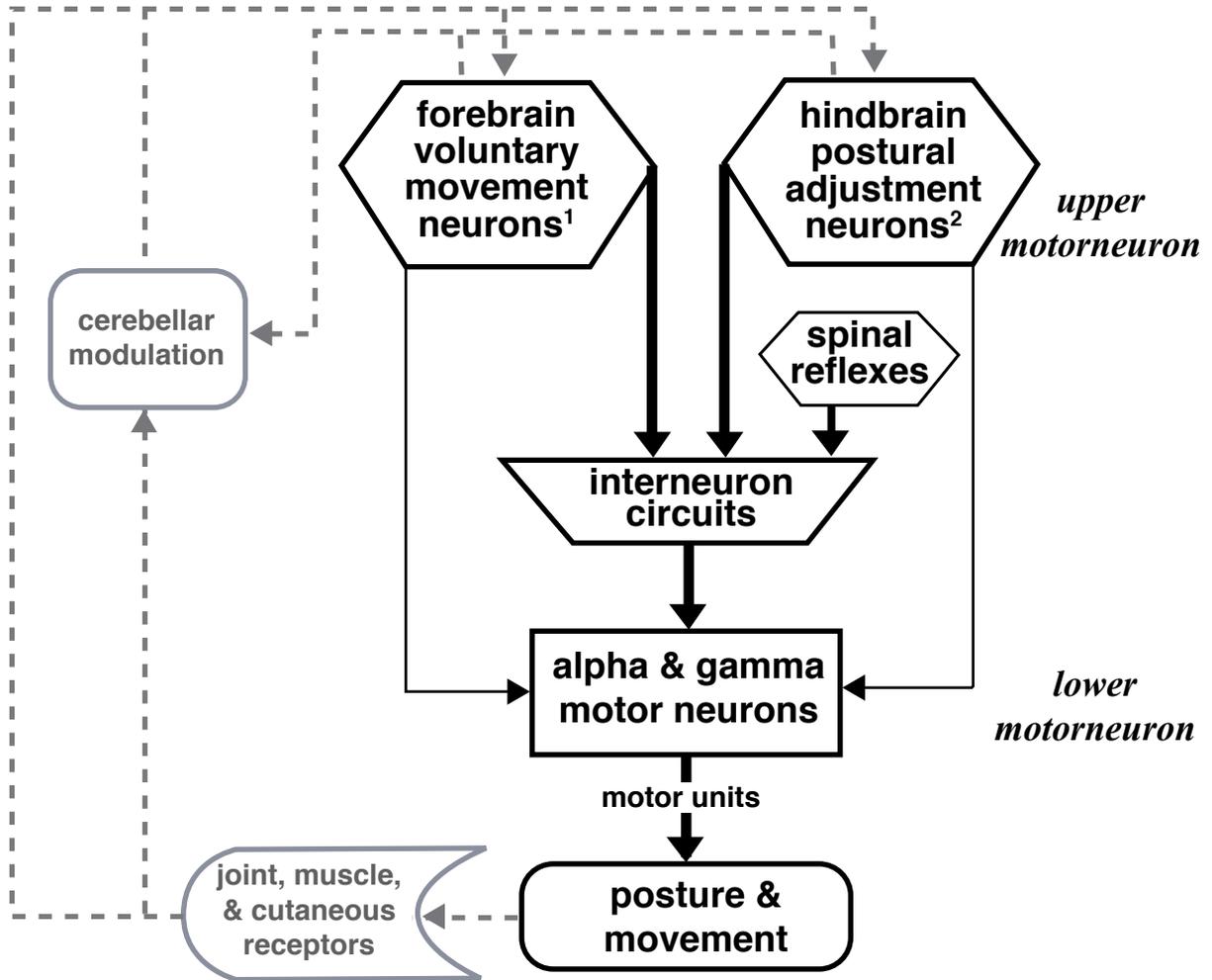
Vestibular nuclei *discussed previously*

Two descending tracts: lateral vestibulospinal tract— which also drives standing posture, & medial vestibulospinal tract (m.l.f.)— which controls neck muscles.

Note: vestibular nuclei also utilize the two reticulospinal tracts to adjust muscle tone.

ADDENDUM

Movement and Posture Overview



Note:

- 1 = Forebrain circuits involving basal nuclei and pyramidal & extrapyramidal tracts, and
- 2 = Hindbrain reticular formation and vestibular nuclei, plus midbrain tectum.

Comments:

- Posture and movement result from excited alpha and gamma motor neurons. A motor unit is one alpha motor neuron plus all of the muscle fibers it innervates.

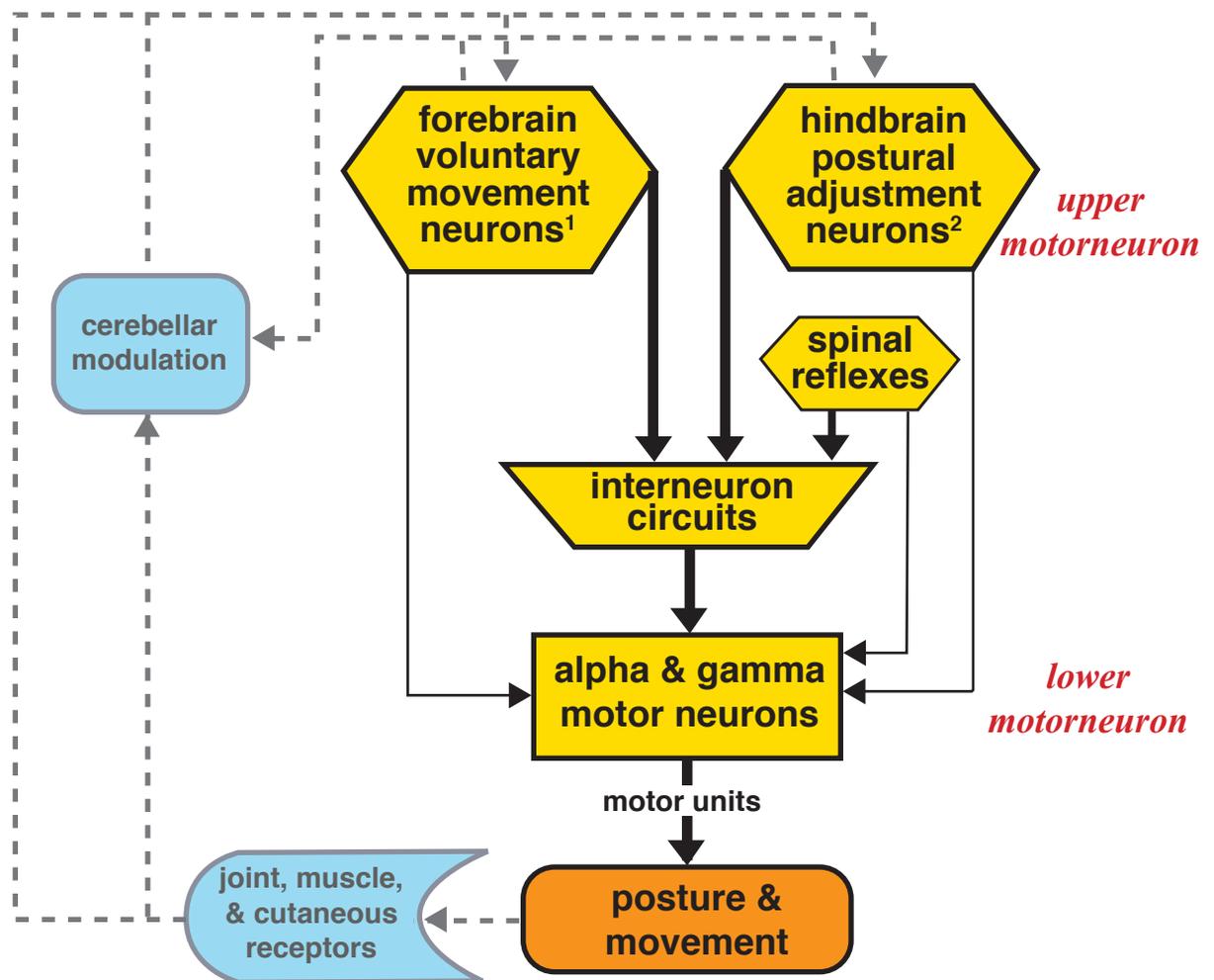
- Interneurons "hardwire" motor neurons so that logical movement patterns are produced (e.g., synergists are excited & antagonists are inhibited). Thus, nearly all descending pathway axons synapse on interneurons. However, a minority of descending axons synapse directly on motor neurons (e.g., vestibulospinal axons; also, a minority of pyramidal tract axons synapse directly on motor neurons that innervate digits in primates and raccoons).

- Thus both voluntary and reflex pathways compete for control of the same interneurons and motor neurons. To gain control of motor units, voluntary neurons must suppress reflex neurons, and vice versa.

- While movement is underway, feedback from proprioceptors influences ongoing neuronal activity in motor centers to effect desired movement goals. The cerebellum influences neuronal activity in the initiating motor centers (the cerebellum continuously modulates neuronal activity, based on information about motor commands and proprioceptive feedback about position and acceleration).

ADDENDUM

Movement and Posture Overview



Note:

1 = Forebrain circuits involving basal nuclei and pyramidal & extrapyramidal tracts, and

2 = Hindbrain reticular formation and vestibular nuclei, plus midbrain tectum.

Posture & Movement

In veterinary neurology, abnormalities of posture & movement are more important than sensory disorders because animals readily express motor behavior but hardly at all report their feelings.

Preview: Posture/Movement Hierarchy

Spinal Cord and Cranial Nerve Motor Nuclei

Local reflex—useful response to a stimulus (determined by local interneuron circuits).

Hindbrain

Standing posture—excitation of alpha & gamma motor units of extensor muscles (driven by spontaneous activity of reticular formation & vestibular neurons).

Equilibrium—maintaining normal position of eyes, head, & body (vestibular system).

Midbrain

Orientation—orienting head/eyes/ears toward abrupt visual/auditory stimuli (tectum).

Specific movements—moving individual joints (via red nucleus and rubrospinal tract).

Forebrain

Inherent movement sequences—species-specific patterns of posture/movement/gait (basal nuclei interacting with thalamus & motor areas of cerebral cortex).

Learned movements—including learned movement sequences performed too rapidly for sensory feedback (involves pattern generation in premotor cerebral cortex).

Brain Structures Concerned with Posture & Movement

-----*Hindbrain*

Reticular Formation

Anatomy: network (mixture) of gray & white matter, found throughout the brainstem
— gets synaptic input from collateral branches of ascending tracts (e.g., spinothalamic tract)

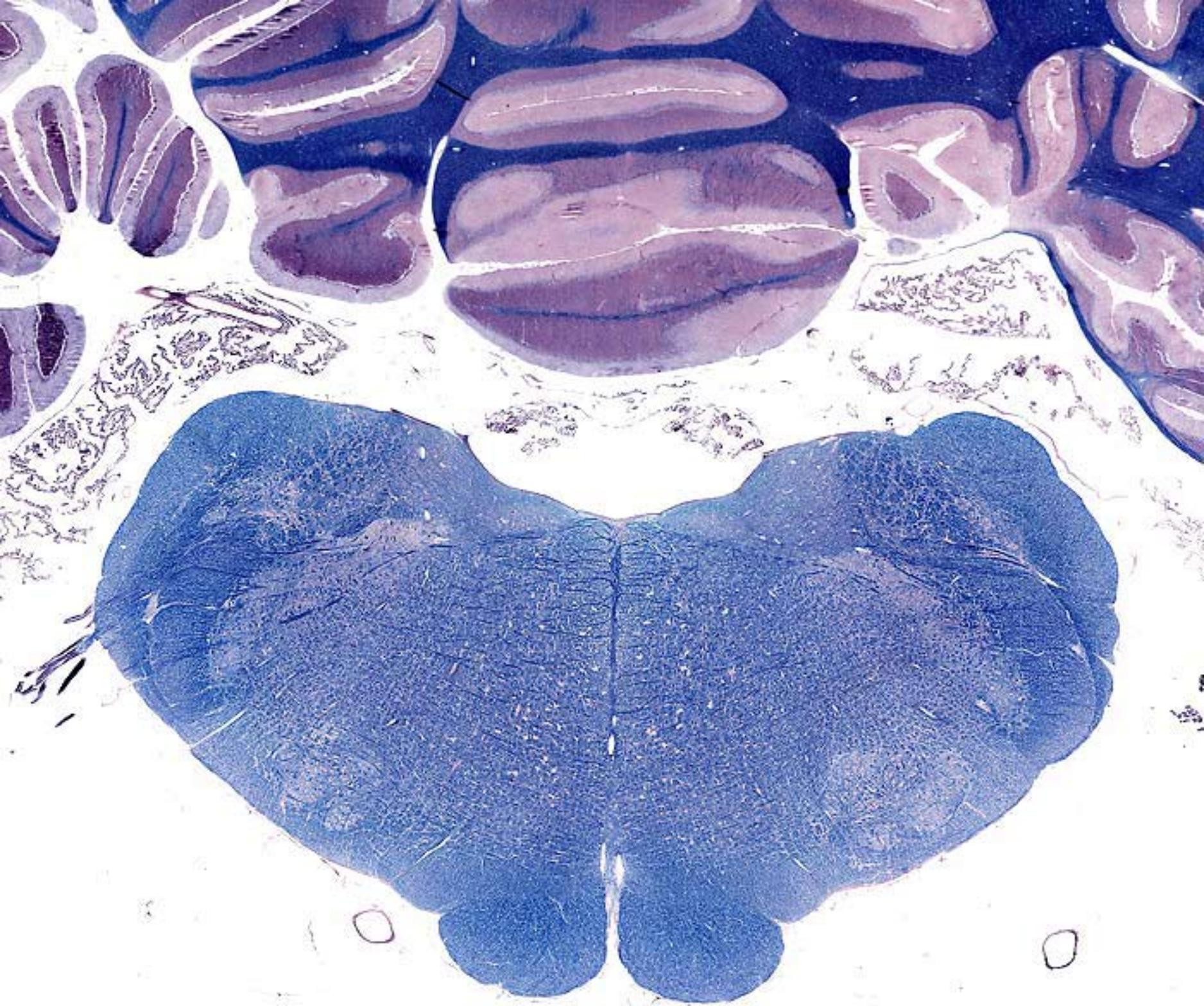
Physiology: spontaneously active neuronal circuits; perform several major functions:

1. Ascending system to alert cerebral cortex (via non-specific thalamic nuclei) vs. coma
2. Vegetative centers: regulate heart rate, respiration, digestion, micturition, etc.
3. Standing posture and muscle tone via two pathways to alpha & gamma neurons:
 - pontine reticulospinal tract — arises from neurons located laterally in pons & medulla
 - dominant and spontaneously active; runs ipsilaterally in the ventral funiculus
 - activates alpha & gamma neurons to extensor muscles of proximal joints
 - medullary reticulospinal tract — arises from neurons located medially in medulla
 - inhibits neurons to extensor mm. & excites neurons to flexor mm.;
 - not spontaneously active — driven by cerebral cortex to preset movement posture
 - descends bilaterally in the lateral funiculus

Vestibular nuclei *discussed previously*

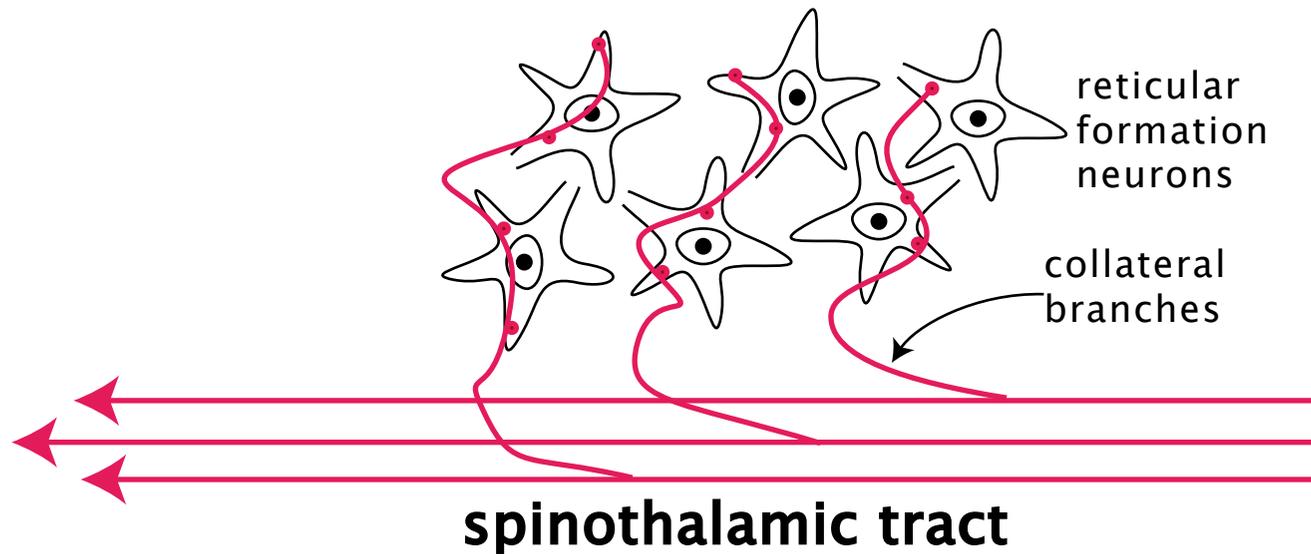
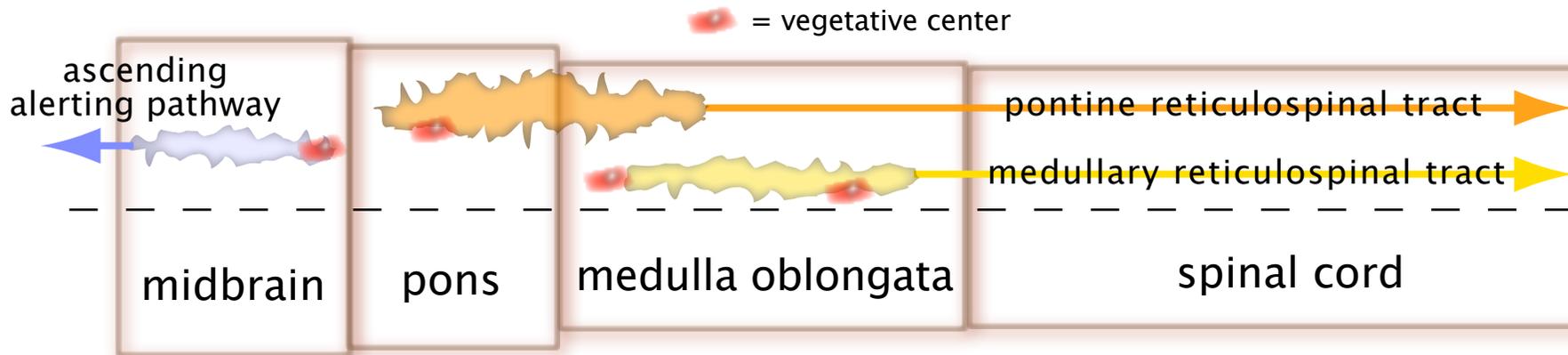
Two descending tracts: lateral vestibulospinal tract— which also drives standing posture, & medial vestibulospinal tract (m.l.f.)— which controls neck muscles.

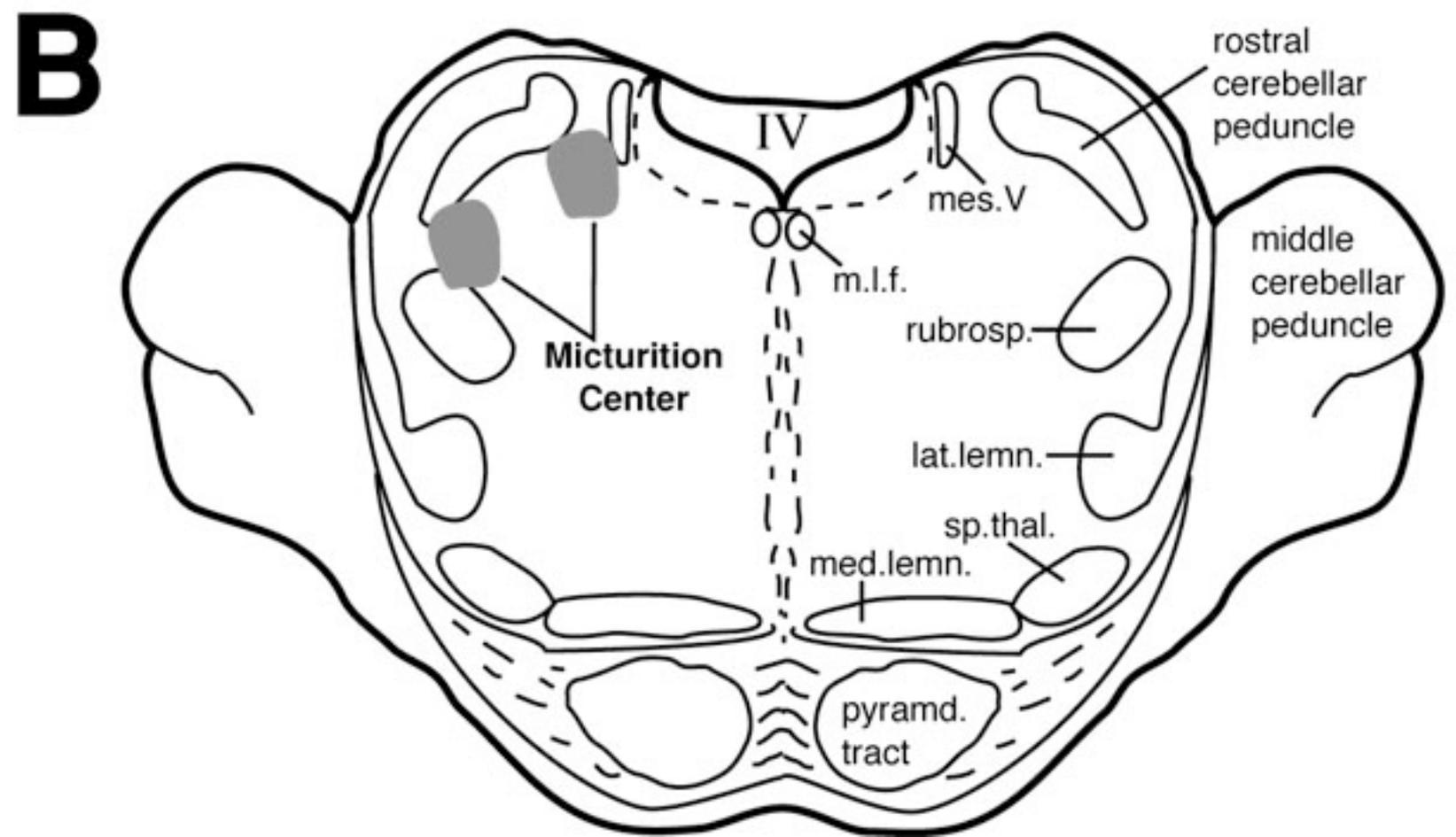
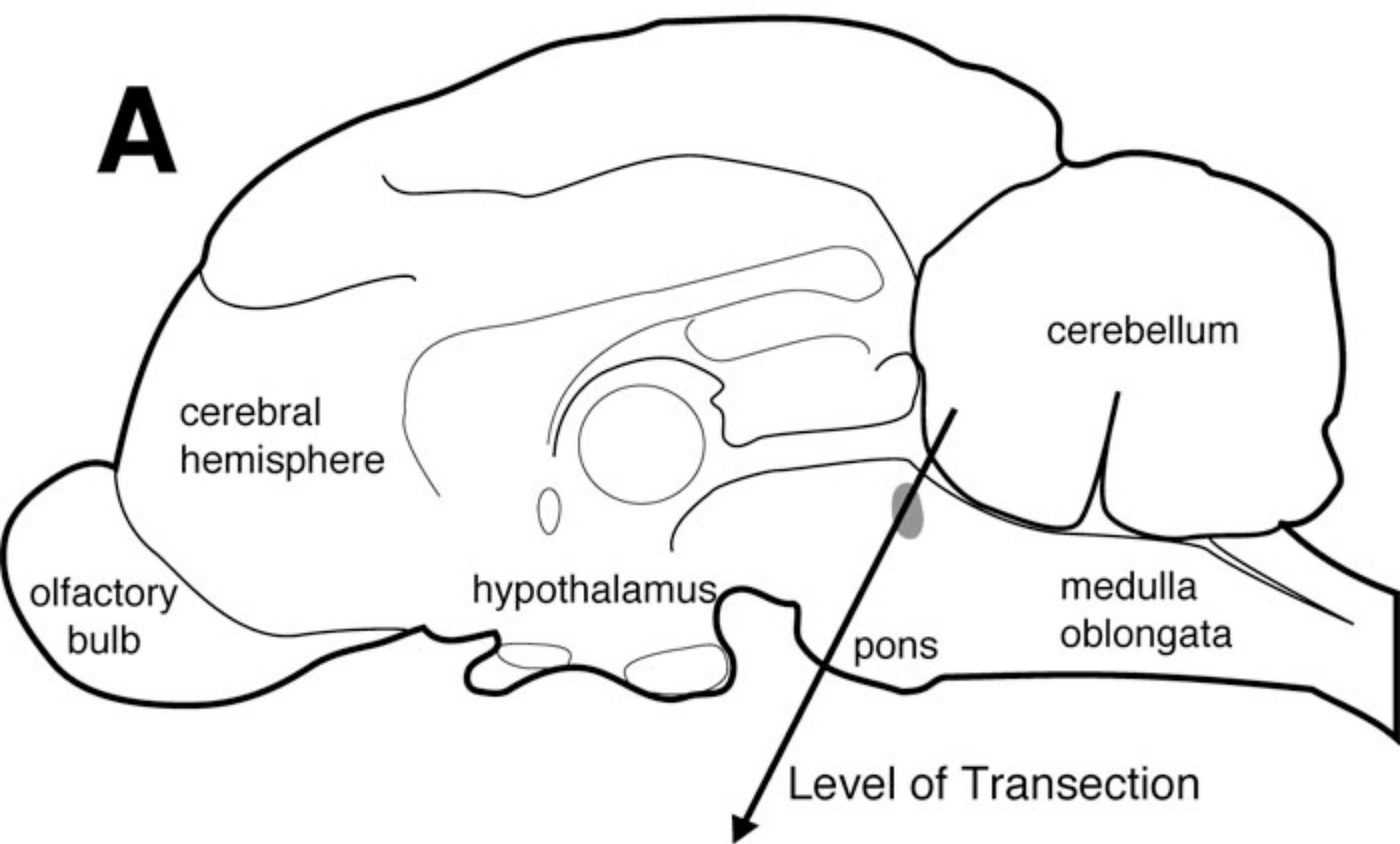
Note: vestibular nuclei also utilize the two reticulospinal tracts to adjust muscle tone.

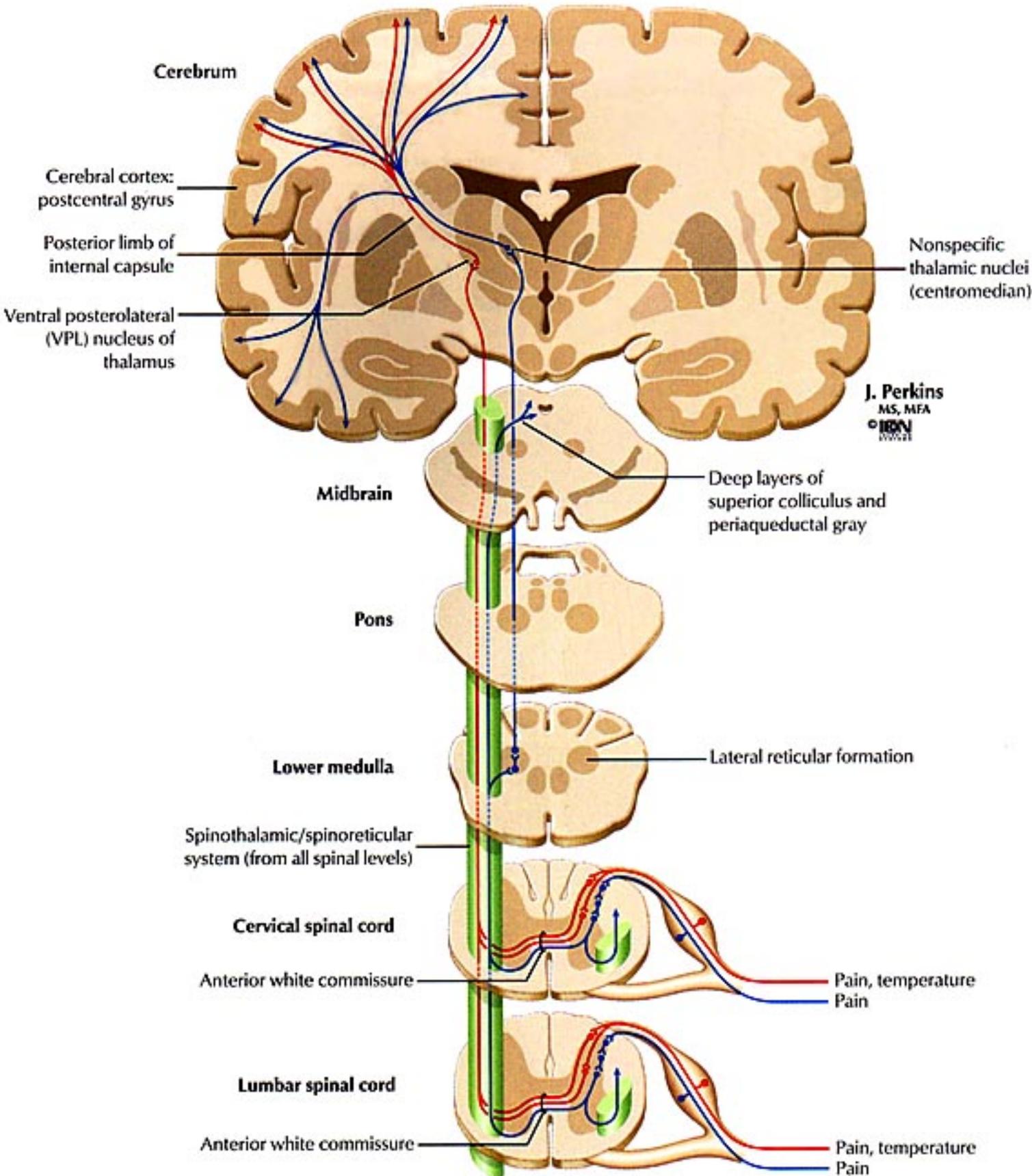


SCHEMATIC

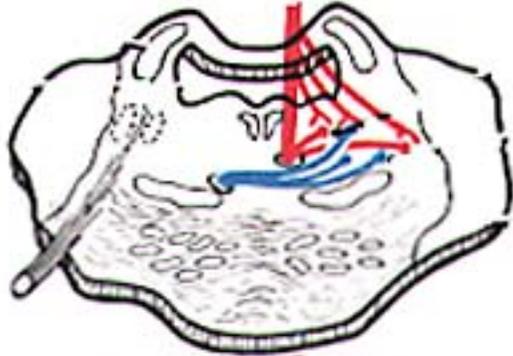
Midbrain, Pontine & Medullary Reticular Formation



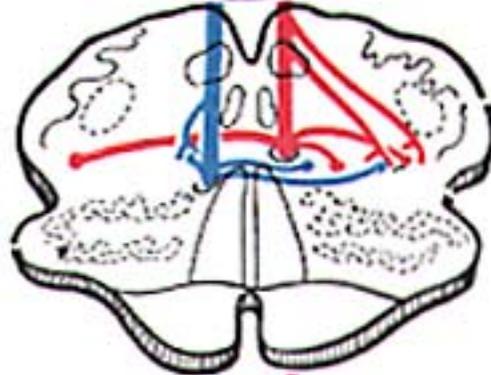




pons



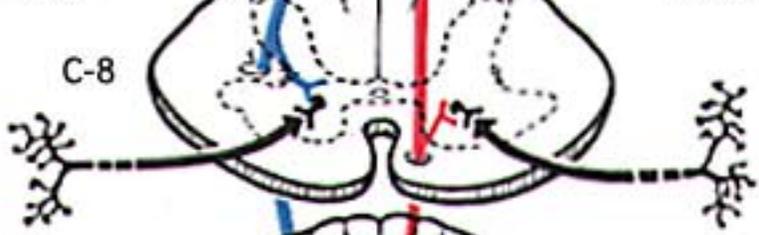
medulla



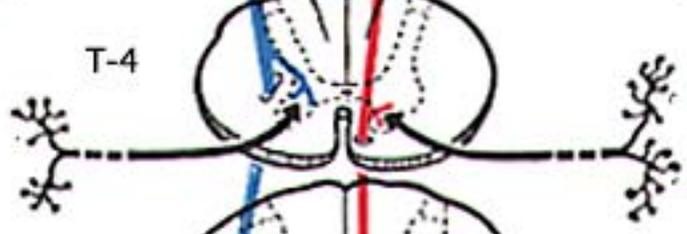
Medullary reticulospinal tract

Pontine reticulospinal tract

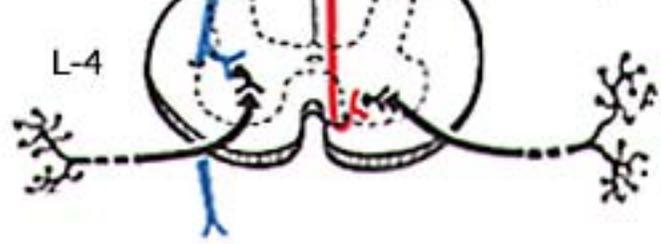
C-8



T-4



L-4



Posture & Movement

In veterinary neurology, abnormalities of posture & movement are more important than sensory disorders because animals readily express motor behavior but hardly at all report their feelings.

Preview: Posture/Movement Hierarchy

Spinal Cord and Cranial Nerve Motor Nuclei

Local reflex—useful response to a stimulus (determined by local interneuron circuits).

Hindbrain

Standing posture—excitation of alpha & gamma motor units of extensor muscles (driven by spontaneous activity of reticular formation & vestibular neurons).

Equilibrium—maintaining normal position of eyes, head, & body (vestibular system).

Midbrain

Orientation—orienting head/eyes/ears toward abrupt visual/auditory stimuli (tectum).

Specific movements—moving individual joints (via red nucleus and rubrospinal tract).

Forebrain

Inherent movement sequences—species-specific patterns of posture/movement/gait (basal nuclei interacting with thalamus & motor areas of cerebral cortex).

Learned movements—including learned movement sequences performed too rapidly for sensory feedback (involves pattern generation in premotor cerebral cortex).

Brain Structures Concerned with Posture & Movement

-----*Hindbrain*

Reticular Formation

Anatomy: network (mixture) of gray & white matter, found throughout the brainstem
— gets synaptic input from collateral branches of ascending tracts (e.g., spinothalamic tract)

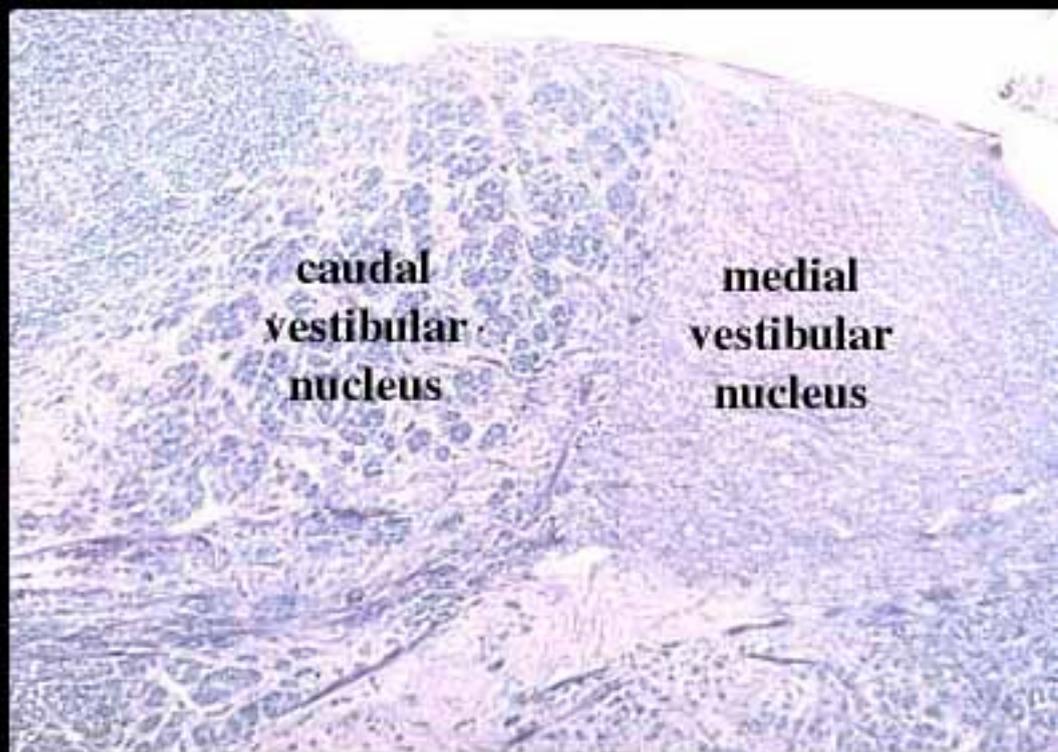
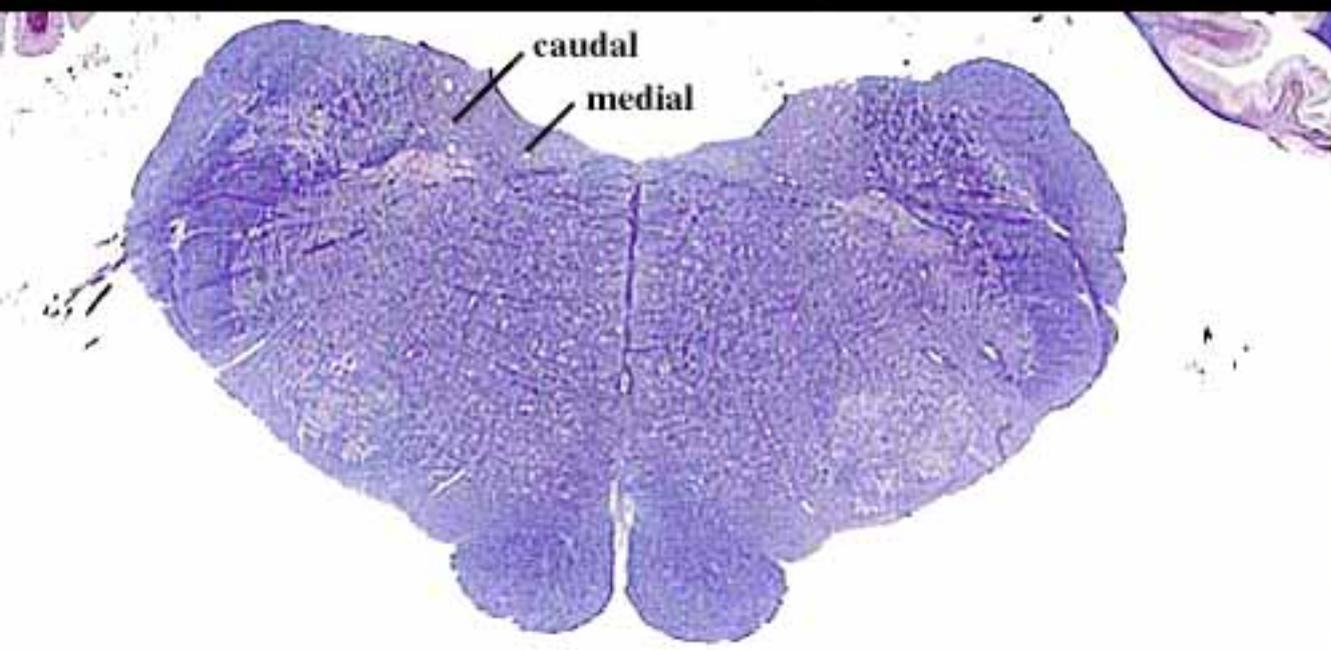
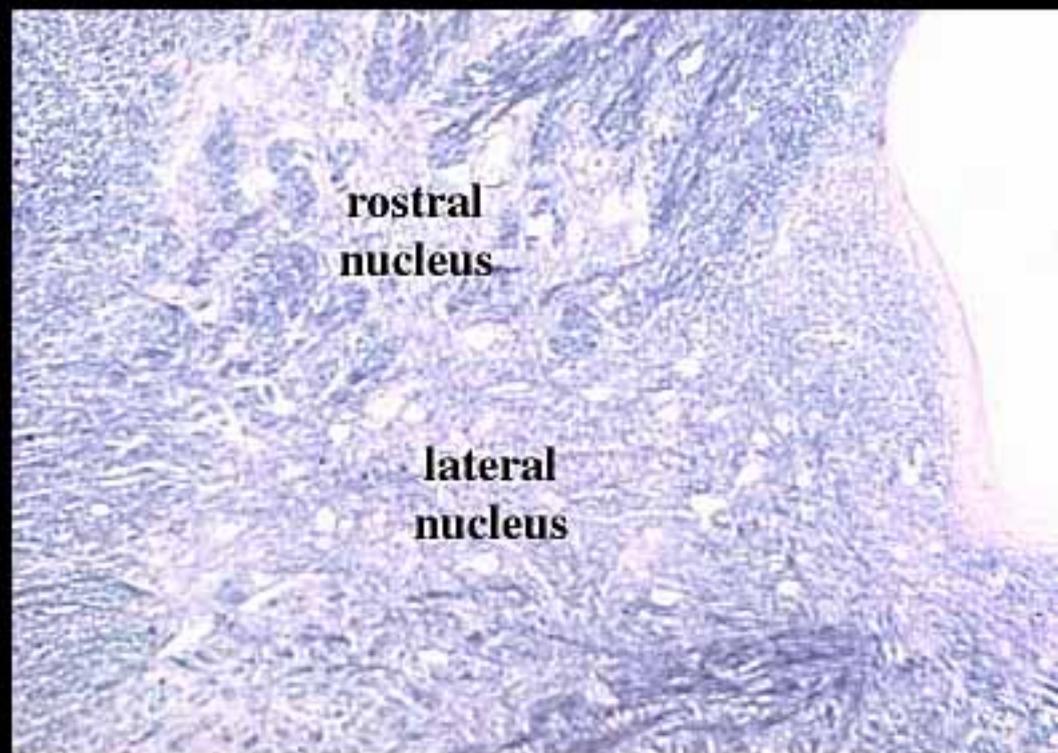
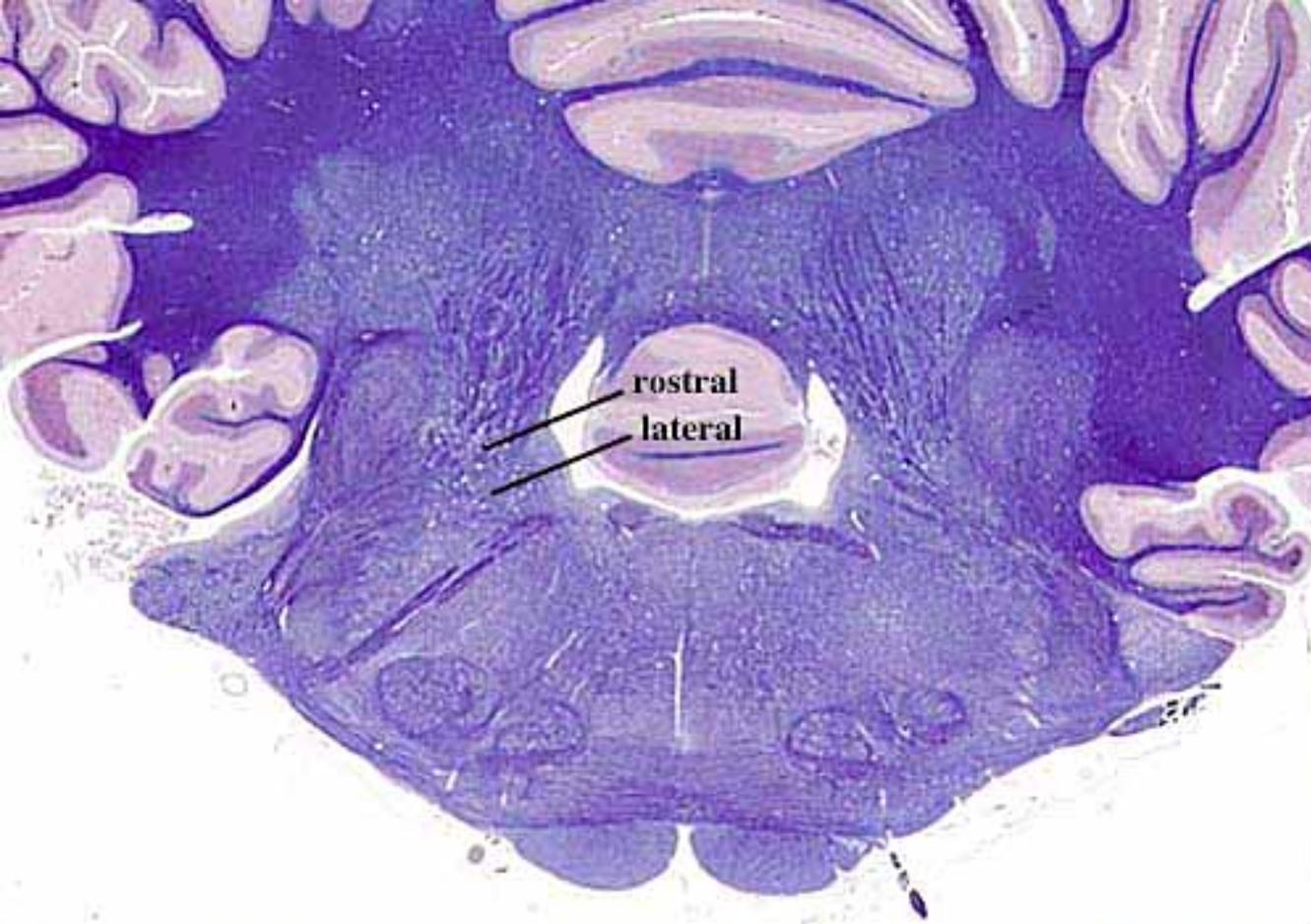
Physiology: spontaneously active neuronal circuits; perform three major functions:

1. Ascending system to alert cerebral cortex (via non-specific thalamic nuclei) vs. coma
2. Vegetative centers: regulate heart rate, respiration, digestion, micturition, etc.
3. Standing posture and muscle tone via two pathways to alpha & gamma neurons:
 - pontine reticulospinal tract — arises from neurons located laterally in pons & medulla
 - dominant and spontaneously active; runs ipsilaterally in the ventral funiculus
 - activates alpha & gamma neurons to extensor muscles of proximal joints
 - medullary reticulospinal tract — arises from neurons located medially in medulla
 - inhibits neurons to extensor mm. & excites neurons to flexor mm.;
 - not spontaneously active — driven by cerebral cortex to preset movement posture
 - descends bilaterally in the lateral funiculus

Vestibular nuclei *discussed previously*

Two descending tracts: lateral vestibulospinal tract— which also drives standing posture, & medial vestibulospinal tract (m.l.f.)— which controls neck muscles.

Note: vestibular nuclei also utilize the two reticulospinal tracts to adjust muscle tone.

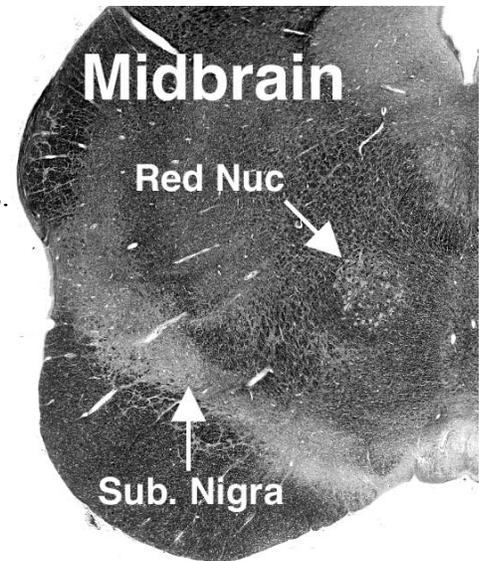


Red nucleus

Large neurons of the red nucleus gives rise to the rubrospinal tract—the principal descending tract for voluntary movement in domestic animals. (Rubrobulbar fibers go to cranial nerve motor nuclei. Other (small) neurons project to the olivary nucleus.)

The red nucleus is merely a collection of projection neurons. Axons from the motor area of cerebral cortex synapse on large projection neurons in the red nucleus and control their activity.

The rubrospinal tract decussates in the midbrain and descends in the dorsal half of the lateral funiculus. Rubrospinal fibers synapse on spinal interneurons and produce independent movements of shoulder/hip; elbow/stifle; and carpus/hock (not digits).



Tectum (*tectum = roof of the midbrain*)

Two tracts arise from deep neurons of the rostral colliculus:

1. tectospinal fibers—descend to the cervical spinal cord (for head turning);
2. tectobulbar fibers—to cranial nerve nuclei that control ear & eye movement. Via horizontal (pons) and vertical (midbrain) gaze centers within the reticular formation, the tectum reflexly shifts the eyes (saccadic movement) to focus on novel stimuli within the visual field. Note: saccade = quick eye movement used to shift focus to new visual feature; perception occurs at stops between saccades.

Substantia nigra

A dorsal region (pars compacta) contains dopaminergic neurons that project to the striatum (caudate nucleus & putamen) to facilitate movement initiation. In primates, the neurons are pigmented and their degeneration results in Parkinson's disease, which features decreased movement activity.

A ventral region (pars reticulata) functions like the globus pallidus (see below). It tonically inhibits brainstem motor nuclei such as those in the tectum. The inhibition is lifted by descending input from the striatum (disinhibition).

Subthalamus

The subthalamic nucleus participates in basal nuclei circuits. The nucleus contains glutamatergic output neurons that excite the globus pallidus which tonically suppresses movement activity. (Physiologically the nucleus generates bursts of neuronal spikes, implying a pacemaker role in rhythmic movements.) Damage to the subthalamus results in hyperkinetic movements such as hemiballismus.

Anatomically, the term "Basal Nuclei" refers to non-cortical gray matter of the telencephalon. There are five major nuclei (and several additional ones).

The major nuclei may be anatomically sub-grouped in different ways:

1] Striatum = caudate & putamen; named for the gray matter striations in the intervening internal capsule.

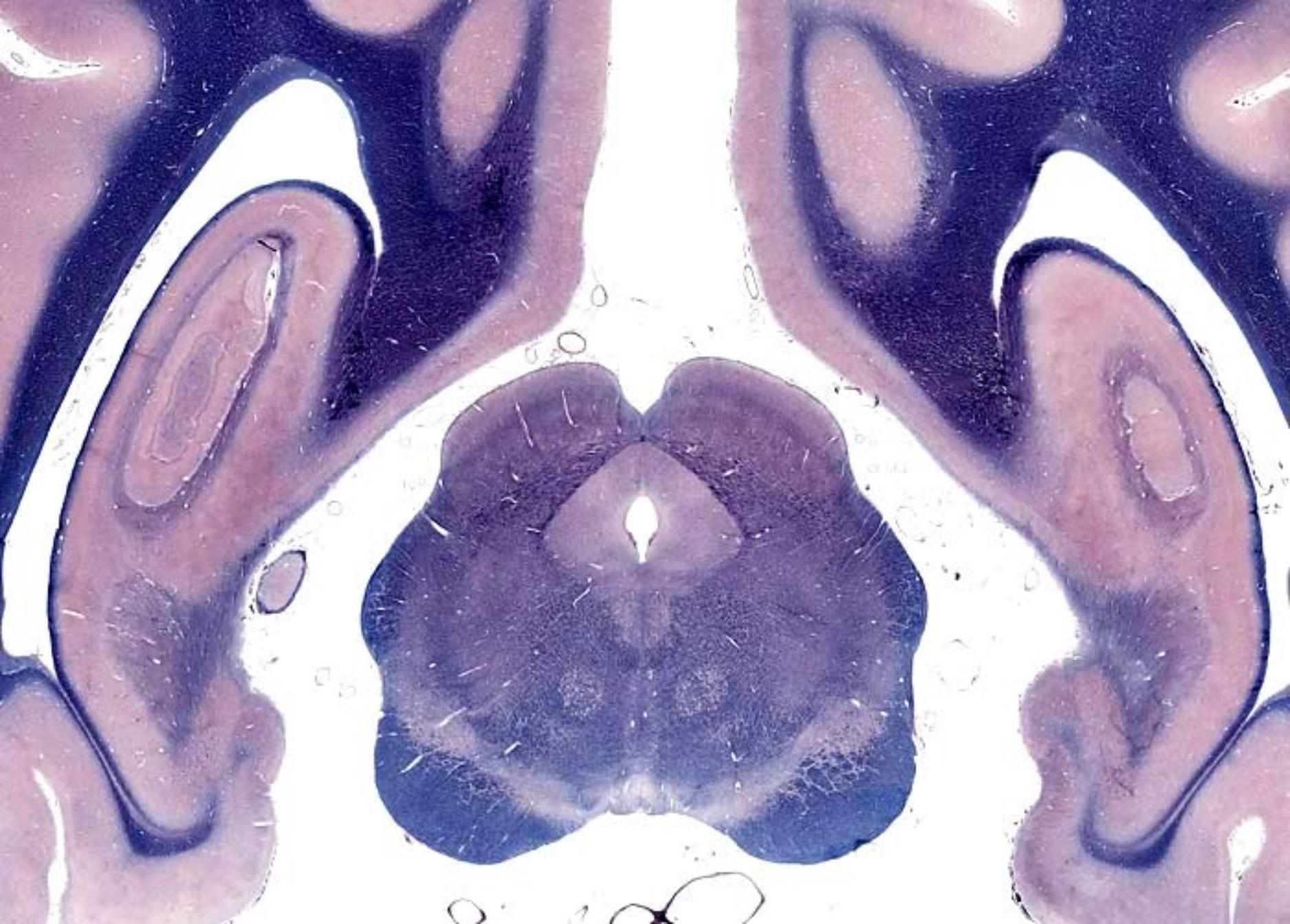
2] Lentiform nucleus = putamen & globus pallidus; together they have the shape of a lens.

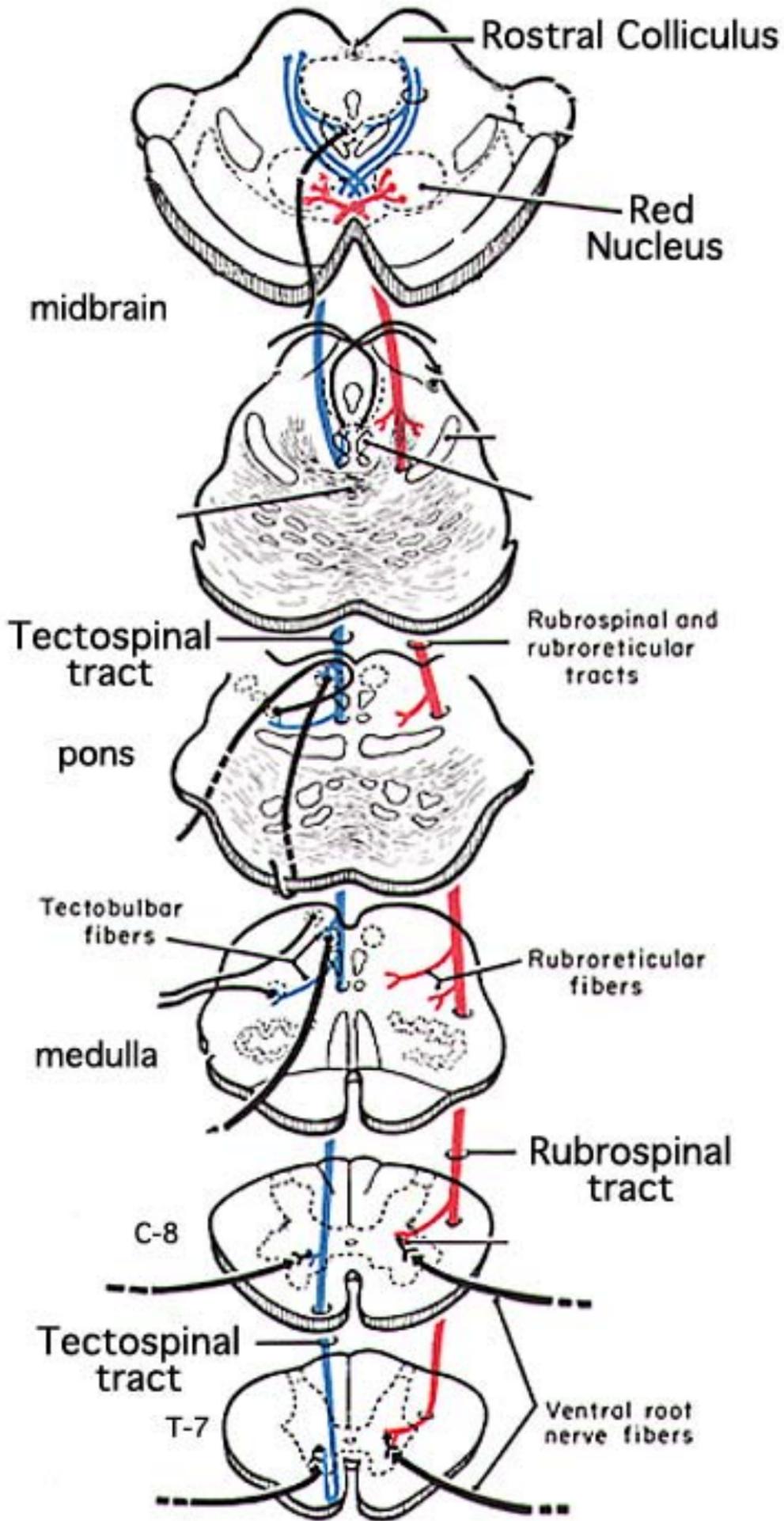
Physiologically, only caudate, putamen, and globus pallidus play a motor role. They participate in a circuit that includes two brainstem nuclei: substantia nigra and subthalamus.

**Lentiform
Nucleus**

Caudate
Putamen
Globus Pallidus
Amygdaloid
Clastrum
etc.

Striatum



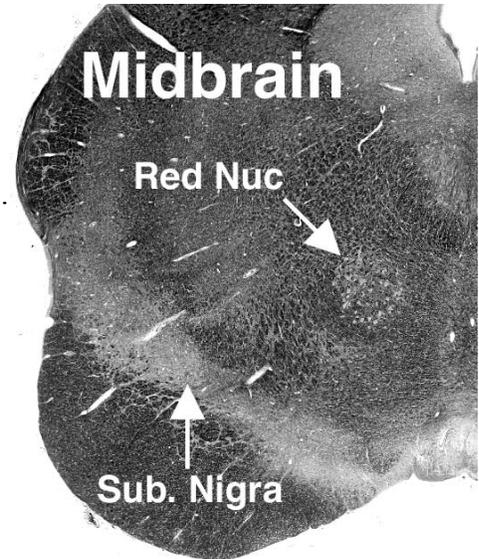


Red nucleus

Large neurons of the red nucleus gives rise to the rubrospinal tract—the principal descending tract for voluntary movement in domestic animals. (Rubrobulbar fibers go to cranial nerve motor nuclei. Other (small) neurons project to the olivary nucleus.)

The red nucleus is merely a collection of projection neurons. Axons from the motor area of cerebral cortex synapse on large projection neurons in the red nucleus and control their activity.

The rubrospinal tract decussates in the midbrain and descends in the dorsal half of the lateral funiculus. Rubrospinal fibers synapse on spinal interneurons and produce independent movements of shoulder/hip; elbow/stifle; and carpus/hock (not digits).



Tectum (*tectum = roof of the midbrain*)

Two tracts arise from deep neurons of the rostral colliculus:

1. tectospinal fibers—descend to the cervical spinal cord (for head turning);
2. tectobulbar fibers—to cranial nerve nuclei that control ear & eye movement. Via horizontal (pons) and vertical (midbrain) gaze centers within the reticular formation, the tectum reflexly shifts the eyes (saccadic movement) to focus on novel stimuli within the visual field. Note: saccade = quick eye movement used to shift focus to new visual feature; perception occurs at stops between saccades.

Substantia nigra

A dorsal region (pars compacta) contains dopaminergic neurons that project to the striatum (caudate nucleus & putamen) to facilitate movement initiation. In primates, the neurons are pigmented and their degeneration results in Parkinson’s disease, which features decreased movement activity.

A ventral region (pars reticulata) functions like the globus pallidus (see below). It tonically inhibits brainstem motor nuclei such as those in the tectum. The inhibition is lifted by descending input from the striatum (disinhibition).

Subthalamus

The subthalamic nucleus participates in basal nuclei circuits. The nucleus contains glutamatergic output neurons that excite the globus pallidus which tonically suppresses movement activity. (Physiologically the nucleus generates bursts of neuronal spikes, implying a pacemaker role in rhythmic movements.) Damage to the subthalamus results in hyperkinetic movements such as hemiballismus.

Anatomically, the term "Basal Nuclei" refers to non-cortical gray matter of the telencephalon. There are five major nuclei (and several additional ones).

The major nuclei may be anatomically sub-grouped in different ways:

- 1] Striatum = caudate & putamen; named for the gray matter striations in the intervening internal capsule.
- 2] Lentiform nucleus = putamen & globus pallidus; together they have the shape of a lens.

Physiologically, only caudate, putamen, and globus pallidus play a motor role. They participate in a circuit that includes two brainstem nuclei: substantia nigra and subthalamus.

Lentiform Nucleus

Caudate

Putamen

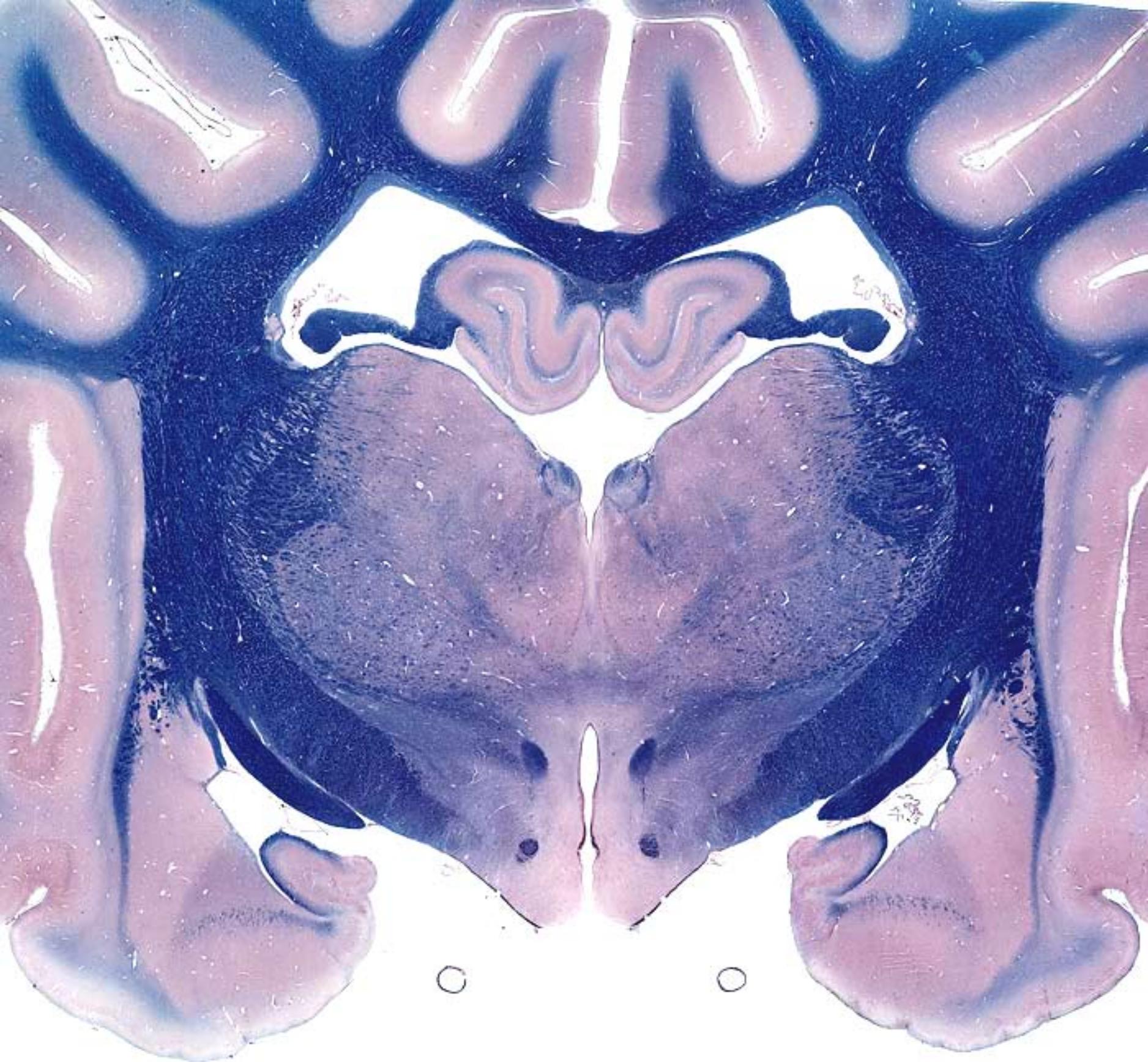
Globus Pallidus

Amygdaloid

Clastrum

etc.

Striatum

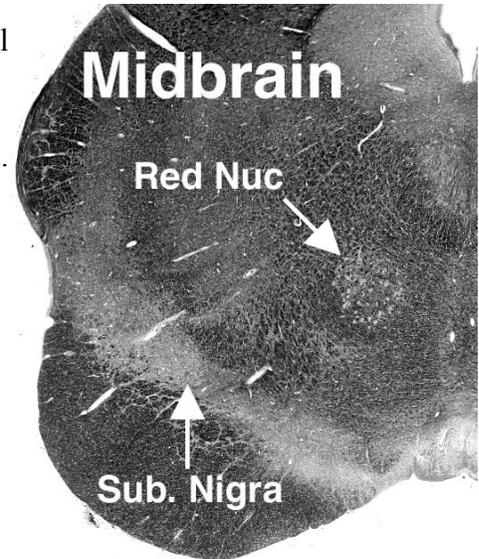


Red nucleus

The nucleus gives rise to the rubrospinal tract—the principal descending tract for voluntary movement in domestic animals. (Rubrobulbar fibers go to cranial nerve motor nuclei.)

The red nucleus is merely a collection of projection neurons. Axons from the motor area of cerebral cortex synapse on neurons of the red nucleus and control their activity.

The rubrospinal tract decussates in the midbrain and descends in the dorsal half of the lateral funiculus. Rubrospinal fibers synapse on spinal interneurons and produce independent movements of shoulder/hip; elbow/stifle; and carpus/hock (not digits).



Tectum (*tectum = roof of the midbrain*)

Rostral & caudal colliculi give rise to two tracts

(which arise from rostral colliculus):

1. tectospinal fibers—descend to the cervical spinal cord (for head turning);
2. tectobulbar fibers—to cranial nerve nuclei that control ear & eye movement.

Substantia nigra

A dorsal region (pars compacta) contains dopoaminergic neurons that project to the striatum (caudate nucleus & putamen). In primates, the neurons are pigmented and their degeneration results in Parkinson’s disease. Ventral (pars reticulata) and lateral regions contain pacemaker neurons that receive input from the striatum. The neurons send output to the thalamus; also, they inhibit the pars compacta.

Subthalamus

The subthalamic nucleus participates in basal nuclei circuits and thus functions in movement regulation. (The nucleus contains glutamatergic output neurons and GABA interneurons.) Physiologically the nucleus generates bursts of neuronal spikes that imply a pacemaker role in rhythmic movements. In primates, the subthalamus is hyperactive in Parkinson’s disease and lesions of the subthalamus result in hemiballismus.

Basal nuclei (Basal Ganglia)

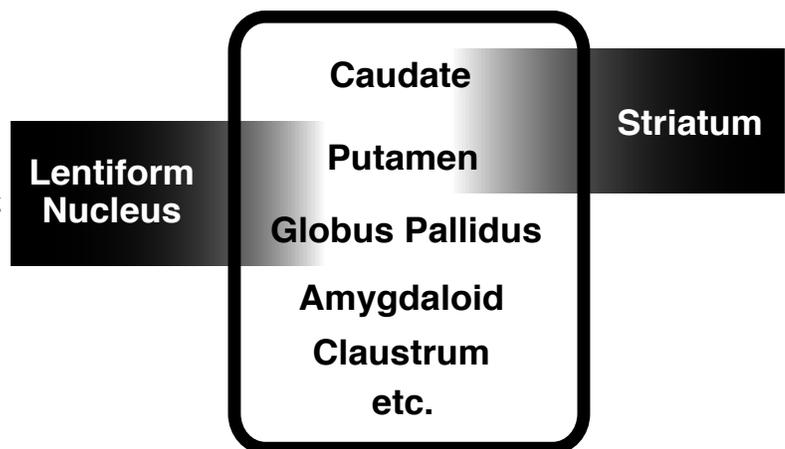
Anatomically, the term "Basal Nuclei" refers to non-cortical gray matter of the telencephalon. There are five major nuclei (and several smaller ones).

The major nuclei may be anatomically subgrouped in different ways:

1] Striatum (caudate & putamen; named for the gray matter striations in the intervening internal capsule).

2] Lentiform nucleus (putamen & globus pallidus; together they have the shape of a lens).

Physiologically, only caudate, putamen, and globus pallidus play a motor role, which involves the substantia nigra and subthalamus as well.



**Lentiform
Nucleus**

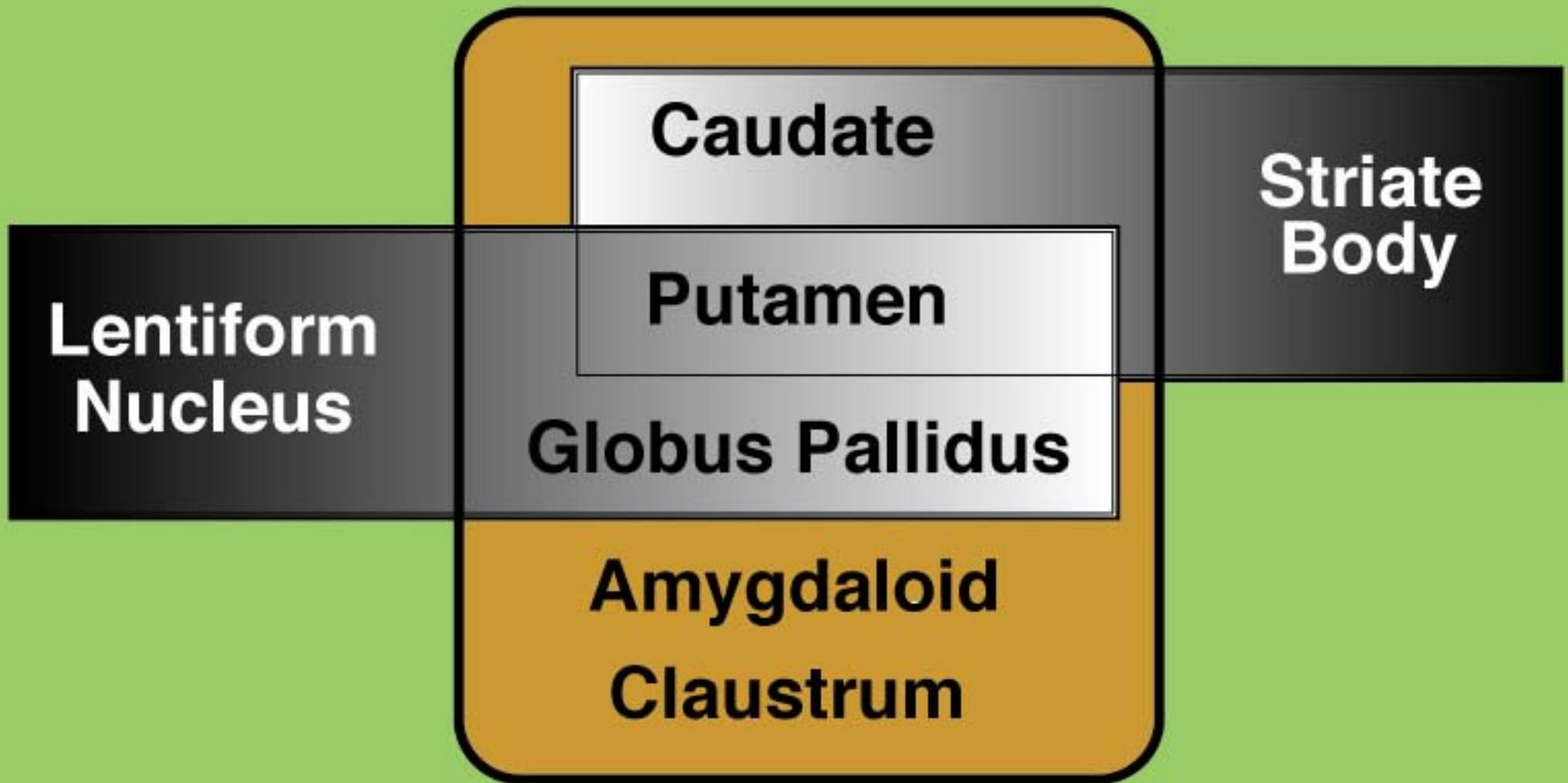
Caudate

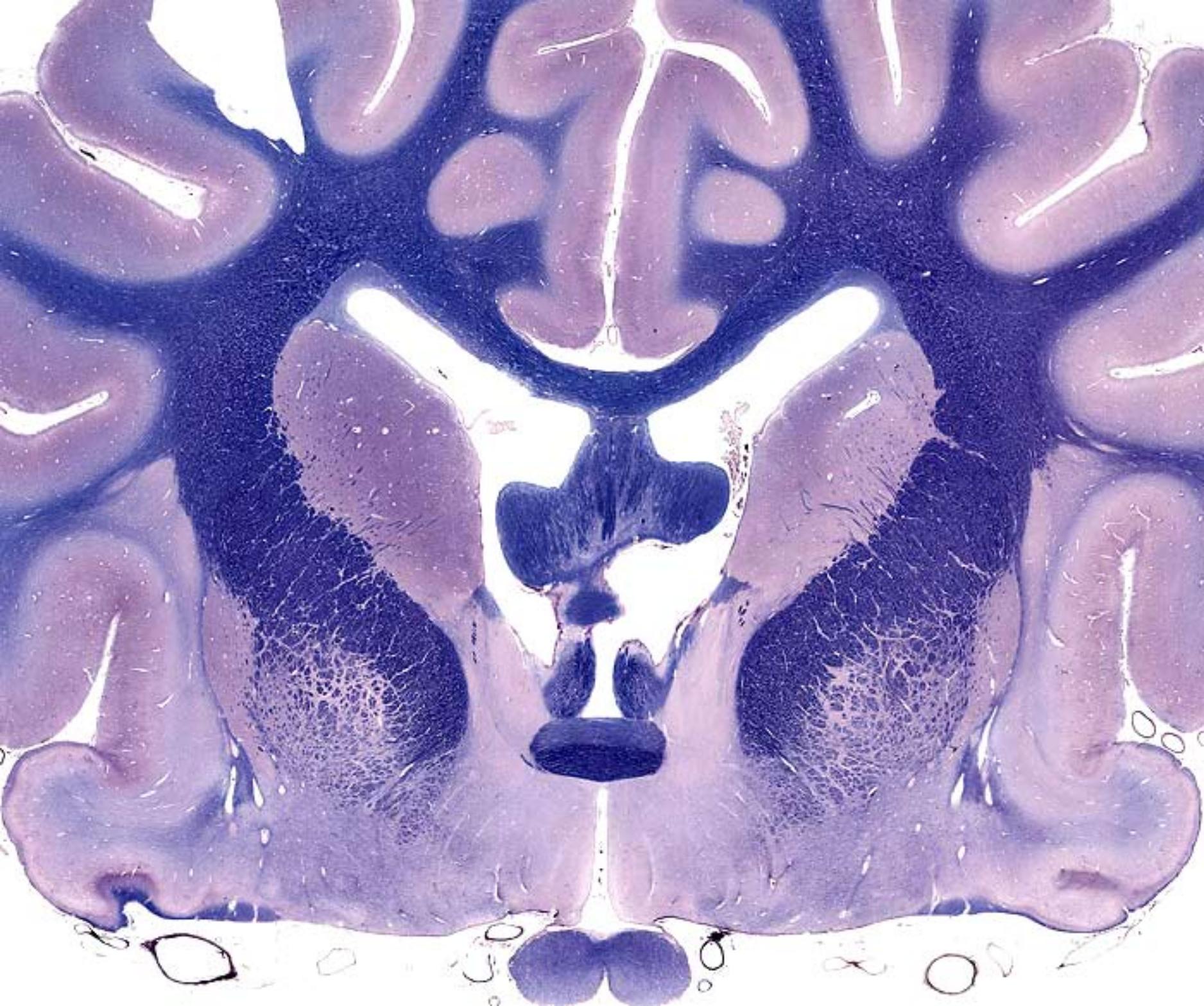
Putamen

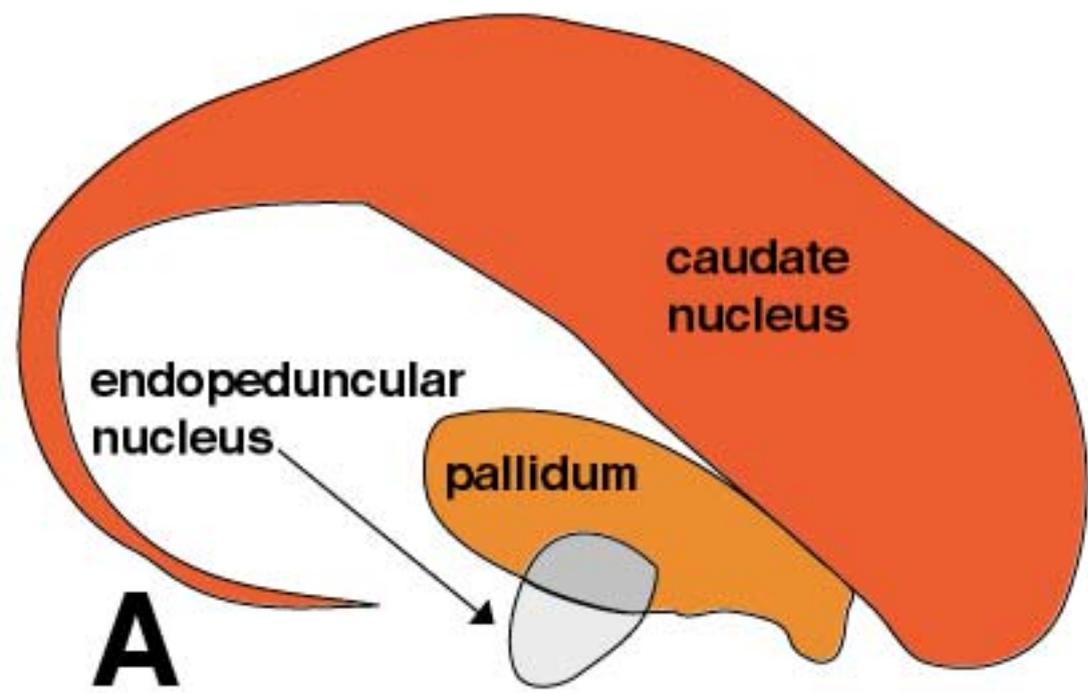
Globus Pallidus

**Amygdaloid
Claustrum**

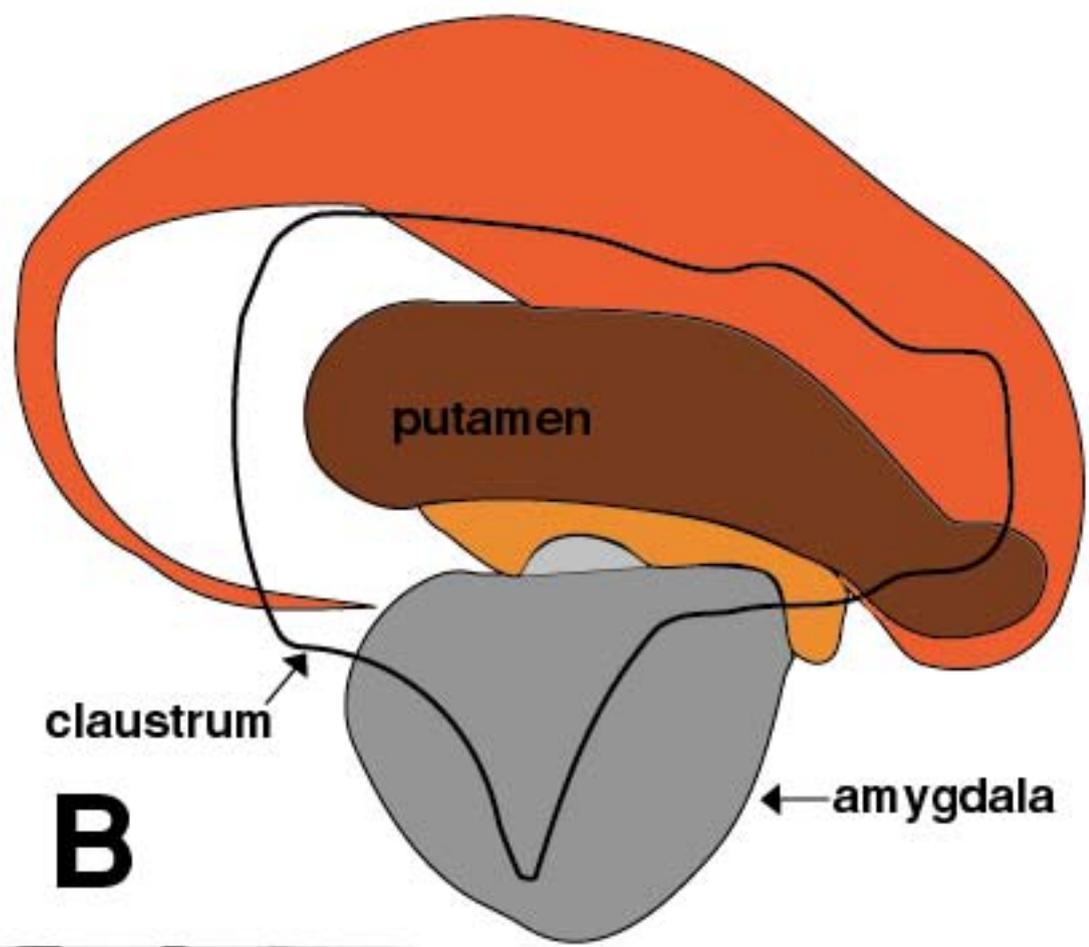
**Striate
Body**



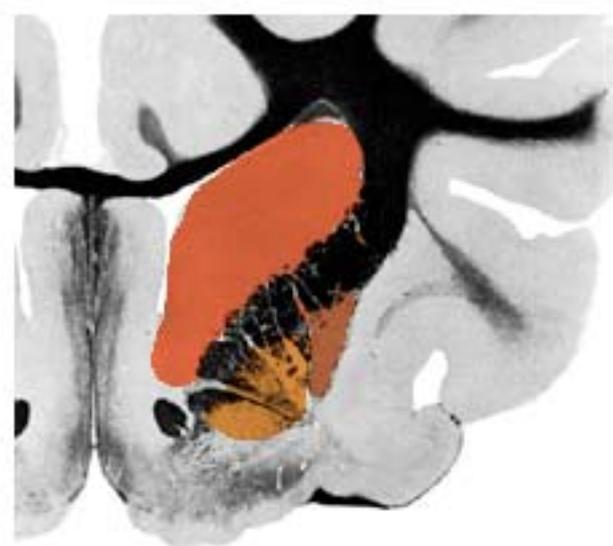
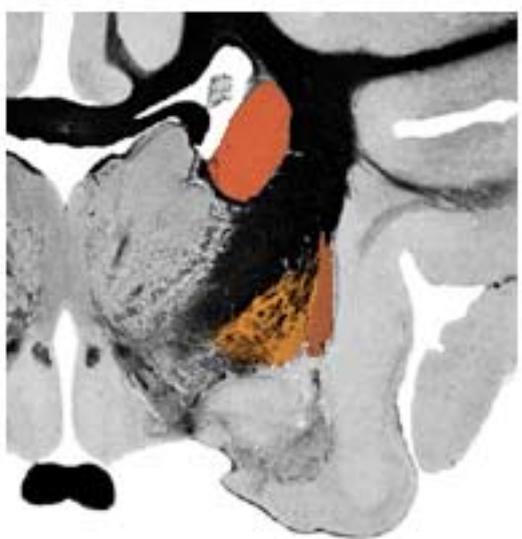




A



B



Basal nuclei (Basal Ganglia)

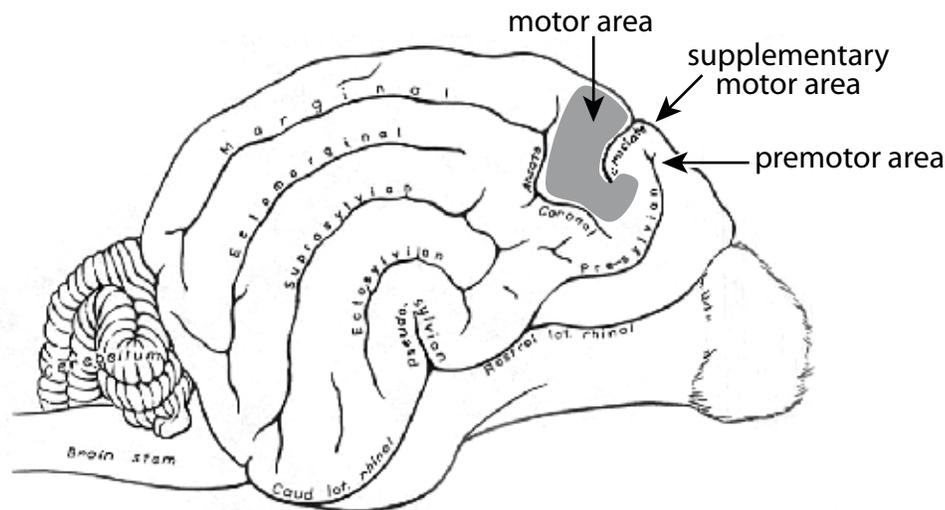
Although they do not give rise to descending tracts, basal nuclei regulate voluntary movement onset and cessation. They facilitate initiation of selected movements and suppression of non-selected movements. They participate in a circuit interposed between non-motor cortex precipitating the movement and motor cortex executing the movement.

The non-motor areas of cerebral cortex do not communicate their movement needs directly to the motor cortex. Instead the cortices are linked by a circuit involving basal nuclei and thalamus, including also subthalamus and substantia nigra (see following Basal Nuclei Circuit diagram).

In general the globus pallidus is tonically active in suppressing movement. Excitation of the globus pallidus by the subthalamus facilitates movement suppression. When non-motor cerebral cortex excites the striate body, the caudate and putamen specifically inhibit neurons in the globus pallidus (and subthalamus). This specific disinhibition enables movement initiation, by releasing excitatory thalamic neurons.

Functionally, there seems to be two separate circuits: one, involving caudate nucleus is active in selecting & assembling movements; the other, involving putamen regulates amplitudes & durations of movements.

Damage to basal nuclei impairs movement onset (hypokinetic syndromes) or movement cessation (hyperkinetic syndromes). Such dyskinesias are more evident in primates and people. In domestic animals, lesions of the basal nuclei produce circling toward the damaged side, pacing, and muscle hypertonia.

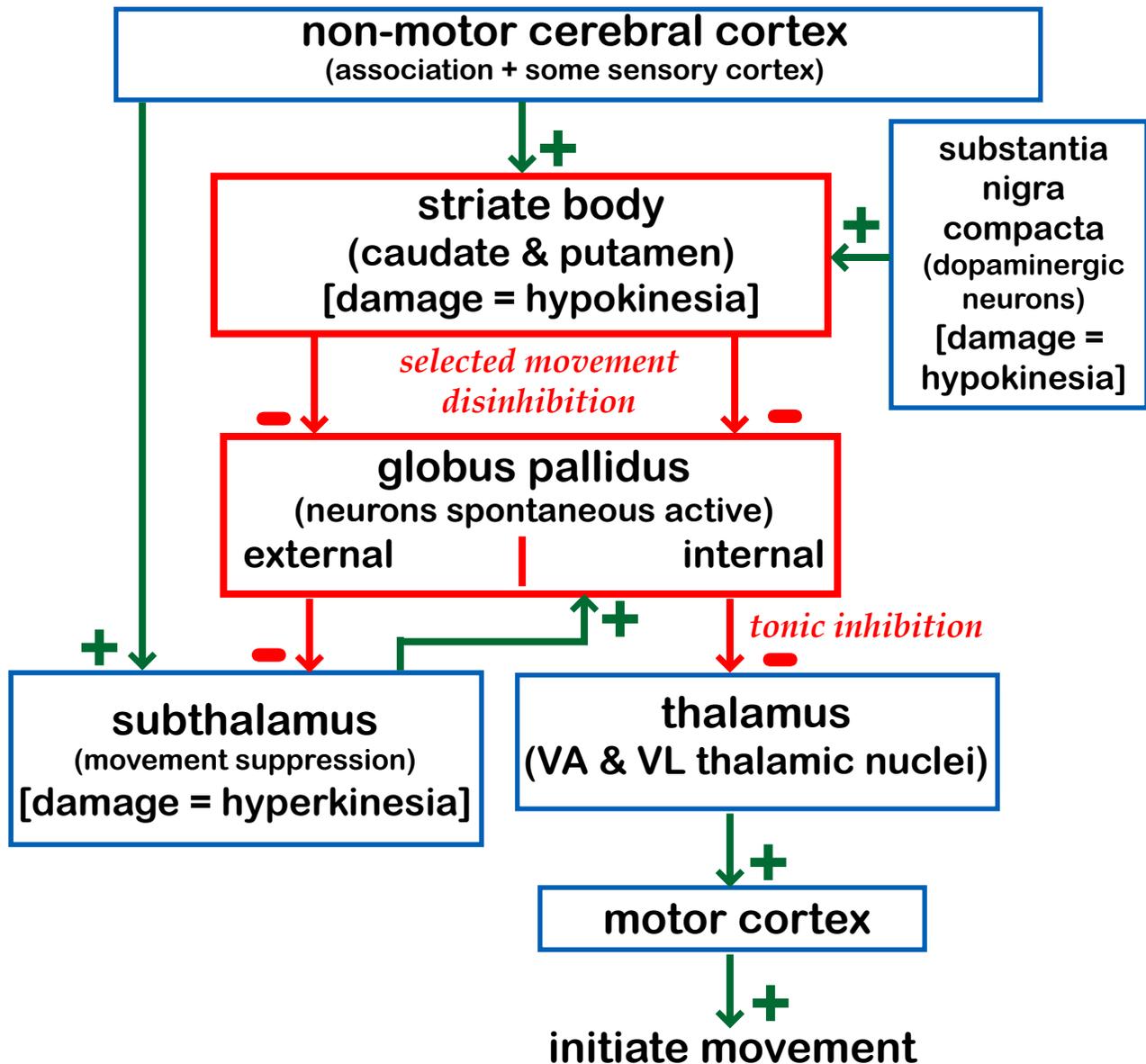


Cerebral cortex

The motor area of the cerebral cortex executes voluntary movement; other motor-related areas of cortex are involved in planing and selecting voluntary movements in response to cues:

- *motor area*—located around the cruciate sulcus; principal source of output for all voluntary movement; it is the main origin of two descending pathway systems: a direct pyramidal & an indirect extrapyramidal (see below). Each output neuron of the motor cortex innervated multiple muscles (movement organization).
- *premotor area*—located in frontal lobe rostral to the motor area; required for generating learned, rapid-sequence movements (motor pattern generator circuits).
- *supplementary motor area*—located medial to premotor area; active when thinking about a proposed movement; projects to the motor area

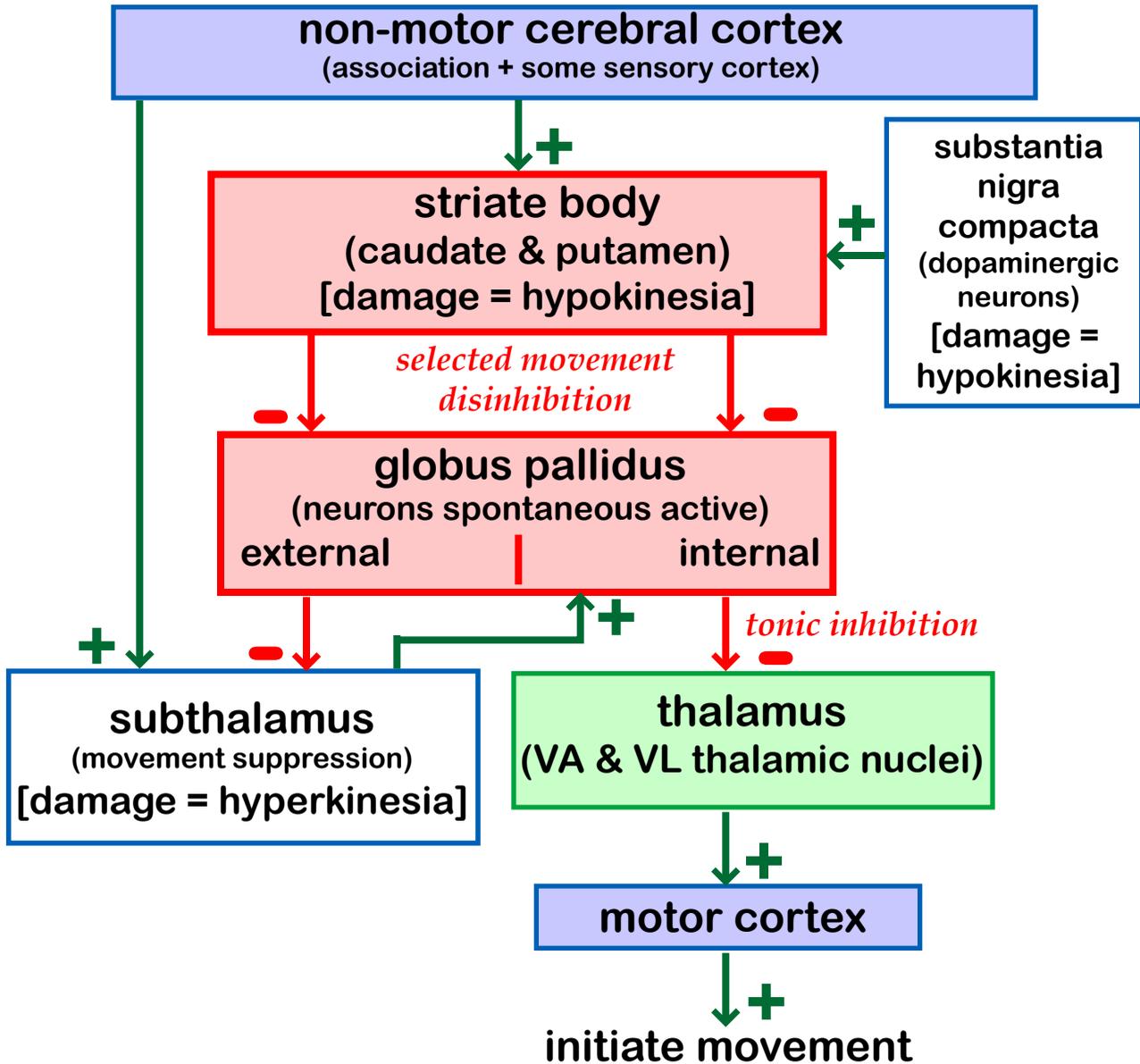
Basal Nuclei Circuit

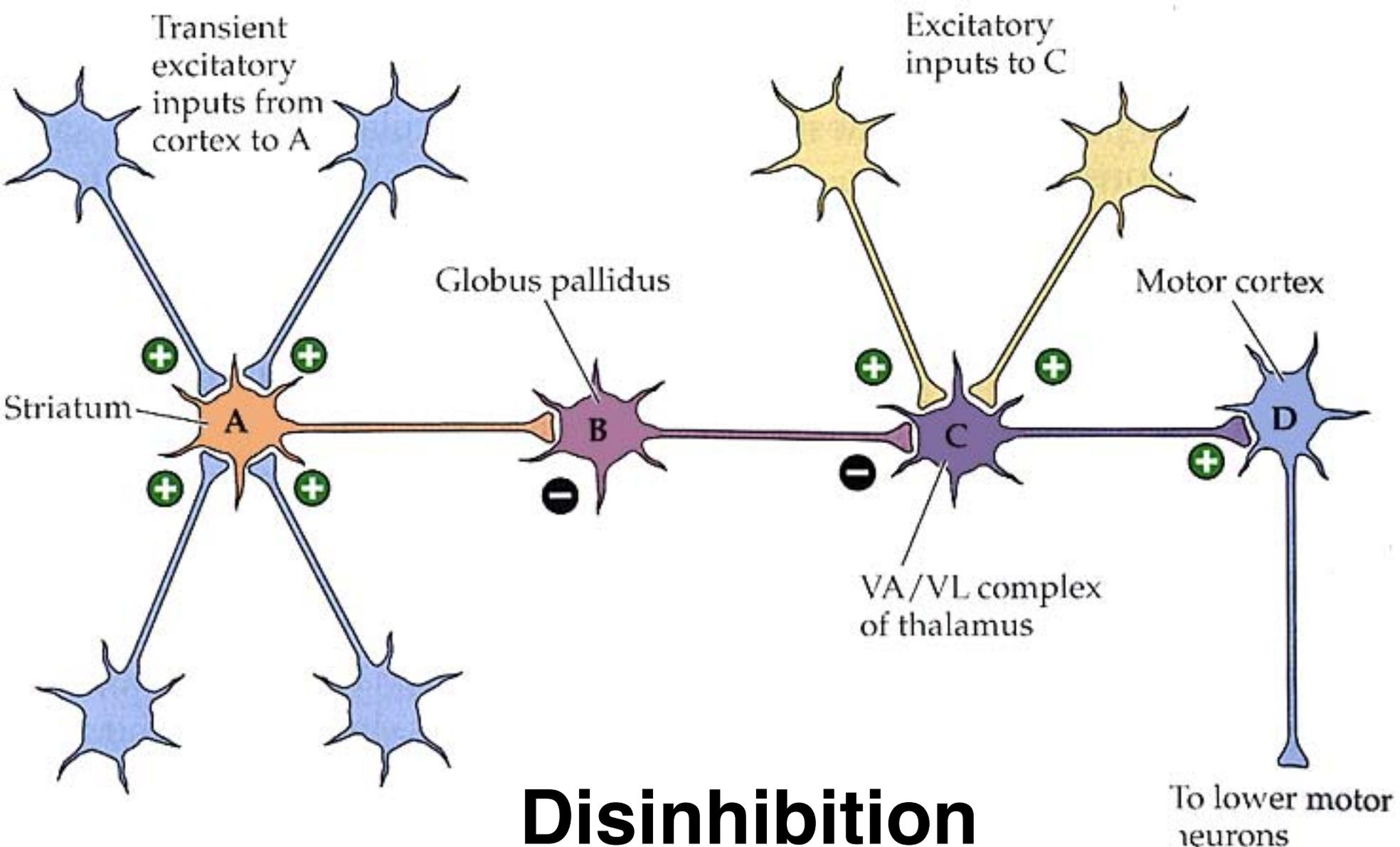


Notes:

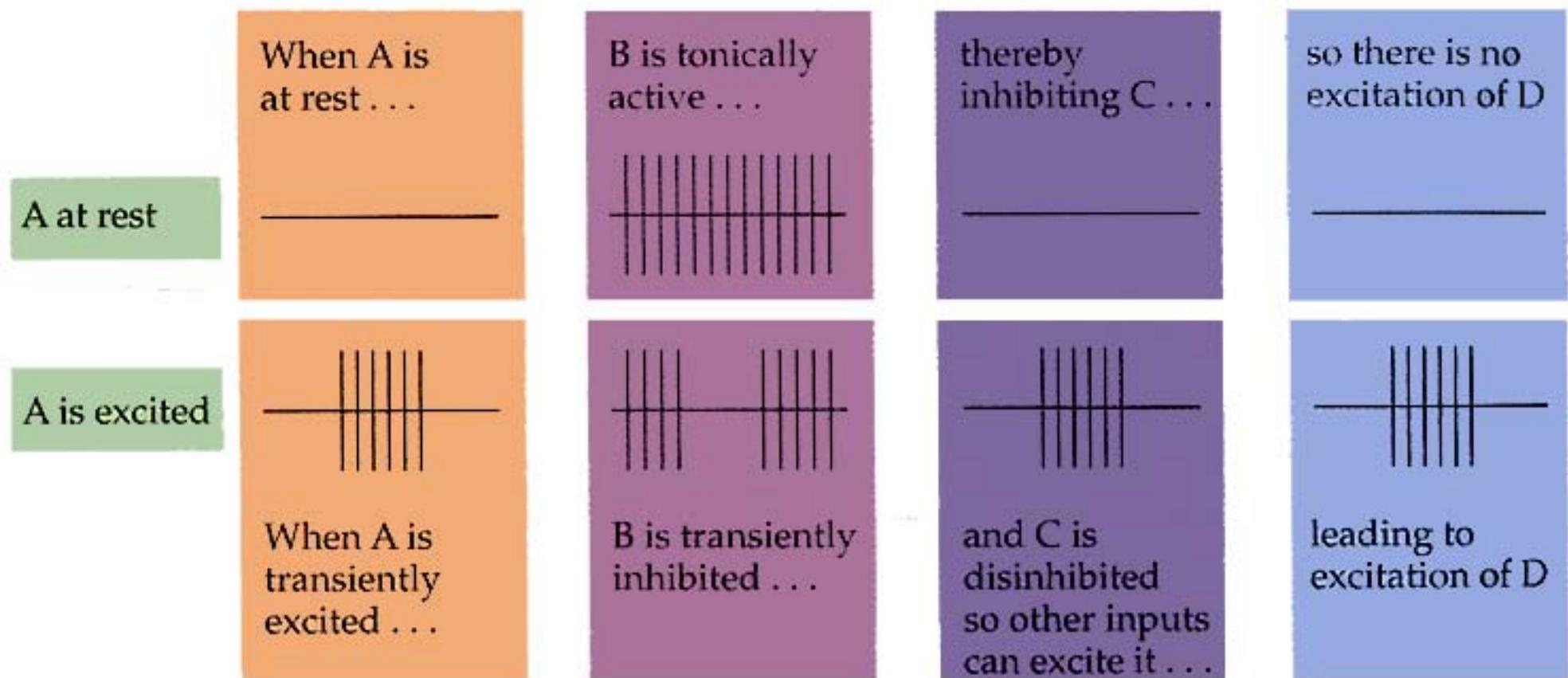
- the non-motor cerebral cortex communicates movement needs to the motor cortex via basal nuclei - thalamic circuits.
- tonic activity of the internal globus pallidus suppresses movements in general; re-inforced by the subthalamus, this inhibition suppresses non-selected movements.
- selected movements are allowed (initiated) by a process termed selective disinhibition, as cerebral cortex selectively excites inhibitory neurons of the striatum that selectively inhibit tonic neurons of the globus pallidus.
- inhibitory neurons (-) are GABAergic; excitatory neurons (+) are glutamatergic; substantia nigra dopaminergic neurons are excitatory via D_1 receptors.

Basal Nuclei Circuit





Disinhibition



Basal nuclei (Basal Ganglia)

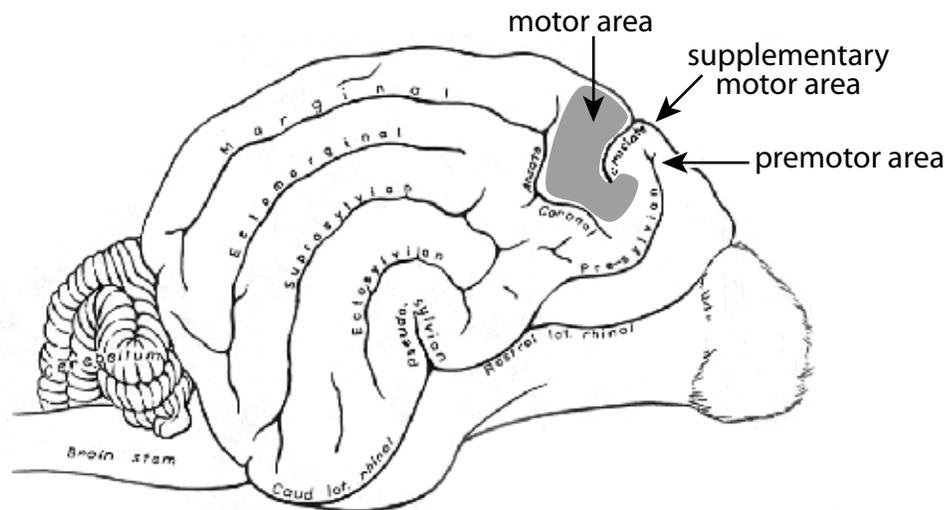
Although they do not give rise to descending tracts, basal nuclei regulate voluntary movement onset and cessation. They facilitate initiation of selected movements and suppression of non-selected movements. They participate in a circuit interposed between non-motor cortex precipitating the movement and motor cortex executing the movement.

The non-motor areas of cerebral cortex do not communicate their movement needs directly to the motor cortex. Instead the cortices are linked by a circuit involving basal nuclei and thalamus, including also subthalamus and substantia nigra (see following Basal Nuclei Circuit diagram).

In general the globus pallidus is tonically active in suppressing movement. Excitation of the globus pallidus by the subthalamus facilitates movement suppression. When non-motor cerebral cortex excites the striate body, the caudate and putamen specifically inhibit neurons in the globus pallidus (and subthalamus). This specific disinhibition enables movement initiation, by releasing excitatory thalamic neurons.

Functionally, there seems to be two separate circuits: one, involving caudate nucleus is active in selecting & assembling movements; the other, involving putamen regulates amplitudes & durations of movements.

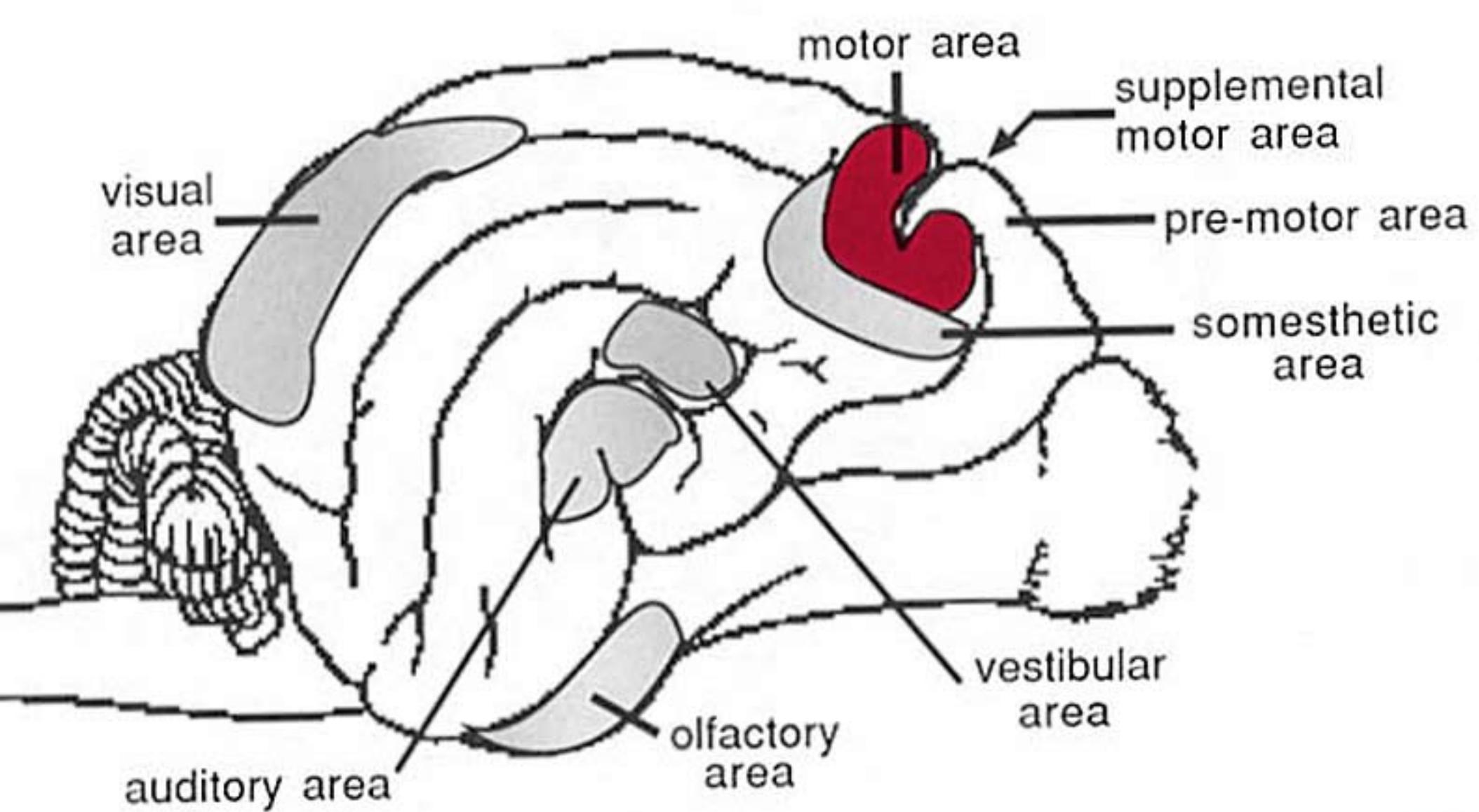
Damage to basal nuclei impairs movement onset (hypokinetic syndromes) or movement cessation (hyperkinetic syndromes). Such dyskinesias are more evident in primates and people. In domestic animals, lesions of the basal nuclei produce circling toward the damaged side, pacing, and muscle hypertonia.



Cerebral cortex

The motor area of the cerebral cortex executes voluntary movement; other motor-related areas of cortex are involved in planing and selecting voluntary movements in response to cues:

- *motor area*—located around the cruciate sulcus; principal source of output for all voluntary movement; it is the main origin of two descending pathway systems: a direct pyramidal & an indirect extrapyramidal (see below). Each output neuron of the motor cortex innervated multiple muscles (movement organization).
- *premotor area*—located in frontal lobe rostral to the motor area; required for generating learned, rapid-sequence movements (motor pattern generator circuits).
- *supplementary motor area*—located medial to premotor area; active when thinking about a proposed movement; projects to the motor area



Descending pathways for voluntary movement fall into two categories:

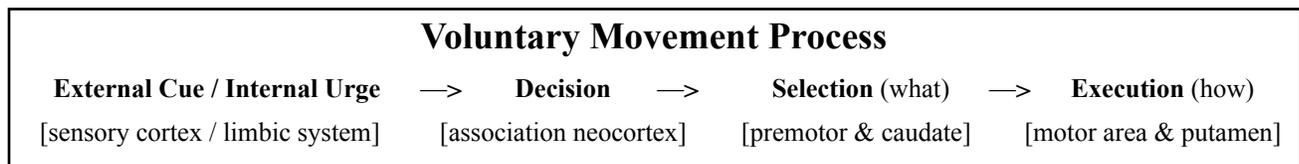
1] **Pyramidal tract** = a direct connection from motor and premotor areas of cerebral cortex to efferent neurons, generally via local interneurons. Axons travel in pyramids of the medulla oblongata.

Most axons (85% in human) decussate at the medullary-spinal junction and form the lateral corticospinal tract which terminates in lateral motor nuclei of ventral horn enlargements. The remaining axons form the ventral corticospinal tract which crosses at the level of termination in the spinal cord and innervates medial motor nuclei (postural muscles). Thus, the pyramidal tract controls particularly musculature of the manus and pes; it is especially concerned with precise movements of individual digits (also lips and tongue).

Note: Some corticospinal axons affect projection neurons of ascending pathways to enable the cerebral cortex to modify sensory traffic on its way to the thalamus and cortex. These axons come from sensory areas of the cortex.

2] **Extrapyramidal tracts** = the term applied to all other (non-pyramidal) voluntary movement tracts. Under the direction of cerebral cortex (motor area), these tracts control proximal musculature and thus generate relatively coarse components of posture/movement/locomotion. Naturally, this system is most important in domestic animals. The principal extrapyramidal tracts are: rubrospinal tract, pontine reticulospinal tract, and medullary reticulospinal tract.

Note: a specific voluntary movement, e.g., a feline paw swipe involving rubrospinal or pyramidal tracts, requires an associated postural adjustment (involving reticulospinal or rubrospinal tracts) that often must precede the specific action. Thus voluntary movements require multiple tracts.



Veterinary Clinical Considerations:

Upper Motor Neuron Damage: (in order of mild to severe deficits)

Loss of only pyramidal tract: paresis (partial paralysis or weakness) of manus & pes; inability to move digits and lips independently & rapidly; deficient tactile placing evident at curbs & on stairs; clinical hopping deficits.

Loss of motor cortex: disappearance of learned movement skills; spastic paralysis (absence of voluntary movement capability, plus hypertonic limb extension due to release of pontine reticular formation suppression).

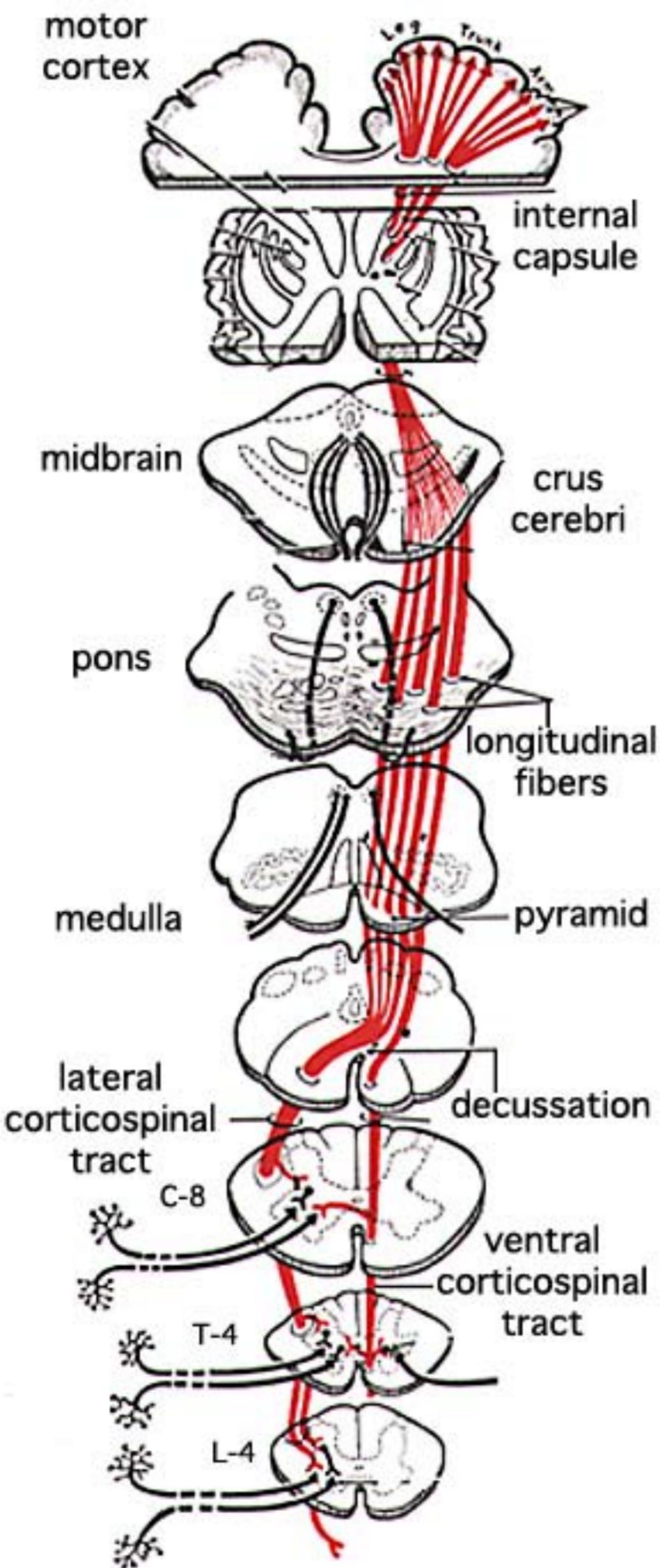
Loss of whole forebrain (= midbrain animal): persistent standing posture but could exhibit phasic actions (sitting, stepping, etc.) if prodded to do so; capable of righting reactions to restore standing posture.

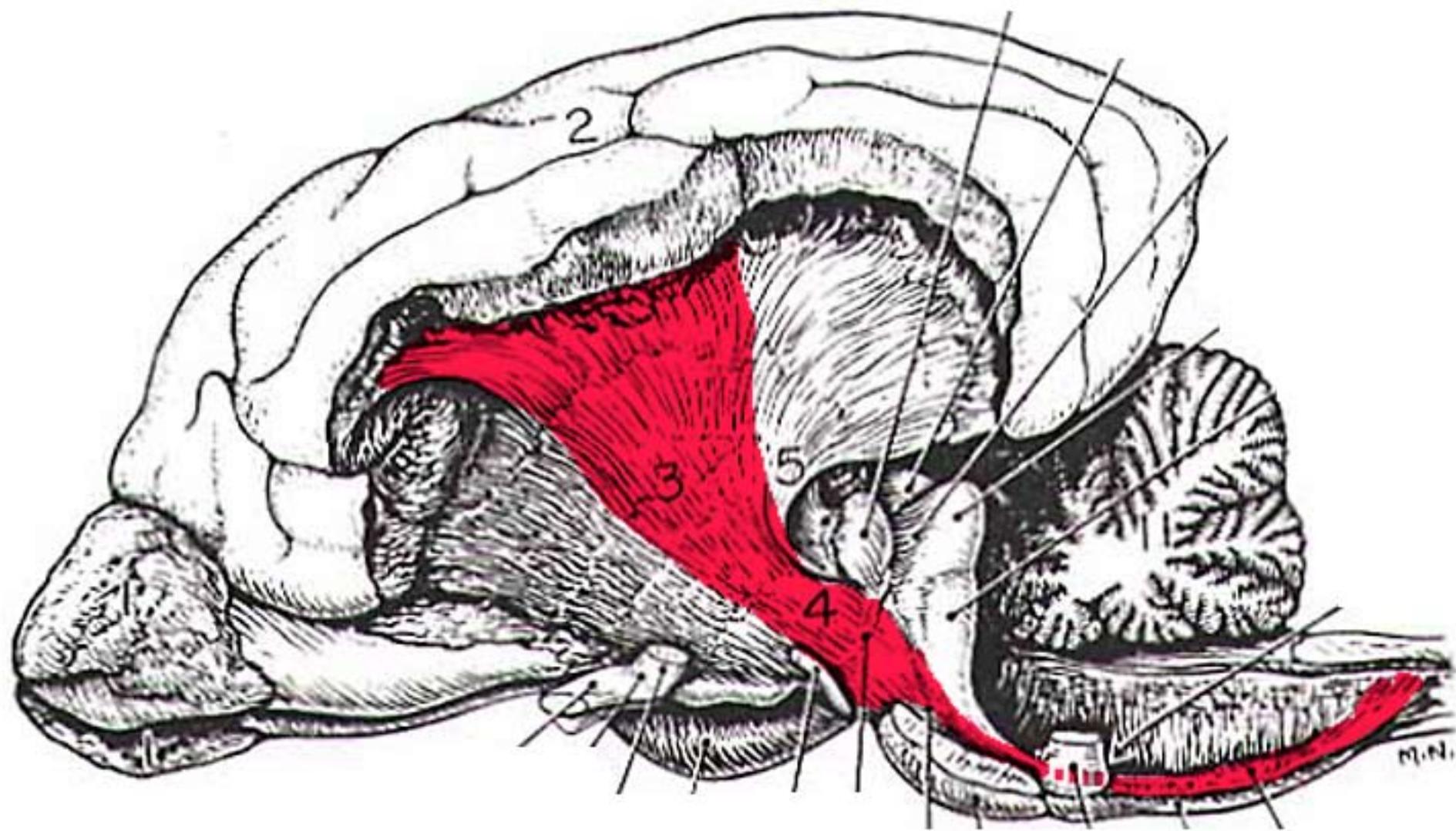
Loss of forebrain & midbrain (= hindbrain animal): limbs rigidly extended constantly in a "saw-horse" attitude (decerebrate rigidity); no locomotion or righting capability; tonic neck reflexes present (postural adjustments initiated by neck proprioceptors).

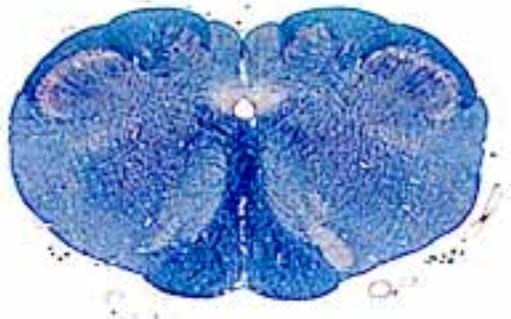
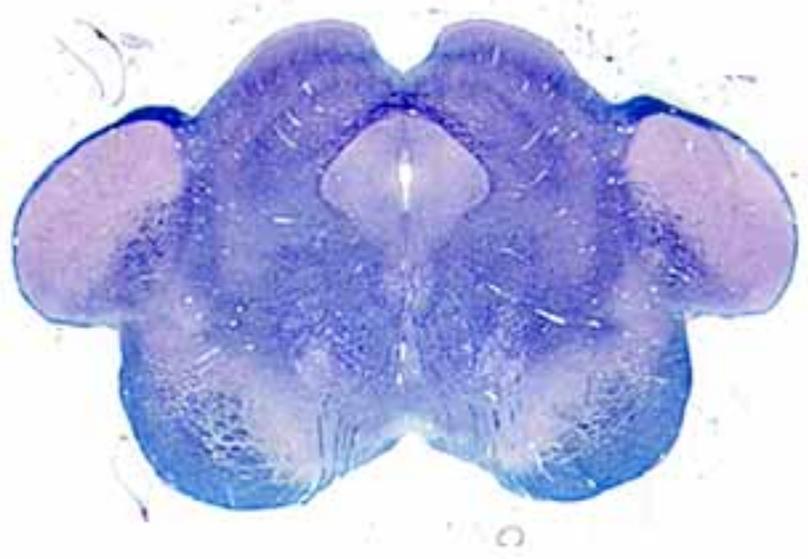
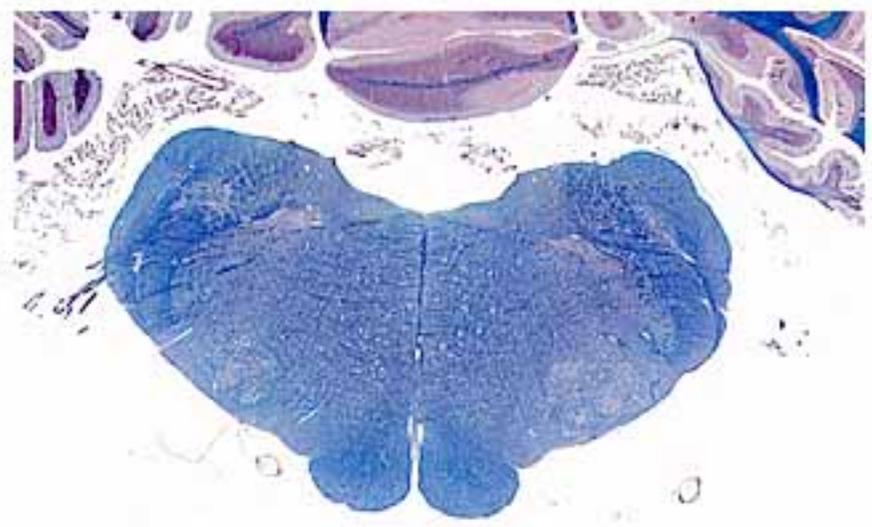
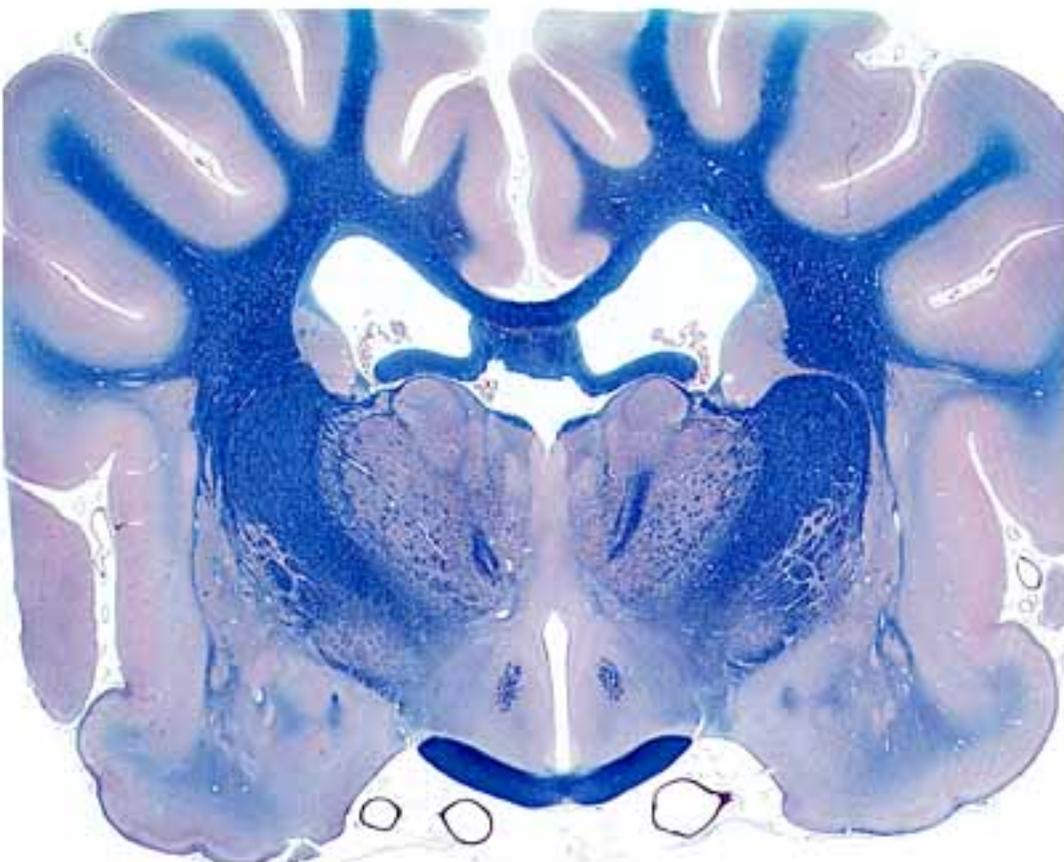
Loss of whole brain (= spinal animal): temporary areflexia may be evident with abrupt injury (spinal shock); paralysis without spasticity; local spinal reflexes intact; crossed extension accompanies the withdrawal reflex.

Lower Motor Neuron Damage:

Spinal cord or peripheral nerve damage: paralysis and areflexia (flaccid paralysis); denervation atrophy of skeletal muscles with time.







Descending pathways for voluntary movement fall into two categories:

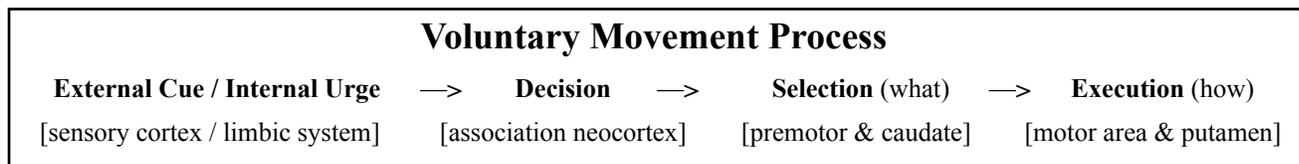
1] **Pyramidal tract** = a direct connection from motor and premotor areas of cerebral cortex to efferent neurons, generally via local interneurons. Axons travel in pyramids of the medulla oblongata.

Most axons (85% in human) decussate at the medullary-spinal junction and form the lateral corticospinal tract which terminates in lateral motor nuclei of ventral horn enlargements. The remaining axons form the ventral corticospinal tract which crosses at the level of termination in the spinal cord and innervates medial motor nuclei (postural muscles). Thus, the pyramidal tract controls particularly musculature of the manus and pes; it is especially concerned with precise movements of individual digits (also lips and tongue).

Note: Some corticospinal axons affect projection neurons of ascending pathways to enable the cerebral cortex to modify sensory traffic on its way to the thalamus and cortex. These axons come from sensory areas of the cortex.

2] **Extrapyramidal tracts** = the term applied to all other (non-pyramidal) voluntary movement tracts. Under the direction of cerebral cortex (motor area), these tracts control proximal musculature and thus generate relatively coarse components of posture/movement/locomotion. Naturally, this system is most important in domestic animals. The principal extrapyramidal tracts are: rubrospinal tract, pontine reticulospinal tract, and medullary reticulospinal tract.

Note: a specific voluntary movement, e.g., a feline paw swipe involving rubrospinal or pyramidal tracts, requires an associated postural adjustment (involving reticulospinal or rubrospinal tracts) that often must precede the specific action. Thus voluntary movements require multiple tracts.



Veterinary Clinical Considerations:

Upper Motor Neuron Damage: (in order of mild to severe deficits)

Loss of only pyramidal tract: paresis (partial paralysis or weakness) of manus & pes; inability to move digits and lips independently & rapidly; deficient tactile placing evident at curbs & on stairs; clinical hopping deficits.

Loss of motor cortex: disappearance of learned movement skills; spastic paralysis (absence of voluntary movement capability, plus hypertonic limb extension due to release of pontine reticular formation suppression).

Loss of whole forebrain (= midbrain animal): persistent standing posture but could exhibit phasic actions (sitting, stepping, etc.) if prodded to do so; capable of righting reactions to restore standing posture.

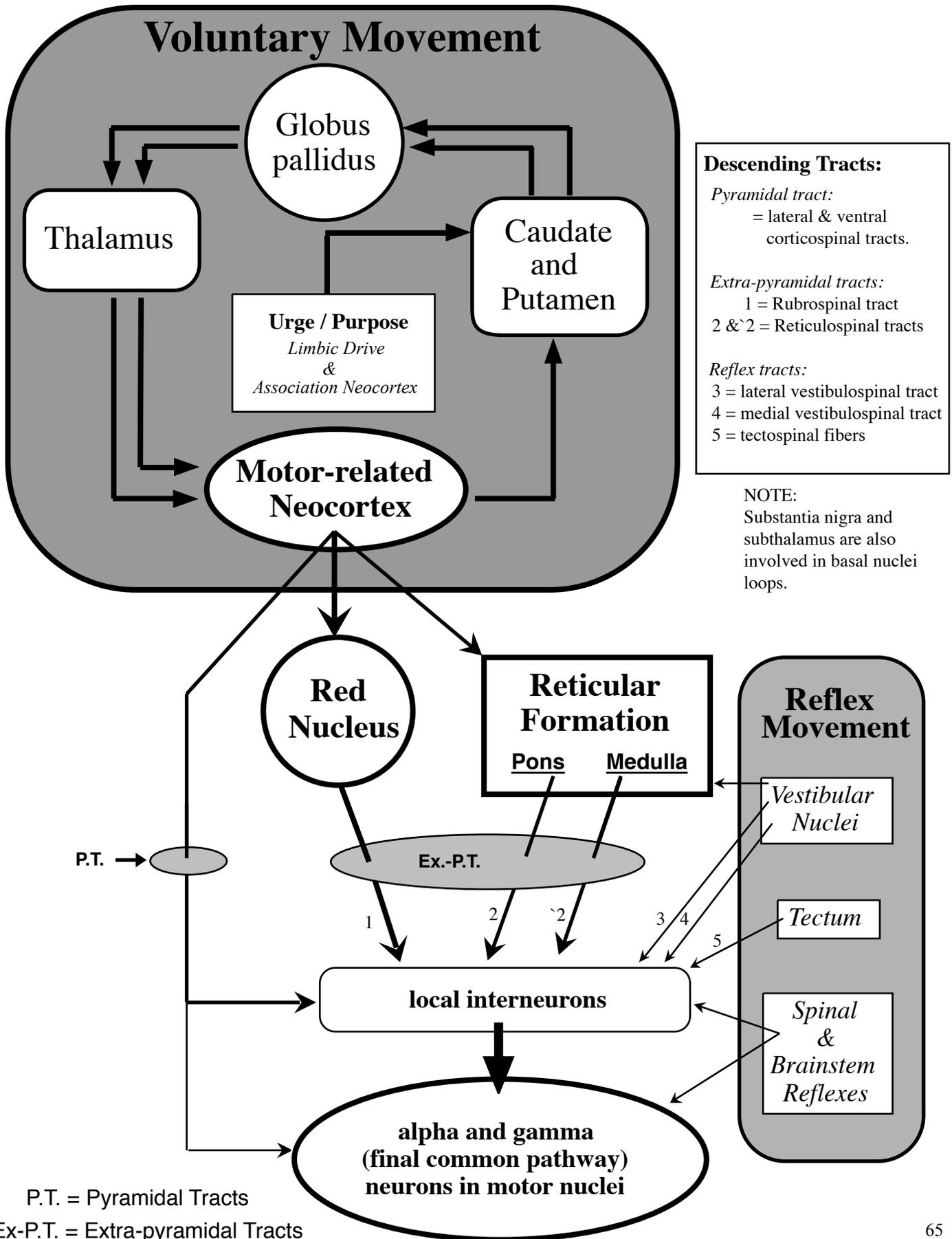
Loss of forebrain & midbrain (= hindbrain animal): limbs rigidly extended constantly in a "saw-horse" attitude (decerebrate rigidity); no locomotion or righting capability; tonic neck reflexes present (postural adjustments initiated by neck proprioceptors).

Loss of whole brain (= spinal animal): temporary areflexia may be evident with abrupt injury (spinal shock); paralysis without spasticity; local spinal reflexes intact; crossed extension accompanies the withdrawal reflex.

Lower Motor Neuron Damage:

Spinal cord or peripheral nerve damage: paralysis and areflexia (flaccid paralysis); denervation atrophy of skeletal muscles with time.

Movement Initiation Schema



Movement Initiation Schema

