The Leg Cross Flexion-Extension Reflex: Biomechanics, Neurophysiology, MNRI® Assessment, and Repatterning

Elvin Akhmatov, MA, Ph.D. Student, Orlando, FL, USA; Jakub Buraczewski, PT, MNRI® Core Specialist; Denis Masgutov, Director of SMEI , Poland


Elvin Akhmatov


Jakub Buraczewski


Denis Masgutov

Introduction

Two separate reflexes, Phillipson's Withdrawal and Leg Cross Flexion-Extension, are easily confused because they have similar motor patterns and are elicited by stimuli that can appear to be alike and usually manifest at the same time. The authors' purpose is to distinguish clearly between these two reflexes and to present detailed information on the one they refer to as the *Leg Cross Flexion-Extension Reflex*. The other reflex, often confused with Leg Cross Flexion-Extension, goes by several names: Phillipson's Withdrawal, Phillipson's Leg Flexion, Crossed Extensor, and Leg Withdrawal Reflex, among others. For clarity in this paper, the other reflex will be referred to as Phillipson's Withdrawal. On the neurophysiological level, these two reflex patterns present the work of two different nerve tracts – tactile and proprioceptive, activated and processed by different receptors.

The Leg Cross Flexion-Extension Reflex is extremely important for overall sensory-motor integration, motor programing and control. Cross-lateral or reciprocal activities such as crawling, walking, running, climbing, swimming, and jumping depend on maturation and integration of this reflex pattern. It also plays a crucial role in the 'training' of proprioceptive nerve pathways (vs. sensory-tactile) and activating the spinal-cortical tract. It is an invaluable resource for supporting neurosensorimotor-proprioceptive integration in physical rehabilitation of children and adults with developmental deficits and/or injuries affecting motor coordination and abilities. However, neither the reflex, its developmental and rehabilitative importance, nor its biomechanics and neurophysiological ascending and descending pathways is clearly or adequately defined in current literature. This study presents a new understanding of this complicated reflex and describes its biomechanics and the neurophysiological tracts of its ascending and descending pathways. The authors will also interpret MNRI® techniques for practical application that enhance precision and accuracy in the treatment of dysfunctional Leg Cross Flexion-Extension and affect other lower limb motor patterns.

Apart from the work of Masgutova and her associates, other researchers, to the knowledge of the authors, have not studied Leg Cross Flexion-Extension in children or persons with neurodeficits. In contrast, a simple search yields a wealth of information on Phillipson's Withdrawal. Two examples are 1) "The flexors in the with-

drawing limb contract and the extensors relax, while in the other limb, the opposite occurs" (Rod, Trent, Tate, 1992; http://en.wikipedia.org/wiki/Withdrawal_reflex), and 2) "when a person steps on a nail, the leg that is stepping on the nail pulls away, while the other leg takes the weight of the whole body" (Ellrich, Steffens, Schomburg, 2000; Solomon, Schmidt, Adragna, 1990; http://en.wikipedia.org/wiki/Crossed_extensor_reflex). According to the authors of this article, important developmental implications of this contralateral motor pattern are missing from these descriptions and thus will be described in detail here.

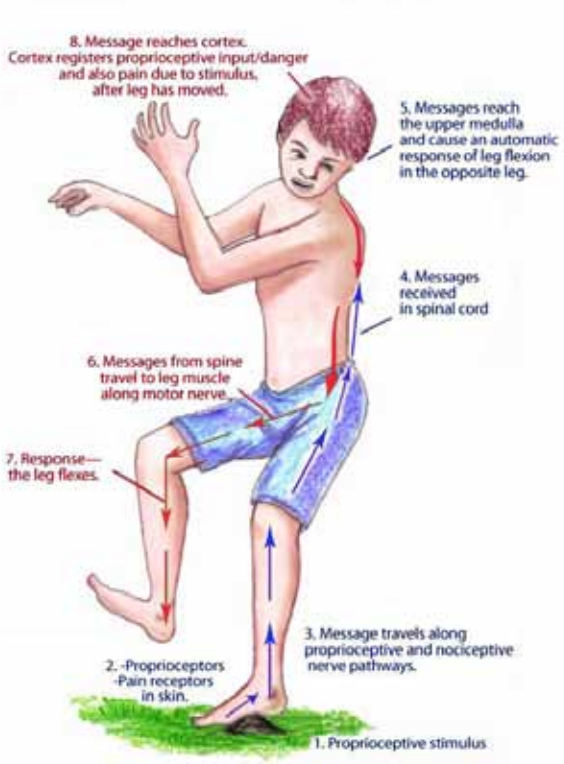


Figure 1. Leg Cross Flexion-Extension Reflex

I. The Leg Cross Flexion-Extension Reflex

Suppose you are moving along with a mature cross-lateral walking pattern when your right foot unexpectedly lands painfully on a sharp object. There are two possible scenarios: 1) your body weight is not yet on this leg and you have the chance to withdraw it, or 2) your body weight is already on this leg and if you try to withdraw it, you fall down. In scenario one, your sensory perception stimulates the tactile nerve pathway, and in scenario two the proprioceptive pathway acts in priority. In both cases the response to pain is present, and your body automatically provides shock absorption to lessen the pain by 'unweighting' the right foot with slight knee and plantar flexion and toe extension. This is a 'withdrawal'. In the case of Phillipson's Withdrawal pattern, the extension of the opposite leg is immediate.

In contrast, with Leg Cross Flexion-Extension the left (opposite) leg does not immediately extend. At this point in your gait, your left foot has just left the ground. It is impossible to withdraw your right foot from the sharp object without falling down. The slight withdrawal of your injured right foot causes a loss of equilibrium, which stimulates extension in that leg and returns that foot to the sharp object to regain balance. Thus, in order to prevent a fall, your reflex system requires that you briefly step down harder on the sharp object to support your body weight while your other leg completes its step, which it does first with flexion, then extension.

Contrary to the descriptions of Phillipson's Withdrawal, in Leg Cross Flexion-Extension, the leg where the stimulus is given extends, pushing against the stimulus rather than withdrawing. Not until your left foot touches the ground can your body weight be transferred to it, freeing you to lift the right foot away from the painful object. Pushing down hard on a sharp/unsafe object hurts more, but rapid flexion and extension of the opposite leg enables you to quickly transfer your body weight to the left foot and step away with the hurt right foot. This is the protective motor pattern of the Leg Cross Flexion-Extension Reflex. These two reflexes use different biomechanics and respond through different neural pathways.

Figure 2. Trigger of Leg Cross Flexion-Extension Reflex in an infant.

In a one or two month old infant the initial phases of this sequence of movements are absent. There is no loss and regaining of equilibrium in the supine infant, though there can be some initial very slight and very brief flexion of knee and foot. Unlike the withdrawal reflex, the Leg Cross Flexion-Extension Reflex is elicited not by pain (tactile nociceptors in the skin), but by deep pressure (proprioceptors in the joints, tendons, and muscles) on the center of the plantar surface of the foot. This proprioceptive stimulus on one foot simultaneously activates automatic extension of the same side leg (pushing against the stone) and flexion in the opposite knee and hip, with slight medial adduction, followed

immediately by lateral abduction, and then extension of that leg, stepping forward and to the side. This same circuitry, developed in infancy, later saves children and adults from possible injury, as described above, when they step unexpectedly on a sharp or possibly harmful object. In this complicated reflex we see how wisely nature has provided for our protection.

The circuit of this response is a complicated one activated by parallel pathways in all levels of the central nervous system, reaching the spinal cord, medulla oblongata, pons, mesencephalon, thalamus, and cortex. The input is processed in sequential and parallel ways in the spinal cord, ascending (sensory) pathways in the pons, and descending (motor) pathways in the mesencephalon, where nerve fiber crossover takes place, and in all these areas of the nerve system simultaneously (Haines, 2002; Lundy-Ekman, 2002). R. Magnus demonstrated experimentally that the processing of input and formation of the circuit of this reflex happens mainly on level of the medulla oblongata (Magnus, 1926).

The motor response is processed mainly by the spinal cord and extrapyramidal nerve net system of the brain stem. Activation of this circuit is essential for development of lower motor neuron pathways and myelination of their axons, especially on the level of the medulla oblongata.

It is very important that this reflex develop at the proper time. Normally it appears in the 12th week of gestation (St. Campbell, 2006; Ronin-Walknowska, Masgutova, 2006) and is active in utero and up to 1–2 months after birth (Barasznev, 2001). By 1–2 months of life it must be integrated, which means that its sensory-proprioceptive-motor circuitry is established for sequential movements of the legs, and works in a stable and timely way.

Figure 3. Stepping on an object with the whole body weight causes automatic flexion and, next, extension of the knee and hip in the other leg.

Sensory-Motor Structure

◆ *Position for eliciting the reflex:* Supine, standing, or walking

◆ *Proprioceptive stimulus:* On pressure point in the middle of the plantar surface of the foot (see stone in Figure 1). Note: In infants of 0-2 months pressure also on the lateral and medial vastus muscle fascia just above the knee (quadriceps; right leg, see Figure 4).

◆ *Proprioceptive stimulus:* Initially deep touch activates the proprioceptive receptors in the foot of a supine infant. In later development the corresponding receptors are activated (infant, child, and adult) standing, stepping, walking, and marching (see Figure 4).

◆ *Motor response:* The leg receiving the stimulus extends and opposite leg flexes in all joints; the knee moves laterally with slight adduction, then abduction, and finally extension.

Protective Meaning

• Protection from injury to the foot stepping on a potentially harmful object

• Transfer of body weight to the other leg to prevent a fall

Facilitating Reflexes

• Automatic gait
• Amphibian
• Bauer crawling
• ATNR (holding postural control)
• Galant
• Balancing
• Segmental rolling
• Spinning.

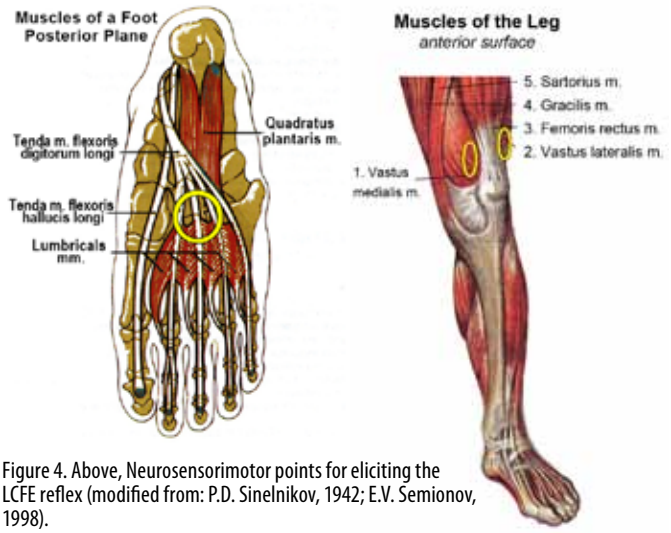


Figure 4. Above, Neurosensorimotor points for eliciting the LCFE reflex (modified from: P.D. Sinelnikov, 1942; E.V. Semionov, 1998).

Developmental Meaning

- Differentiation and coordination of legs
- Precursor to walking and running
- Walking, marching, running, swimming, skipping, jumping
- Cognitive development
- Speed of perception and thinking.

Opposing Reflexes

- Trunk extension (including lower limbs)
- STNR
- Moro
- Spinal Perez
- Landau
- Fear Paralysis.

Figure 5, Step 1. Stepping on the object (stone)

Figure 5, Step 2. Flexion of the knee and hip of opposite leg

Figure 5, Step 3. Adduction of the knee laterally leading orientation of the body to side and forward

Figure 5, Step 4. Extension of the opposite leg to save the hurt foot.

Effects of Non-Integration

In children with neuro-motor problems, muscular hypo/hypertension, cerebral palsy and other deficits, the Leg Cross Flexion-Extension Reflex is usually delayed and/or pathological. It is also poorly developed in children with autism and selective mutism. This is why skills requiring leg differentiation, shifting body weight from one leg to another for cross-lateral movements and balance control are all challenging for them. Additional effects of non-integration include:

- Poor neural maturation of lower motor neurons
- Hyper- (ADHD) or hypo-activity
- Dysfunctions of the Automatic Gait and Crawling Reflexes
- Postural problems: chronic inclining of the body to one side or another while standing, or instability due to poor antigravity skills
- Poor ability to cross the midline and inefficient cross-motor patterns
- Excessive muscle tension causing fatigue
- Delay in skipping, walking, swimming, jumping, and climbing skills
- Slow transition from perception to action
- Poor cognitive development: comparison, classification, prioritization, calculation, and analysis
- Incorrect overlapping of tactile and proprioceptive input and chaotic processing due to homologous crawling and walking patterns.

II. Biomechanics of the Leg Cross Flexion-Extension Reflex

Activation and maturation of this reflex motor pattern in infancy orients the brain-body system to its later protective function when the walking child or adult encounters a potentially harmful object under foot. The biomechanics of the Leg Cross Flexion-Extension Reflex involve complex antigravity phenomena related to body weight distribution on the feet and the defensive response of reorienting body weight and re-locating it to a safer spot when gravity pulls one foot down on a painful object. Clearly the motor response can be activated from either side. For the description below, please assume that the 'stone' is under the right foot as in Figures 5, Step 1, 2, and 3.

Sequence of Movements in the Leg Cross Flexion-Extension Motor Pattern

Part 1-a. Plantar flexion of the right foot provides shock absorption—Figure 5, Step 1.

Muscle Groups	Main Muscles *(contracts)*	Opposing Muscles *(lengthens)*	Facilitating Muscles *(contracts)*
Foot *(plantar flexion for shock absorption)*	Quadratus plantae *(contracts)* Lig plantare longum *(contracts)* Flexor hallucis longus	Extensor digitorum brevis	Lumbricales Extensor hallucis longus Interosei plantares
Knee and tibial area, calf *(slight knee flexion)*	Gastrocnemius Triceps surae Popliteus Flexor digitorum longus Flexor hallucis longus	Extensor digitorum longus Peroneus longus Peroneus brevis Tibialis anterior	Soleus Fascia cruris Plantaris

Part 1-b. Extension of right leg towards stimulus (stone)—to regain equilibrium.

Muscle Groups	Main Muscles	Opposing Muscles	Facilitating Muscles
Knee and tibial area, calf *(extension)*	Gastrocnemius Tricepts surae Popliteus Flexor digitorum longus Flexor hallucis longus Tendo calcaneus (Achillis) *(lengthens)*	Extensor digitorum longus Peroneus longus Peroneus brevis Tibialis anterior *(contracts)*	Soleus Fascia cruris Plantaris *(lengthens)*
Thigh *(leg extends with slight knee extension)*	Rectus femoris Quadratus femoris (Vastus medialis and lateralis) Adductor longus Adductor minimus Gracilis *(contracts)*	Biceps femoris Adductor magnus Semimembranosus Semitendinosus *(lengthens)*	Tensor fasciae latae Extensor digitorum longus Extensor digitorum brevis Extensor hallucis longus *(contracts)*

Part 1-c. Extension of left (opposite) hip, knee, and foot with slight medial adduction —Figure 5, Step 2.

Muscle Groups	Main Muscles	Opposing Muscles	Facilitating Muscles
Calf	Tibialis anterior Peroneus tertius Extensor digitorum longus Extensor hallucis longus *(contracts)*	Gastrocnemius Soleus Flexor hallucis longus Flexor digitorum longus Tibialis posteriori Peroneus longus Peroneus brevis *(lengethens)*	Gastrocnemius Popliteus *(contracts)*
Thigh	Quadricieps femoris Femoris rectus Adductor brevis Sartorius *(contracts)*	Semimembranosus Semitendinosus Gracilis Biceps femoris Adductor magnus *(lengthens)*	Adductor brevis Adductor longus Tensor fasciae latae *(contracts)*
Hip	Psoas major Iliacus Gluteus medius Gluteus minimus Gracilis *(contracts)*	Gluteus maximus Piriformis Lig. sacrospinale Lig. sacrotuberale *(lengthens)*	Tensor fasciae latae Iliopsoas Obturatorius internus *(contracts)*

Part 2. Lateral abduction of flexed left knee—Figure 5, Step 3.

Muscle Groups	Main Muscles *(adducts and contracts)*	Opposing Muscles	Facilitating Muscles
Thigh, hip	Gluteus maximus Gluteus medius Tensor fasciae latae Sartorius Piriformis *(abducts and contracts)*	Adductor magnus Adductor longus Adductor brevis Gracilis Pectineus *(adducting, opposing to abduction and lengthening)*	Rectus femoris Gluteus maximus Gluteus minimus *(abduct and contracts)*

Part 3. Extension of left (opposite) leg in hip, knee, and foot—Figure 5, Step 4.

Muscle Groups	Main Muscles *(lengthens)*	Opposing Muscles *(contracts)*	Facilitating Muscles *(lengthens)*
Hip	Psoas major Iliacus Gluteus medius Gluteus minimus Gracilis	Gluteus maximus Piriformis Lig. sacrospinale Lig. sacrotuberale	Tensor fasciae latae Iliopsoas Obturatorius internus
Thigh	Quadricieps femoris Femoris rectus Adductor brevis Sartorius	Semimembranosus Semitendinosus Gracilis Biceps femoris Adductor magnus	Adductor brevis Adductor longus Tensor fasciae latae
Calf	Tibialis anterior Peroneus tertius Extensor digitorum longus Extensor hallucis longus	Gastrocnemius Soleus Flexor hallucis longus Flexor digitorum longus Tibialis posteriori Peroneus longus Peroneus brevis	Gastrocnemius Popliteus

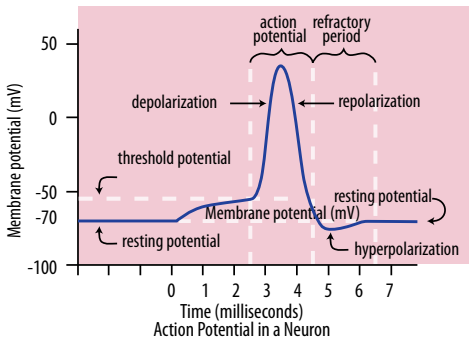
Part 4. Release of foot from stimulus: flexion of right hip, knee, and foot with slight medial adduction—Figure 5, Step 4.

Muscle Groups	Main Muscles	Opposing Muscles	Facilitating Muscles
Calf	Tibialis anterior Peroneus tertius Extensor digitorum longus Extensor hallucis longus *(contracts)*	Gastrocnemius Soleus Flexor hallucis longus Flexor digitorum longus Tibialis posteriori Peroneus longus Peroneus brevis *(lengthens)*	Gastrocnemius Popliteus *(contracts)*
Thigh	Quadricieps femoris Femoris rectus Adductor brevis Sartorius *(contracts)*	Semimembranosus Semitendinosus Gracilis Biceps femoris Adductor magnus *(lengthens)*	Adductor brevis Adductor longus Tensor fasciae latae *(contracts)*
Hip	Psoas major Iliacus Gluteus medius Gluteus minimus Gracilis *(contracts)*	Gluteus maximus Piriformis Lig. sacrospinale Lig. sacrotuberale *(lengthens)*	Tensor fasciae latae Iliopsoas Obturatorius internus *(contracts)*

With the release of the right foot from the stimulus, the protective function of the reflex is accomplished.

III. The Neuro-Sensory-Motor Pathways of the Leg Cross Flexion-Extension Reflex

Figure 6-1. Electrical currents in nerve transmission (changes from -70 mvt at rest to -55 mvt).



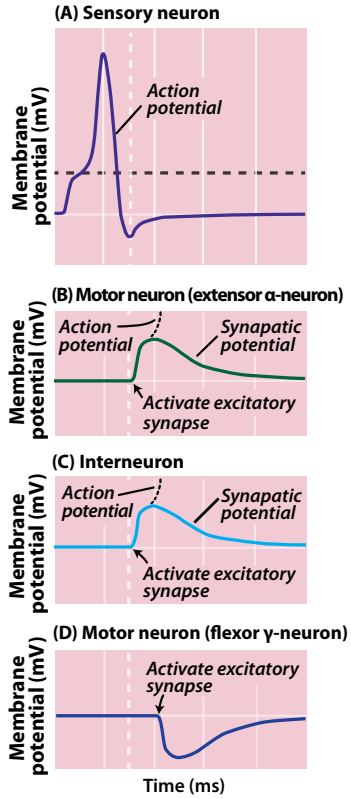
Afferent nerve pathway

A sensory-proprioceptive stimulus (on the right foot arch) generates action potentials that set off a Na+-sodium bio-chemical cascade creating electrical currents (from -70 mvt at rest to -55 mvt) to start the transmission. These currents travel via the bulbo-thalamo-cortical tract to the cortex (Figure 6-1).

Figure 6-2 presents the cellular mechanisms of action potential generation in a sensory receptor and synaptic potentials triggering conducted signals in motor and inter-neurons: A) Action potential measured in a sensory neuron; B) Postsynaptic triggering potential recorded in an extensor motor neuron (α-neuron); C) Postsynaptic triggering potential in an interneuron (γ-neuron); D) Postsynaptic inhibitory potential in a flexor motor neuron (γ-neuron) (Purves, Augustine, Fitzpatrick, et al., 2001; Haines, 2006).

The afferent tract, running from the plantar surface of the foot to the gyrus post-centralis in the motor cortex, provides sensory-proprioceptive sensation to the brain (Haines, 2006; Cohen, 1999; Barker, Barasi, Nil, 2008). It consists of three orders of neurons, working at different levels and all interacting simultaneously to process the sensory-proprioceptive input involved in this reflex:

Fig 6-2 (A, B, C, & D) Action potential generation in sensory and motor neurons.



1. Axons of first order sensory neurons (connected with receptors as described above), enter the dorsal horn of the lumbar spine, travel along the fasciculus gracilis and end in the nucleus gracilis.

2. Axons of second-order sensory neurons start at the same level of the spinal cord, follow ipsilaterally to the lower olives of the medulla oblongata, and continue through the spinobulbo-thalamocortical tract to the medial lemniscus. There they transmit nerve impulses across (decussate) to the other side of the brain.

3. Third-order sensory neurons located in the thalamus direct their axons to the cortex via the thalamo-cortical tract, follow to the corona radiata and end in the postcentral gyrus in the motor cortex (Haines, 2006; Cohen, 1999).

Afferent nerve pathway in more detail

Ascending and processing of the sensory-proprioceptive input in the Leg Cross Flexion-Extension Reflex pattern takes place at several levels of the nerve system.

Lower limb

A sensory-proprioceptive stimulus to the tendon receptors (Golgi, Spindle cell, Meissner, Merkel, and Rufini cells) on the plantar surface of the right foot (Golgi tendons, quadratus plantae muscle) triggers receptors and dendrites of the sensory neurons, reaching their cell bodies in the spinal ganglion, continuing through their axons (proximal nerve fibers closer to spinal cord), following along the fasciculus gracilis and the nucleus gracilis and finally entering the posterior horn of the lumbar spine. Axons providing input on this level are called first-order neurons.

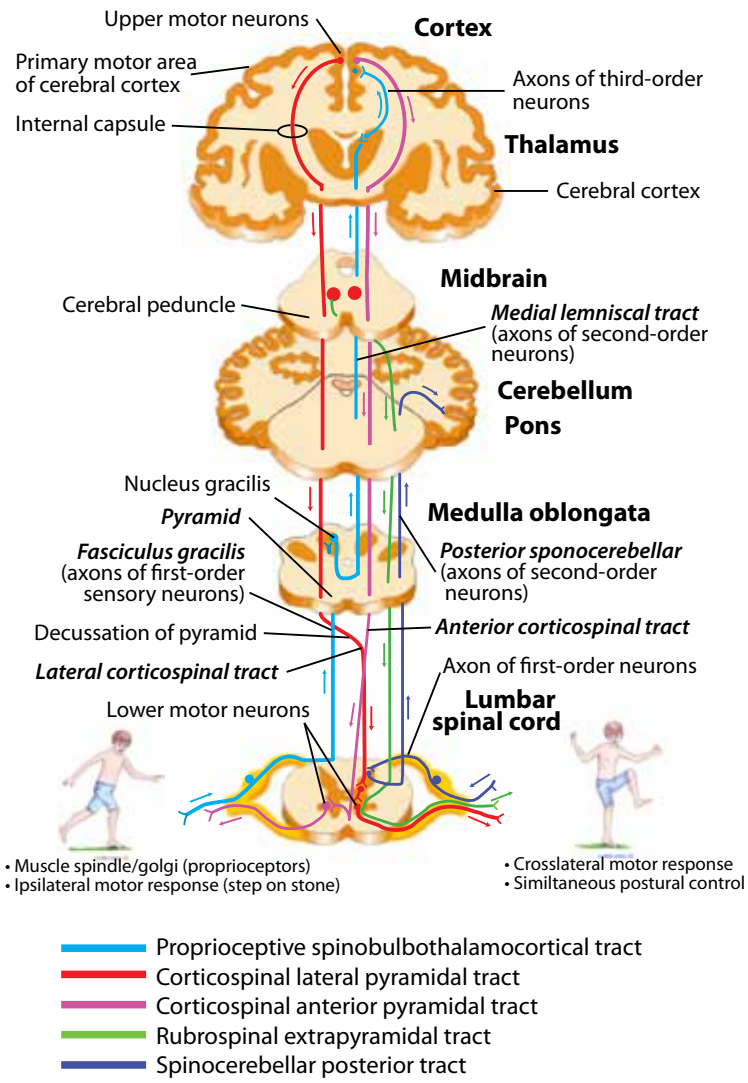


Figure 7. Ascending and descending nerve pathways of the Leg Cross Flexion-Extension Reflex pattern

Spinal Cord

First-order sensory neurons enter the dorsal horn of the lumbar spine, travel along the fasciculus gracilis, and end in the nucleus gracilis. In the middle of the spinal cord their sensory axons connect through synapses with dendrites of two motor neurons, whose axons follow across the spinal cord to the anterolateral horn, forming two separate descending pathways. So on this level, the proprioceptive input is transmitted in two directions:

• One pathway is formed by an α-motor neuron connected directly or through an excitatory interneuron with the sensory neuron. In the synapse, where the sensory and motor neurons connect, excitatory neurotransmitters such as substance P, glutamate, and CGPR (calcitonin gene-related peptide) are released to increase the action potential in the α-motor neuron and its interneuron.

• The other pathway is formed by the γ-motor neuron, connected with sensory neuron indirectly through an interneuron, whose synapse serves for the release of inhibitory dopamine and

GABA neurotransmitters. These neuro-mediators control the speed and strength of the action potential by inhibiting the impulse in order to regulate the tone in the intra- and extrafusal muscle fibers of both agonist (facilitating) and antagonist (opposing) muscles.

In synapses at the ends of axons of both α- and γ- motor neurons, where they connect with muscle fibers, the acetylcholine neurotransmitter is released to support the transmission of action potential to muscle fibers. The α-motor neuron in part 2 of Leg Cross Flexion-Extension causes the leading agonist muscle (left leg hamstring) to contract, and the γ-motor neuron inhibits and regulates the antagonist muscle (quadriceps, supporting lengthening in the same left leg). This muscle tone regulation results in flexion of the left leg. Other elements of this motor pattern occur thanks to similar interactions between sensory and motor neurons in their complex circuitry.

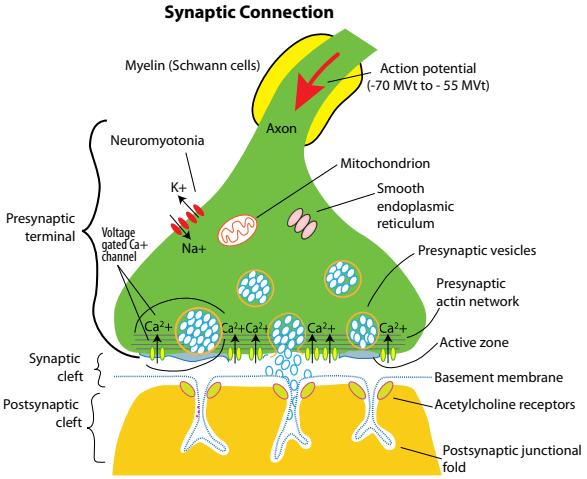


Figure 8. The mechanism of connectivity in a synapse.

Note 1: Synaptic connectivity results from currents of directed potential created biochemically by the Na+ and K+ ions (D.E. Haines, 2006, 2013; H. Cohen, 1999). Synaptic connectivity includes the work of an inhibitory synapse, which produces effects opposite to those of the excitatory synapse: the action of the transmitters on the receptor sites is to open those ionic channels which hyperpolarize the postsynaptic membrane. Typically these are the K+ and Cl- channels. The chemical gradients of these two ions are such that K+ diffuses outward while Cl- diffuses inward. This combination of ionic fluxes hyperpolarizes the membrane so that the internal potential becomes even more negative than the resting state. An inhibitory postsynaptic potential (IPSP) is established thanks to that. The potential is called inhibitory because the membrane potential is even farther from the excitation threshold than in the resting state (D.E. Haines, 2006; H. Cohen, 1999; R. Barker, S. Brasi, M. Nil, 2007). This process must correlate with the stimulus. The inhibitory mechanism in synaptic connectivity is a part of the nervous system functioning that contributes to:

- *regulation of sensory input and processing*
- *slowing down the flow of sensory information (especially in γ-motor neuron)*
- *control of overlapping sensory input*
- *transition of short-term memory into long-term cellular memory.*

Note 2: Dysfunction in reflex circuitry can happen in any of its parts: receptors (poor development of specific receptors and their interlinks), connection of dendrites with the axon though the cell body of a sensory neuron, in action potential (conductivity of electrical potential, Na-sodium relations, mitochondrial deficiencies), neurotransmitters at synapses (pre-synaptic, synaptic or post-synaptic processes), the motor neurons, and also effector organs (muscles and glands). Lack of adequate neural regulation in the work of a sensory neuron and its connection with a motor neuron is caused basically by imbalance between excitatory sensory and inhibitory neurotransmitters released in the interneuron, which affects the tone and coordination of opposing muscles. This kind of imbalance affects the quality of reflex circuit function, challenging the processing of input and blocking the control circuits of the reflex in the brainstem and diencephalon.

Medulla and Pons (Brainstem)

Part of the proprioceptive input in the Leg Cross Flexion-Extension Reflex travels to the medulla through axons of second-order neurons via the bulbothalamocortical tract. This afferent tract provides proprioceptive sensation to the cortex. These neurons cross contra-laterally in the lower olives of the medulla oblongata and continue to travel via the bulbo-thalamic tract to the medial lemniscus and finish in the ventroposterior thalamic nucleus. Repetitive eliciting of this reflex in our infancy and childhood allows for myelination of the axons of second-order neurons in the medulla and pons. This is the part of the brain that deals with integration of the neuro-structural aspect of a reflex and its function of protection and survival. Thus survival strategy, the ability to process danger on the sensory level and to supply grounding and stability in dangerous situations when the body is destabilized, results from the work of this reflex in the brainstem. Input from this reflex also links the brainstem (medulla, pons, and midbrain) with the cerebellum and thalamus. This level of the brain completes the processing, preparing the physical action of the Leg Cross Flexion-Extension motor pattern to provide immediate protection: stepping harder on the stone (right leg) and flexing the left leg and extending

it to regain stability on safer ground. This most important part of processing Leg Cross Flexion-Extension input is automatic and entirely unconscious.

Thalamus

Third-order neurons are located in thalamus and direct their axons to the cortex along the thalamo-cortical tract. Input from the Leg Cross Flexion-Extension Reflex that reaches this level is processed here and coordinated with sympathetic nervous system responses to activate the stress axis (alarming the organism about danger and activating an increase of stress hormones). Third-order neurons form one third of the posterior limb of the internal capsule, next they follow to the corona radiata and finish in the post central gyrus, where fourth order neurons are located. The thalamus controls the circuit of the Leg Cross Flexion-Extension Reflex performed at the level of the medulla and pons. It determines the relative danger or safety of a stimulus and then regulates sympathetic activation, muscle tone and motor activity/reactivity accordingly.

Sensory Cortex

Third-order neurons from the thalamus follow along the same thalamo-cortical tract via the corona radiata and finish in the post central gyrus, where they reach the fourth order neurons. This final level of the brain controls the voluntary work of all the main muscles in the body and, according to I. Sechenov (1961), serves as the physiological basis for the psychological process of creating an 'image' of the stimulus: "Is it a stone? Is it a piece of wood, a pen?" Here the brain uses its executive functions to analyze sensory input. The whole cortex becomes involved in decoding and analyzing the input, organizing subsequent output to the muscles and making any movement more precise, goal oriented, and meaningful.

Efferent/descending nerve pathway

There are two principal groups of descending tracts: one serves for postural/gross motor coordination and control, and the other for voluntary control of fine motor activity. Descending tracts classified as postural/gross motor tracts in the Leg Cross Flexion-Extension Reflex control automatic responses of the skeletal muscles to extend one leg (right foot on the stone), and to flex, abduct, and extend the opposite leg (left).

The first group of tracts involves the purely automatic motor response and is generated from the lumbar spine, where descending innervation takes place in its different segments. The excitation of motor neurons activates the anterior leg muscles (quadriceps) for movement and depolarizes the contralateral motor-neurons of the hamstring muscles (biceps, semitendinosus, and semimemranosus), with simultaneous inhibition of motor neurons innervating antagonistic muscles creating the possibility for one leg to extend (right; led mainly by the quadriceps), and other leg to flex, then abduct and extend (left; led mainly by the biceps).

The second group of descending tracts delivers motor information from the high brain to lower motor neurons of the brainstem or spinal cord and is more involved with voluntary control. These descending tracts are formed by upper motor neurons that: a) arise in the cerebral cortex and descend to the brainstem; b) synapse with lower motor neurons and/or interneurons in the brainstem and spinal cord. They would be involved in conscious direction of the left leg forward, backward or to the side, depending where the safest place to land might be.

Control circuits of the Leg Cross Flexion-Extension Reflex operate from the basal ganglia, cortex, and cerebellum, which is responsible for planning automatic responses and adjusting to external circumstances on different levels of the nerve system.

Descending motor pathways through different levels of the nerve system

Motor Cortex

Descending motor pathways in Leg Cross Flexion-Extension present sequential and simultaneous work of the whole pyramidal and extrapyramidal nerve systems. The pyramidal system involves the corticospinal tract (lateral and anterior-pyramidal tracts), which governs rapid voluntary movements at the distal ends of limbs. They originate in the primary (Broadmann's area 4 – motor cortex) and secondary (area 6 – premotor cortex) motor cortex and the parietal lobe (areas 1, 2, 3 – somatosensory cortex). This tract decussates in the pypramids and descends as the lateral corticospinal tract to its destination: an inter- or α-motor neuron. Branches of this tract are present in: the cerebral cortex, basal nuclei, red and olivary nuclei, and the reticular formation.

This part of the descending pathway involves the pyramidal upper motor neurons, called first order motor neurons because they are unable to leave the central nervous system. Multi-synaptic, they connect with lower motor neurons to send messages to the muscles. Upper motor neuron lesions can lead to dysfunctional Leg Cross Flexion-Extension responses, as happens in individuals with CP or brain damage.

Note: 80% of the pyramidal cells are located on the precentral gyrus in the frontal lobe (motor strip), 20% of the pyramidal tract fibers originate in the postcentral gyrus of the parietal lobe (areas 1, 2, and 3). Pyramidal tract fibers begin their descent from the cortex (corona radiata) before forming the internal capsule. They are direct and monosynaptic; their axons do not synapse with other cells until they reach their final destination in the brainstem or spinal cord. Because they connect directly from the cortex to the lower motor neurons, messages can be transmitted to the periphery very rapidly.

Subcortical and Extrapyramidal tracts (diencephalon and brainstem)

The efferent output from the cortex travels via the frontopontine tract and next along the rubrospinal tract. These extrapyramidal tracks include:

- tectospinal tracts
- vestibulospinal tracts
- rubrospinal tracts
- anterior, medial, and lateral reticulospinal tracts

The tectospinal tract is involved in the control of neck muscles. It originates in the midbrain (in the brainstem) and ends in the spinal nerves, thus governing the lowered head position in the Leg Cross Flexion-Extension Reflex pattern.

The vestibulospinal tract runs from the vestibular nuclei (lower pons and medulla) to the spinal nerves. It is involved in balance. (Note that all of these tracts receive input from the cerebellum). In Leg Cross Flexion-Extension this tract serves to keep the body in equilibrium until the legs react contra-laterally.

The rubrospinal tract passes through the red nucleus, carrying messages from the cerebellum to the spinal nerves. Information in this part of the tract flows from the superior cerebellar peduncle to the red nucleus and to the spinal nerves, allowing for somatic motor or skeletal muscle control and regulation of muscle tone for posture and voluntary movement. In Leg Cross Flexion-Extension this track will control the direction and tone of opposing muscles to make possible this extremely complex motor response.

The reticulospinal tract runs from the reticular nuclei of the pons and medulla to the spinal nerves. It is involved in somatic (voluntary) motor control, like the rubrospinal tract, and also plays an important role in the control of autonomic (involuntary) functions in the Leg Cross Flexion-Extension Reflex such as the level of stress hormones released by the sympathetic/parasympathetic system, and regulation of breathing, heart rhythm, blood pressure).

Thalamus

The efferent fibers from the cortex and subcortical centers (including control circuits of the Leg Cross Flexion-Extension Reflex) of extrapyramidal system follow along the frontopontine, vestibulospinal, and rubrospinal tracts. After their cross-over (Foreli decussation) in the red nucleus they reach γ-motor neurons.

Control circuits of the Leg Cross Flexion-Extension Reflex reach the cortex, basal ganglia, and cerebellum which:

- regulate the excitation or inhibition of the lower motor neurons
- partially determine intensity of muscle contraction
- adjust the motor activity according to sensory-proprioceptive input.

Midbrain

Our study leads us to suppose that in the midbrain (tegmentum, pre-tectum, tectum) the descending pathway of the Leg Cross Flexion-Extension Reflex provides automatic 'assessment' of relative danger/safety and adjusts cortical programing to find a safer spot on the ground, causing abduction in the hip joint in part 3 of the pattern to move the knee and foot forward and away from the source of harm or danger.

Pons

Superior to the medulla, the pons consists of white matter with four cranial nerves attached to it. This part of the CNS includes tracts that conduct signals from the cerebrum down to the cerebellum and medulla, and tracts that carry sensory signals up into the thalamus, thus connecting the cerebellum to the pons and midbrain. Some nuclei in the pons relay signals from the forebrain to the cerebellum; others deal primarily with respiration, bladder control, hearing, equilibrium, eye movement, facial expressions, and posture. Through these connections the pons participates in control of equilibrium (vestibular system), coordination of motor, visual and auditory systems, breathing, and possible bladder activation when the Leg Cross Flexion-Extension response is triggered.

Medulla oblongata

This level of the brain is crucial for development of the Leg Cross Flexion-Extension Reflex and the timely emergence of crawling, walking, running, jumping, marching, and swimming in children, thus we describe it in more detail. The medulla oblongata is the inferior part of the brainstem between the spinal cord and the pons. It consists of: olives, pyramids, the roots of four cranial nerves (CN XII, IX, X, XI), the inferior cerebellar peduncle and the fourth ventricle.

The anterior of the medulla has two vertical bulges called pyramids. They contain axons of the corticospinal tract projecting from the cerebral cortex to the spinal cord. In the lower part of the medulla some of these fibers cross each other at the anterior median fissure known as the decussation of the pyramids (also called low motor extrapyramidal cross-over). Other fibers, the external arcuate fibers, originate from the anterior median fissure above the decussation of the pyramids and run laterally across the surface of the pons.

Beside the pyramids are two small oval lumps called olives formed by nerve fibers that connect them to the pons and the cerebellum. They have a role in motor learning.

The fourth ventricle, containing cerebrospinal fluid, forms the dorsal surface of the superior part of the medulla, where the four cranial nerves are rooted, and extends within the inferior part.

The task of the medulla oblongata in Leg Cross Flexion-Extension is control over the very complex physiological circuits and bio-dynamics of cross-lateral responses (crawling, walking, running, marching, jumping, climbing, swimming), also muscle tone, posture, respiration, heartbeat, and blood pressure. Patients with brain damage can still have functioning bodies, as long as the medulla oblongata is working. The Leg Cross Flexion-Extension test can show the level of medulla oblongata function in both healthy and persistent immobile states.

Spinal Cord

At the level of the anterior horn of the spinal cord the output follows via the frontopontine, vestibulospinal, and rubrospinal tracts, crossing over in the area of the red nucleus (Foreli) to reach the α- and γ-motor neurons of flexor and extensor muscles of contralateral limbs.

Muscular and Motor Response

Joints are controlled by two opposing sets of muscles, extensors and flexors. Thus, when a muscle spindle is stretched and the stretch response is activated, the opposing muscle group must be inhibited to prevent it from working against the resulting contraction of the homonymous muscle. This is accomplished by an inhibitory interneuron in the spinal cord.

At this level the motor neurons carry acetylcholine neurotransmitters: α-motor neurons release excitatory acetylcholine to cause a tonic contraction of protagonist muscle fibers; at the same time γ-motor neurons release inhibitory acetylcholine to cause lengthening of the antagonist muscle fibers.

In Leg Cross Flexion-Extension, one branch of the descending pathway innervates the α-motor neuron causing the homonymous muscle (same side quadriceps) to contract, producing the leg extension movement in the reflex. The other branch innervates the inhibitory interneuron, which in turn innervates the synapses connecting to the opposing muscle (same side hamstrings). Because the interneuron is inhibitory, it prevents the opposing α-motor neuron from firing, thereby protecting the opposing muscle against over-contraction and damage. The reciprocal excitation-inhibition innervates two opposing sets of muscles in synchrony: flexors and extensors causing the needed motor responses in the legs.

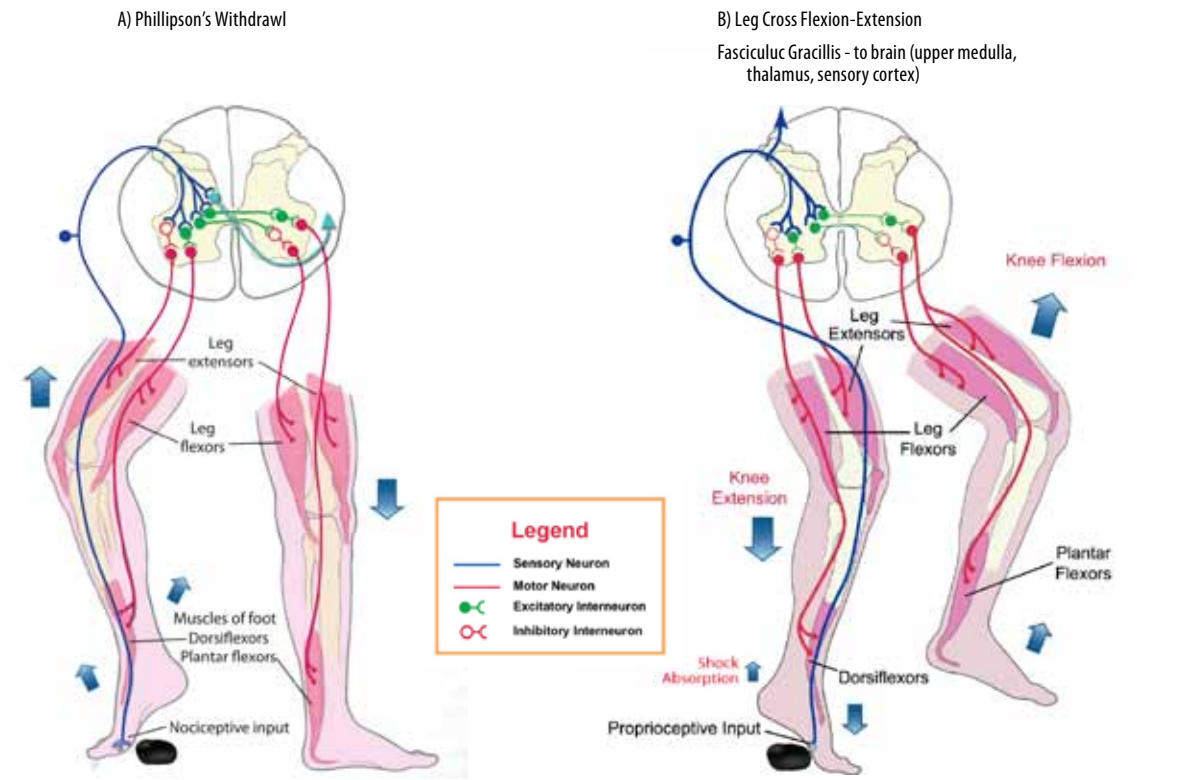
Note: the reticular activating system (RAS), a group of neurons coming from the thalamus and following along the brainstem, regulates arousal and inhibitory mechanisms and provides survival/protection in stress (stone under foot). It is composed of several neuronal circuits connecting the brainstem to the cortex. It plays the role of a filter for defining a stressor and its components and regulates thalamo-cortical activity and corresponding behavioral states. When the sensory input is 'not safe' the RAS system activates automatic survival responses, regulates the flow of sensory input, posture, equilibrium, motor coordination, head movements, orientation to the vertical plane, and postural adjustments needed for defensive responses. Disorders of the RAS can lead to too much arousal and hyperactive Leg Cross Flexion-Extension, causing constant running and jumping in children and adults. Known for regulation of attention, in the Leg Cross Flexion-Extension response it allows the choice of a safe spot for the opposite foot. There are links between RAS circuits and physiological pain pathways, so when the Leg Cross Flexion-Extension and Phillipson's Withdrawal are mixed, the Leg Cross Flexion-Extension can recruit nociception (pain receptor activation) instead of proprioception and compromise the whole Leg Cross Flexion-Extension circuit, causing poor cross motor coordination for walking and running (hyperactive and hypoactive, immobility), poor balance, frequent falls, injuries, as well as emotional and behavioral challenges.

IV. Neurophysiological Tracts for Leg Cross Flexion-Extension Reflex & Phillipson's Withdrawal

To produce Phillipson's Withdrawal, pain receptors in the skin send a rapid message though the afferent nerve fibers, bringing input to the posterior horn of the spinal cord. The signal then reaches out the ventral/front side of the spinal cord to muscles in both legs causing them to cooperate simultaneously: the leg that stepped on the stone must flex its knee and hip joints to lift the foot off; the opposite leg must immediately bear the full body weight (Andersen, Sonnenborg, Arendt-Nielsen, 2001; Clair, et al., 2009, Mileva, Green, Turner, 2004; see Figure 9). The input is transmitted across from the stimulated side of the body (right foot) to the contralateral anterior horn of the spinal cord and on to synapse with interneurons, which, in turn, excite or inhibit alpha motor neurons leading to the muscles of the contralateral limb (left leg) for extension of that leg and to the same side limb for pulling the injured right foot away from danger, all within 0.5 seconds (Solomon, Schmidt, Adragna, 1990). So, the Phillipson Reflex is elicited by nociceptor stimuli (pain, free nerve end receptors), not proprioceptive as in the Leg Cross Flexion-Extension Reflex. The main area for processing of nociception is at the level of first order neurons in the spinal cord (lumbar level). The biomechanics of this reflex are the following: in the ipsilateral leg (right), the flexors contract and the extensors relax to lift it off the ground. On the contralateral side (left), the flexors relax and the extensors contract to keep the leg extended and stable, since it must suddenly support the entire weight of the body. At the same time, some signals travel up the spinal cord and cause contraction of the contralateral hip and abdomenal muscles to shift the body's center of gravity to the extended left leg. To a certain extent, the coordination of all these muscles, maintenance of equilibrium and feeling of the pain are mediated by the cerebellum and cerebral cortex (Spaich, Arendt-Nielsen, Andersen, 2005). However, the motor response in Phillipson's Withdrawal is generated mainly from the spinal cord, not from the brainstem (medulla oblongata) as in Leg Cross Flexion-Extension.

The Leg Cross Flexion-Extension response is elicited by a proprioceptive stimulus and uses a different nerve

Figure 9. Pathway for A) Phillipson's Withdrawl and B) Leg Cross Flexion-Extension



tract – reaching the posterior horn of the spinal cord and transmitting the input ipsilaterally to the same side for the leg to extend (stepping even harder on the stone), and another sensory axon synapsing with second-order neurons sends the message to decussate (cross over) in the medulla oblongata (brainstem) to activate descending motor pathways in the opposite side to cause flexion, abduction and extension of the left leg so it can step away and ground the body in a safe spot. Thus, Leg Cross Flexion-Extension differs from Phillipson's Withdrawal as it is a more complex pattern, uses different ascending and descending nerve pathways and is processed mainly at a higher level of the CNS - medulla oblongata (Magnus, 1926). It is important to understand this difference as it should determine both the procedure used for repatterning and success of rehabilitation.

Conclusions

The bio-dynamics and neurophysiology of the Leg Cross Flexion-Extension Reflex schema is different from that of Phillipson Withdrawal:

• The Leg Cross Flexion-Extension Reflex is elicited by sensory-proprioceptive receptors. Organization of balance follows in first priority, and then other systems are activated (tactile/pain). The stimulus must reach the level of medulla oblongata for transmission across to the opposite leg. It also will reach the cortex level for organization of balance organization and registration of pain.

• Phillipson's Withdrawal is triggered by a nociceptor stimulus (tactile system/pain). The sensation of pain activates in priority the withdrawal away from the stimulus, and then other systems are activated for organization of balance. Contralateral stimulation of motor neurons in this reflex is reached in priority in the lumbar spine. The sensory neuron also sends signals up higher than the spinal cord to maintain balance and stabilize the body (this is why it is also called the Crossed Extension Reflex). On the level of the cortex this reflex allows the feeling and awareness of pain (Spaich, Arendt-Nielsen, Andersen, 2005).

MNRI® work with the Leg Cross Flexion-Extension Reflex activates pathways transmitting the sensory-proprioceptive stimulus, not those involved with nociception as in Phillipson's Withdrawal. This difference is essential for proper application of neuro-corrective techniques for patients.

V. Practical Application: The Leg Cross Flexion-Extension Repatterning Exercise

There are several reasons to repattern/pattern the Leg Cross Flexion-Extension Reflex pattern in children and adults:

• delay in development of the reflex pattern

• pathology in the reflex pattern caused by brain damage, cerebral palsy or a lesion in lower or upper motor neurons

• faulty connections in sensory and motor axons

• poor work of the alpha- and gamma- motor neurons (inadequate production of the GABA and dopamine, and acetylcholine neurotransmitters)

• nerve transmission mixing the Leg Cross Flexion-Extension and Phillipson's Withdrawal patterns

• negative compensation, trauma, PTSD.

In all these cases the repatterning/patterning of the physiological circuit is suggested. Training this reflex in infancy and childhood promotes myelination of second-order neuronal axons in the medulla and pons. Such training can also strengthen or rebuild these networks whenever they become dysfunctional. Because this part of the brain integrates the neuro-structural aspect of a reflex with its protective function, it is essential for survival strategies. In the case of Leg Cross Flexion-Extension, the ability to process danger on the sensory level and to supply grounding and stability when the body is destabilized, originates in the brainstem.

Processing danger and providing a survival strategy are automatic and entirely unconscious. If these functions are not accomplished efficiently in the brainstem and its midbrain, then higher more conscious levels of the brain are recruited. Development and maturation of higher-level skills then suffer when the cortex must be engaged for protection and survival and is not free to support voluntary, conscious and skillful activity. Children with developmental issues have a particularly urgent need (see Part I: Effects of Non-Integration) for repatterning of the Leg Cross Flexion-Extension Reflex.

The MNRI® Leg Cross Flexion-Extension repatterning exercise, based on its sensory-motor pathway, consists of four steps presented below (Masgutova, Akhmatova, 2004, 2007, 2010).

Figure 10. Points for stimuli on right foot arch and above the knee.

Step 1

Intervention A: Press on the center of the right foot's quadratus plantae muscle to simulate the sensory-proprioceptive stimulus of stepping on an object (See Figures 4 and 10). Hold for 5 – 7 seconds.

Reflex motor pattern: stretching and forced raising of the right arch (Sinelnikov, 1942) caused by stepping on an object.

Intervention B: Press and stretch on lower 1/6th of the quadriceps to trigger extension of the knee and hip (Figure 10). Hold for 5 – 7 seconds.

Reflex motor pattern: extension of right knee and hip, stepping harder on the object

Main muscles: Quadratus plantae, quadriceps, adductors, poplitus. Quadratus plantae, abductor hallucis, flexor digitorum longus and brevis

Opposing muscles: tensor fasciae latae, sartorius, extensor hallucis brevis, extensor digitorum brevis

Optional intervention: Perform steps A and B simultaneously.

Step 2

Intervention: Bring left knee and hip into flexion with slight medial adduction. (see Figure 11) Hold 5-7 seconds.

Reflex motor pattern: Flexion of opposite (left) knee and hip

Main leg muscles: semimembranosus, semitendedinosus, biceps femoris, gracilis, sartorius

Opposing leg muscles: gastrocnemius, popliteus

Facilitating leg muscles: quadriceps

Main hip muscles: psoas major, iliacus, rectus femoris, gluteus medius, gluteus minimus, adductor brevis

Opposing hip muscles: iliopsoas, tensor fasciae latea, pectineus, adductor longus, adductor brevis

Facilitating hip muscles: gluteus maximus, biceps femoris, semimembranosus, semitendinosus, adductor magnus.

Figure 11. Bring the left knee and hip into flexion.

Step 3

Intervention: Rotate the hip, bringing the knee away from the midline. (see Figure 12) Apply traction first between the hip and knee, then between the knee and ankle. Hold each stretch 5-7 seconds.

Reflex motor pattern: Lateral rotation of hip, knee abduction

Main muscles: gluteus medius, gluteus minimus, adductor magnus, piriformis, gemeli, iliopsoas

Opposing muscles: sartorius, gracilis

Facilitating muscles: tensor fasciae latae, rectus femoris, adductor magnus, biceps femoris

Figure 12. Rotate the hip laterally by pulling the knee to the side, stretch from hip to knee and stretch from knee to ankle.

Step 4

Intervention: Extend the flexed knee/hip and stretch the whole leg downward, holding it above the ankle joint. (see Figure 13, below)

Reflex motor pattern: extension and grounding of the flexed knee/hip to support the body's weight on a safe spot.

Main muscles: quadriceps femoris, gluteus maximus, semimembranosus, semitendinosus, adductor magnus

Opposing muscles: gluteus medius, gluteus minimus, adductor brevis

Facilitating muscles: biceps femoris, gracilis, sartorius, psoas major, iliacus, rectus femoris, gluteus medius, gluteus minimus, adductor brevis mm.

Figure 13. Extend and stretch the whole leg.

Step 5

Repeat steps 1 – 4, beginning with stimulus on the left foot.

This exercise can be done 2-3 times on one foot/opposite leg, and then on the other foot/opposite leg. Do a total of 5-7 repetitions, working slowly.

The use of this Leg Cross Flexion-Extension exercise procedure has led, during many years of the MNRI® practice, to highly significant changes in cases of severe dysfunction, such as cerebral palsy and brain damage, allowing them to learn crawling and walking. It allows individuals with autism to reset an improper hyperactive Automatic Gait pattern (toe walking, constant running, and jumping) and learn normal, matured reciprocal walking. It allows children with dyslexia to acquire differentiation functions and athletes to gain precision in their cross-lateral movements. This is a pattern which supports proper neurological pathways and CNS plasticity, and thus makes our gross movements more goal-oriented, controlled, precise, flexible, and easy.

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Our dear patients, children and adults! We wish for you, proper neurophysiological work of the Leg Cross Flexion-Extension Reflex circuit and for it to serve well in your walking, running, climbing, swimming and jumping – in everyday life, sports, and dances. – Authors