A likely mechanism for headache of cervicogenic origin

JOHN REGGARS D.C.*

Abstract: As chiropractors an important and common presentation seen in the clinical setting is cervicogenic headache.

The entrenched vascular or psychosomatic models may not be the most common mechanism of headache. Rather a mechanism involving either joint dysfunction and/or soft tissue dysfunction appears to be a likely cause:

Key Words: Headache, cervical spine, cervicogenic headache, chiropractic, manipulation.

To fully understand the mechanism of cervicogenic headache it is first necessary to identify the pain producing structures of the cervical spine. Listed below are the primary structures involved.

(i) Atlanto-occipital joint

(ii) Atlanto-axial joint

(iii) C2-3 Zygapophyseal joints

(iv) Soft tissues - ie. muscle, ligament tendon and fascia

(v) Dura mater

(vi) Nerve Sheath

(vii) Intervertebral disc

(viii) Arteries

(ix) Veins

As chiropractors an important and common presentation seen in the clinical setting is cervicogenic headache. This may arise from intervertebral joint dysfunction alone, or in combination with functional disturbances of the supporting ligamentous and muscular structures (1).

Despite a longstanding tradition of writing in medical and heterodox circles supporting the notion of cervicogenic headaches, debate still rages regarding the role of the cervical spine in the etiology of various varieties of headache. Recent authors who have supported the validity of cervicogenic headaches and who have produced important, descriptive and explanatory models include Vernon (2), Bogduk (3), Sjaastad (4), Fredricksen (5), Pfaffenrath (6) and Jaeger (7).

Bogduk (8) in his latest research showed that stimulation of the normal zygapophyseal joints by injection of a contrast medium under fluoroscopic control produced a clinically distinguishable characteristic pattern of pain. Only the C2-3 joint produced headaches, the others producing either neck or shoulder pain. Therefore, it would appear that headaches arising from joint dysfunction below the C2-3 level cannot be due to facet irritation alone.

Other mechanisms described by Kovacs (9), Seletz (10) and Skillern (11) provide a plausible explanation for these types of headaches:

- True subluxation of the cervical apophyseal joints producing constriction of the vertebral artery or mechanical irritation of the vertebral sympathetic plexus.
- Uncovertebral joint proliferation producing vertebral artery compromise.
- Restriction and disturbance of joint motion leading to reflex muscle spasm.
- Noxious stimulations from cervical joints and muscles, producing local occipital headache syndrome via reflex irritation of the spinal accessory nerve.
- Trapezius muscle spasm resulting from cervical joint dysfunction production greater occipital neuralgic headache.

Skillern's (11) research discussed the connection between C1 and C2 sensory pathways and the second order neurons of the spinal tract of the trigeminal nerve as a cause of cervicogenic headache. Boake (12) similarly proposed that upper cervical apophyseal joint trauma, resulting in hypomobility could result in cervical headache and that this joint dysfunction may lead to spasm of the cranio-cervical musculature producing 'tension' type headaches.

Vernon (13) categorises vertebrogenic headaches into four groups:

1) Extrasegmental ie. large muscles and fascia
2) Intersegmental ie. anterior and posterior joint structures and the deep short intersegmental muscles.
3) Infrasegmantal ie. structures contained in the intervertebral foramen or the bony environs of the nerve roots.
4) Intrasegmental ie. Spinal cord.

The most likely mechanism relating to the common form of vertebrogenic headache would be a combination of both the 'extrasegemental and intrasegmental models'. That is muscular structures producing pain by themselves and/or joint dysfunction, or joint dysfunction directly causing head pain, and/or with soft tissue dysfunction.

Regardless of the mode the result is vertebrogenic headache. The exact mechanisms involved in these models can therefore be seen to overlap.

The extrasegmantal mechanism relates to the large regional cervico-thoracic and cervico-cranial muscles and with one exception (occipitofrontalis), are collectively referred to by Vernon as "regional craniocervical extensors" (13).

Poor posture and occupational strain including cervical hyperlordosis, occipital extension, anterior weight bearing and constant upright support of active arms, such as adopted by seated office workers "produces a variety of chronic low level, but accumulative stresses that may alter their function and prepare the way for primary or secondary pain states" (13).

Alternatively, cervical joint dysfunction may produce secondary arthrogenic contractions. The resultant accumulative stresses could produce irritation at the tendoperisoteal junctions of the involved muscles leading to "low level chronic foci of tenderness and pain" in the occipital, suboccipital and nuchal regions (13).

*Private Practice: Preston, Victoria 3134, Australia.

Furthermore, the "trigger" point phenomenon described by Travell (14) and others may result from chronic myofascitis which could also be attributed to accumulative low level stress and hypertonicity. These trigger points may result in referred pain patterns to the head. Entrapment of neurovascular structures such as the greater occipital nerve (11) mentioned earlier and those of the thoracic outlet may also result from postural dysfunction and muscle hypertonicity or spasm producing cephalgia (15).

The intersegmental mechanism involves the apophyseal joints, posterior ligaments, uncovertebral joints, intervertebral joints and suboccipital muscles. Emphasis here is on the nociceptors in the apophyseal joints and ligaments as described by Wyke (16). These nociceptors when irritated by mechanical or chemical stimuli may produce segmental muscle spasm or produce a 'sclerotogenous' type of referred pain as a consequence of activation of convergent neurones as described by Bogduk (3), Feinstein (17) and Kellgren (18) et al.

Degenerative joint changes are not essential for nociceptor stimulation but may also accompany joint dysfunction as well as produce cervicogenic headache as previously outlined.

In summary, when one considers the following authors (see below) it can readily be seen that the entrenched vascular or psychosomatic models may not be the most common mechanism of headache. Rather a mechanism involving either joint dysfunction and/or soft tissue dysfunction appears to be a very likely cause:

i) Boake (12) 70% of all headaches are attributable to upper cervical joint dysfunction.

ii) Jirout (14) 90% of individuals with C2-3 dysfunction have headaches.

iii) Bogduk (3) upper cervical hypomobility is the major cause of 'muscle contraction' type headaches.

REFERENCES:

1. Hilderbrant & Argyrakis, Percutaneous nerve block of the cervical facets. Manual Medicine 1986 2:2, 48-52.

2. Vernon H., Spinal manipulation and headaches of the cervical origin. Manual Medicine 1991 6:4, 73-79.

3. Bogduk N., Headaches and cervical manipulation. Med J Aust 1979, 66:65-66.

4. Sjaastad O., Fredricksen T.A., Stolt-Neilsen A. Cervicogenic headache C2 rhizopathy and occipital neuralgia: a connection. Cephalgia 1986; 6:189-195.

5. Fredrickensen T.A. Huadahl H., Sjaastad O. Cervogenic headache clinical manifestation., Cephalgia 1987;7:147-160.

6. Pfaffenrath V., Dandekar R., Pollman W. Cervicogenic headache - the clinical picture, radiological findings and hypothses on its pathophysiology. Headache 1987;27: 495-499.

7. Jaeger B. Cervicogenic headache: relationship to cervical spine dysfunction and myofascial trigger points. Proceedings of the International Headache Society, Florence, Italy 1987. Cephalgia 7: 398-399.

8. Bogduk N., Dwyer, April C., Cervical zygapophyseal joint pain patterns I & II. Spine: 15:6. 453-461.

9. Kovacs A. Subluxation and deformation of the cervical apophyseal joints: a contribution to the etiology of headache. Acta Radiol 1955: 43: 1-16.

10. Seletz E. Headache of extracranical origin C.A. Med 1958 89: 314-319.

11. Skillern P.E. Great occipital-trigeminus syndrome as revealed by induction block. Arch. Neurol Psych 1954;72: 335-340.

12. Boake H.K. Cervical Headache. Can Fam Phys 1972; 75-78.

13. Vernon H. Upper cervical syndrome. Chiropractic Diagnosis and Treatment 1988, Chapter 9, 152-188.

14. Travell J. Mechanical headache. Headache 1967 April: 23-30.

15. Raskin N.H., Howard M.W., Ehrenfeld W.K. Headache as the leading sympton of the thoracic outlet syndrome. Headache 1985; 25: 208-210.

16. Wyke B.D. (1972) Articular neurology - a review. Physiotherapy 58: 94-99.

17. Feinstein B, Langton J.N.K., Jameson R.M., Schiller F. Experiments on pain referred from deep somatic tissues. J Bone Joint Surg (AM) 1954 36: 981-997.

18. Kellgren J.H. On the distribution of pain arising from deep somatic structures with charts of segmental pain area. Clin Sci 1939; 4:35.

19. Jirout J. (1985) Comments regarding the diagnosis and treatment of dysfunctions of the C2-3 segment. Manual Med 2: 16-17.