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Review Article

Cervical Osteoarthritis Pain NeuroMotor Sources and Treatment Opportunities

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Abstract

Painful cervical spine osteoarthritis can be extremely debilitating. Here we highlight some thoughts concerning the possible sensory and motor nerve origins of cervical pain and its remediation, a topic not well articulated as of December 2024, but one with multiple possible clinical and public health implications.

Keywords: Cervical Osteoarthritis; Cervical Muscles; Intervention; Mechanoreceptors; Older Adults; Proprioception

Introduction

Osteoarthritis, a common disease is one characterized largely by pain and dysfunction. Although studied for many decades, its resolution remains challenging at best. This may be because the disease is studied through a focal biochemical and genetic lens in many instances, rather than from a neuromotor perspective given the integrated functioning of the cervical spine comprising seven vertically oriented bone units or vertebrae and their bilaterally associated facet joints. Designed to foster neck joint stability as well as overall spinal mobility its overall structure may be subject to damaging impacts that evoke subnormal neuromotor responses and joint vulnerability.

If so, there may be incremental and immense signs of emergent dysfunction and incessantly painful osteoarthritis joint pathology and movement impairments that is hard to reverse. That is, despite acting in harmony to ensure a variety of surrounding as well as distant muscle reflexes that afford optimal head support, active muscular control mechanisms that enable the ability to pivot the head, afford brain and spinal cord protection, plus optimal neck, arm, hand, locomotor and jaw motions, due to their close anatomical proximity to nerves, the vertebrae and their facet joints if duly damaged may be subjected to excessive impacts readily and incrementally and adversely. This system can also fail if nerves located in the small nerve root exit sites located on the vertebrae are compressed and elicit abnormal impulses or fail to react in the face of impact or sudden perturbations in a well modulated and calibrated timely manner.

Since osteoarthritis is now considered a disease of the whole joint, where its synthetic and degrading processes in cartilage are partly maintained by cyclic motion and contraction generated by muscle, the presentation of osteoarthritis may thus implicate damage to one or more neural sites and neuromotor receptors, including those in the surrounding neck muscles, ligaments, nerve sheaths, tendons, vertebral discs and joint capsules and central nervous system that control muscle [1-3].

Indeed, while structurally and physiologically very effective and responsive at multiple levels under physiological conditions, the spinal neural networks linked to and within the cervical spine joints including its surrounding tissues may function less optimally in multiple ways if injured or subject to the painful joint disease termed osteoarthritis that is almost inevitable with age and often results in nerve-root and spinal cord compression, inflammation and both local and referred pain [1,4,5]. In addition, there may be a loss of vertebral disc height, painful nerve impingement, bone and ligament damage and ensuing bone

and synovial tissue related bioactive substance production and a host of adverse local and central neural responses [6].

This can prove even more disabling if the condition causes or fosters a state of disequilibrium between the anabolic and catabolic normal tissue interactions of the neck joint support tissues, such as cartilage, in favor of noxious catabolic processes as well as abnormal muscle responses and adaptations and joint stiffness or excess mobility that will be increasingly hard to reverse.

Moreover, as time advances, it is common to observe signs of possible progressive and pathological articular changes in joints other than the incident joint, plus associated declines in desirable joint based cellular biosynthetic activities, an increase in pain and possible numbness in the neck and arm as well as neck muscle weakness, limb tingling and changes in central pain processing or central sensitization, which can greatly affect function and the ability to readily pursue a life of independence and high quality. This is partly due to the fact the cervical spine is both weak and fragile and vulnerable to vertically oriented forces and others, as well as injuries of soft tissue (especially in the ligaments and intervertebral discs) that may lead to instability and periosteal reactions and the subsequent formation of new bone [7,8].

In addition to muscle weakness that is found to be a strong neck pain correlate, Ozen, et al., notes this above scenario is likely to occur quite frequently because the cervical spine is an essential region housing many diverse proprioceptive receptors vital for motor control and joint protection [8,9]. However, unraveling the source of neck pain and effectively mitigating this when present is commonly challenging and often seemingly impossible [8] as multiple pain inputs and others may interact at one or more levels of the cervical column cartilaginous vertebral body components. An analogous role applies to their associated ligaments and protective membranous tissue covering the spinal cord and brainstem, plus the dorsal compartment joints of the vertebral arches and their ligaments, mechanoreceptor arrays and muscles [2,8-10].

But do all older adults and younger adults have to suffer unduly from cervical osteoarthritis pain or can some degree of suffering be preempted or attenuated?

In particular, can the ability to isolate the causative factor[s] underlying the presence of chronic neck pain and possible functionally undesirable aspects of osteoarthritis pathology be overcome?

Although not a simplistic task this paper investigates if inroads could be made here as far as its by examining what is known of the morphology, function and clinical relevance of the joint as well as the muscle spindle receptors in the region of the joints of the cervical spine and their central and spinal connections rather than the well established pathological findings of the disease. It was thereby hoped the literature could possibly provide some form of guidance to more ably support a neural based rationale that could be effectively applied to the desired conservative management of cervical osteoarthritis, for example, intervening to normalize the observed muscle trunk imbalance noted in cervical spine pathology cases, with the predominance of extensor over flexor muscles that is associated with functional disability [11,12].

Methods

A broad based scoping biographical review and scan encompassing key aspects rather than details concerning cervical nerve networks and osteoarthritis pain was conducted. Designed to include historically based as well as contemporary observations as observed on the PUBMED, PubMed Central and Google Scholar database sites and housing articles specifically salient using the terms cervical osteoarthritis, muscle and motor control were sought as well as the terms postural and kinaesthetic perceptions that afford posture and stability. The article does not discuss osteoarthritis biology, pharmacology or possible gene therapy, nor any invasive forms of cervical spine intervention. Moreover, it does not present any quantitative data summaries or critical methodological perspectives but all forms of study and study substrates were deemed valid as long as there was a bearing on understanding the complexity and underlying nerve and muscle linkages to osteoarthritis and neck related pain. The term proprioception as used by Ozen, et al., refers to the concept of body part awareness including joint position sense, kinesthesia and the sense of muscle force [9]. The term cervical osteoarthritis was used throughout although the term cervical spondylosis is used to similarly describe a wide range of progressive degenerative changes that affect all the components of the cervical spine of many adults after age 50.

Major Findings

In addition to age as the predominant osteoarthritis determinant, it appears that understanding the role of the joint sensory receptors and their motor connections in this regard could help explain or predict the oftentimes intractable nature of pain produced in the neck region and its possible advancement due to osteoarthritis associated damage [10]. Now seen as a possible cumulative or contributory parameter in the cervical osteoarthritis damage cycle is the role of the extensive cervical articular located receptors, as well as their influence on muscle and muscle degenerative changes. In addition, related neck pain effects can be traced in many cases to mechanoreceptor sources that cause widespread vertigo, dizziness, and/or nystagmus responses. Additionally, locomotor performance disturbances, losses or excess joint range of motion, muscle fatigue and declines in muscle volume, disturbances in proprioception and progressive postural changes may ensue as well [13-18].

Consequently, even though it is the cellular and molecular aspects of osteoarthritis that are studied intently, rather than its neuromotor associations, a wealth of cumulative literature does point to a considerable role for one or more alterations in the extensive potent sensorimotor receptor and impulse transmission systems within and surrounding the neck joints that could provoke unremitting or acute bouts of pain and muscle volume changes [10, 13]. Indeed, the differing albeit functionally diverse joint receptor types that interact statically as well as dynamically to foster facilitatory or inhibitory reflex like influences on the ipsi- as well as the contralateral striated musculature of the neck, trunk and limbs, as well as respiratory muscles in response to changing mechanical stresses on the joint tissues may play a large role in either the maintenance or degradation of the associated joints. Based on many years of electrophysiological and clinical studies, these reflexes are generally considered of extreme importance in the control of posture, joint position, direction, amplitude and velocity, intra-articular pressure changes, gait and respiration [19]. They had further influences on upper extremity limb movement, joint acceleration and deceleration and cortical interactions [19, 20]. As well, among the many mechanoreceptors found to influence postural control and gait, many are nociceptors or pain nerve receptor endings located not only around the cervical joints, but in the adjacent connective tissue coverings of the cervical vertebrae and its ligaments, as well as the adventitia of its related blood vessels [21].

These pain receptors that are normally considered inactive, were observed however to be triggered in the face of excess mechanical deformation and tension, as well as direct mechanical or chemical irritation to induce pain and possible postural and functional changes as found in cervical osteoarthritis [10, 21, 22]. Moreover, these evoked responses were found to not only discharge for long time periods but to have widespread effects on distant tissues and the kinematics of the normal sub adjacent vertebral segments with possible resultant larger than desirable translation displacement in the extension mode and high degrees of motor dysfunction.

Later, McLain, who studied 21 cervical facet capsules taken from three normal human subjects, identified mechanoreceptors in 17/21 specimens and as classified according to the scheme for encapsulated nerve endings established by Freeman and Wyke were found constituted by 11 Type I, 20 Type II and 5 Type III receptors, as well as a number of small, unencapsulated nerve endings and free nerve endings subserving pain [23]. The author strongly suggested a dual role for these receptors in motor control including proprioception and pain sensation and thereby the degree of overall cervical spine functions and stability and integrity.

This supports the conclusion reached in anatomic studies and others that free and encapsulated nerve endings in facet joints as well as nerves containing substance P and calcitonin gene-related peptide are readily stimulated. The onset of inflammation in turn, leads to decreases in the thresholds of these nerve endings in facet capsules as well as elevated baseline discharge rates in the face of excessive deformation of the capsules of lower cervical facet joints [22].

According to Johnson [24] the results of studies examining the innervation patterns of the facet or zygoapophysial joints of the cervical spine were similarly found to be partly innervated by sensory nerves and in addition appeared to travel along sympathetic pathways. These studies also demonstrated that the neuropeptide levels in the cell bodies located within the dorsal root ganglion of these sensory nerves fluctuated according to the physiological state of joint. Additional to the sympathetic nerves accompanying the vertebral artery, the innervation patterns of dural tissue and posterior longitudinal ligament in the upper cervical spine were notable distinctive features of the examined cervical spine innervation extent. Recent data further allude to a role for cervical spine meniscoids or intra articular synovial membrane folds thought to be pain associated and that can be innervated and appear to vary in morphology in the presence of articular degeneration. In a clinical population, moreover, it

appeared associations have been observed between cervical spine meniscoid morphology and the presence of cervical spine symptoms [25]. A parallel change in muscle function and volume plus fat content that may implicate cervical neural processes has also been observed in cases with chronic non specific neck pain [26].

Others have tended to emphasize the importance of proprioceptive mechanisms in neck pain development and progression [14,27]. As per Ou, et al., proprioceptive signals generated from cervical structures appear crucial to normal cervical functions, whereas abnormal proprioception caused by neck pain and other factors may provoke alterations in neural plasticity, cervical muscle recruitment and cervical kinematics [10]. This is important to note and especially to identify because long-term sensorimotor disturbances and neural mal adaptations may contribute to the recurrence and chronicity of neck pain. According to Neuhuber and Zenker, a further noteworthy observation is that the consistent collateralisation pattern of rostral cervical afferents along their whole rostrocaudal course enables their connections to serve a diverse array of precerebellar, vestibulospinal and preoculomotor neurons that have a well-established significance as regards proprioceptive neck afferents that control posture, head position and eye movements [27].

As such, Peng, recommended cases with unrelenting bouts of neck pain should be assessed and managed for cervical proprioceptive impairments and sensorimotor control disturbances as indicated [14]. Chen, who confirmed the existence of receptors in the facet joint capsule indicated that the capsule probably has pain as well as proprioceptive sensory functions [28]. As discussed by Chen and since pain is the main complaint of neck sufferers the existence of pain receptors in the facet joint capsule tissues that are readily provoked must be acknowledged rather than overlooked especially in efforts to avert or minimize their subnormal functions that can collectively or independently heighten a state of extensive, intensive and widespread pain and pain hypersensitivity [16].

Others indicate cases with neck pain tend to exhibit an overall stiffer and more rigid neck motor control pattern than healthy controls and one that that may implicate the joint receptors as well as proprioception. This group may also show signs of a slower movement velocity, as well as an increased degree of head steadiness and a more rigid head trajectory and head motion pattern. Yet, it appeared only neck flexibility demonstrated a significant association with the selectively observed clinical features among those with neck pain. Nonetheless, many factors were not studied and those that were may have altered the selected response patterns due to fatigue or pain or both [29]. Factors such as headaches, balance, walking ability, depression, insomnia and anxiety for example, may have been present and clinically relevant but were not examined or examined thoroughly.

Based on their research of adults with neck pain complaints, Nobe, observed that the activity of the cervical extensor and flexor muscles associated with neck motion increased and that an imbalance in activity between these muscles was generated that was not observed in healthy subjects [17]. In addition, the presence of fibromyalgic-like muscle pain and impaired cervical proprioception that arose especially in the face of muscle fatigue were cited as having a possible and significant bearing on explaining an indirect neck related loss of balance control [30]. Injury to one or more cervical located joint intrinsic and extrinsic sensory receptor sites also appears to have the possible effect on fostering capsular ligament laxity and cervical instability as well as dorsal root ganglion changes that can in provoke the production and discharge of inflammatory chemicals that sensitize joint afferents to mechanical stimulation, neck pain and spinal inflammation [31,32]. Stelion, et al., also explains that if the capsular ligaments are injured and become elongated and exhibit laxity, the ensuing onset of excessive movement between one or more cervical vertebrae can cause nerve irritation and associated symptoms of vertigo, tinnitus, dizziness, facial and arm pain, plus migraine headaches. This can also cause muscle spasm, joint movement crepitation, and/or paresthesia in addition to chronic neck pain.

Additionally, due to the apparent stimulation of the sensory nerve network supplying the C1-T3 dorsal root ganglia, Ohton, et al., who studied cases with cervical facet and whiplash lesions noted this group sometimes experienced diffuse neck pain, as well as shoulder pain, rather than localized pain [33]. Others have implied cervical pain could also arise independently or collectively consequent to the excitation of those mechanoreceptors located in the cervical intervertebral discs that may be damaged at one or more neck sites [34]. In any event, cases demonstrating a higher pain intensity than others regardless of cause show they have significant joint position sense defects that could due to the abnormal stimulation of the sensory nerve network supplying one or more facet joint capsules or alternately from disuse atrophy of the surrounding muscles that may decrease the ability to detect displacements joint control and muscle force generation. Reddy, et al., noted neck extensor muscle endurance a

vitally influential factor for maintaining optimal cervical spine function during prolonged tasks was often observed to be defective along with position sense in those with chronic neck pain [35]. Moreover, there is often not only a loss of joint sense but also evidence that directional and velocity sensorimotor receptors that guide joint movements may be impaired and misinterpreted by the central nervous system on receipt thereof in cases with cervical spine damage [14,36]. These inputs that may extend from the facet capsular receptors and others and thereby to muscle spindle outputs may also impair vision and balance as well as heightening pain and headaches and a reduced ability to render timely accurate postural adjustments during certain neck movements [37-42]. In addition, vestibular abnormalities such as vertigo may arise if abnormal cervical proprioceptive discharges originating in the cervical joint, muscles, vertebral discs, tendons and tendon junctions and ligaments remain undetected, especially those located in the C1-3 upper cervical regions [40,43,44].

Over time, there may also be associated changes in local muscle control associated inputs to articular mechanoreceptors in response to changing stresses and strains of neck movements, alongside irreversible losses of cervical joint position sense, eye movement control and postural stability. As well, reports of muscle fatigue, bone strain, dizziness and gait unsteadiness may follow along with pain regardless of originating site of dysfunction [9,41,45] in the face of increasing pain and the heightened static and dynamic sensitivity declines of the adjacent and related muscle spindles of the less efficient muscles and their reflex responses, which may yet involve or stem from damaged or destroyed joint sensory receptors [41].

As a result, Lin, et al., conclude that the highly varied and influential essential features of postural control system that protect joints may warrant high degrees of attention in efforts to avert or minimize damaging alterations to the neck joints other than age [46]. This includes the control of cervical joint stability, performance-based balance, posture and cervical proprioception. Alternately, a failure to appreciate and obtain accurate related clinical understandings can be predicted to generate long lasting pain problems, radiculopathy-irritation and/or compression of the nerve root and/or myelopathy, high levels of activation of certain muscles but not others, muscle damage and inflammation, sleep disturbances and dizziness [43,44,46-48].

Neuromotor inefficiency could also be a significant causative as well as a pathogenic factor that may worsen in those with high pain, limited functional ability, gait challenges and the adoption of sedentary behaviors that impact joint nutrition and lubrication. Moreover, without due attention, it is possible the ability to attenuate joint impact will decline progressively, especially in the older adult with signs of age related muscle protein losses, thus adding to the spread of the disease along with a cascade of ill effects that may not otherwise be inevitable.

Discussion

Can cervical spine osteoarthritis be prevented or mitigated and if not is this a situation one should simply accept rather than act on? Years of study devoted to examining this issue and others plus efforts to tease out causes of neck pain other than age show many believe neck osteoarthritis disability is not inevitable but understandable and actionable. In this regard, data have tended to increasingly point to a possible moderating influence and interaction between neural based impulses and joint biomechanics in the region of the cervical spine among other health related factors. On the whole, it now appears increasingly challenging to argue against the need to better understand cervical spine degeneration mechanisms plus the idea that the whole motor system may be implicated [49].

In particular, mounting evidence points to a role for the disruption of normal sources of proprioceptive activity and their responses as these may affect head, limb, eye and lower limb functions as well as cervical spine integrity [50]. In addition to pain, it appears sensory alterations of any type that have a bearing on muscle force generation can induce as well as explain headaches, brachalgia or arm aching of compressive and/inflammatory origin, as well as motor system and motor neuron alterations, such as muscle hyperactivity or excess weakness and muscle imbalance found in cases with varying degrees of osteoarthritis cervical alterations.

There may also be a variety of other muscle reactions that induce contractures, joint instability and a reduced irritability threshold and pain. Sensory abnormalities arising in weak or atrophied muscles or deranged ligaments may induce further impairments if they fail to repeatedly exert timely and well modulated motor responses to perturbations that in turn lead to the gradual or acute attrition of one of more cervical spinal structural elements, including its supportive ligaments [48].

Other data show that these subnormal sensorimotor responses are not arbitrary but manifest clinically in typical patterns of muscular response in the face of cervical joint osteoarthritis presence as measurable alterations in agonist antagonist neck muscle balances [49] and with some muscles being over reactive and others under reactive. This situation commonly obviates the attainment of what is normally an inherently generated ideal cervical joint posture and set of responses and consequent movements or non movements designed to foster joint protection. In turn and in the face of prolonged muscle imbalances, one can thus expect increasing bouts of uneven joint loading, increased stresses on some joints and soft tissues, additional pain and possible alterations in muscle afferent inputs and afferent neural traffic patterns that engender joint inflammation, vertigo and further cervical proprioception deficits [9,40,49-51]. It is also possible to observe changes in gait that emanate from severe forms of disc herniation [48].

In early groundbreaking work, Gracovetsky and Farfan, very well established orthopedic scientists listed the essentials of a healthy joint [52]. They included 1) having an intact sensory system; 2) CNS coordination; 3) muscle responsiveness and the well-timed and modulated integration actions thereof to maintain structural integrity. In this regard, not only must muscles be capable of well timed and appropriately modulated contractions, but without the correct amount and rate of tension joint destruction would almost be assured.

However, as a result of deafferentation, age and/or a lack of appropriate articular sensory feedback processes, reaction time as well as force generating reactions may be altered within the muscular system such that repetitive impulse loading of poorly protected joints is likely to manifest during activity with dire consequences. Over time exposure to perpetual and abnormal joint stresses may induce joint degenerative changes that become rapidly progressive or chronic at some point. Moreover, if remediation is suboptimal or not forthcoming and carefully integrated in consideration of the diverse morphology, joint and muscle nerve supply of the diseased or painful neck joints their functionally beneficial relationship to head posture and movement control may wane, even if the local cervical pain relief is forthcoming via injections or surgical intervention [53]. Even here, additional deterministic factors that may also have an influence and include injury, injury severity, injury location and extent, overall health status, body mass factors, age and overall general prognosis and upper cervical muscle and golgi tendon organ position sense receptor status [54,55].

In light of the above, it is clear no single remedy can uniformly induce cessation of the disease or pain remittance readily and completely. Since surgery is a last resort and may not be completely without risk, non pharmacologic therapies used with some success for some time include but are not limited to:

- Thermotherapy, laser, cupping therapy and ultrasound applications [56,79]
- A combination of high-intensity laser and exercise therapy [57,58]
- Electroacupuncture, acupuncture and percutaneous neuromuscular stimulation [59,60,68,80]
- Manipulation and mobilization therapies conducted manually as well as kinesio-taping, massage and controlled skeletal traction, head and neck position sense as well as movement retraining, balance and strengthening exercises [11,12,24,48,61,62]

In all cases, treated or untreated, the affected individual should probably avoid sudden exertion, excess muscle or capsular stretching or repetitive movements even though often advocated as beneficial [16,31,32]. This is because, in part, excessive cervical facet joint capsule stretch can activate nociceptors that lead to prolonged neural after discharges and can cause damage to axons in the capsule or stretched receptor at other vulnerable sites causing possible symptoms of persistent pain [22]. In other cases, short term periods of immobilization, rather than mobilization may prove beneficial as well [48].

In cases that have indeed advanced to central pain sensitization levels, cognitive behavioral therapy and appropriate patient education and physical therapies such as transcutaneous electrical stimulation believed to foster endorphin production and possibly dopamine ameliorating processes may be helpful as well [7,73,74]. However, in comparison to the possible benefits of insightfully applied exercises and electromagnetic therapy, a failure to apprehend the importance of consistent joint protection and desirable ergonomic home and workplace strategies, as well as the application of psychological interventions used in isolation is likely to be prove less impactful than not [67]. Masking pain with opioids, injectables and various corticosteroids as well as prolonged use of neck supports should be avoided as far as possible [48]. Evidence does point however to possible cumulative consequent to joint protection education, good nutrition, stress reduction, pillow adjustments, behavioral therapy

and nutraceuticals found to reduce joint inflammation [75,76].

In all instances and until more research is forthcoming, it appears safe to say that very careful understandings and analyses of the possible sources of cervical pain in any region that encompass the nerve network sources in the cervical region will potentially enable the development of a rational patient specific and oriented selection of treatments and although likely to differ widely these are expected to prove additive in benefits, rather than not.

Moreover, based on what we know about the painless origins of most osteoarthritis forms, as well as oftentimes poor radiographic ability to always detect osteoarthritis accurately, those who complain of neck pain should be monitored prospectively. This is to minimize the probable occurrence of any long term loss or disturbance of the neck mechanoreceptor inputs and outputs including gait, as well as postural disturbances, dizziness, subnormal reflex adjustments, including ocular reflexes that govern postural equilibrium positional and movement awareness and responsiveness to perturbations, along with progressive vertebral disc attrition that appear hard to initially detect and reverse without surgery [48,62].

There is also emerging evidence regarding possible more complex innervation networks and implications than perceived earlier, plus largely unexplored psychological as well as neuromotor ramifications thereof that may apply to cervical derangement and destruction processes in its own right as well as its disability [63]. In the interim, it can be argued that managing a client's neck pain complaints and enhancing their pain coping skills, along with extensive diagnostic follow ups will yiled functional benefits even in the face of any irreversible pathology [64-68]. This appears imperative to consider in light of the immense suffering incurred otherwise by many and that may not be directly linked to or commensurate with the degree of observable pathology [69,70].

In the interim, it seems apparent that nerves supplying the neck region form a multi directional linkage and degree of impacts in the realm of cervical osteoarthritis and its most common symptom, namely pain. In addition to local joint dysfunction, disturbances in locomotor function may follow a loss of deep neck region proprioception, as may vertigo and nystagmus, along with life quality declines that warrant exploration [8,71,72]. Conservative intervention can yet help here and although no one modality appears best each should be examined more thoroughly and systematically [76-80].

Conclusion

Although this report provides but a brief overview and snapshot and is not a quantitative or systematic one, based on a 25 year study by the author of this topic and many clinical years of practice, it appears safe to say:

- When attempting to understand the nature of painful cervical osteoarthritis, its diverse origins, including the role of sensory receptors in the cervical spine and their cortical influences that have considerable functional significance should not be overlooked
- Combination therapies applied carefully over time and efforts to address remediable risk factors such as injury and obesity are more likely to succeed than singular approaches implemented on a single occasion or sporadically
- The same ideas also pertain in the event invasive strategies are sought and enacted
- Careful clinical assessments, follow-ups and interventions can help avert a sizeable degree of suffering as well as health threats to many older adults as well as younger adults who sustain neck injuries and/or osteoarthritis pathology
- Those experiencing chronic unrelenting cervical pain and degeneration determinants should be carefully assessed as well
- Psychological interventions alone, while probably ineffective, may greatly enhance overall cervical spine injury and disorder outcomes

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In sum, in all cases, it appears more can be done and solutions to mitigating cervical pain and possible osteoarthritis development and its multiple health challenges may prove highly beneficial physiologically as well as lowering costs. Indeed, as per Ferreira and de Luca we assert that cervical osteoarthritis encompasses more than just pain and its prevention has immense ramifications for the wellbeing and independence of older people within the community, including a marked life quality impact [65]. It is however, a potent disabler that directly threatens efforts to support healthy ageing locally and globally, thus more should be done here in our view to examine the role of neuromotor correlates and related opportunities to mitigate osteoarthritis cervical pain.

Conflict of Interests

The authors declare that they have no conflict of interest in this paper.

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