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Diagnosing and treating chronic musculoskeletal pain based on the underlying mechanism(s)

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A B S T R A C T

Until recently, most clinicians considered chronic pain to be typically due to ongoing peripheral nociceptive input (i.e., damage or inflammation) in the region of the body where the individual is experiencing pain. Clinicians are generally aware of a few types of pain (e.g., headache and phantom limb pain) where chronic pain is not due to such causes, but most do not realize *there is not a single chronic pain state where any radiographic, surgical, or pathological description of peripheral nociceptive damage has been reproducibly shown to be related to the presence or severity of pain*. The primary reason for this appears to be that both the peripheral and central nervous systems play a critical role in determining which nociceptive input being detected by sensory nerves in the peripheral tissues will lead to the perception of pain in humans. This manuscript reviews some of the latest findings regarding the neural processing of pain, with a special focus on how clinicians can use information gleaned from the history and physical examination to assess which mechanisms are most likely to be responsible for pain in a given individual, and tailors therapy appropriately. A critical construct is that, within any specific diagnostic category (e.g., fibromyalgia (FM), osteoarthritis (OA), and chronic low back pain (CLBP) are specifically reviewed), individual patients may have markedly different peripheral/nociceptive and neural contributions to their pain. Thus, just as low back pain has long been acknowledged to have multiple potential mechanisms, so also is this true of all chronic pain states, wherein some individuals will have pain primarily due to peripheral nociceptive input, whereas

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in others peripheral (e.g., peripheral sensitization) or central nervous system factors ("central sensitization" or "centralization" of pain via augmented pain processing in spinal and brain) may be playing an equally or even more prominent role in their pain and other symptoms.

Introduction

The notion that chronic pain should be treated based on the underlying mechanisms present in each individual rather than the disease causing the pain is not new. It was first raised nearly two decades ago by Mitchell Max, and later by Clifford Woolf and others [1,2]. However, these authors opined that we should do this in the future; this manuscript suggests that we might finally have made enough scientific progress in the pain field to begin to implement these techniques in clinical practice. Making this distinction is critical clinically as both the drug and nondrug therapies that will work for any given patient with chronic pain might be much better guided by a nuanced view of the mechanisms of their pain rather than knowing which of these "diagnoses" the patient is suffering from.

Fig. 1 briefly describes at least three different underlying mechanisms that can be operative in chronic pain states: peripheral/nociceptive, (peripheral) neuropathic, and central neuropathic, or "centralized" pain. Some authors prefer to use the term "neuropathic pain" for any pain of neural origin, whereas others prefer to reserve this term for conditions where there is identifiable damage to the nervous system. We acknowledge this and prefer to use the term "centralized" pain to refer to the fact that the central nervous system (CNS) (rather than the peripheral nervous system) is prominently involved in maintaining the pain. This distinction between peripheral neuropathic pain (where peripherally directed therapies such as topical treatments, injections, and/or surgery might be helpful, and should be considered) and centralized pain (where these are generally not options) is extremely important.

Of note, although specific diagnoses are noted in Fig. 1 as being considered peripheral/nociceptive, peripheral neuropathic, or centralized, this is meant to indicate the primary underlying mechanism for pain in each of these diagnoses. Again, the emphasis of this manuscript is that some individuals with

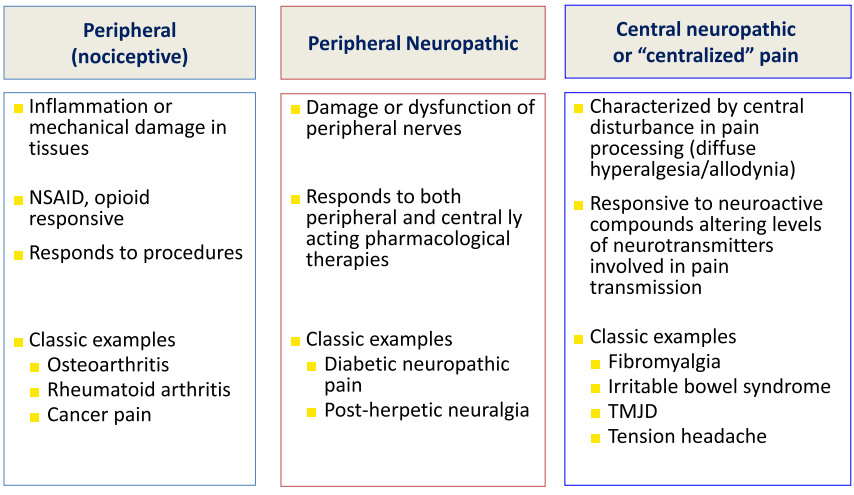

Fig. 1. Mechanistic characterization of pain.