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Damage to the pyramidal tracts is necessary and sufficient for the production of the pyramidal syndrome in man

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ABSTRACT

The causal role played by damage to the pyramidal tracts in the production of spastic hemiplegia in man has been hotly debated over the past hundred years. Two broad streams of thought have emerged from this dispute. The first, which is grounded on the clinicopathological schools of Jean-Martin Charcot (1825–1893) and Paul Flechsig (1847–1929), claimed that the four cardinal signs of hemiplegia, namely (i) paralysis, (ii) spasticity, (iii) hyperactive phasic muscle reflexes ("tendon jerks") and (iv) the sign of Babinski, are caused by injury or dysfunction of the pyramidal tracts. The second school, championed by John Farquhar Fulton (1899–1960) and Derek Denny-Brown (1901–1981), reflects the increasing influence of experimental neurology on clinicopathological concepts after World War II. According to this school, most elements of the pyramidal syndrome are caused by the added release or injury of extrapyramidal structures at different levels of the forebrain and brainstem. Most symptoms of spastic hemiplegia were thus interpreted as signs of extrapyramidal (*e.g.*, reticulospinal) release or damage. However, consensus on which symptoms of spastic hemiplegia were due to pyramidal or extrapyramidal changes was never reached. To add to this uncertainty, a number of clinicopathological cases that supported the old view were sporadically published over the same period. The purpose of the present essay is to provide clinicoanatomic perspective to the neurological literature in support of the hypothesis that damage to the pyramidal tracts is a necessary and sufficient condition for the production of the complete pyramidal syndrome in man.

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Introduction

The goal of this article is to present evidence to support the hypothesis that damage to the pyramidal tracts (PyrTs) is a necessary and sufficient condition for the production of the classical pyramidal syndrome in man. A corollary of the hypothesis is that when the PyrTs are bilaterally damaged in the cerebral hemispheres or brainstem the syndrome of double (bilateral) hemiplegia ensues. As discussed elsewhere, severe and complete bilateral hemiplegia equals the locked-in syndrome [1]. For the purposes of this article, strict definitions of "PyrTs" and "pyramidal syndrome" will be adopted. To circumvent the hazards of drawing inferences from other species when dealing with the difficult issue of the cerebral organization of movement in man only human clinicopathologic material will be considered for analysis. This claim is grounded on the assumption that the human nervous system is qualitatively unique from a neurobehavioral and phylogenetic perspective [2]. Therefore, no attempt will be made to trace parallels concerning the neurobehavioral organization of movement in humans and nonhuman species.

The pyramidal concept

The rise and fall of the pyramidal concept

Current knowledge on the anatomy of the PyrTs is the result of painstaking research spanning at least the past four centuries [3]. Beginning with (*a*) the description of the medullary pyramids in the seventeenth and eighteenth centuries [4] and (*b*) the recognition that the decussation of their fibers is the substrate of the millennial observation that injuries of one side of the head lead to seizures or paralysis of the opposite side of the body [5], the clinical and anatomic concepts of the pyramidal syndrome and tracts, which had hitherto developed along independent lines, became interwoven [6]. This convergence was due to the success of the clinicoanatomical method, which was established as a powerful analytic tool in the second half of the nineteenth century [7].

Beginning with the work of Ludwig Türck (1810–1868), further elaborated by the schools of Jean-Martin Charcot (1825–1893) and

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Paul Flechsig (1847–1929), the lateral and anterior PyrTs were thoroughly described in cases of degenerative and focal diseases of the brain [8–11]. The broadening of the pyramidal concept to encompass "aberrant" pyramidal fibers [12] promoted the idea that the segmental somatomotor neuronal pools of the brainstem and spinal cord were directly innervated by projections from circumscribed cortical areas whose injury resulted in contralateral paralysis [11,13] or motor fits [14]. On the anatomical side, the PyrTs comprised the collection of motor fibers with a strict cortical origin that traversed the medullary pyramids and terminated in the motor nuclear apparatus of the brainstem and spinal cord. On the clinical side, a stereotyped constellation of signs regularly accompanied the degeneration of the PyrTs no matter at which point of the forebrain or brainstem the damage took place [15]. These signs affected the side of the body contralateral to the supraspinal lesion and the same side of the body of the secondary degeneration in the dorsolateral column of the spinal cord. The clinicoanatomic association between degeneration of the PyrTs and the syndrome of permanent contralateral spastic hemiplegia with hyperactive tendon jerks and the sign of Babinski was so constant that before long it became known as the "pyramidal syndrome" [16], implying a predictable clinicoanatomical association, or, on purely phenomenological grounds, the syndrome of "pure motor hemiplegia" [17]. At the turn of the twentieth century, neurology had finally put an end to the old puzzle concerning the clinicoanatomical substrate of crossed hemiplegia. Or so it seemed (Fig. 1).

The "pyramidal concept" (the conceptual blending of syndrome and tract), however, was not meant to last. In the first half of the twentieth century, physiologists and anatomists attempting to reproduce the signs and symptoms of ordinary clinical practice in the laboratory [18] increasingly voiced criticisms against the old view. At a time when the fascination with phylogenetic explanations and the methods of experimental medicine set the stage for fashionable pathophysiological concepts, the biological gaps that distinguish the human nervous system were systematically underappreciated [19]. Coincident with the rise of interest in the projections of the cortico-strio-tegmentospinal motor fiber systems subsumed under the rubric "extrapyramidal" [20] by anatomically minded physiologists [19,21] and clinicians [22–24] the pyramidal concept was eventually assimilated into the extrapyramidal system [25] or swiftly dismissed as an artifact of the clinicoanatomic

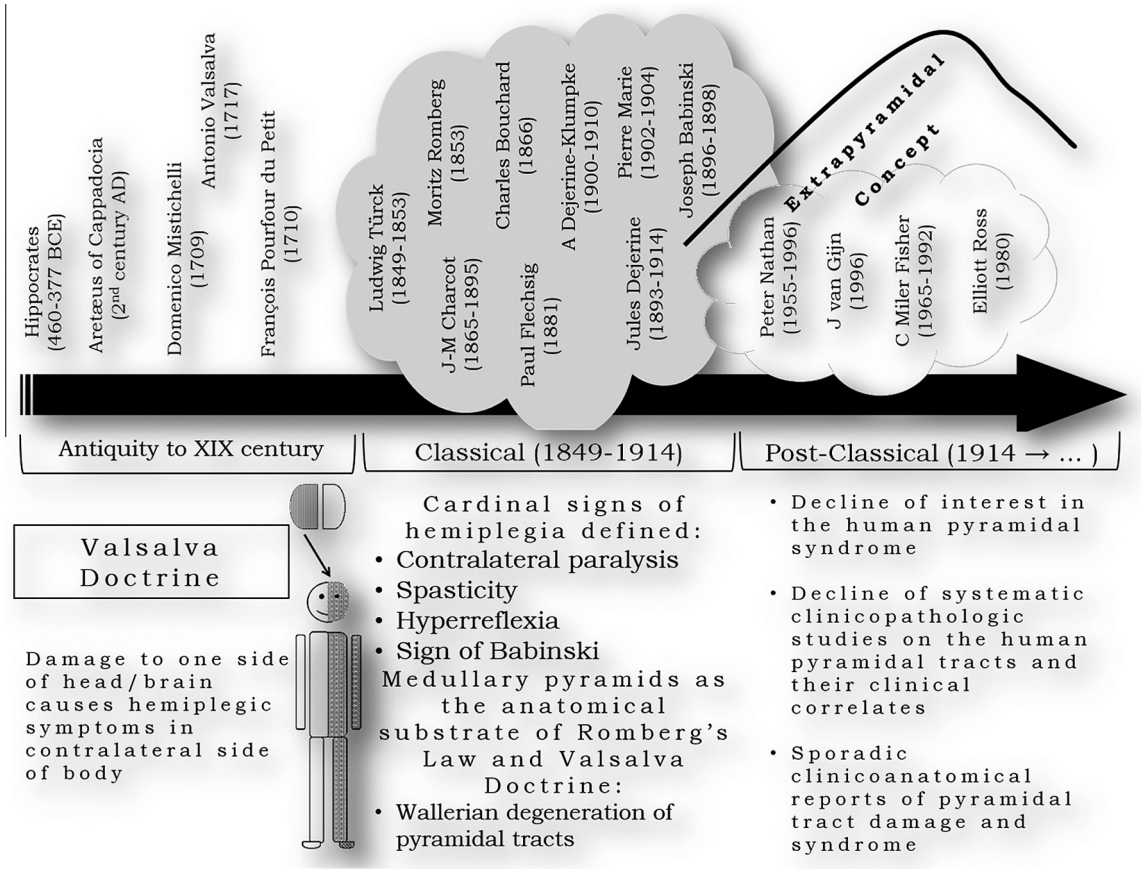

Fig. 1. Epochs in the evolution of knowledge about the clinicopathologic substrates of human hemiplegia. In the first period (from Classic Antiquity to the mid-nineteenth century), injuries in one side of the head/brain were noted to produce paralytic or convulsive symptoms in the opposite side of the body. The second period begins with Ludwig Türck's work on the degeneration of the pyramidal tracts secondary to unilateral cerebral damage and ends in 1914 with Jules Dejerine's monumental *Sémiologie des Affections du Système Nerveux*. During this period, the decussation of the medullary pyramids was established as the anatomical substrate of Romberg's Law of Crucial Conduction and the Valsalva Doctrine. The cardinal signs of human hemiplegia were also clearly described and related to the degeneration of the pyramidal tracts regardless of the level of damage in the central nervous system. These two accomplishments (pathological and clinical) gave rise to the "pyramidal concept", one of the most distinctive achievements of the Golden Age of the clinicopathological method. The third period is marked by the inception of the extrapyramidal concept, which crystalized in the first half of the twentieth century as a reflection of the progress in experimental neurophysiology in nonhuman species. The fourth period overlaps the third. It is marked by a regrettable loss of interest in the anatomical organization of the human pyramidal tracts and a decline in the rate of *postmortem* investigations on the clinicoanatomical correlates of the pyramidal syndrome. This neglect paralleled the rise of the extrapyramidal concept, a reflection of the outstanding progress of animal experimentation and the increasing popularity of animal models as surrogates of the human condition. The extension of the extrapyramidal system concept to humans was a major factor in the divergence of clinical neurology from neuropathology, which in the early days were often combined in the same physician. The division in periods and their representative authors are arbitrary and reflect the author's personal views. The numbers below each author represent the years of critical publications on the pyramidal concept.

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