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The “primitive reflexes” are those that appear and disappear in an expected order during infant development.

They are important signs of a normally developed nervous system when present at the right stage of development. Likewise, abnormal absence of an expected reflex at a given stage of infancy, or the presence (i.e., the abnormal return) of a reflex implies some dysfunction within the nervous system.

Primitive reflexes are inborn actions originating in the central nervous system.

They are exhibited by normal infants and, contrary to other authorities, this author believes them to be demonstrably intact in adults also, in response to particular stimuli. Most observers believe that these reflexes disappear as a child moves through normal development, but it seems more appropriate that they are modulated by the functioning of the frontal lobes.

Older children and adults with atypical neurology (primarily thought to be those with cerebral palsy) may retain these reflexes, and they may re-appear in adults due to certain neurological conditions including, but not limited to, dementia, traumatic lesions, and strokes. However, their atypical display may also be seen in certain functional disorders.

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Receptor Based Solutions™ for Your Tonic Neck Reflex
Taking up the “Fencing Posture”

The neck receptors give rise to a primitive reflex—the Tonic Neck Reflex (TNR)—found in newborn humans. Books say that the TNR normally vanishes by the child's first birthday, but that appears to be functionally and demonstrably untrue. The TNR persists throughout life and is a fundamental tool for neurological examination.

The TNR is fundamental to and inseparably fused with balance; they reinforce each other in their effects on the upper extremities and oppose each other in their effects on the lower extremities. However, the aspects of steadiness seem to be the more dominant reflex for lower extremity patterning.

The TNR arise via stimulation of joint receptors in the neck (especially around the atlantooccipital and atlantoaxial joints) when the head is inclined forward, backward, or sideward, or rotated to either side. That is, **turning the head to the left should cause a functional facilitation of the extensors on the side toward head rotation and the flexors on the side opposite head rotation. This rule applies to both upper and lower extremities.**



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Case Study: Linda (47yo) had been having lower back pain for the past 2 weeks. It started after lots of bending and twisting, then reaching overhead to stock the shelves in her store. She had also been having some vague whole-head headaches.

During Linda's exam, both the psoas major muscles appeared to be able to meet the demands of manual muscle testing. However, relative to the TNR, the psoas major displayed a functional facilitation on the side toward head rotation, and a functional inhibition on the side opposite head rotation.

Palpation of the lower cervical spine indicated pathological motion. Challenging the cervical spine in one direction and treating it accordingly brought about the expected psoas display.

Discussion: Relative to the TNR rule, we know that Linda's psoas display was “other than human.” Its dysfunction indicated that a main low back stabilizer was working contrary to its original programming, and that may indicate a neck problem.

A functional TNR display indicates that low back stability is the result of cervical receptor stimulation—cervical spinal motion. Further, it suggests

that upper body performance may be more primary than lower body movement; the lower body follows upper body.

In the absence of TNR influence, the postural demands of bipedal posture would be impossible. While a person can certainly move around despite TNR dysfunction, their optimal performance level would be less than human.

Further, the TNR is not an isolated part of the human neurological design. Afferent signals also rise to the cerebellum, midbrain, and cortex. Faulty input at these levels has a high probability of relating to motor errors and structural problems.

Summary: TNR display does indeed persist beyond the toddler stage. It is a fundamental part of normal human movement and displays itself both functionally and dysfunctionally. The functional display encourages neurological stability while the dysfunctional display means certain performance error.

(Cont from front column)

An individual with cerebral palsy and typical intelligence can learn to suppress these reflexes, but the reflex might resurface under certain conditions such as during an extreme startle reaction. Further, a dysfunctional input secondary to movement error can also cause a lack of inhibitory controls of these reflexes.

Reflexes may also be limited to those areas affected by the atypical neurology, such as individuals whose cerebral palsy affects only their legs retaining the Babinski reflex but having normal speech in individuals with hemiplegia, the reflex might be seen in the foot on the affected side only. Conversely, a cortical deafferentation may also cause a similar but Babinski-like response in the absence of frank neurological pathology. However, these cases make the point that the reflexes remain on display but are more properly modulated by higher order neurological development.

Primitive reflexes are also tested with suspected brain injury to evaluate frontal lobe function. If they are not being modulated properly, they are called "frontal release signs." Research potentially indicates that atypical primitive reflexes may be a likely early warning sign of an autistic spectrum disorder.

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For more information about Dr. Allen's two books—*What Your Brain Might Say if It Could Speak* and *Receptor Based Solutions*[™]; *Functional Neurology Every Doctor Should Know*—go to www.receptorbasedsolutions.com.



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