## **Ipsilateral hemiparesis**

Establishing the neurological localization doctrine for the contralateral hemispheric control of motor functions in the second half of the 19th century, researchers faced the challenge of recognizing false localizing signs, in particular, paradoxical or ipsilateral hemiparesis (IH). Despite tremendous progress in current methods of neuroradiological and electrophysiological exploration, a complete understanding of this phenomenon has yet to be attained.

The authors researched the well-described cases of hemiparesis/hemiplegia ipsilateral to an intracranial lesion published in the scientific literature in the pre-MRI era (before 1980). A comprehensive review of the physiopathological mechanisms proposed for paradoxical hemiparesis throughout this period, as well as the pathological evidence substantiating them, is provided.

A collection of 75 patients with hemiparesis/hemiplegia ipsilateral to the primary intracranial lesion reported between 1858 and 1979 were eligible for analysis. Most cases occurred in adults with supratentorial, slowly developing, extraparenchymatous mass lesions, such as neoplasms (38%) or chronic subdural hematomas (36%). Physiopathological theories proposed by the neurologists who investigated IH can be grouped into 4 major concepts: 1) lack of anatomical decussation of the corticospinal tract; 2) impaired functional activation of the contralateral hemisphere by the lesioned dominant hemisphere through the callosal connections; 3) Kernohan notch phenomenon, or mechanical injury of the contralateral cerebral peduncle against the free edge of the tentorium; and 4) cerebrovascular dysfunction involving the contralateral hemisphere owing to kinking and mechanical flattening of the carotid artery contralateral to the primary intracranial lesion.

IH represents a still underdiagnosed paradoxical neurological phenomenon. With the aid of modern neuroradiological and neurophysiological methods, Kernohan's peduncle notch mechanism has been confirmed to cause IH in many of the cases reported in recent decades. Nevertheless, alternative functional and/or vascular mechanisms must be investigated further for unexplained IH cases, in particular for transitory IH without evidence of peduncle injury <sup>1)</sup>.

Patients with acute ischemic stroke were prospectively examined. Ipsilateral hemiparesis was defined as hemiparesis ipsilateral to recent stroke lesions. Patients with ipsilateral hemiparesis were examined with functional neuroimaging studies including transcranial magnetic stimulation (TMS) and functional MRI.

RESULTS: Of 8360 patients, ipsilateral hemiparesis was detected in 14 patients (0.17%, mean age 71±6 years, eight men). Lesions responsible for the recent strokes were located in the frontal cortex in three patients, corona radiata in seven, internal capsule in one, and pons in three. These lesions were located along the typical route of the corticospinal tract in all but one patient. Thirteen patients also had a past history of stroke contralateral to the recent lesions; 12 of these had motor deficits contralateral to past stroke lesions. During TMS, ipsilateral magnetic evoked potentials were evoked in two of seven patients and contralateral potentials were evoked in all seven. Functional MRI activated cerebral hemispheres ipsilaterally in eight of nine patients and contralaterally in all nine.

CONCLUSIONS: Most patients with ipsilateral hemiparesis had a past history of stroke contralateral to the recent one, resulting in motor deficits contralateral to the earlier lesions. Moreover, functional neuroimaging findings indicated an active crossed corticospinal tract in all of the examined patients. Both findings suggest the contribution of the uncrossed corticospinal tract contralateral to stroke lesions as a post-stroke compensatory motor system<sup>2)</sup>.

1)

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