Defining post-stroke pain: diagnostic challenges

Recently, a new grading system for central post-stroke pain (CPSP) was proposed, which might be used to distinguish patients with stroke who have central neuropathic pain from patients who have peripheral pain.1 Accordingly, for a CPSP diagnosis, all other causes of pain have to be excluded. Although this criterion has its purpose for defining CPSP as a separate entity, a too rigorous distinction between central and peripheral post-stroke pain might have drawbacks as well. Most importantly, by strictly following the proposed grading system, central pain mechanisms could be missed or even disputed in patients with other types of post-stroke pain. This possibility is particularly relevant as “mixed” pain and pre-existing pain are common after stroke.2 For this reason, we would like to emphasise that peripheral nociceptive pain after stroke might coincide with symptoms characteristic of CPSP. To lend support to our concern, we present recent data on post-stroke shoulder pain (PSSP).

PSSP is commonly localised to the affected upper extremity and regarded as peripheral nociceptive pain. However, unsatisfactory treatment and the frequent occurrence of persistent pain suggest a role for other mechanisms. To try to understand the possible central mechanisms that underlie PSSP, we used some parts of the diagnostic assessment for neuropathic pain in 19 patients with chronic PSSP, none of whom could be classified as having CPSP.3 Several sensory abnormalities overlapped with those observed in CPSP. Of particular interest was the high prevalence of abnormal spinothalamocortical tract function in patients with PSSP (15 of 19) compared with pain-free stroke patients (13 of 29), as abnormal function of this tract has been implicated in CPSP. Moreover, supportive criteria for a CPSP diagnosis, such as touch or cold allodynia (four of 19) and the absence of a primary relation with movement (seven of 19), were common in patients with PSSP, and PSSP was associated with abnormal sensory function in the unaffected side.

Our data strongly suggest that central pain mechanisms have an essential role in post-stroke pain, even in patients who cannot be classified as having CPSP. Therefore, central pain mechanisms should be assessed in all patients with post-stroke pain and treatments used for patients with CPSP might also be appropriate for patients with other forms of post-stroke pain. We hope that the CPSP grading system will not prevent clinicians and researchers in the fields of neurology, rehabilitation, and pain medicine from regarding and treating central pain mechanisms in patients with post-stroke pain who do not fulfil the criteria for CPSP.

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In conclusion, we believe the diagnosis of CPSP depends on a combination of history and clinical findings, in particular the sensory examination. Neuropathic PSSP is not excluded by the proposed definition.

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Erratum
Bonati LH, Jongen LM, Haller S, et al; for the ICSS-MRI Study Group. New ischaemic brain lesions on MRI after stenting or endarterectomy for symptomatic carotid stenosis: a substudy of the International Carotid Stenting Study (ICSS). Lancet Neurol 2010; 9: 353–62. In this Article (published Online First on Feb 26, 2010), Annet Waajier should have been listed as Annet Waaijer. This correction has been made to the printed Article in this issue, and to the online version as of March 15, 2010.