International Journal of Chronic Diseases & Therapy (IJCDT)
ISSN: 2572-7613

Can Cervical Osteoarthritis Pain be Treated More Successfully? An Umbrella Review of its Neural Sources and Treatment Opportunities

Research Article

Ray Marks*

Osteoarthritis Research Center, Unit 2, Box 5B, Willowbrook-Charnwood Postal Depot, Markham, Ont L3T, 5H3, Canada.

Abstract

In the mature adult, the cervical spine designed to afford both mobility and stability is highly vulnerable to injury and the development of painful osteoarthritis. 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 Vertebrae; Chronic Neck Pain; Intervention; Mechanoreceptors; Older Adults; Proprioception.

Introduction

Although largely well designed and 'serviceable' for many decades without any major complication, the integrated functioning of the cervical spine comprising seven vertically oriented bone units or vertebrae, and their dorsally situated bone projections and bilateral associated facet joint freely moving synovial articulations vital for the provision of neck joint stability as well as overall spinal mobility may be subject to injury and progressive degradation as well as age related diseases. As such, even though structured to foster function and to safely house the upper regions of the spinal cord, and extending from the base of the head to the thorax, the seven cervical vertebral bodies separated by shock absorbing restraining intervertebral cartilaginous discs may fail to do this if damaged or diseased. If so, there may be incremental and immense signs of emergent dysfunction and incessantly painful osteoarthritis joint pathology and movement impairments. That is, despite acting in harmony to ensure a variety of surrounding as well as distant muscle reflexes that afford optimal head support, the ability to pivot the head, 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 do so readily and incrementally and adversely. This system can also fail if nerves located in the small nerve root exit sites located on the vertebrae, as well as the facet joint articulations are impacted mechanically, as well as biochemically, and structurally and thus elicit abnormal impulses or fail to react in the face of impact or sudden perturbations in a well modulated timely manner.

Since osteoarthritis is now considered a disease of the whole joint, its presentation may also implicate damage to other neural sites as well, including those in the surrounding neck muscles, ligaments, nerve sheaths, tendons, vertebral discs and joint capsules, and central nervous system disturbances [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].

***Corresponding Author**:
Ray Marks,
Osteoarthritis Research Center, Unit 2, Box 5B, Willowbrook-Charnwood Postal Depot, Markham, Ont L3T, 5H3, Canada.
Tel: +1-905-889-2725
E-mail: rm226@columbia.edu/Dr.RayMarks@osteoarthritisreserachcenter.com

Received: December 15, 2024
Accepted: December 23, 2024
Published: December 30, 2024

Citation: Ray Marks. Can Cervical Osteoarthritis Pain be Treated More Successfully? An Umbrella Review of its Neural Sources and Treatment Opportunities. *Int J Chronic Dis Ther.* 2024;9(3):154-162.

Today, and as population's age, this health challenge is a major concern because with the presence of persistent cervical pain older adults may experience a marked reduction in their ability to function physically and confidently. This can prove even more disabling if their 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 that will be increasingly hard to reverse.

Moreover, even in the younger adult who has incurred a neck injury, as time advances, it is common to observe signs of possible progressive and pathological articular changes, declines in desirable joint based cellular biosynthetic activities, an increase in pain and possible numbness in the neck and arm as well as neck stiffness and muscle weakness, 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 reaction and the subsequent formation of new bone [7, 8].

Ozen et al. [9] notes this above scenario is likely to occur quite frequently because the cervical spine is one of the major affected areas in axial spondyloarthritis. They suggest this may be partly due to it being an essential region housing many diverse proprioceptive receptors vital for motor control and joint protection. However, unraveling the source of neck pain and effectively mitigating this when present is commonly challenging as multiple pain inputs and others may interact at one or more levels of the cervical column cartilaginous vertebral body components, plus their associated ligaments and protective membranous tissue covering the dorsal and ventral horns of the spinal cord and brainstem, plus the dorsal compartment joints of the vertebral arches and their ligaments, mechanoreceptor arrays and muscles [2, 9].

Osteoarthritis, which is increasingly common may thus not only be impacted or develop due to aging factors, but to alterations in the bone or neural tissues in response to injury and/or its severity. Indeed in our view many older adults and younger adults do not have to suffer unduly from cervical osteoarthritis pain as multiple determinants exist that in our view are actionable or can be preempted or attenuated. Many too, can avoid surgery or bouts of injections and narcotic usage through the adoption of one or more preventive measures, and efforts to carefully tease out the role played by neuromotor mechanisms in the disease process.

However, if severe and unrelenting, or overlooked and unchecked, those inherent motor control interactions that normally protect the individual from joint dysfunction may fail to display those adaptive neural actions that would otherwise foster optimally efficient and timely well modulated motor movements and posture. This in turn, may further induce an even more serious condition termed cervical myelopathy - the most common cause of spinal cord impairment among older people and one to avoid if possible because affected cases typically present with multiple signs of gait dysfunction as well as hand impairments, adverse neural tract signs of dysfunction, and often a rapid neurological decline. Moreover, even if only mildly impacted, a degree of cervical-spine sensorimotor dyscontrol that becomes chronic is strongly associated with a recurrence or perpetuation of neck pain and possible heightened rates of joint dysfunction [3, 5, 11, 12].

Yet, when compared to other more topical health issues, osteoarthritis is often neglected and less frequently viewed as a serious health condition and one producing an untold burden and costly outcome on adults of all ages, especially the older adult. Involving the presence of focal or complete lesions of the articular cartilage lining of one or more joints, such as those in the neck region, as well as various degrees of bone and ligament attrition and remodelling, inflammation and immense disability, the condition can progress at remarkable rate, even though a role for preventative efforts to counter these changes is being discussed in anticipation of its promise.

In particular, the ability to isolate the causative factor[s] underlying the presence of chronic neck pain and possible functionally undesirable preventable degrees of osteoarthritis pathology is thus of considerable import given the failures over the years to overcome these disabling conditions. Especially challenging in this regard is accurate and precise isolation and detection of any lesion and its underlying causes. Yet, many measures commonly used are often shown to be less than accurate or reliable, or ecologically valid as far as representing what actually emerges during 'real time' daily actions and encounters [5]. Also, very few studies adequately represent the important role played by the processes of kinesthesia and proprioceptively derived body awareness sensations and their highly selective attributes that underpin the elicitation plus the integration of information from multiple tissue sources such as the skin, joints, muscles, and tendons and joints in our view. Their projected influences and role in mediating or moderating joint stability and load distribution in the cervical spine of those with neck pain may hence be overlooked or underestimated, thus impactful therapies may not be attempted and unsafe approaches fail to be avoided [5].

However, even though limited, a study of 135 cases with neck pain recently revealed subnormal findings with respect to direction-specific repositioning tests generated during flexion, extension or rotations that implied the possible influence of subnormal central and peripheral sensorimotor adaptations [5] plus the presence of poorly integrated information from the surrounding neck joint and muscle receptors [9]. Findings may also have implicated suboptimal co-ordination of several key muscles in this regard [2] and that can be altered structurally and functionally by age, osteoarthritis or other joint diseases, trauma or some form of associated joint dysfunction [6] such as abnormal side to side as well as rotary movements of the neck [3, 5] plus head posture [5] and shoulder girdle misalignments and does not discount changes in the integrity of periarticular ligaments and muscles in the presence of disease.

Indeed ligament structures in the intervertebral formamina or intervertebral formanins ligaments of the cervical spine that could be assumed protective in maintaining the position, shape, and function of nerve roots under normal conditions could aggravate the symptoms of cervical nerve root radicular pain associated with other pathological conditions such as osteoarthritis and types of nerve compression [13]. This appears noteworthy to consider, because as explained by Peng [14] chronic neck pain regardless of origin can cause structural and functional impair-

ments of several articular tissues such as the muscles of the neck; the excessive activation of pain evoking receptors located in degenerative cervical discs and facet joints that can likely produce a large number of erroneous sensory or stimulatory signals as well as joint biochemistry alterations. In addition, Sung [15] proposes problems that occur in the ligaments or muscles of the upper cervical spine can cause a form of proprioceptive confusion, and possible subsequent inaccuracy of information to the vestibular nucleus, resulting in abnormal motor reactions or adaptations that can lead to cervicogenic dizziness. There may also be measureable instances of extreme hypersensitivity of the neck neural nociceptors that are evoked readily by even the most modest movement and/or thermal stimuli rather than those directly associated with the disease specifically [16].

Indeed, despite years of research, how to reverse chronic neck pain or even attenuate this effectively is not a simplistic task and its persistence has specific as well as far reaching personal and socioeconomic impacts that have been discussed for some time without resolution [17].

In an effort to try to meet the increasing challenges faced by older adults with osteoarthritis, the purpose of this paper was to investigate if inroads could be made here as far as its presence in the neck or cervical region. The focus was on reviewing 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 craniocervical disorders of biomechanical origin such as cervical osteoarthritis.

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, articles specifically salient using the terms cervical afferent pathways, cervical osteoarthritis, chronic neck pain, and motor control were sought. The terms postural and kinaesthetic perceptions that also served as key words were used and followed that described by the authors. These data were taken to represent the neural attributes of the conscious awareness of static joint position, as well as movement sense and direction plus velocity as involved in everyday joint movements and protection, as well as posture and stability. The article does not discuss the role of osteoarthritis biology, pharmacologic and possible gene therapy, or any invasive forms of cervical spine intervention or the biochemistry of osteoarthritis degenerative processes. Moreover, it does not differentiate between the differing articular receptors specifically as aspects of the total neck or cervical spine nerve network and its importance as regards neck pain and injury in the absence of a cervical osteoarthritis diagnosis. All years of study were acceptable as were all forms of study and study substrates as long as there was a bearing on understanding the complexity and underlying nerve linkages to osteoarthritis and neck related pain. The term proprioception that refers as per Ozen et al. [9] to the awareness of body parts including joint position sense, kinesthesia, and the sense of muscle force is increasingly thought relevant to understanding the pathology of chronic pain and joint dysfunction. 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 [18]. A detailed overview on cervical spine anatomy and its implications is located at the Cleveland Clinic website: https://my.clevelandclinic.org/health/articles/22278-cervical-spine.

Key Findings

Since the inception of efforts to understand neck motion and its dysfunction when it arises, it has been clear that understanding the role of the joint sensory receptors and their 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. While the study of the nerves and their ramifications and functions in the neck region was initially largely examined in isolation, and in animal models under anesthesia or on tissue samples of deceased animals or surgically removed human tissues it is now seen as a possible parameter of note to pursue further in clinical efforts to better understand the nature of cervical damage commonly affecting one or more cervical vertebrae in many older adults as well as younger adults. Such research seems quite urgent if we consider the possible impact of neck injuries on the extensive cervical articular located receptors alone, plus their possible related neck pain effects on the induction and perpetuation of vertigo, dizziness, and/or nystagmus responses. Additionally, locomotor performance disturbances and losses or excess joint range of motion, muscle fatigue and disturbances in proprioception as well as muscle nerves and their vertebral attachments may be provoked in cases with cervical pain and osteoarthritis quite readily and can lead to movement evoked postural changes, falls and further injury and debility [16].

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 dysfunction as predicted to some degree by early studies performed by Wyke and Palacek [19]. This group repeatedly found as with all mammalian joints those of humans were supplied by four basic albeit differing functionally diverse joint receptor types that interacted 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. These reflexes were considered of extreme importance in the control of posture, joint position, direction, amplitude and velocity, intra-articular pressure changes, gait and respiration and were also found to influence the reactivity of the ocular as well as the mandibular muscles [19]. They had further influences on upper extremity limb movement, joint acceleration and deceleration, and cortical interactions [19, 20]. In addition, some cervical nerves supplying the surrounding muscles or running through

muscle and their vertebral attachments have been implicated in posture control [11]. As well, among the many mechanoreceptors found to influence postural control and gait, many were 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 [23] 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. 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.

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 [14, 27] have tended to emphasize the importance of proprioceptive mechanisms in neck pain development and progression. According to Neuhuber and Zenker [27] more specifically, the consistent collateralisation pattern of rostral cervical afferents along their whole rostrocaudal course enables their connections to 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.

As such, Peng [14] recommended cases with unrelenting bouts of neck pain should be assessed and managed for cervical proprioceptive impairments and sensorimotor control disturbances as indicated. Chen [28] who confirmed the existence of receptors in the facet joint capsule indicated that the capsule probably has pain as well as proprioceptive sensory functions. 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. Nonethelesss, 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 [17] 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. 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 bearing on explaining an indirect neck related loss of balance control [30]. Injury to a cervical located joint also appears to have the possible effect on fostering capsular ligament laxity and cervical instability as well as dorsal root ganglion changes in inflammatory provoking chemicals that sensitize joint afferents to mechanical stimulation, neck pain, and spinal inflammation [31, 32].

Ohton et al. [33] who studied patients with cervical facet and whiplash lesions noted this group sometimes experienced diffuse neck pain, headaches, arm, and shoulder pain that was conceivably due to the stimulation of the sensory nerve network supplying the facet joints and derived from the C1-T3 dorsal root ganglia. As well, some cervix nerves entered the paravertebral sympathetic nerve trunks and reached the dorsal root ganglia at multi segmental levels.

As discussed by Bogduk et al. [34] cervical pain could also arise from receptors located in the cervical intervertebral discs that may be damaged at one or more neck sites. In addition, recent research showing proprioception is impaired in subjects with cervical spondylosis offers an additional explanation wherein a higher pain intensity correlates with greater cervical joint position sense defects. Reddy et al. [35] further proposed neck extensor endurance a capacity somewhat vital for maintaining optimal cer-

vical spine function during prolonged tasks was often observed to be defective along with position sense in those with chronic neck pain. Moreover, there is 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 findings may also interact and thereby explain observations from the clinic where patients may voice concerns about their vision and balance as well as referred pain and headaches [37-39] and a reduced ability to render timely postural adjustments during certain neck movements [40].

Sufficient anatomic and neurophysiological research also points to damage to either the joint capsules or nerves or cortical pathways and cerebellum that can indeed induce neck muscle tone alterations and possible movement dysfunction, postural abnormalities, and widespread pain [19, 41, 42]. In addition, vestibular abnormalities such as vertigo may arise if abnormal cervical proprioceptive discharges originating in the cervical joint, muscles, tendons and tendon junctions, and ligaments remain undetected, especially those located in the C1-3 upper cervical regions [43, 44].

In turn, prolonged dysfunction of one or more of the cervical sensory receptors can markedly alter the normal integration of well timed and modulated sensorimotor control responses that protect joints and render movement efficient at low energy cost. Over time, there may also be associated changes in cervical joint position sense, eye movement control and postural stability, reports of dizziness and unsteadiness along with pain regardless of originating site of dysfunction [9, 41, 45] and caused by cervical degenerative disease where the elevation of inflammatory cytokines, may stimulate the mechanoreceptors in degenerated discs thereby evoking peripheral sensitization. As well, abnormal cervical proprioceptive inputs from the mechanoreceptors may be transmitted to the central nervous system, resulting in sensory mismatches with vestibular and visual information in the face of increasing pain and the heightened sensitivity of the adjacent and related muscle spindles [41].

As a result, Lin et al. [46] conclude that specific aspects of the postural control system may warrant attention in efforts to avert or minimize damaging alterations in the control of joint stability, performance-based balance, posture, and cervical proprioception, and long lasting pain problems, radiculopathy-irritation and/or compression of the nerve root and/or myelopathy, sleep disturbances, and dizziness [43, 44, 46-48].

Discussion

Overview and commentary

Years of study devoted to uncovering the intricacies of the articular neurology of the cervical spine in various invertebrate and vertebrate models plus efforts to tease out causes of neck pain other than age, have tended to point to a strong interaction between neural based impulses and joint biomechanics among other health related factors. On the whole, it is now 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 this regard, 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 particular, in addition to pain there may sensory alterations, headaches, brachalgia or arm aching of compressive and/inflammatory origin,, as well as motor system and motor neuron alterations. There may be a variety of muscle reactions that are derived from muscles around deranged neck joints that may elicit muscle spasm and various degrees of reflexive muscle dysfunction, contractures, or alternately, muscle hypotonia, weakness, 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. These reactions tend to systematically elicit measurable alterations in agonist antagonist neck muscle balances [49] and with some 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 [49] that engender joint inflammation, and further cervical proprioception deficits [9, 50, 51]. It is also possible to observe changes in gait that emanate from severe forms of disc herniation [48].

Gracovetsky and Farfan [52] listing the essentials of a healthy joint 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 as proposed by Salo [53].

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].

Intervention opportunities

In light of the above, it is our view that to advance the well being and life quality of those with neck pain and/or chronic osteoarthritis lesions of one more cervical spinal origins, 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 including thermotherapy, lasers, and ultrasound may be helpful [56]. A combination of high-intensity laser therapy and exercise therapy may further provide substantive pain relief [57] as may exercises that build on proprioceptive neural facilitation understandings [58].

Likewise, acupuncture especially electroacupuncture appears to be a further promising pain alleviating approach as well one that seems to work well when applied incrementally and should be explored further along with the application of percutaneous neuromuscular stimulation [59, 60, 68]. In all these cases there may be a high chance of improving upon current successes by insightful efforts to selectively stimulate those neck receptors that are suppressed, while deactivating those that are triggered excessively and as advocated in accord with the Gate Control Theory of pain production and amelioration.

Johnson [24] alluded to a possible salient role for reducing pain via manipulation that was based on debatable but possible effects of manual therapy on the function of the sympathetic nervous system and a possible associated change in autonomic activity and pain relief. As Johnson outlined, much of the focus here has been directed towards the descending inhibitory influences of the sympathetic nervous system on the spinal cord in order to explain the immediacy of effect observed with manipulation-induced analgesia and in our view is a potentially valuable one to explore.

Manipulation and mobilization therapies conducted manually as well as massage and controlled skeletal traction and strengthening exercises should proceed cautiously in our view however, especially if the case in point has had recent analgesic injections or is using narcotics, skeletal muscle relaxants, and exhibits frailty, although it may help quite effectively when carried out by a skilled professional [48, 61]. The affected individual should probably avoid sudden exertion, excess muscle or capsular stretching or repetitive movements even though touted to be beneficial [16, 31, 32] and may require immobilization rather than mobilization [48].

A role for vitamin C and possibly other supplements that build cartilage and bone tissues-that may be a potent source of neck pain [75], as well as those that reduce joint inflammation, such as turmeric should be explored. In addition, a parallel role for joint protection education, neck supports and assistive devices as indicated may prove beneficial. Emotional issues that can impact pain intensity and are treatable should be duly addressed as well.

In cases of central pain sensitization, cognitive behavioral therapy and appropriate patient education and physical therapies such as transcutaneous electrical stimulation believed to foster endorphin production and possibly dopamine and electroacupuncture pain ameliorating processes may be helpful as well [7, 73, 74].

However, in comparison to the possible benefits of insightfully applied Tai Chi and Qigong type exercises and electromagnetic therapy [67] believed to impact posture and pain safely, a failure to carry out consistent joint protection and desirable ergonomic home and workplace strategies, as well as the application of psychological interventions used in isolation are less likely in our view to prove impactful and may not address the sources of neck dysfunction readily and significantly. Masking pain with opioids, injectables, and various corticosteroids as well as prolonged use of neck supports should be avoided as far as possible [48].

In all instances, and until more research, including anatomical as well as radiological, neural, cellular and molecular aspects of clinical discomfort or cervical joint disease is forthcoming, and high attention is paid to evaluation processes thereof [76], it appears safe to say that very careful understandings and analyses of the possible sources of cervical pain in any region are paramount to the development of a rational selection of treatments and although likely to differ widely are expected to prove additive in benefits, rather than not.

Moreover, based on what we know about the painless origins of most osteoarthritis forms, it appears that even if no observable or measurable evidence of any joint lesion prevails this situation 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 [62] and reverse without surgery [48].

There is also emerging evidence regarding possible more complex innervation networks and implications than perceived earlier [63], plus largely unexplored psychological ramifications of neck neural origins that may hasten cervical derangement and destruction in its own right as well as disability. Thus managing pain, and enhancing pain coping, as well as extensive diagnostic follow ups along with efforts towards mitigating any negative associated affect as much as possible in its own right may not only have functional benefits even in the face of irreversible pathology, but appears imperative, in all intervention attempts [64, 66] 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].

Older adults with this condition as well as their providers who may simply believe the disease is inevitable, and that thoughts of reversing this condition are 'heresy', may yet be able to show improvments by a dual effort to respond favourably to suboptimal neural and possible linked intracellular signals, all factors considered.

Other pathological features of cervical osteoarthritis such as various degrees of joint capsular and synovial membrane thickening, joint inflammation, ligament and tendon damage, and muscle pathology and atrophy, may indeed respond favourably to targeted treatments, thus allaying chondrocyte cell death and ensuing joint

destruction processes.

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 [76-78].

Key Conclusion

Although this review 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 afferent signals arising from the joint and muscle receptors and their cortical influences and 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.
- To avert a widespread potentially increasing public health threat as well as to optimal individual health, longevity and life quality among many older as well as younger adults prevention and early intervention appear paramount.
- Those experiencing chronic unrelenting cervical pain and degeneration determinants should be carefully assessed as well.
- In all cases solutions to mitigating cervical pain and possible osteoarthritis development these health challenges more intensive and groundbreaking research, possibly using artificial intelligence and the ability to map nerves and their ramifications in three dimensions along with tissue and muscle spindle and synovial fluid assays and cartilage biomechanical properties is recommended.

Currently, it appears safe to predict less harm will emerge to aging adults as well as health systems and costs if all cases reporting persistent neck discomfort are subject to systematic physical exams, plus sustained monitoring and screenings plus interventions that are best suited for the condition and enacted both insightfully and with fidelity so as to avert a multitude of cascading biochemical and biomechanical disturbances and their possible untold costs.

In addition to lifting limits on access to therapies that may require prolonged therapeutic efforts and resource access, public and local campaigns to promote safe driving, sports, workplace, and environmental safety, and general awareness and the importance of preventing "cervical spine locomotive syndromes" including chronic neck pain may be warranted.

In the interim, in agreement with Ferreira and de Luca [65] we assert that cervical osteoarthritis encompasses more than just pain, and has immense ramifications for the wellbeing and independence of older people within the community, including a marked life quality impact. At present though, despite its global burden, spinal pain in this group is often poorly assessed, managed, and followed up. In addition, knowledge of safe and effective treatment strategies are lacking perhaps because of the common exclusion of older people in the realm of clinical research. It is however, a potent disabler of; and its physical and personal impact directly threatens efforts to support healthy ageing locally and globally. More should consequently be done here in our view as the use of narcotics by poorly treated older adults with neck pain has enormous ramifications in its own right. Other factors such as a role for obesity, lifestyle and genetic factors should be further explored as well.

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