eISSN 1303-1775 • pISSN 1303-1783

Review Article

An Essay on the Human Corticospinal Tract: History, Development, Anatomy, and Connections

Published online 24 May, 2011 © http://www.neuroanatomy.org

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Received 24 March 2011; accepted 19 May 2011

ABSTRACT

The corticospinal tract is arguably the most important descending tract in the central nervous system. It has been recognised since the seventeenth century, but an accurate description of its pathway in humans is still elusive. The advancement of studying methods including magnetic resonance modalities has lead to great leaps in our understanding of the pathway.

It is evident that the tract has an extensive origin in the cerebral cortex, and follows a fairly straight and predictable course in the brain and spinal cord. Although it is asymmetric in most, the asymmetry itself is independent of hand-dominance. The tract modulates motor functions and its terminations therefore either synapse on lower motor neurons or interneurons. © **Neuroanatomy. 2011; 10: 1–4.**

Key words [Corticospinal Tract] [Anatomy] [Fibre Somatotopic Arrangement] [Upper Motor Neuron] [Lower Motor Neuron] [Reflex Arc]

Introduction

The corticospinal spinal system, one of the most important descending tracts in the central nervous system, has been recognised since the seventeenth century. An accurate delineation of the complete tract, however, is only recently emerging with the advent of functional magnetic resonance imaging, like diffusion-tensor imaging. Serving a number of motor functions essential for our voluntary movements, the exact pathway it follows is of great interest to neurophysiologists, neuroscientists, neurologists and neurosurgeons alike, as it will allow a better understanding of the lesions involving it such as infarcts and tumours. This mini-review will therefore describe the pathway this tract follows in the brain and spinal cord and allude to its connections.

Historical Background

An historical review by Nathan and Smith [1] traces mapping of the corticospinal tract back to Willis who, in 1664, described the medullary pyramids. Work was later done to follow the tract both superiorly and inferiorly, but it was not until 1851 when Türck first adequately traced the fibres distally and found two separate tracts: the lateral corticospinal tract, and the anterior corticospinal tract. However, he did not establish that both the lateral and anterior tracts were divisions of the same pathway, a connection that was later established by Charcot in his rather accurate description of the corticospinal (or pyramidal) tract [2].

Most of this early work in humans was done through the post-mortem study of samples with specific lesions that were associated with a reduction in the size of the related tracts. Other studies, mostly in non-human animals, employed retrograde and anterograde labelling using silver staining, horseradish peroxidase, fluorescent and immunolabelling, as well as viral trans-neuronal markers [3-5] that could not be used in living human subjects. Recently, the use of diffusion tensor imaging (DTI) has greatly advanced the non-invasive mapping of this tract in humans [6-8].

Origin and Development

The corticospinal tract, as its name implies, originates from the cerebral cortex, and in particular from the layer V cells (Betz, or pyramidal, cells) [9]. Over 60% of the fibres are from the primary motor area, the premotor area, and the supplementary motor area of the frontal lobe [10,11]. Other fibres take origin from the primary sensory area, the parietal cortex, the parietal operculum [4], and, at least in macaque monkeys, from the cingulate gyrus [12]. One study, involving children up to the age of 17 years [13], found that the corticospinal tract originated from both the pre- and post-central gyri in 71.4% of patients, while it took origin purely from the post-central gyrus in 7.1% emphasising the substantial non-motor origins of the tract.

Studying the development of the corticospinal tract provides the best explanation for the aetiology of

this distribution of origin, and its variation among individuals. In the rat and non-human primate models, the corticospinal tract has been shown to stem origins from a much larger area of the cortex during intrauterine development, including the occipital lobe, than in the adult animal. Selective elimination of these projections as the animal grows and develops results in the maturation of the corticospinal tract [14,15], an observation extended onto human foetuses [3,16].

Pathway Followed by The Corticospinal Tract

The cells belonging to CST project their axons through the *centrum semiovale* and follow a predictable, fairly straight, course through the cerebral cortex [17]. These run in the *corona radiata* lateral to the posterior half of the lateral ventricle, with fibres representing the lower limb being most posterior, those representing the face most anterior, and those of the arm in between [18]. Another study by Song [19] showed that the fibres ran more medially posteriorly and more laterally anteriorly, maintaining the previously mentioned arrangement.

The fibres then descend in the posterior limb of the internal capsule beginning anteriorly, and shifting posteriorly with more caudal sections, but occupy the anterior portion of the posterior half for the most part of their course [7,20,21]. As in the *corona radiata*, the fibres here maintain their somatotopic organisation with those representing the hand anterior to those representing the foot. Some studies have shown these as anterolateral, while others showed them as anteromedial [7,22].

The corticospinal tract then enters the midbrain through the cerebral peduncle, where it occupies the middle third of the *crus cerebri* [23,24]. The fibres of the tract are arranged here somewhat differently with those representing the face being most medial, those representing the hand in the middle, and those representing the foot lateral [5,24,25]. They then course through the anterior pons, the *basis pontis*, with the fibres representing the hand in the anteromedial portion, and those representing the foot posterolateral to them [26]. After that they travel through the anterior medulla where they form the medullary pyramids on either side of the midline (after which the tract is also named the pyramidal tract) [1]. A study by Kwon et al. [28] concludes that the fibres here too maintain a somatotopic arrangement whereby those representing the hand are medial to those representing foot. It should be noted here that fibres controlling movements of the face leave the tract to synapse on their nuclei in the brainstem and do not enter the spinal cord.

At the caudal medulla, right near its junction with the spinal cord, most fibres in the pyramids decussate. The rest of the fibres, as many as 10 to 25% [28], do not and enter the spinal cord on the ipsilateral side to their origin. In the spinal cord, the fibres form two major tracts: the lateral corticospinal tract, and the anterior (or ventral) corticospinal tract. Fibres in the lateral tract are of contralateral origin, and descend in the posterior part of the lateral funiculus. It correlates closely to the denticulate ligament in the cervical spine, where it can be found just posterior to its insertion [29]. The ascending spinocerebellar tract, however, affects this correlation at various levels. This lateral tract can be detected all through to the lumbosacral spine [30]. Fibres of the anterior tract, on the other hand, are mostly of ipsilateral origin, and descend in the anterior column of the cord close to the midline on either side of the median fissure [28,30]. The caudal extent of this tract varies markedly and depends on its size, with termination being reported anywhere between the thoracic and the sacral spine [30].

The synaptic termination of these tracts is in the spinal grey matter. Fibres of the lateral corticospinal tract synapse either directly on lamina IX neurones (anterior horn cells) of the contralateral side to their origin (ipsilateral to their side of descent in the spinal cord), or on interneurones of layers V to VIII within this same side. The anterior corticospinal tract, in contradistinction, decussates to terminate on the contralateral anterior horn grey matter at the respective spinal levels, which means that although it descends on one side, the effect it exerts is on the opposite side of the body. [4,30,31]

There exist yet two other corticospinal tracts, albeit less well defined than those previously described. The first is an ipsilateral lateral corticospinal tract that has been described in non-human primates, and also in humans, which continues in the lateral funiculus of the spinal cord and terminates on ipsilateral neurones down to the cervical spine [9,30,32,33]. The other is a contralateral anterior corticospinal tract that travels close to the midline in the anterior column of the spinal cord that can be seen as far as the C6-C7 segment [30].

Throughout its course, asymmetry has been described in the corticospinal tract. For example, a study by Herve et al. [34] showed that there exists asymmetry that is hand dominance-dependant in the pre-central gyri. Another study by Westerhausen et al. [35] found asymmetry at the level of the internal capsule ascribed to the corticospinal tract, although it failed to demonstrate a direct correlation to hand dominance. Yet another example is seen in the spinal cord, in around three quarters of examined cords, where Nathan et al. [30] demonstrated in their study that the asymmetry seen here is a result of more corticospinal fibres on the right side than those on the left in almost three quarters of asymmetric cords, an observation noted in both lateral and ventral tracts that was independent of hand-dominance.

Connections of The Corticospinal Tract

The corticospinal tract therefore is essentially bundles of axons; the cell bodies from which they stem reside in the cerebral cortex. These neurones are referred to as upper motor neurones (UMN) as they descend from the cortex, or from the brainstem in other tracts, to synapse on neurones within the brainstem or spinal cord [4]. The fibres derive their myelin sheaths from outgrowing processes from oligodendrocytes [36], and have an average conducting velocity of 65 to 80 m/s [37-39]. They utilise aspartate and glutamate as neurotransmitters [9]. Damage to UMN's results in the upper motor neurone syndrome, whereby the motor area for which the neurones are responsible exhibits paralysis (or paresis), hypertonia, hyperreflexia, clonus, up-going plantar reflexes (Babinski's sign) and spasticity [40].

This is in contrast to neurones from the brainstem or spinal cord that arise from the anterior horn lamina IX (anterior horn cells), known as lower motor neurones (LMN). These neurones synapse directly on groups of muscle fibres (motor units), and receive input from UMN's in addition to other neurones (e.g. interneurons) [9]. Myelination in LMN's occurs via indentation into Schwann cells, and not through oligodendrocytic processes [41]. There are two types: the larger (and therefore faster conducting) alpha motor neurones that synapse on extrafusal muscle fibres causing effective muscle contraction, and the smaller, slower, gamma neurones that synapse on intrafusal muscle fibres in the muscle spindle. The neurotransmitter for these neurones is acetylcholine. Damage to LMN's results in the muscle area supplied by these fibres exhibiting paralysis (or paresis), areflexia (or hyporeflexia), hypotonia, and flaccidity along with fibrillation of the denervated muscle fibres, or fasciculations in case the nerve was damaged but not completely severed [9].

The Reflex Arc

The LMN's, in addition, play a pivotal role in reflex arcs involving skeletal muscles. These are rapid responses in which sensory input stimulates the anterior horn cell directly (monosynaptic reflexes) or indirectly through interneurones without interference from the cerebral cortex, which still receives sensory input. It should be noted, however, that the neurones involved in these reflexes still receive input from higher centres [40] partly through the corticospinal tract. Two such examples are the myotatic stretch reflex and the autogenic inhibition reflex.

The myotatic reflex starts with stretching of the muscle fibre, as would occur when a tendon is tapped. This is sensed by nuclear bag fibres responsible for detecting the change, and rate of change, in muscle length, and by the nuclear chain fibres that are also responsible for detecting the change in fibre length, both of which are found in the muscle spindle [42,43,44]. Group I-a and group II sensory neurones carry the signal, and enter the grey matter of the spinal cord via the posterior horn and synapse on anterior horn alpha and gamma neurones and therefore directly excite them [44]. The afferent neurones also synapse, through interneurones, on motor neurones controlling the antagonistic muscle group to inhibit them (reflex inhibition) [44,45]. This results in contraction of the stretched muscle shortening it. The function of the gamma motor neurone is more to stimulate intrafusal fibres' contraction keeping them taut ready to receive further stimulation [44], while alpha neurones are responsible for effective contraction.

The autogenic inhibition reflex, on the other hand, involves the Golgi tendon organs that respond to tension within the muscle or direct stimulation of the tendon [46,47]. These send their sensory input via I-b fibres that synapse on I-b inhibitory interneurones that inhibit the efferent motor neurones, thus decreasing the tone within the muscle [48], while motor neurones of the antagonistic muscle group are stimulated through stimulatory interneurones.

Conclusion

In conclusion, the corticospinal tract originates from multiple areas in the cerebral cortex, and follows a rather predictable, although slightly variable and asymmetric, course in the cortex, brainstem, and spinal cord. Its neurones synapse on lower motor neurones and interneurones to effect precise motor movements. These lower motor neurones in turn synapse on muscle fibres directly, and receive input from sensory neurones in addition to that from the corticospinal tract and interneurones, and are involved in reflex arcs that are essential for our maintenance and ambulation.

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