ARTICLE IN PRESS

TECHNIQUES IN REGIONAL ANESTHESIA AND PAIN MANAGEMENT I (IIII) III-III



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Cervical spine pain related to the facet joints

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ARTICLE INFO

Keywords: Cervicalgia Cervical facet syndrome

ABSTRACT

Neck pain is a common diagnostic entity, with a lifetime prevalence of between 65% and 80%. Appreciation of the role of the cervical facet joints in the etiology of cervical spine pain is paramount to providing sustained pain relief for individuals suffering from degenerative and posttraumatic neck pain. Studies have demonstrated that approximately 60% of patients who sustain whiplash-type rear-end motor vehicle collisions would have pain that results from the facet joints alone, or in conjunction with the cervical intervertebral disks. An appreciation of the anatomical foundation for the development of these painful conditions includes knowledge of the dual, overlapping innervation of each cervical facet joint with contributions from levels at, and above the joint. Medial branch nerves invest the joints; are held closely adherent to the articular pillars by tendons of the semispinalis capitis muscles; and can be treated using local anesthetic nerve blocks followed by radiofrequency (RF) procedures for prolonged benefit. Nerves in facet joints contain modified nociceptors, including silent nociceptors, low-threshold mechanoreceptors, and mechanically sensitive nociceptors. Nerves within facet joints are both free and encapsulated and contain Substance P and calcitonin gene-related peptide. Treatment approaches must address these diverse anatomical and physiological phenomena to provide the highest level of interventional therapy. Large, well-conducted studies have demonstrated the efficacy and safety of providing short-term symptomatic pain relief using cervical facet medial branch nerve blocks. Continuous-energy thermal lesioning RF ablation techniques of the cervical medial branches may produce pain relief that persists for up to 12 months in two-thirds of patients so treated. A systematic review of well-conducted studies recently published confirmed that the evidence in favor of using RF ablation is level II for the longterm effectiveness of RF neurotomy and facet joint nerve blocks in managing cervical facet joint pain. Imaging for performing these procedures is mandatory to assure success and to minimize adverse events from occurring. Fluoroscopy is a standard imaging technique, but ultrasound and even computed tomography scan guidance have been documented to be satisfactory in properly trained interventionalists. The cervical facet joints with their medial branches represent a reliable target for directing interventional therapies aimed at addressing nociceptive type pain, albeit with a neurogenic component. Future studies would reflect our evolving appreciation of these intricate anatomical networks of innervation and their role in the etiology of chronic headache and neck pain.

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http://dx.doi.org/10.1053/j.trap.2016.10.002 1084-208X/© 2015 Elsevier Inc. All rights reserved.

Introduction

Neck pain and cervical spine pain are common pain management issues faced not only in the United States but also on a global scale. For example, Great Britain has a yearly incidence of roughly 12 cases per 1000 persons, Canada estimates a lifetime prevalence of 67%, and the Netherlands describes 2% of primary care visits being related to pain emanating from the cervical spine.^{1,2} Multiple studies have demonstrated a lifetime prevalence of cervical pain of 65%-80% with a chronicity prevalence of 26%-44% that affects patients in the United States.³ This leads to many millions of dollars not only in medical expenses but also in lost wages from nonattendance to work, unemployment or underemployment, and disability. It is worth mentioning that neck pain is nearly as prevalent as low back or lumbar spine pain, may be underrepresented and underreported, and moreover both cervical and lumbar spinal pain can occur concomitantly.

A number of structures can contribute to spinal pain including the cervical intervertebral disks, the dorsal root ganglia, fascia, ligaments, muscles, and the zygapophyseal or facet joints, which are present from C2-C3 caudally. Indeed, pain from CO-C1 (atlanto-occipital joint) or C1-2 (atlanto-axial joint) (Figures 1 and 2) can be sources of headache and neck pain, but have been considered to be somewhat controversial sources of pain as well owing to the absence of a true joint capsule or a true neural target. Increasing evidence points to the zygapophyseal joint as a major contributor to spinal pain. This is represented in the cervical spine by 54%-60% of patients with chronic spinal pain and by 15%-45% of patients with chronic lumbar spine pain.⁴ Patients most frequently develop cervical spine pain after an inciting event, including whiplash injury or posttraumatic arthropathy, or postspinal surgical pain; however, degenerative changes are a recognized etiology as well. With whiplash injury when the patient has the forceful hyperextension followed by acute flexion the zygapophyseal joint can be disrupted or injured. Studies have shown in this scenario that the zygapophyseal joint can be fractured, can develop intra-articular hemorrhage, or undergo rupture of the joint capsule.⁵ Degenerative models attempt to

hypothesize the zygapophyseal cascade involving a 3 joint complex structure of 2 vertebral bodies, the intervertebral disk and the zygapophyseal joints, in an aging and structural description.³ Furthermore, there are 3 distinct types of nociceptors (specialized pain receptors) found within the joint capsule of the true zygapophyseal joints. These include silent nociceptors, low-threshold mechanoreceptors, and mechanically sensitive nociceptors.⁶ Nerves found here are both free and encapsulated and contain Substance P (SP) and calcitonin gene-related peptide (CGRP). CGRP is produced in both peripheral and central neurons. It is a potent peptide vasodilator and can function in the transmission of pain. CGRP is derived mainly from the cell bodies of motor neurons when synthesized in the ventral horn of the spinal cord and may contribute to the regeneration of nervous tissue after injury. SP is an undecapeptide member of the tachykinin neuropeptide family. It is a neuropeptide, acting as a neurotransmitter and as a neuromodulator. SP can be released from the peripheral terminals of sensory nerve fibers in the skin, muscle, and joints. It is proposed that this release is involved in neurogenic inflammation, which is a local inflammatory response to certain types of infection or injury occurring to the cervical facet joints. These then are the 2 primary chemical mediators of nociception involved in cervical facet joint-mediated pain and dysfunction. Pain related to the cervical zygapophyseal joints and the A-O and A-A joints is typically described as being dull and achy, continual, and located in the suboccipital and posterior neck regions.7 Indeed, Bogduk et al have carefully mapped out the overlapping innervation on the head, neck, and shoulders related to the cervical zygapophyseal joints using a provocation maneuver involving neural stimulation.7

Anatomy

The cervical spine is an intricate structure of soft tissue and bony structures that allow for stability and mobility of the head and neck. The cervical spine is composed of 7 vertebrae with intervertebral foramina found at every level to accommodate exiting spinal nerves, excluding the atlas (C1) and



Fig. 1 – (A and B) Skeletal anatomy of atlanto-occipital (A-O) and atlanto-axial (A-A) joints. The A-A joint flexes, extends, and laterally rotates the head up to 60°. (Photo courtesy: Kenneth D. Candido, MD) (Color version of figure is available online.)

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