# Motor Systems

The control of voluntary movements is complex. Many different systems across numerous brain areas need to work together to ensure proper motor control. We will start a journey through these areas, beginning at the spinal cord and progressing up the brain stem and eventually reaching the cerebral cortex. Then to complete the picture we'll add two "side loops," the basal ganglia and the cerebellum. This is relatively difficult material because the pathways are complex and our understanding of the nervous system decreases as we move up to higher CNS structures. Please keep up and complete all of the practice questions, and you will really enjoy this section. Remember, this material is important for you as future physicians because many of your patients will exhibit signs and symptoms associated with motor diseases.

SPINAL LOWER MOTOR NEURONS

Lower motor neurons (**LMNs**; also called **alpha motor neurons**) are directly responsible for the generation of force by the muscles they innervate. A **motor unit** is a single LMN and all of the muscle fibers it innervates. The collection of LMNs that innervate a single muscle (triceps) is called a **motor neuron pool**.

You should remember from the spinal cord and brain stem module that LMNs that control proximal and distal muscles are found mainly in the **cervical** and **lumbosacral** segments of the spinal cord. At these levels, the LMNs are distributed such that neurons controlling **axial** muscles lie **medial** to those LMNs controlling distal muscles, and motor neurons controlling **flexors** lie **dorsal** to those controlling extensors. In the thoracic cord below T1, the LMNs are associated only with axial musculature.
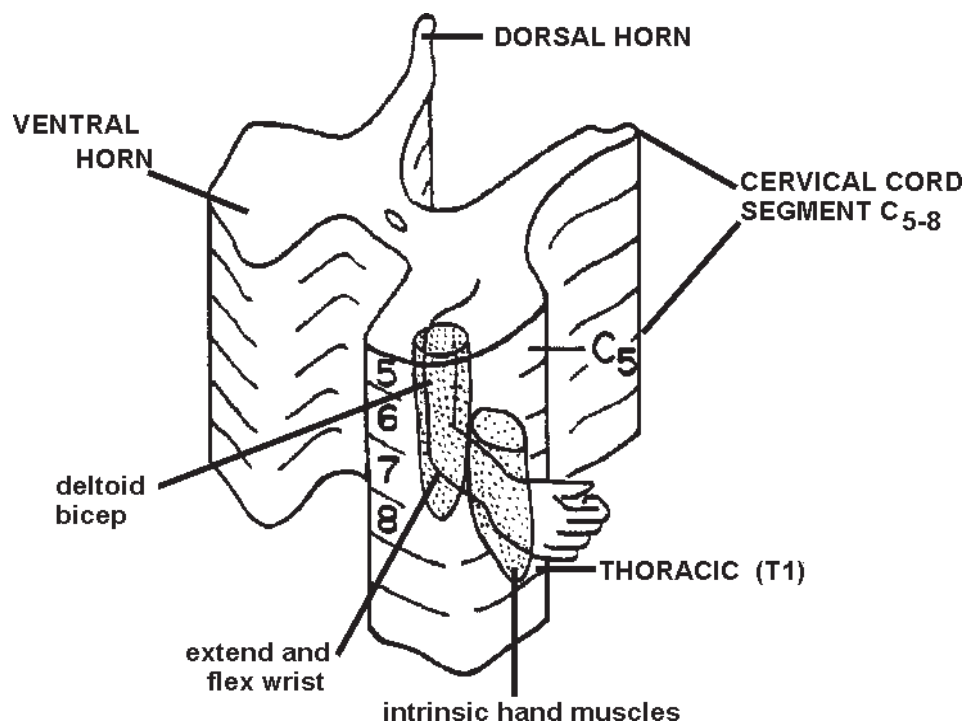
LMNs respond to, and are therefore controlled by, inputs from three sources: dorsal root ganglia, spinal interneurons (cells that do not project out of the area), and projections from higher centers such as the brain stem and cerebral cortex.

Stretch Reflex

Reflexes are short latency, relatively automatic responses to sensory stimulation. Spinal reflexes are a basic building block of movement.   Dorsal root inputs provide the sensory input for spinal reflexes, and the LMNs provide the motor output pathway.

One of the simplest and best studied reflexes is the stretch reflex - stretch a muscle and the reflex circuit leads to contraction of the same muscle. Stretch reflexes work to resist lengthening of a muscle. They are functionally efficient because they allow weight-bearing muscles to adjust to a changing load at the level of the spinal cord, without the information having to go all the way up to cortex for a decision. This is accomplished with a **muscle spindle** (our old friend from spinal cord and Physiology lectures) and a few neurons. You should recall the Ia and II fibers in the dorsal roots. **Ia fibers** are associated mainly with the nuclear bag intrafusal fibers and carry information regarding the **length** and **change in length** of the muscle. The **II fibers** are primarily concerned with the **constant length** of the muscle and thus bring information into the spinal cord from the nuclear chain fibers. You recall from the spinal cord module that these fibers synapse on cells in Clarke's column and accessory cuneate nucleus, which give rise to the spino- and cuneocerebellar axons. The Ia and II fibers make additional synapses that are important in stretch reflexes. Stretch of the intrafusal muscle fibers increases activity in the Ia and II fibers, which excite alpha LMNs. This results in contraction of the extrafusal muscle fibers in the same (**homonymous**) muscle. This is a **monosynaptic reflex**, in that there is only one synapse between the incoming information and the outgoing signal to contract the muscle. This monosynaptic reflex takes about 1 ms; the synaptic delay between the sensory input and the LMN is between 0.5 and 0.9 ms. In the cat, a single Ia fiber makes excitatory connections with all homonymous motor neurons. This Ia **divergent** signal produces a very strong excitatory drive to the muscle within which it originates. This is called **autogenic excitation**. The Ia fiber also makes monosynaptic connections with LMNs that innervate muscles that are synergistic to (cooperate with) the homonynous muscle.
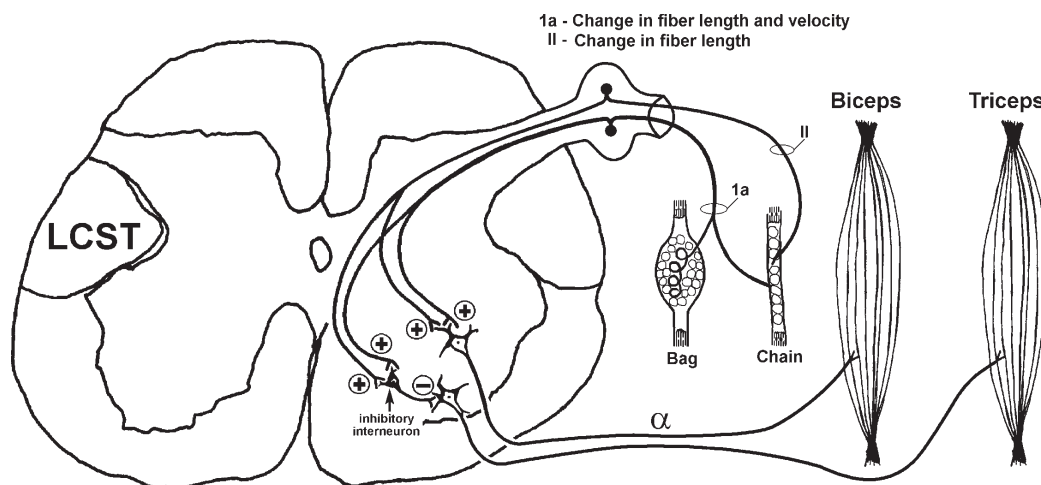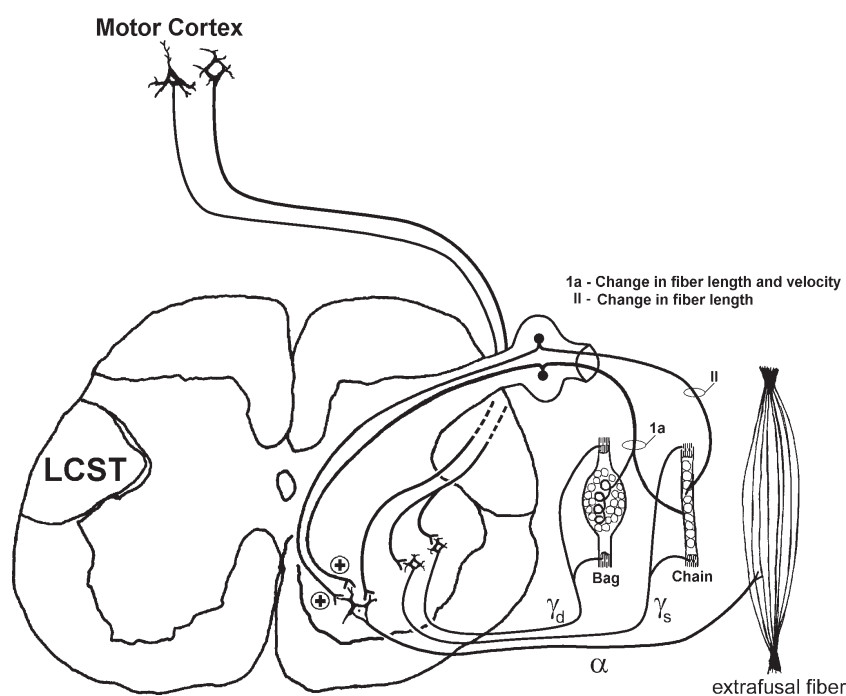

The incoming Ia fiber also synapses on a **Ia inhibitory interneuron** that in turn inhibits LMNs that innervate the antagonist muscles (stretch biceps——inhibit triceps; notice this is **NOT** a

monosynaptic reflex). This is called **reciprocal innervation**. Ia inhibitory interneurons are also important for coordinating voluntary movements, as corticospinal axons make direct excitatory connections with LMNs and also send a collateral to the Ia inhibitory neuron. Thus, the cortex does not have to send separate messages to the opposing muscles.

The Ia interneuron is one source of inhibition in the spinal cord. Another important source of inhibition that acts on this circuit is the **Renshaw cell**. Renshaw cells are excited by collaterals (branches) of motor neuron axons and then the Renshaw cells inhibit those same motor neurons. This recurrent inhibition provides negative feedback to the motor neuron. Increases in activity in the motor neuron increase inhibition from the Renshaw cell. This helps to stabilize the activity of the motor neuron and prevents sustained periods of extremely high

activity, which could produce muscle tetanus. The Renshaw cell also inhibits the Ia interneuron associated with the antagonist muscle. The Renshaw cell shown acts to inhibit biceps muscle contraction and dis-inhibit (effectively increase) triceps muscle contraction. Thus, Renshaw cells, like some other spinal interneurons discussed below, *adjust in a complementary fashion the activity of opposing muscle groups.*
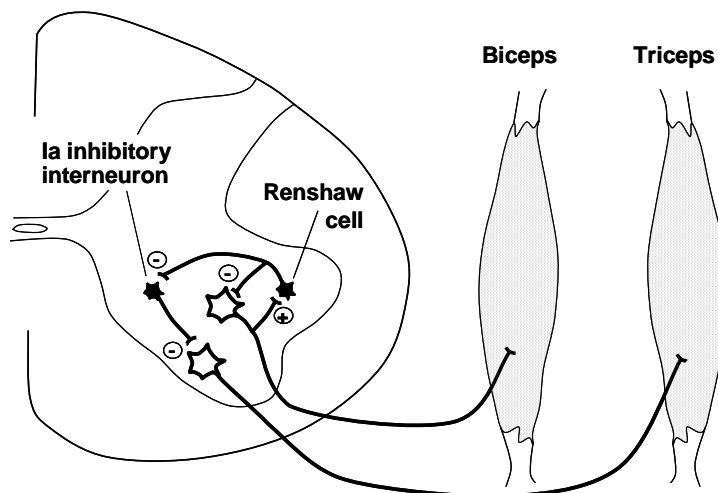
The intrafusal muscle fibers in the muscle spindles can be controlled by the small gamma efferent neurons that lie in the ventral horn. Gamma efferents that innervate the **bags** are called **gamma dynamics** while those targeting the **chains** are **gamma statics**. These gamma efferent cells, which are influenced by descending cortical and brainstem inputs, bias the intrafusal muscle fibers in order to ensure that the intrafusal muscle fibers are always signaling information to the brain. For instance, contraction/shortening of the extrafusal muscle will put slack on the intrafusal fibers, and they will stop telling the brain about the length of the muscles. This is bad! To get around this, the cortical input activates **both** the alpha and gamma LMNs. This is called **co-activation**. Activation of the alpha LMN leads to contraction of the extrafusal muscle while activation of the gamma LMN tightens up the intrafusal muscle fibers so the spindle retains its sensitivity. Thus, gamma efferent information adjusts the sensitivity of spindles for different conditions.

Inverse Stretch (myotatic) Reflex

   Ib fibers carry information from **Golgi tendon organs** (**GTOs**). Each GTO is comprised of collagen fibers intertwined with a Ib fiber. Thus, stretching of the tendon "squeezes" the Ib fiber, and it begins to fire. While muscle spindles are sensitive to changes in length of a muscle, GTOs are most sensitive to **changes in muscle tension** and thus signal the **force** in a muscle. GTOs provide the nervous system with precise information about the **state of contraction** of the muscle.

   The inverse stretch reflex is also known as the **inverse myotatic reflex** or **Golgi tendon reflex**. This reflex involves Ib afferents, **Ib inhibitory interneurons**, and LMNs. Increased firing of the Ib results in the **inhibition** of the **homonymous** muscle (**autogenic inhibition**).
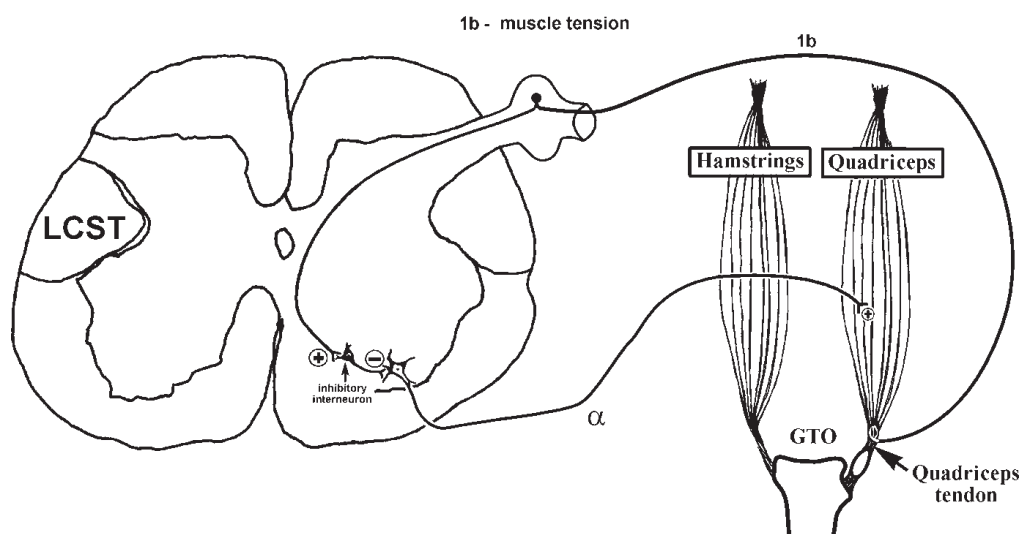
   The inverse stretch reflex is **polysynaptic**, meaning that it involves more than one synapse. This reflex is therefore slower than the stretch reflex. However, the inverse stretch reflex can override the stretch reflex. If there is a very large stimulus, such as a strong blow to the patella tendon with a hammer, the quadriceps muscles will contract due to the stretch reflex. To prevent damage to the tendon due to the muscle pulling too hard on it, the inverse stretch reflex is initiated by increasing tension in the tendon, and the contraction of the muscle is inhibited. The inverse stretch reflex is therefore damping down the effect of the stretch reflex.
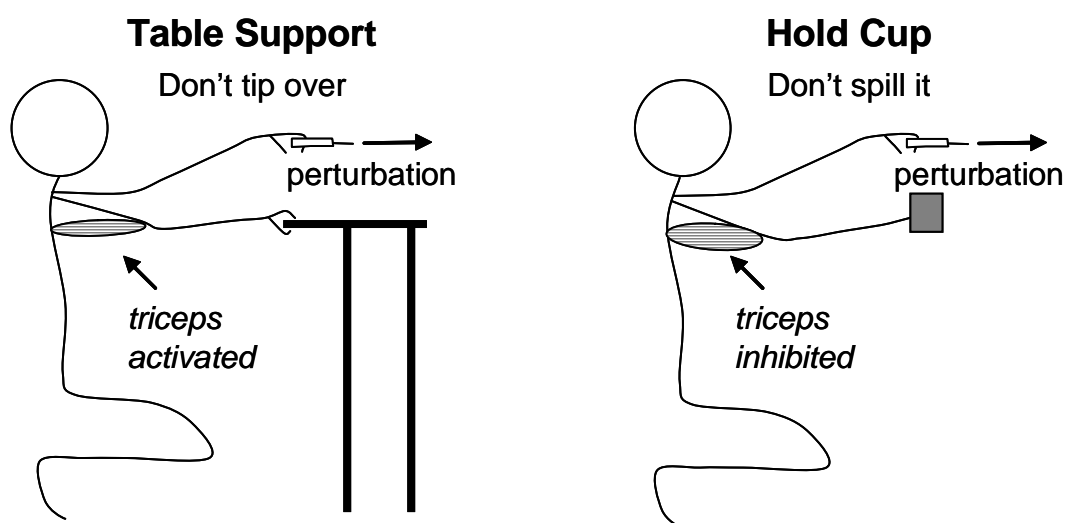
Flexion/withdrawal reflex

Pain and temperature fibers in the dorsal roots (Cs and deltas) play a role in the **flexion reflex**, also known as the **withdrawal reflex** (you already know the pathway over which this pain and temperature information reaches cortex via the ALS, etc.). At the spinal level, this reflex responds to noxious stimuli, such as a hot object on the skin. The result is a rapid flexion, a protective withdrawal mechanism that moves the body part away from the noxious stimulus. Another example of this reflex is stepping on a pin. The pin will be sensed by delta and C fibers, which synapse with a series of excitatory and inhibitory interneurons to produce a flexion response. The excitatory interneurons excite LMNs to the hamstring, while the inhibitory interneurons inhibit LMNs to the quadriceps (**reciprocal inhibition**).
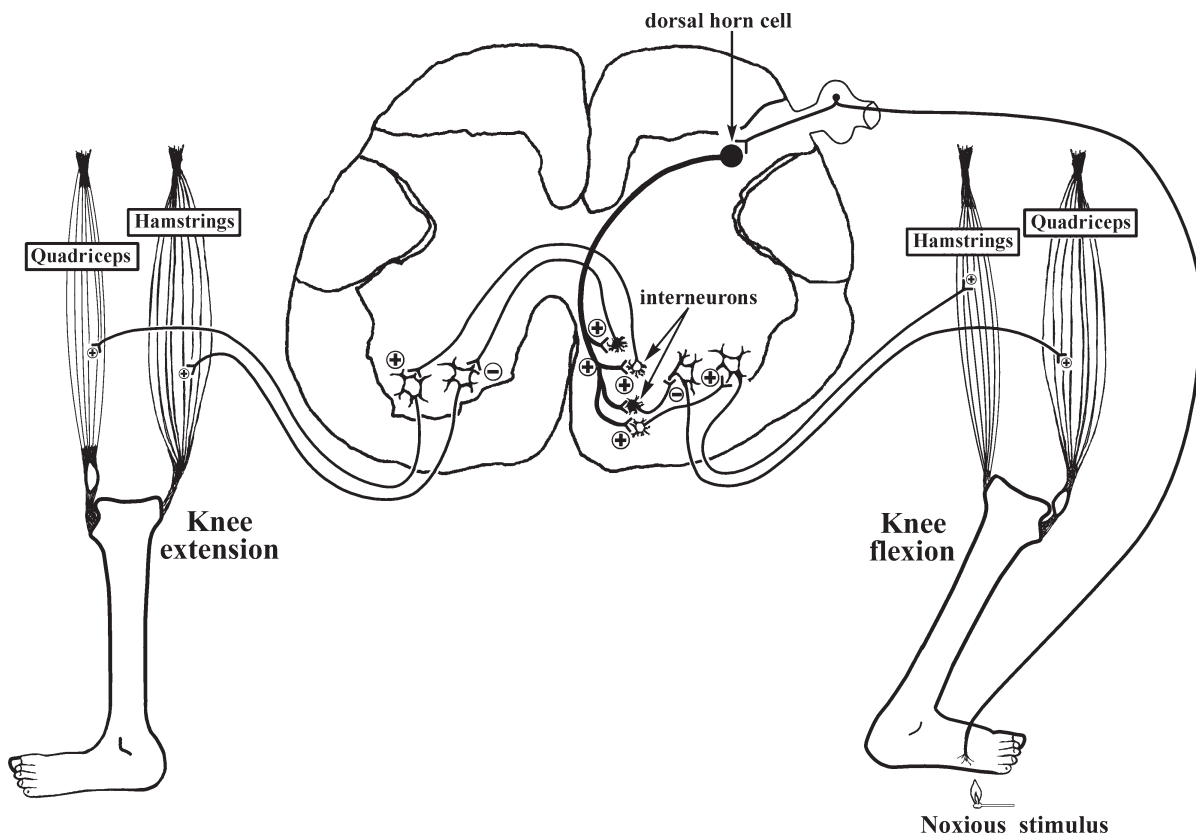
The **flexion reflex** is often accompanied by a **crossed extension reflex** acting on the contralateral limb. Using the example of stepping on a hot match, the crossed extension reflex would brace the other leg, helping to maintain balance. In this case, excitatory interneurons excite LMNs innervating the contralateral quadriceps, while inhibitory interneurons inhibit LMNs that project to the contralateral hamstrings.

You can see from the flexion/crossed extensor reflex that flexion withdrawal is a **complete**, albeit simple, **motor act**. While this reflex is reasonably stereotyped, the spatial extent and force of muscle contraction is dependent upon stimulus intensity. For instance, a moderately painful stimulus will result in the production of a moderately fast withdrawal of your finger and wrist, while a real painful stimulus results in the forceful withdrawal of the entire limb. Thus reflexes are not simply repetitions of a stereotyped movement pattern, but instead are **modulated** by properties of the stimulus.

It is important to understand that reflexes are **adaptable** and control movement in a purposeful manner. For example, a perturbation (a disturbance of motion, course, arrangement, or state of equilibrium) of the left arm can cause contraction of the opposite elbow extensor in one situation (right arm used to prevent the body from being pulled forward) but not in the situation where the opposite (right) arm holds a cup and the perturbation causes an inhibition of opposite elbow extensor. Also, don't forget that spinal reflexes are functionally efficient because they enable adjustments of movements to be made at the level of the spinal cord, without having to involve (and wait for) decisions from higher levels of the motor systems.

Table Support
Don't tip over

perturbation

*triceps
activated*

Hold Cup
Don't spill it

perturbation

*triceps
inhibited*

SPINAL CORD NEURONAL NETWORKS (CENTRAL PATTERN GENERATORS)

You now understand some basic reflexes involving the LMNs. Let's move "up" slightly in the motor "hierarchy," in particular, to neuronal networks in the spinal cord that generate rhythmic alternating activity. A **central pattern generator** (**CPG**) is a neuronal network capable of generating a rhythmic pattern of motor activity. Normally, these CPGs are controlled by higher centers in the brain stem and cortex. A simple spinal cord circuit is shown. This circuitry underlies **alternating** flexion and extension --- when some cells are active, the others are inhibited. These cells lie in the ventral horn on the same side of the spinal cord and include flexor and extensor motor neurons, together with their associated interneurons. Descending inputs from higher levels provide continuous input to the excitatory interneurons. However, because of random fluctuations in excitability or other inputs, one side of the circuit initially dominates and inhibits the other. Let's say for example that excitatory interneuron #1 turns on first. It will not only excite the **flexor** LMN and cause flexion, but it will also turn on inhibitory interneuron #2, which inhibits excitatory interneuron #2 so that the **extensor** LMN is inhibited. Inhibitory interneuron #2 also inhibits itself, so the flexion will switch to extension when the inhibitory interneuron #2 stops firing. Remember, the tonic inputs are exciting excitatory neuron #2. This will mean that now excitatory interneuron #2 will fire and excite inhibitory interneuron #1, which inhibits excitatory interneuron #1, and the pattern continues. You do not have to understand the details of such circuits. What you need to know is that there are circuits of cells in the spinal cord, composed of LMNs and interneurons, that generate basic patterns of locomotion.
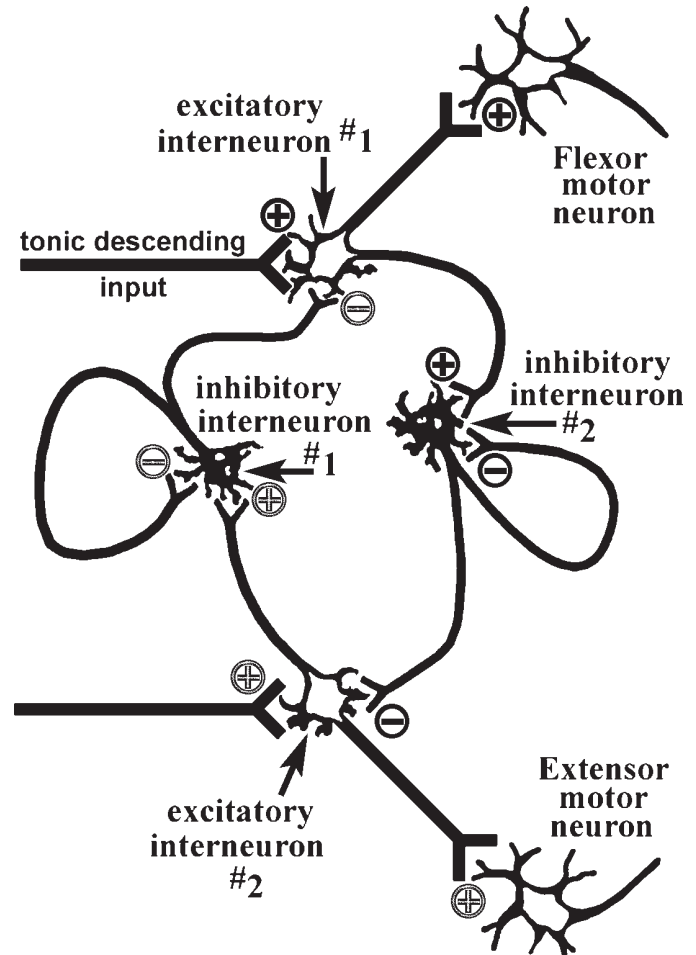
Sometimes these CPGs can be activated below the level of a spinal cord transection. For instance, if a quadriplegic's hips are extended, spontaneous uncontrollable rhythmic movements (flexion and extension) of the legs occur. Moreover, if babies are held erect and moved over a horizontal surface, rhythmic stepping movements take place. Granted, these stepping rhythmic activities in babies involve sensory input, but the circuitry for the rhythmicity of the movements is in the spinal cord. These two examples indicate that basic neuronal circuitry for locomotion is established genetically.

An important point to remember is that descending inputs (e.g., from brainstem or cortex) can act on these spinal circuits to modify their associated movements and even initiate the movements.

INFLUENCE OF "HIGHER" CENTERS UPON LMNs

At this point in our examination of the motor systems we know that there is reflex and central pattern program circuitry within the spinal cord and that certain reflexes and movements can occur using this intrinsic circuitry of the spinal cord.

The next level of organization in the motor systems "hierarchy" is descending inputs from a number of different nuclei in the brain stem and primary motor cortex to the spinal cord circuitry. Descending pathways to the spinal cord can be divided into **dorsolateral** and **ventromedial** systems based upon which spinal cord funiculus the pathway travels in and the termination pattern of its axons in the spinal cord grey. These two systems target different musculature.
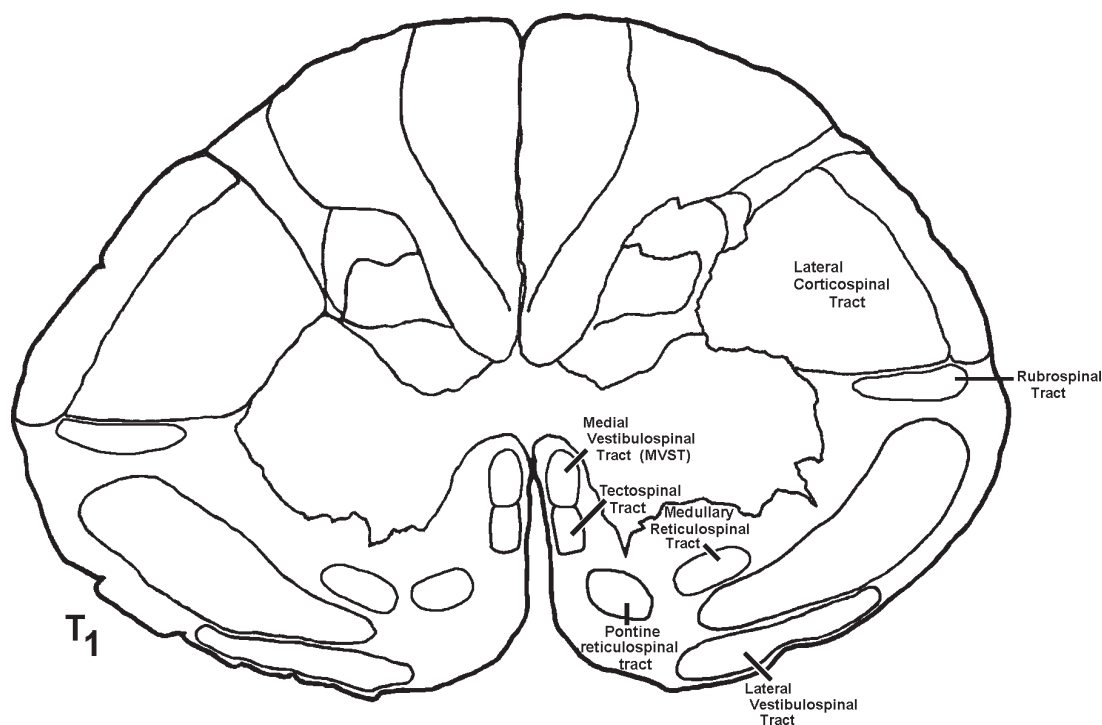
Dorsolateral system—control of distal musculature

You already know about the lateral corticospinal tract (**LCST**), and this is one of two dorsolateral pathways. The LCST is especially important for the control of **distal musculature** and for steering extremities and manipulating the environment. The other pathway in the dorsolateral system is the rubrospinal tract, which arises from our old friend the "ruber-duber." The rubrospinal tract courses next to the LCST within the **lateral** funiculus and is also important for the control of distal limb musculature. Axons of the dorsolateral system reach the LMNs that lie more **laterally** in the ventral horn. They also terminate in only **one or two** spinal segments; this precise targeting by individual axons allows precise control over muscle groups. Thus the dorsolateral pathways are involved in the finer motor control of the distal musculature (finger movements for example).

Lesions involving both dorsolateral pathways (LCST and rubrospinals) result in the inability to make fractionated (independent) movements of the wrist or fingers. Moreover, voluntary movements are slower
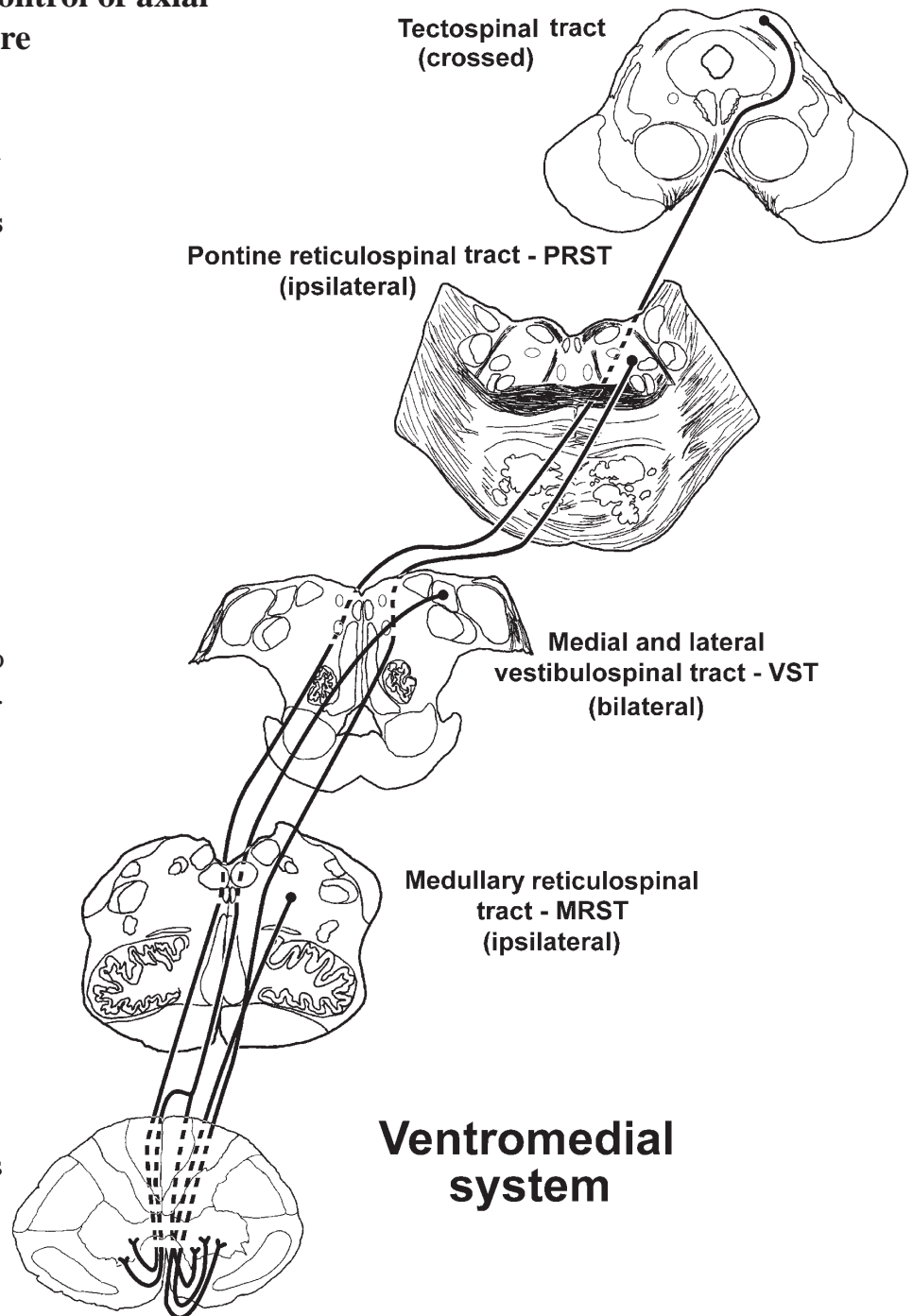
397

and less accurate. However, the patient still has the ability to sit upright, and stand with relatively normal posture (via functioning pathways in the ventromedial system discussed next).

A lesion of only the LCST initially resembles a combined lesion of the LCST and rubrospinal tract, but there is considerable recovery of function over time. The principle remaining deficit is inability to move the fingers independently (fractionated movements). Evidently, the functioning rubrospinal tract accounts for the recovered function of the wrist but it does not contribute to independent finger movements.

Ventromedial system—control of axial and proximal musculature

You already know that the **tecto**- and **vestibulospinal tracts** (**MVST** and **LVST**) travel in the **ventral funiculus** and terminate on LMNs that control **axial** and **proximal** musculature. Thus, they keep the head balanced on the shoulders as the body navigates through space, and the head turns in response to new sensory stimuli.

The **pontine** and **medullary reticulospinal** tracts (**PRST** and **MRST**) also travel in the **ventral** funiculus. (These pathways were not presented in the spinal cord and brain stem lectures, so you have not forgotten them). The **PRST**, which lies medial to the MRST in the ventral funiculus, arises from cells in the pons that lie around and near the PPRF (paramedian pontine reticular formation at the level of the abducens nucleus and motor VII; level 5). In contrast, the **MRST** has its origin from cells dorsal to the inferior olive (level 3). The reticular formation consists of those cells in the brain stem that do not

Tectospinal tract (crossed)

Pontine reticulospinal tract - PRST (ipsilateral)

Medial and lateral vestibulospinal tract - VST (bilateral)

Medullary reticulospinal tract - MRST (ipsilateral)

Ventromedial system

comprise the sensory and motor nuclei that you so carefully learned earlier in the course.

The **PRST enhances** the **anti-gravity** reflexes of the spinal cord. Thus, this pathway **excites** upper limb flexors and lower limb extensors. This is an important component of motor control, since most of the time the activity of ventral horn cells maintains, rather than changes, muscle length and tension. **PRST** cells are **spontaneously** active but they also receive excitatory input from the cerebellum (soon to be discussed) and the **cerebral cortex**

The **MRST** has the **opposite** effect of the PRST, in that it **inhibits anti-gravity** reflexes of spinal cord (**inhibits** upper limb flexors and lower limb extensors). MRST cells, like those in the PRST also receive excitatory input from the cerebellum and the **cerebral cortex**.

All of these **ventromedial** pathways (MVST, LVST, TST, MRST, PRST) innervate LMNs and interneurons that control **axial** and **proximal** limb muscles. Moreover, the axons in the ventromedial pathway distribute collaterals to **many segments** along their distribution in the spinal cord (for the coordination of intersegmental movements). You can see that such a distribution is not well suited for the discrete control of a few muscles but rather for **control of groups of muscles involved in posture and balance**.

Lesions of the ventromedial pathways in animals result in difficulty in **righting** to a **sitting** or standing position and immobility of the body axis and proximal parts of the extremities. However, the hands and distal parts of the extremities are still active, because the descending pathways controlling LMNs that innervate more distal muscles travel in the dorsolateral part of the spinal cord.

Don't forget the important organizational rule for of the descending motor pathways in the spinal cord:

**dorsolateral system = distal musculature
ventromedial system = axial and proximal musculature**

You now know the main descending motor pathways. One important point to remember is that there is more than one way for cortical information to reach the spinal cord; damage to one pathway can sometimes be compensated by a surviving pathway. There are smaller pathways I have not discussed that follow the ventromedial-dorsolateral organizational rule. For example, a small percentage of corticospinal axons travel in the **ventral** funiculus. Unlike their famous LCST cousins that travel in the lateral funiculus and influence distal musculature, these ventral funiculus axons target spinal neurons related to axial and proximal muscles. This example is only meant to reinforce the dorsolateral:distal, ventromedial:axial/proximal distinction. We won't discuss this particular ventromedial corticospinal tract further.
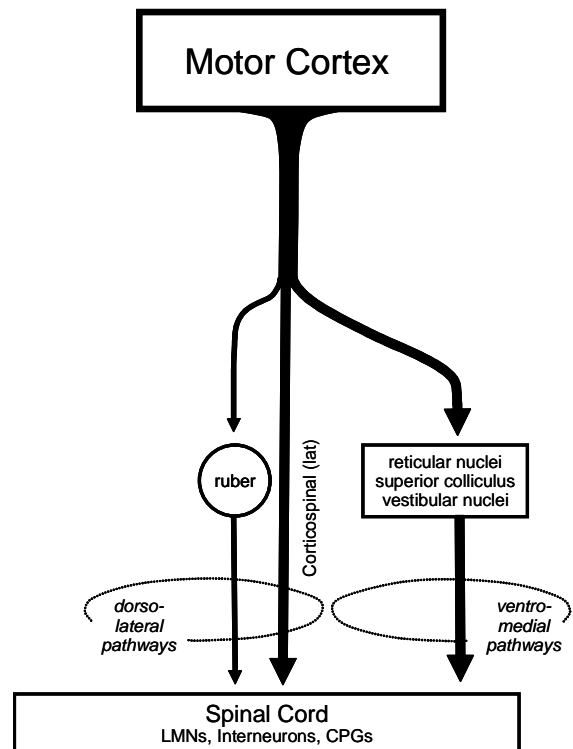
PRIMARY MOTOR CORTEX

Motor cortex acts on the spinal and brainstem motor neurons and pathways to produce the more elaborate **voluntary movements**. **Primary motor cortex** provides the descending signals to **execute** the movements.
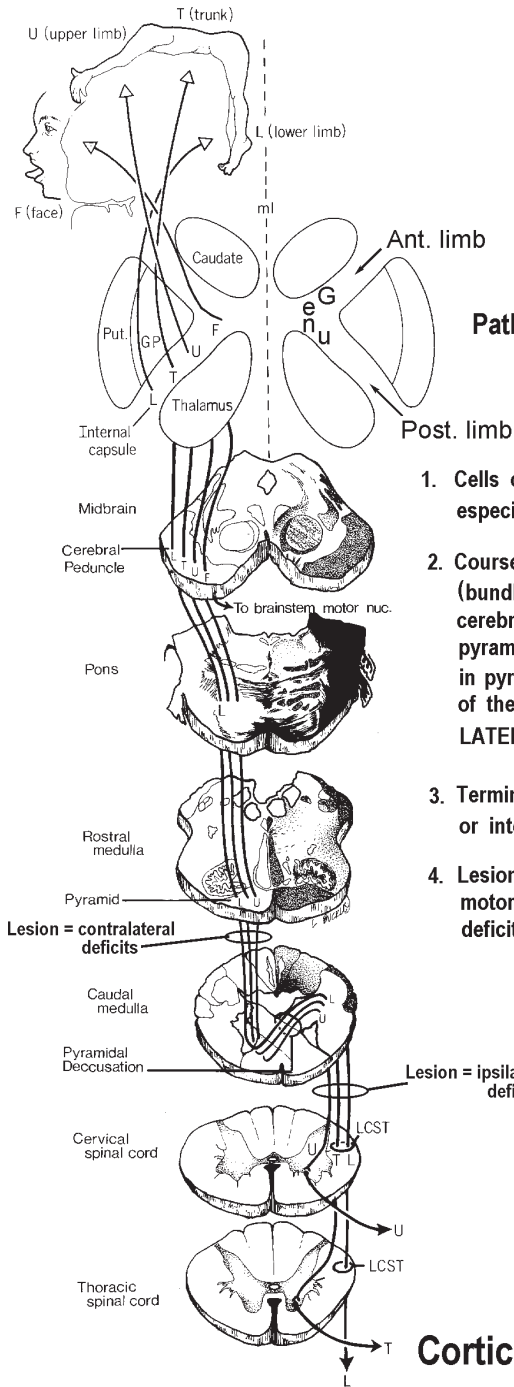
The **primary motor cortex** is also called **area 4** of Brodmann and **motor I** (**MI**). Corticospinal cells in area 4 project directly to the lower motor neurons (LMNs) in the spinal cord and brain stem and tell these LMNs, and in turn the muscles they innervate, what to do. **Cells in MI are the classic upper motor neurons** (**UMNs**). MI is located in the precentral gyrus, which lies in front (rostral) of the central sulcus. MI occupies most of this gyrus along both its lateral and medial surfaces and is bound caudally by the central sulcus.
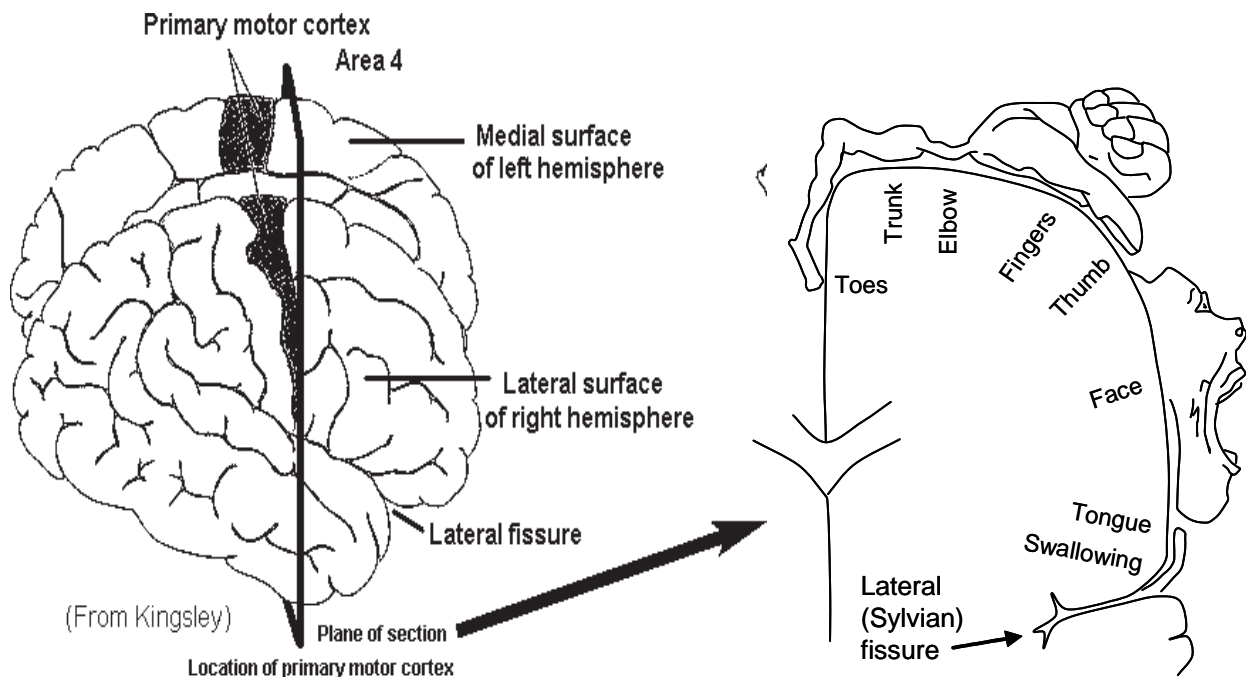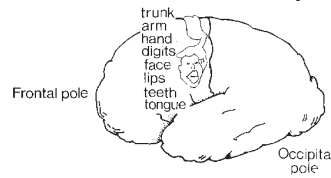

Human motor map. *Left: The entire lateral surface of the* ***right*** *hemisphere is seen while only the dorsal and medial aspect of the left hemisphere is visible. A coronal (or frontal) section is cut through area 4 or MI. Right: The map of the body (homunculus) as represented on the precentral gyrus in this brain section. Medial is to the left, lateral to the right.*

Somatotopic organization of MI

In the late 1950s Wilder Penfield studied the somatotopic organization of MI in patients by stimulating MI with relatively large electrodes (the patients gave their permission to do this as part of their surgery). He found a topographically organized representation of the entire head and body, which is schematically shown above and to the right as the classic homunculus (*L. little man*). The head and forelimbs are represented laterally, and the hind limb and feet medially. The cortical areas allotted to different bodily regions are of unequal size, with the degree of representation proportional to the amount of skilled movement required of the respective part of the body. Thus, in humans the face (particularly the tongue and lips used in speaking), thumb, and hand receive a disproportionately large representation, giving these structures a correspondingly exaggerated

T (trunk)
U (upper limb)
L (lower limb)
F (face)
ml

Caudate
Put.
GP
Thalamus
Internal capsule

G
e
n
u
F
U
T
L

Ant. limb
Post. limb

Representation of the body on the precentral gyrus of the LEFT cerebral hemisphere

trunk
arm
hand
digits
face
lips
teeth
tongue

Frontal pole
Occipital pole

Pathway involved in voluntary control of the *contralateral* arm, trunk, and leg.

Midbrain
Cerebral Peduncle
To brainstem motor nuc.
Pons
Rostral medulla
Pyramid
Lesion = contralateral deficits
Caudal medulla
Pyramidal Decussation
Lesion = ipsilateral deficits
Cervical spinal cord
LCST
Thoracic spinal cord
LCST
U

1. **Cells of origin** - several areas of the cerebral cortex, especially motor cortex (area 4).

2. **Course of axons** - traverse the internal capsule (bundle of axons surrounding the diencephalon), cerebral peduncle of midbrain, basilar pons and pyramid of medulla. Axons cross at caudal medulla in pyramidal decussation and enter the lateral funiculus of the spinal cord, where they comprise the LATERAL CORTICOSPINAL TRACT (LCST).

3. **Termination** - axons end either on lower motor neurons or interneurons in the spinal cord.

4. **Lesions** - rostral to pyramidal decussation = contralateral motor deficits, caudal to pyramidal decussation = ipsilateral deficits.

motor cortex

spinal cord
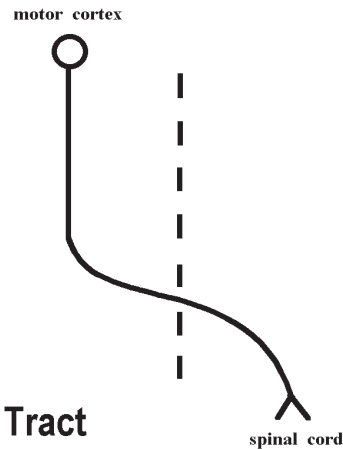
Corticospinal Tract

appearance. Movements of limbs evoked by stimulation of MI are strictly contralateral, but some movements of the face (remember corticobulbars) may be bilateral.

The main outputs of MI are the now familiar corticospinal and corticobulbar pathways, which arise from cells that are pyramidal in shape and lie in layer 5 of MI (most of the cerebral cortex has 6 layers, 1 being most superficial and 6 the deepest). **The largest corticospinal cells are called Betz cells**.

Several corticospinal axons are shown in the drawing above as they course through the posterior limb of the internal capsule, the cerebral peduncle, pyramids of the medulla and pyramidal decussation. Once in the spinal cord, these corticospinal axons comprise the LCST. You will learn more about the internal capsule in a future lecture. For now, all you need to know is that the internal capsule is 1) a large fiber bundle that contains axons going to and coming from the cerebral cortex,

2) that it has an anterior and posterior limb separated by a genu (*L. bended knee*) and 3) that the **corticospinal tract axons course through the posterior limb, while the corticobulbars are found in the genu**.

Physiology of corticospinal neurons

Much of what we know about the physiology of cells that project into the corticospinal tract comes from single neuron recordings in awake monkeys trained to make specific movements. Corticospinal neurons were studied in a classic experiment in which monkeys were trained to move a handle by flexing and extending the wrist (a nice distal motor task). The monkey was cued by a light to either flex or extend its wrist. The experiment revealed several basic properties of corticospinal neurons:

First, corticospinal neurons are sensitive to the **direction of movement**. Some are most active for wrist extension. Others, as illustrated in the figure, are most active for wrist flexion. Second, the MI corticospinal response **precedes** the muscle electromyographic (EMG) activity, as expected since it is producing the motor command for that movement. Third, corticospinal activity is **related to the force** required to make the movement. The cell produces more action potentials with the addition of resistance that requires more force to make the movement. Conversely, the neurons are less active in conditions when the movement is assisted, requiring less force (not illustrated). Finally, the same neuronal activity is seen even when the movement is done repeatedly many hundreds of times (i.e., there is no adaptation or habituation of the cellular activity).

Corticospinal activity correlates with muscle force
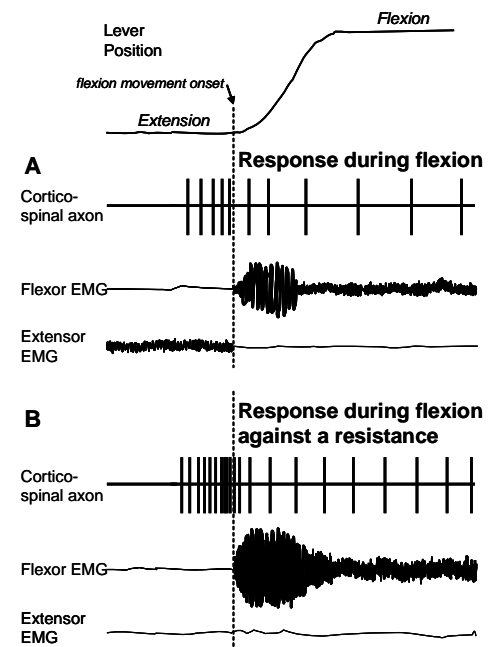
Functions of MI

Studies of the type illustrated have shown that corticospinal cells are telling LMNs in the spinal cord what to do. In fact, corticospinal cells convey a lot of details about amplitude, force and direction of limb movement. In other words, MI neurons are NOT leaving these decisions up to the LMNs, as LMNs are too dumb. Thus, MI cells act on the LMNs (and in turn certain muscles) to determine specific aspects of the movement: what direction to move, how far to move (amplitude) and how much force to use. The corticospinals in MI are specifying some pretty basic stuff instead of just sitting back and saying "grab that glass of water, arm and hand." Remember, MI's contributions to motor control include controlling the number of muscles and movement forces and trajectories. Thus, MI cells are mainly concerned with the actual *execution* of the movements.

# Primary Motor Cortex - Execution of Movements

Effects of lesions of MI

You already know from spinal cord and brainstem lectures that **MI lesions** affect the **contralateral** body. Relatively small lesions affect localized parts of the body because MI is topographically organized (remember the homunculus). With larger lesions there will be hemiplegia. Much of the homunculus is devoted to the hand, so skilled, independent movements of the fingers are often affected. Also, a Babinski sign would be present if the lesion involves UMNs of the lower limb.

An important consequence of multiple descending pathways is that a surviving pathway can sometimes compensate for the loss of another pathway. A nice example of this is the partial recovery of wrist and hand function that follows a complete lesion of LCST. The corticorubrospinal pathway appears to assume some of the jobs formerly done by LCST (although it isn't much help when it comes to the more refined movements of the fingers). As you might expect, very little recovery occurs if the lesion involves the LCST **and** the corticorubrospinal pathway.

Separate cortical neurons generally comprise the beginning of each of the descending motor pathways. This means that one set of cortical neurons gives rise to the corticospinals, another set to the corticorubers, another set to the corticoreticulars, etc. Thus, a small lesion in motor cortex could affect cells of just one projection, producing symptoms associated with only that pathway.

However, you need to know that <u>lesions of motor cortex are usually large - large enough to affect cortical neurons of the direct (e.g., LCST) and indirect (e.g., corticorubrospinal, corticoreticulospinal) descending pathways</u> involved in voluntary motor control. This leads to compromised voluntary movement and the classic signs associated with UMN lesions. You already know these. Don't forget them!
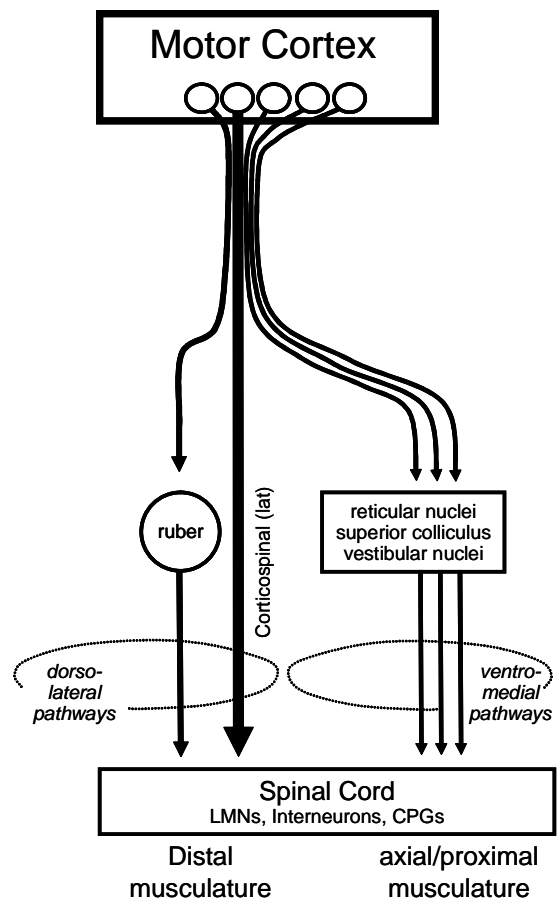

Classic signs of UMN lesion:
weakness, hypertonia, hyper-reflexia, spasticity, Babinski sign and NO muscle atrophy

Cerebral Arteries

Blood Supply of MI

Area 4 or MI receives its blood supply from the **Middle Cerebral Artery** and **Anterior Cerebral Artery**. Note that the anterior cerebral artery feeds the part of MI that extends down the medial surface of the hemisphere, which controls the lower limbs. The middle cerebral artery feeds the lateral surface of the hemisphere, which contains the upper limb and head portions of MI.

Corpus callosum

Caudal   **Medial View**   *Rostral*

posterior cerebral artery

anterior cerebral artery

Lateral View

middle cerebral artery
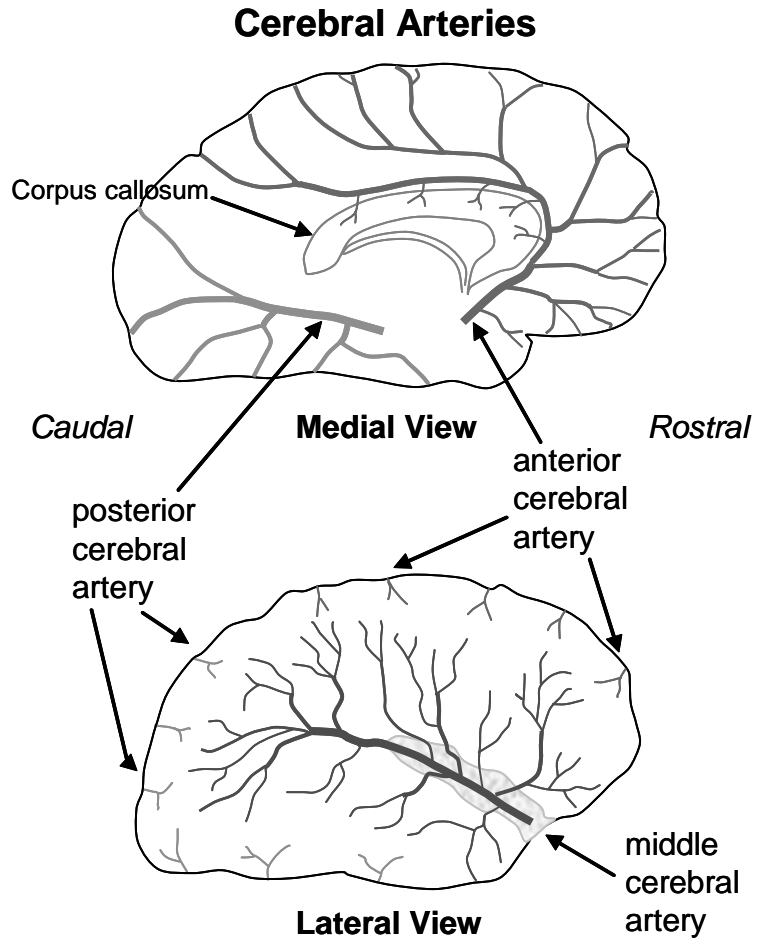
AFFECTED ARTERY	AREA AFFECTED	SIDE
Ant. Cerebral	Lower limb	Contra
Mid. Cerebral	Upper limb and Head	Contra

MOTOR ASSOCIATION/PREMOTOR CORTICAL AREAS

Studies in the late 1800's showed that electrical stimulation of frontal cortex elicited movements of body parts. We now know this happens most easily in MI. Stimulation of MI requires the lowest intensity current to evoke a movement. This probably reflects the direct projection of MI corticospinal neurons to LMNs. MI-stimulated movements are relatively simple and involve a single joint, consistent with the role of MI in the execution of simple movements.

Electrical stimulation of cortex immediately rostral to MI also elicits movements, but with important differences. The current intensity has to be higher to produce an effect, and the movements are more complex, involving multiple joints, such as reaching or grasping movements. The areas in front of MI associated with more complex motor function are called **motor association** or **premotor areas.**

Premotor cortex is involved with making **complex, multiple-joint movements** and in the **planning** of movements. This is accomplished largely by telling MI what movements to execute. The total size of these premotor areas is considerably larger than area 4. These areas prove to be particularly important in the control of human voluntary movement and are often compromised in diseases. The two premotor areas we will talk about are the **supplementary motor area** (**SMA**) and the **lateral premotor area (PMl)**. Remember, both are part of cortical **area 6**.
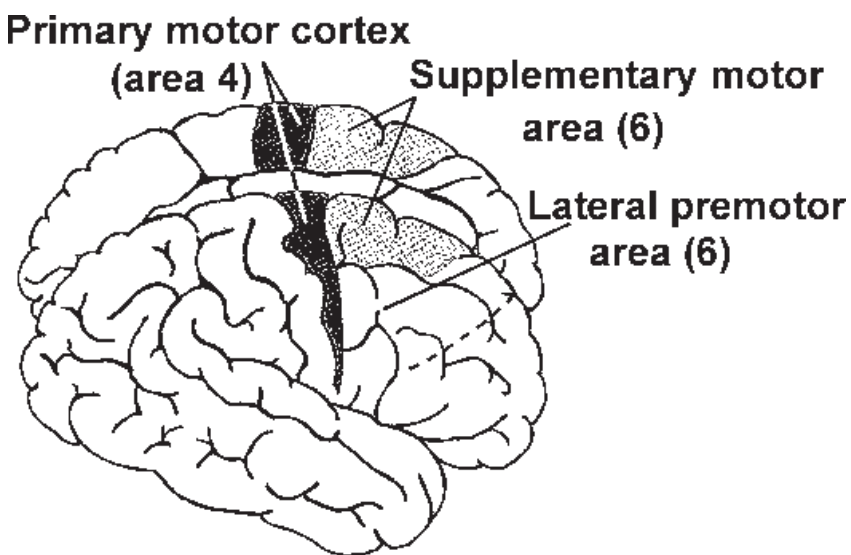
Lateral premotor area: sensory motor interactions

The **lateral premotor area** (**PMl**) lies on the lateral convexity of the hemisphere. Many motor actions are responses to visual or auditory cues, and cells in PMl are active during such **externally cued** movements. For instance, you see an apple (external cue), and you reach out and grasp it. Pathways involved in this sensorimotor transformation (seeing it is "sensory" and reaching for and grasping it is "motor") are seen in the next figure. The visual information (apple) falls first on the retina, and the retinal signal eventually reaches the primary visual cortex in the back of the brain. From visual cortex, the visual information is conveyed rostrally and bilaterally to what is termed the **posterior parietal cortex** (**PPC**) in areas 5 and 7. The PPC helps to transform visual information about the **location** of the apple in extrapersonal space into the direction of a **reaching** movement. Moving a body part relative to another body part is an example of a movement in **intrapersonal** space, while movements in **extrapersonal** space are movements executed in a three dimensional coordinate system fixed by points in the environment —whew! Information from PPC is conveyed (via several pathways, but we won't sweat the details) to PMl. PMl acts on MI which conveys the descending information to LMNs to execute the reaching movement.
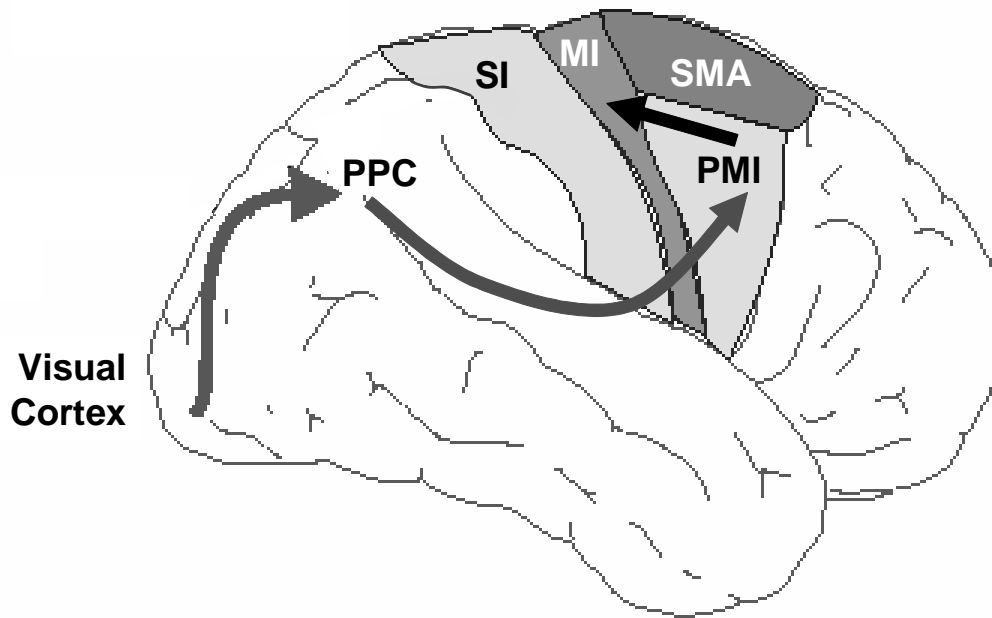
Lesions of the premotor cortex result in **apraxia**, which is **an inability to perform a purposeful movement (especially skilled) in the absence of any significant paralysis, sensory loss, or deficit in comprehension**. As you would expect, the apraxia that results from lesions in PMl involves deficits in **sensory-motor integration**: there is failure to "find" the appropriate movements when presented with particular sensory cues. For example, monkeys can be trained to turn or pull a handle depending upon whether the handle is red or blue. If there is a lesion of PMl, the monkey can still turn and pull the handle in other conditions (MI is OK), but they can not **associate** the correct movement with the correct **external visual cue** (colors). PMl lesions are also associated with deficits in **ability to reach and grasp objects.**

Supplementary motor cortex: internally generated movements and movement sequences

The supplementary motor area (SMA) is located immediately rostral to area 4, and includes the caudal one-third of the medial, dorsal, and lateral surfaces of the superior frontal gyrus. SMA is involved in **internally generated movements**. For instance, if you suddenly have the "internal need" to reach out and snap your fingers (this is NOT precipitated by an external cue), the memory/ motor patterns necessary for this movement are encoded in cells within the SMA. Cells in the SMA will start to fire and, in turn, send the instructions for the movement to the workhorse, MI, so that the proper muscles are moved with the correct amount of force and speed (execution of the movement). Interestingly, the two SMAs (right and left) are interconnected by cortico-cortical fibers, and, unlike MI, the activity in the SMA is usually **bilateral**. So, during the movement of the right arm/hand, cells in both SMAs start firing, but only cells in the left MI fire. Cells in SMA fire before those in MI but both SMA areas will be firing during the movement. Interestingly, if you just **imagine** reaching out and grabbing something in front of you, cells in SMA will increase firing bilaterally!!! This is consistent with SMA's involvment in internally generated movements.

SMA is also involved in the programming of **movement sequences**. The role of the SMA in **sequencing** of movements is exemplified nicely by data from experiments in which monkeys were

trained to do three movements; push, pull or turn a manipulandum (a joystick like you use with video games) in four different orders (e.g. push, turn, pull, would be one choice). Cells were found that increased their firing to a particular sequence (turn, push, pull) but not to a different sequence (turn, push, turn). Other SMA cells were active during other sequences. In the above task with the manipulandum, the visual appearance of the apparatus (an external cue) gave no clue as to which movement to make or even the order of the movements (unlike the case in movements involving PMl).
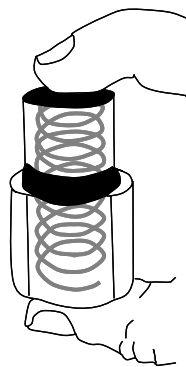
Bottom line: SMA is big on **internally generated sequences of movements.**

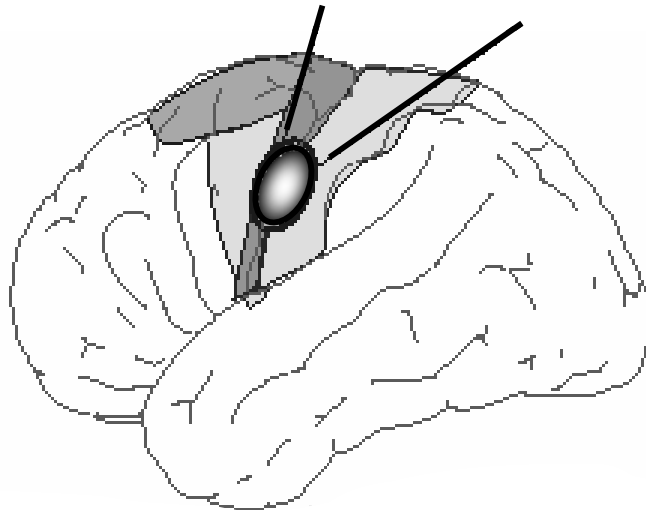## Motor Cortex function revealed by imaging cerebral flood flow

Additional information about SMA and its differences from MI are provided by studies that look at the regional cerebral blood flow **(rCBF)** during motor tasks. In these studies an increase in rCBF means the cortical area is more active. In one rCBF study, subjects are asked to make movements that differ in complexity. The simplest motor task requires subjects to make fast flexions of the index finger against a spring-loaded movable cylinder. This leads to increased rCBF in the finger area of the **contralateral MI** and SI (primary somatosensory cortex). The increase in rCBF in the somatosensory cortex reflects activation of peripheral tactile and proprioceptive receptors caused by the finger flexions. More importantly, *no higher cortical motor area (like PMl or SMA) exhibits an increase in rCBF in this task, just MI.*
 Thus, MI is the primary motor cortical area involved in the execution of simple repetitive movements.
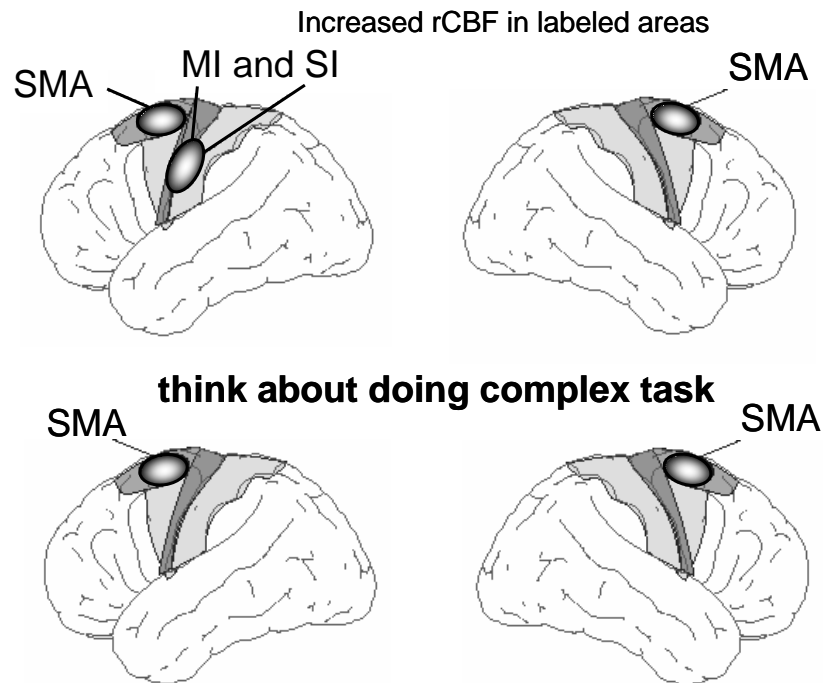

Simple Repetitive Movements

Increased rCBF
in MI and SI

complex motor sequence task

Increased rCBF in labeled areas

SMA   MI and SI   SMA

think about doing complex task

SMA   SMA

        When the same subjects are asked to carry out, from **memory, sophisticated** and **complex**
movements, pre-motor cortical area(s) also exhibit an increase in rCBF. For example, the thumb, in
quick succession, must touch the index finger twice, the middle finger once, the ring finger three
times, and the little finger two times. Then, with the thumb opposed to the little finger, the whole
sequence is reversed!!! (Don't worry, you will not be asked to do the sequence for the boards). This
is a complex series of movements guided by memory and carried out in **intrapersonal** space
(moving a body part relative to another body part). Again, there is increased rCBF in the
contralateral MI and SI (a bigger area since more fingers are involved). In addition there is
increased blood flow within **SMA bilaterally**. Thus, a complex task that consists of a **sequence** of
remembered movements involves the activity of both **SMAs** and MI and SI contralateral to the
active fingers. Of course, MI is needed for the execution. When the subject is asked to just **imagine**
the finger sequence movement, SMA lights up bilaterally, consistent with its role in the planning of
internally generated sequences. However, MI is inactive (no execution, no MI activity).

        Lesions of SMA lead to **apraxia**, which we have already defined for PMl lesions as the
inability to produce purposeful movement even in the absence of muscle weakness or sensory loss.
There is **NO** paralysis, only problems in planning. As you would expect, the apraxia following SMA
lesions involve a **deficit in performing sequences** of movements. For instance, monkeys who had
learned how to open a latch box by opening three different latches in a particular **sequence** are
greatly impaired following either uni- or bilateral lesions of SMA. Remember, the two SMAs are
connected across the midline. A lesion of one affects the proper functioning of the other. For
instance, a lesion of the **right** SMA means the **right** MI is not getting proper planning information.
This affects the **left** arm/hand. In addition, the **left** SMA has lost important input from the **right**
SMA so the **left** SMA input to the left **MI** is not normal. In the end**, bi-manual coordination** is
affected.

The premotor areas are more specialized than the "execution" area, MI. Both SMA and PMl are involved in **planning** of complex movements. However, the premotor area involved depends on the type of movement and whether the impetus for the movement is generated internally (SMA) or based on an external cue (PMl). **The movement can be the same, but the areas that activate and tell MI which muscles to turn on for execution differ**.
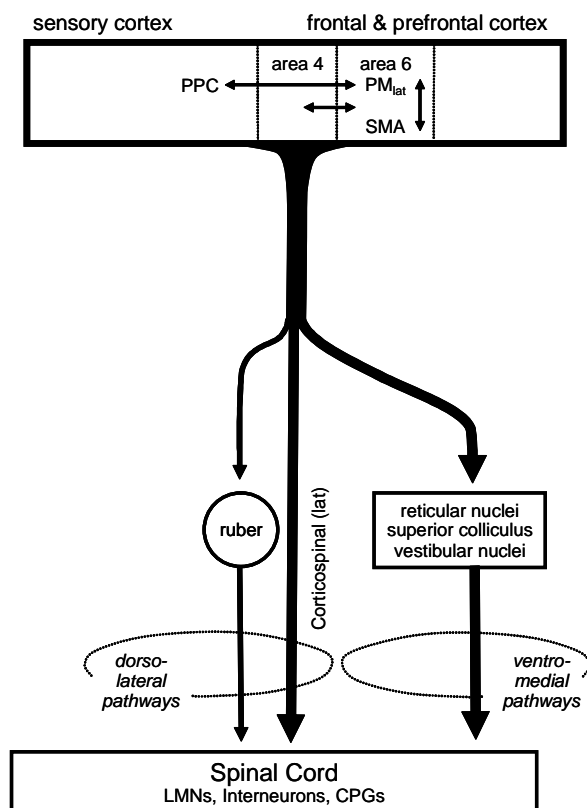
IN SUMMARY

MI - descending commands to execute basic movements
Ventro-medial pathways - axial/proximal musculature
Dorso-lateral pathways - distal musculature

Premotor cortex - planning of complex movements
SMA   - sequences, bimanual coordination
- internally-generated movements
PMl   - sensory-motor associations
- externally initiated and guided movements