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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.[1] 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[2] 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[1] 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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Authors' reply

In their thoughtful letter, Roosink and colleagues raise the point that central pain mechanisms might be overlooked in patients with stroke who have pain if our proposed definition for central post-stroke pain (CPSP) is used. The essential point here is how we define central pain mechanisms. It is important to distinguish between central neuropathic pain and central mechanisms. When the nociceptive system is activated, physiologically short-lasting neuroplastic changes occur in the CNS. In persistent pain disorders, the molecular and cellular changes are more profound and sometimes irreversible, whether due to inflammation or a lesion of the nervous system. In neuropathic pain, there is damage to the somatosensory systems, causing peripheral and central neuroplastic changes that can sometimes be permanent. In inflammatory or simple nociceptive pain disorders, the somatosensory system is essentially intact, but it is in a state of heightened excitability that gradually returns to normal when the inflammation subsides.

Specific sensory testing could be used to clarify whether there is a loss of sensory input to the nervous system, but such testing can be misleading. The abnormalities mentioned by Roosink and colleagues in assumed spinothalamic functions, such as temperature and pinprick response, are not necessarily an indication of central neuropathic pain. Central neuropathic pain requires a loss of

function in a body part corresponding to the affected brain territory, whereas abnormal positive or negative spinothalamic sensory functions might be seen in both inflammatory and neuropathic disorders.

In our experience, many patients with CPSP have a combination of both inflammatory and neuropathic pain elements. When located in the same area, it can be difficult to distinguish between them. In certain cases of post-stroke shoulder pain (PSSP), the pain is clearly nociceptive but in other cases the pain mimics that seen when CNS structures are damaged.

Is it important to differentiate between neuropathic and nociceptive pain? We think so. Admittedly, available treatments for central neuropathic pain are only partially effective and have dose-limiting side-effects, but new compounds that target specific sites implicated in neuropathic sensitisation events are emerging.

Does our grading system exclude a central pain diagnosis in some cases of PSSP? In our opinion, patients with PSSP who have sensory abnormalities in the shoulder area corresponding to the lesion fulfil our proposed criteria for CPSP if there is no other obvious pathological abnormality in the shoulder that can fully explain the pain.

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.