**How low can you go? Decreasing pain through exercise induced hypoalgesia**

We all know that exercise is going to make us feel better, whether we’re injured, healthy, in our prime, or on a decline. The problem begins when a patient is no longer able to continue or even begin exercise due to pain. So the question becomes, how do we get our patients to move again without as much pain? Let’s consider a patient with acute or sub-acute pain. Regardless of the body part affected, they have likely tried a myriad of OTC approaches possible including thermo-therapy, topical creams, TENs units, and possibly even a prescription for pain killers. By these means they have only been receiving half of the potential pain-relieving effects.

Exercise induced hypoalgesia (EIH) is characterized by a temporary increase in pain threshold and tolerance during and following exercise.1 It is hypothesized that these effects are due in part to a noxious stimulation of A-delta and C fibers, which utilize the gate theory of pain modulation.2 This however, cannot fully explain the EIH phenomenon because the introduction of exogenous opioids alone does not entirely replicate the physiological effects seen in studies. Both Koltyn et al. and Gajsar et al. sought to quantify changes in the endocannabinoid system, specifically 2-AG and 2-OG. In short, studies observed an increase in both 2-AG and 2-OG following isometric exercises which were not seen in the control which received exogenous opioid injections.1,3 The author concluded a systemic circulation of these pain modulating compounds is responsible for the other half of the EIH response.

In a recent study, Holden et al. attempted to compare the acute effects of isometric and dynamic resistance exercises on pain during a single limb decline squat in those with patellar tendinopathy. The results indicated that there was temporarily a significant change in perceived pain and pain-pressure threshold of the patellar tendon.4,5 Interestingly, the control group performing dynamic resistance showed an identical change in pain-pressure threshold. Furthermore, there was a systemic effect on pain-pressure threshold which was seen in the tibialis anterior, suggesting a central means of pain modulation in favor of EIH.4

If we look back at basics, we learned that isometrics can be useful for reducing perceived pain as well as pain-pressure threshold.4 Just look at the fundamentals of PNF where isometric contractions of either the agonist or antagonist will decrease perceived pain. More recently the concept of functional range conditioning has expanded on that thought to include angular isometric holds at end range to decreased pain and improve motor control at end ranges. If we consider taking this centrally mediated pain-relieving approach, these patients should perform sets of isometrics at or around the painful area as the above articles suggest. The patient could also attempt a tolerable whole body active warm-up so they can take advantage of EIH. Furthermore, we can even consider using exercises that are less provocative for pain and increasing the total load so they finish at a higher RPE. By doing so, we will induce EIH but also decrease fear associated with activity. With the shift in our field for patient empowerment to manage their own symptoms, this approach can be critical in steering away from passive treatments and more towards active ones. This can open possibilities including better tolerance to manual therapy and their therapeutic exercises. Just remember that these effects are temporary and may be blunted in patients with chronic pain.6 This makes patient education a necessity regarding this useful window of time where we can make progress towards improving their functional mobility.

Reference:

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