How important are perturbation responses and joint proprioception to knee osteoarthritis?

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OSTEOARTHRITIS (OA) is the most common form of arthritis and one of the leading causes of pain and disability worldwide (8). The knee is the most affected lower limb joint, with lifetime prevalence of symptomatic knee OA estimated to be 44.7% (10). Although knee OA is characterized by degenerative and regenerative changes of the cartilage, bone, and surrounding tissues, it is the symptoms of knee OA, rather than radiological features, that correlate more strongly with the disability experienced in daily life (3).

In this issue of the Journal of Applied Physiology, Kumar et al. (7) present data that reject the hypothesis that, compared with controls, people with knee OA would show a diminished response in knee kinematics and muscle activation patterns in response to a novel mechanical perturbation during walking and would show poorer adaptation in knee kinematics and muscle activation patterns over repeated perturbations. In responding to an external perturbation during walking, the knee OA and control groups responded similarly to the first perturbation and adapted similarly to repeated perturbations. These findings were despite the knee OA group having significant joint pathology, pain, muscular weakness, poorer mobility, and altered gait patterns. This study failed to show deficits in joint proprioception that have previously been seen in people with knee OA. These results challenge the existing literature that links joint stability and proprioception to the disability and pathogenesis of knee OA.

The study by Kumar et al. (7) is based on concerns regarding the contribution of joint stability to articular health—the idea that altered sensory input may impair the muscular activity to stabilize the joint, leading to altered mechanical loading and subsequent joint damage. There is good evidence that altered joint loading during gait may contribute to the development and progression of knee OA (1). For example, larger-than-normal knee adduction moments, which load the medial articular surface of the tibiofemoral joint during the stance phase of gait, have been associated with the presence, severity, and progression of knee OA (1).

Kumar et al. (7) challenged joint stability during walking with a translating platform underfoot. Sensory information from the foot and lower leg muscles is likely to have potentially influenced the initial response and subsequent adaptation, whereas proprioceptive signals from the knee are unlikely to have had a major role in this task. It is therefore not entirely surprising that no differences were observed between knee OA and control groups in the response and adaptation to the perturbations. Many sources of afferent input are available to detect perturbations during gait and to drive adaptation to repeated perturbations. Evidently, people with knee OA retain the ability to use a variety of sensory information, including from the foot, ankle, and hip, as mentioned by the authors, and, of course, vision and vestibular sense.

Investigations involving proprioception in knee OA must be performed with careful consideration of the neural mechanisms underlying the tests (11). In contrast to numerous previous reports that proprioceptive deficits exist in people with knee OA (2), no between-group differences were found in proprioception, measured as movement detection thresholds in flexion and extension of the knee. It is likely that the lack of difference here was due to the testing procedure. No conditioning contractions were performed to control for the effects of muscle thixotropy (11). Detection thresholds will have been elevated for the rather slow movement (0.5°/s) during the proprioception test compared with the rapid perturbations applied during walking in the remainder of the experiment (11). Moreover, testing the joint within the mid-range, rather than near the limits of movement range, would minimize any contribution from joint receptors to the sensation of movement; instead muscle spindles will have predominated (11). Finally, the measurements were made in a passive condition, which, contrary to traditional thinking, is a state in which the muscle spindles may be more sensitive to detecting movement and any influence of weaker muscles in people with OA would have been eliminated (11).

With improved methods Kumar et al. (7) may well have detected proprioceptive deficiencies in the knee OA group, but small differences in laboratory tests of proprioception are unlikely to impact on functional abilities (2, 4). Despite the large literature implicating proprioception in the pathogenesis of OA and clinical interventions based on proprioceptive training for treatment of knee OA, the link between proprioception and disease severity is currently very weak. Felson et al. (5) failed to identify a significant association between proprioceptive deficits and incident knee OA in a 3-year prospective study of 2,243 subjects. Indeed, no study has provided evidence to support a clinically significant role for proprioceptive deficits in the progression of knee OA. Baert et al. (2) reported that the proprioceptive deficit identified in their sample of people with knee OA was not associated with measures of functional ability, balance, and strength. These findings raise the question: how important is the loss of proprioception to function in knee OA? Kumar et al. clearly demonstrate that the ability to adapt to perturbations during walking is not affected in people with knee OA. Indeed, the authors note “compensatory strategies may be sufficient to allow people with knee OA to maintain stability when challenged during walking.” Interestingly, however, the heightened incidence of falls for people with knee OA suggests that there may be some deficit in the maintenance of dynamic stability (13). The mechanisms for this are...
not resolved, but reduced muscle strength appears to be a key factor (13).

Muscle strength deficits have long been recognized as having an important influence on the development of knee OA and the progression of symptoms (12). Exercise interventions to increase strength are also effective in improving symptoms (14). However, contrary to common conjecture, the improvement of knee OA symptoms from strength training occurs without any demonstrated change in joint mechanics (6, 9). Despite this, Kumar et al. (7) suggest that their findings might apply to the prescription of exercise that is intended to challenge the ability to respond to perturbations and, in so doing, modify the loading of joints. It has recently been highlighted that some pragmatic decisions are warranted in any further investigations of exercise interventions for knee OA: a paper in BMJ found that by 2002 there was sufficient evidence to support the efficacy of exercise interventions yet, since that time, minimal evidence for how this practice may be modified or refined for improved outcomes (14). It does not seem that exercise designed to challenge proprioception will be any more effective for the management of knee OA than other forms of exercise. The new data presented by Kumar et al. (7) suggest that so called “proprioceptive training” for people with knee OA is not warranted.

DISCLOSURES

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