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A review of corticospinal tract location at corona radiata and posterior limb of the internal capsule in human brain

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Abstract. The corticospinal tract (CST) is the most important motor pathway in the human brain, therefore, the accurate estimation of the CST state following brain injury would enable us to predict the motor outcome or enable accurate surgical planning. The corona radiata (CR) and the posterior limb of the internal capsule (PL) are important locations because they are related to poor motor outcome. In this study, we reviewed the available literature regarding the location of the CST at the CR and PL in the human brain. The results of this review indicate that the CST is located in the posterior portion of the CR and PL. However, a direct comparison of the results of previous studies would be impossible because many of the previous studies did not define the exact boundary or analytic standards used to locate the CR or PL. Therefore, further complementary studies that define these in detail are warranted.

Keywords: Corticospinal tract, motor function, diffusion tensor imaging

1. Introduction

The corticospinal tract (CST) is the major neuronal pathway that mediates voluntary skilled movements in human brain [10,42]. The preservation or recovery of the CST is mandatory for good recovery of impaired motor function in the patients with brain injury [1,7, 15]. The accurate estimation of the CST state following brain injury would enable us to predict the sequelae of motor weakness or to set up the scientific management strategy [9,22,27]. This information also could be useful in accurate surgical planning for the patient with brain tumor or vascular anomaly [2]. On the other hand, the corona radiata (CR) and the posterior limb of internal capsule(PL) are important locations because these areas are related with poor motor outcomes, for example, Shelton et al. reported that 75% of the patients with cortical infarct showed isolated movement recovery of upper extremity although only 5.9% of patients with CR and/or PL infarct did [29,36].

In the current study, we reviewed the literature on the CST location at the CR and PL in human brain.

2. The evaluation methods used to locate the CST

Previous studies have used various methods to locate the CST in the human brain [4,6,12,16–21,24,28,34, 35,39–41]. In earlier studies, invasive methods such as dissection of the brain or direct electrical stimulation were used [6,12,16,17,28,35], however, the development of radiologic techniques such as brain CT and MRI enabled more recent studies to be conducted using noninvasive methods [18–20,39,40]. In particular, recent advances in diffusion tensor tractography (DTT), which is derived from a diffusion tensor image (DTI),

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have allowed visualization and localization of the CST at the subcortical level in three dimensions [23,25,30, 32]. Although DTT appears to be a revolutionary tool for studying neural pathways at the subcortical level, it can lead to erroneous results because this technique can be operator-dependent or provide mathematical results that do not reflect the actual anatomy of the brain [18, 19,30]. Functional MRI (fMRI) is capable of precisely identifying cortical activation sites due to its excellent spatial resolution at the cortex [14,31], therefore DTT has been used in conjunction with fMRI to allow more accurate localization of the CST [14,31]. In addition, the combination of DTT with intraoperative brain stimulation, such as the subcortical motor-evoked potential recording technique which can distinguish between a motor pathway from a CST or a non-CST by analyzing the characteristics of the motor-evoked potential [34], subcortical direct motor fiber stimulation technique [4, 24] or cortical motor mapping technique [5], has also been used in more recent studies.

3. The location of the CST at the corona radiata

There have been several studies conducted that have described the location of the CST in the CR [21,26,37, 38,41]. In 1996, Tohgi et al. measured the relative distance to the center of an infarct from the anterior pole to the posterior pole of the lateral ventricle in patients with CR infarct using brain CT. The results of this study revealed that the centers of infarcts associated with dominant dysarthria/upper/lower extremity weakness were located at an average distance of 0.33/0.45/0.49, respectively [38]. However, unlike other brain MRI studies, the brain CTs used in this study were taken using the orbitomeatal line as a standard. Inoue et al. reported the CST location in 13 patients with CR infarct in 2001 using a three-dimensional anisotropy contrast method derived from diffusion-weighted MRI. Seven patients with upper extremity dominant motor dysfunction had an infarction located in the middle one third of the CST on a color map [21]. In addition, a patient with lower extremity dominant motor dysfunction had an infarction in the posterior one third of the brain. Furthermore, 5 patients with equal motor dysfunction in the upper and lower extremities had an infarction in both the middle and posterior one thirds of the CST. However, the results of this study did not clarify the exact locations of the CSTs on the color maps.

There have been two conventional brain MRI studies conducted in an attempt to localize the somatotopy of CST at the CR by comparing the characteristics of motor weakness with the location of the lesion [26,37]. In 2005, Kim et al. measured the distance between the center of the infarct and the posterior pole of the lateral ventricle (CP) and the distance between the anterior and posterior poles of the lateral ventricle (AP) using T2-weighted MRI [26]. In their study, the CP/AP ratio (CP/AP X 100%) was used to assess the antero-posterior localization of lesions. The centers of the infarct in patients who showed bulbar/arm/leg dominant motor weakness were located an average of 44.4%/35.8%/28.7%, respectively. Another MRI study was conducted by Song Y, in which the relative antero-posterior and medio-lateral location of the lesions were located using T2-weighted MRIs of patients who showed isolated motor weakness limited to the arm, leg, or bulbofacial muscles due to a CR infarct [37]. He measured the the longitudinal distance between the most lateral points of the anterior and posterior horns of the lateral ventricle, and between the center of the lesion and the most lateral point of the posterior horn of the lateral ventricle, the horizontal distance between the gray matter margin of the insula cortex and the wall of the lateral ventricle, and between the center of the lesion and the wall of the lateral ventricle. The average longitudinal distances in patients with bulbofacial/arm/leg weakness were 0.49/0.40/0.30 respectively, and the average horizontal distances were 0.68/0.66/0.50 respectively. These results indicate that the location of the lesions associated with isolated bulbofacial, arm, and leg weakness showed anterolateral-to-posteromedial distribution.

Recently, a DTT study was conducted to evaluate the location of a CST in the CR and PL. In that study, Yamada et al. reported anatomical landmarks that would allow predicting the course of the CST through the CR and PL [41]. The results of their study showed that the central portion of the CST deviated from the line drawn between the primary motor cortex and midway between the anterior commissure-posterior commissure by only average 1.8 mm on the sagittal view.

4. The CST location at the posterior limb

Traditionally, due to the influence of Charcot and Dejerine it was believed that the location of the CST within the PL was in the anterior portion [8,11]. However, this concept began to change after electrical stimulation studies were conducted during sterotaxic brain surgery [3,6,13]. Currently, it is generally accepted

that the CST is located in the posterior portion of the PL, although the detailed location has not yet been elucidated [6,12,16,18,28,35,40]. In 1965, Bertrand et al. published detailed anatomical maps generated using a stereotaxic electrical stimulation study that showed that the CST was located in the anterior 8 mm of the posterior half of the PL [6]. In addition, Englander et al. [12] and Hanaway et al. [16] reported that the CST was located in the third quarter of the PL (anterior to the posterior border of the lenticular nucleus) at the midthalamic level and posterior half of the PL using an autopsy study of stroke patients respecitively. Conversely, a normal brain dissection study demonstrated that the CST entered the rostral PL at the anterior half of the PL in either the first or second quarter [35]. In that study, the CST was shown to progressively shift into the posterior half of the PL in the caudal portion, and then pass into the third quarter at the low thalamic level. In some brains, however, it occupied a portion of the fourth quarter at this level. Another newborn brain dissection study showed that the CST was located in the middle of the PL, in the rostral portion at the level of the interventricular foramen, and in the posterior third of the PL at the level of the caudal portion at the subthalamic nucleus [28]. In addition, in 1994, a T2-weighted MR imaging study reported that the CST was located at the posterior portion of the PL [40].

Several studies regarding the location of the CST at the PL have been conducted since the introduction of DTI combined with DTT [18,20,39]. Holodny et al. reported DTT study that the CST was located in the third quarter of the PL and that most of the CST for hand were located lateral and slightly anterior to CST for the foot [18]. In addition, several other studies were conducted to evaluate the somatotopy of the hand and foot, as well as asymmetry according to handedness [20,39]. Westerhausen et al. studied CST asymmetry at the internal capsule according to handedness using DTI and found that there were no CST asymmetries according to handedness [39]. In addition, Ino et al. studied the topographical relation of hand and foot fibers of the CST using DTT and fMRI results. The results of their study were similar to those of a study conducted by Holodny et al. [18], but there was interindividual variability. However, this study was limited because the number of subjects was small (7 subjects) and they drew the fMRI activation area without coregistration. The two other DTI studies conducted to date used the probalistic mapping technique to show that the fiber tract from the primary motor cortex was located at the posterior portion of the PL [33,43].

5. Conclusions

We reviewed the available literatures on the location of the CST at the CR and PL in the human brain and found that the CST appears to be located in the posterior portion. However, to date, the detailed location has not been accurately elucidated. In addition, a direct comparison of the results of the previous studies is impossible because many of these studies did not declare the exact anatomical boundaries of CR and PL, or did not adopt the standards for analysis that can be applied easily in clinical field [6,12,16–18,20,21,28,35,38–41]. These problems make it difficult to use the results for clinical application. Therefore, further complementary studies that define the anatomical boundary and the benchmarks for analysis accurately are warranted. Using DTT in combination with other modalities such as fMRI, TMS or direct electrical stimulation would be useful for investigating the localization of the CST in the human brain.

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