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Editorial comment

Conditioned pain modulation: A robust phenomenon?

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Pain may be attenuated by another painful stimulus at another part of the body. Diffuse noxious inhibitory control (DNIC) is the concept describing the modification of convergent neurons by heterotopic stimulation mediated by descending pathways from the caudal medulla (see [1,2] for reviews). When elicited in humans, this phenomenon has been labelled 'conditioned pain modulation' (CPM [3]). In the laboratory study situation, the primary pain is induced by a test stimulus (TS), and the potential inhibition of this pain is produced by a painful heterotopic conditioning stimulus (CS).

The animal studies of DNIC have been interpreted as the presence of diffuse and selective powerful control of wide dynamic range neurons [1]. The effect of this control may be altered body representation which may amplify the transmission of one specific input and inhibit other inputs, e.g. the primary pain (in the laboratory: the TS).

There is a need to determine if the mechanisms behind CPM are mechanisms that play a major role in preventing chronification or maintenance of pain. Indeed, one recent study reported that testing CPM may identify patients at risk of developing chronic pain [3].

However, CPM effects seem to show large variation between studies. The effects of CPM may depend of both modality of test stimulus (i.e. nature of primary pain) and magnitude and nature of the conditioning stimulus [2]. The only study reporting predictive potential of CPM response administered heat as both TS and CS [3].

The study of Oono et al. [4] addresses the important issues of reliability of the CPM phenomenon by testing intra-individual and inter-individual variation of CPM. Furthermore, they report magnitude of CPM as a function of CPM-stimulus modality and region of testing the TS. They tested the following conditioning stimuli: cold-pressor pain (CPP), tourniquet pain, and mechanical pressure pain by stimulation of the craniofacial region. Test stimuli were pressure-pain threshold and -tolerance and pain intensity from masseter, forearm, and leg. Since previous studies of CPM-effects have reported varying results, the present study of CPM with different stimulus modalities is an important contribution to the field.

One of the findings of this study was that hand immersion in 2–4 °C water for 10 min (Cold Pressor Pain) was the most efficient conditioning stimulus. The VAS during this cold exposure was 9 on a 10 cm VAS scale. It seems possible that attention, arousal, effort, and sympathetic nervous system activation may contribute to the CPM effect. Hence, factors other than the afferent traffic from noxious heterotopic stimulation *per se* may contribute to the observed CPM in conscious humans.

Pain is a warning that signals threat or malfunction. Nociceptive inputs are amplified, modified, and/or inhibited at several levels of the central nervous system by a multitude of mechanisms. Understanding the mechanisms that modify nociception and pain is pivotal to understanding chronic pain and pain syndromes with seemingly little peripheral cause of pain. So far, it seems that CPM in humans may be mediated by several mechanisms. Further studies of combination of modalities and dose–response relationships are needed in order to conclude how the CPM phenomenon is related to DNIC observed in animal experiments and whether CPM effects are predictors of chronification. The study by Oono et al. is a significant contribution to this field of pain research.

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