Pain and functional disability

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ABSTRACT

The pain and functional disability in headache is tentatively presented with respect to the displayed causal relationships, beginning with the nociceptor. The concept is presented that primary deep pain must stem from a pain generator (PG), of which the free C-fiber ending together with the extracellular space constitute the (inflammatory) nociceptor; and that neurogenic pain is a secondary, enhancing measure. Headache is considered to be a result of various PGs in pain-sensitive structures implicated in head locomotion. Headache occurs when nociceptive volleys from either the cephalic or cervical levels traverse the locomotor centres of the head in the upper cervical medulla and are realized as referred pain. Some clinical aspects are presented.

Pain and functional disability

Deep pain including headache is a personal experience belonging to the conceptual world. The apparent multitude of releasing factors have traditionally been dealt with in a descriptive way, which presently obliges the clinician to choose between some 120 different kinds of headache representing the final result of unclarified releasing circumstances (1). When one is dealing with a headache patient one is also dealing with a classifying system, which cannot tell the clinician or the patient exactly what is going on.

Behind the clinical, descriptive frontlines of neurology throughout this century, the basic sciences have continued to accumulate data which may allow some semantic clarifications, by using an approach starting with the cause to reach the effect and not vice versa. With the help of some of these data and their associated philosophy, I will attempt to present headache with due consideration given to the natural course of events as the background to causal relations.

During the course of evolution, bodily injury has assumed a key position among selection pressures affecting survival. Mechanically sensitive channels are found in virtually all cells including bacteria. Many of these are non-specific cation channels, which in principle could be used in a nociceptor to produce a depolarizing generator potential during mechanical strain (2).

In solid, multicellular coexistence this cellular property becomes useless, as the cells are unable to move away from a registered noxious milieu. The adaptive measure then taken is the evolution of a supervising neural system to defend a diseased area, at best with attention simultaneously given to the survival of the entire coexistence. This could be one of the basic biological principles underlying the creation of a nervous system, which is believed to issue from microtubuli, also known from bacteria as the first specific structures to sense and to move (3). Nociceptors develop in the extracellular space to assure the best possible survival of its lodging cells in creatures belonging to the moving animal kingdom.

The prototype of the vertebrates, the prochordate, as represented by the lancelet, is characterized by its bilateral, symmetrical structure. The entirely unmyelinated nervous system consists essentially of a spinal cord. It has virtually no brain since, with the lack of specific remote senses, there is no need for one. However, a supra-segmental control is assured by a few, poorly differentiated neurons around the rostral end of the neural tube corresponding to the later 3. ventricle. The first structures to differentiate from the neural crest are the nociceptive C-fibers and the sympathetic nerve fibers (4).

Man belongs to the phylum Chordata. We have inherited this entirely autonomous internal defence system. The signals from our C-nociceptors cannot be traced above the diencephalic level (5), being well hidden by the tremendous growth of a telencephalic structure to take care of our volitional control of ex-

teroceptive signals from the developed remote senses.

The large head that characterizes humans represents the enlarged, rostral part of the spinal column, a solid entity which physiologically continues to participate in vertebrate control systems, very likely dissociations between autonomous and volitional interests. It behaves autonomously like a segment, and segments must work together as a functional unit. If a pain generator (PG) cannot be pacified by nociceptively-induced pain and muscular defence in the diseased segment, more segments become involved. This is monitored by the spinal cord and is supposedly executed via C-fiber noceffectors. The initial inflammatory pain becomes enhanced by neurogenic pain (6). In this context the locomotor apparatus obliges the head and the cervical spine to work as a functional unit. All pain-sensitive structures involved in or by head locomotion can only be immobilized to the necessary degree by an induced functio laesa of the cervical spine or, eventually, by an arrest of voluntary bodily activity.

The autonomic control of the cervicocephalic region is neurobiotactically assured by convergence of the descending spinal tract of the trigeminal nerve (7-9) and its homologous tract of Lissauer (10) on the locomotor centre of the head at C2 (Fig.1). An intracranial pain generator and a pain generator in the lower cervical spine strong enough to involve upper cervical segments, can both cause headache according to the principles of referred pain (11-13). It is no wonder that Frykholm at the Cervical Pain Symposium in 1971 (14) could report a few patients of his own treated for cervicogenic migraine, who turned out to have brain tumors.

A tentative definition of headache in man might be that headache is referred pain due to traversing C-fiber volleys in a posterior horn belonging to the centre of head locomotion at C2, and coming from a primarily initiated depolarization triggered by the meeting of any noxious agent with a nociceptor consisting of a free C-fiber ending and its lodging in the extracellular space. Other vertebrate species may have another arrangement with different consequences, but the principle remains the same. In the frog, for example, with its armoured fixation of the head to the trunk, the descending spinal tract of the trigeminal nerve ends in the lumbar intumescens. Automatic escape movements of the head are assured by the hind legs. If the message is nociceptive it may help to immobilize them, and the animal will suffer from lumbalgia.

Tissues at high risk with respect to survival have the highest density of C-fibers. Increasing importance is given to the tissues in this order: muscle, fascia, tendon, joint capsule, ligament and periost (15). Thus, the transverse ligament of C1 and the posterior disc walls are highly potent pain generators.

In the brain stem the nociceptive signals are dispersed into spino-reticular pathways (5). Thus we have no reason to believe that autonomous medullo-segmentary pain defence reactions at any given level, mono- or bilaterally, can be mobilized from intracerebral neuronal activity. This means that psychogenic headache, *sensu strictiori*, does not exist, and one can put a question mark next to the currently presumed initiation of hemicrania (migraine) by afflicted intracerebral neurons for unknown reasons (16).

If one has confidence in this layout of headache, one may be convinced that the main bulk of headaches are cervicogenic in nature. However, the diagnosis should be made with precaution and reservation, as long as we represent a minority in this field. Nevertheless, the concept of cervicogenic hemicrania with or without vertebro-basilar implications can be adopted without restraint as long as no proof to the contrary exists.

The cervical spine is a tool for movements and posturing of the head. One may have a lot of broken, rusty tools at one's disposal, which doesn't matter if they are not utilized. Thus, doctors for instance may have relatively few movement problems, while female cleaners often have to quit their jobs while still in their 40's. In later years most of us stiffen progressively in the cervical region due to entrapment of spinal nerve root sleeves and vertebral arteries caused by encroachment or adherence related to the unco-vertebral regions (17). Sustained awkward head positions or a sudden twist may create a periarterial PG leading to cervicogenic hemicrania or a painless perturbation of the blood flow in a

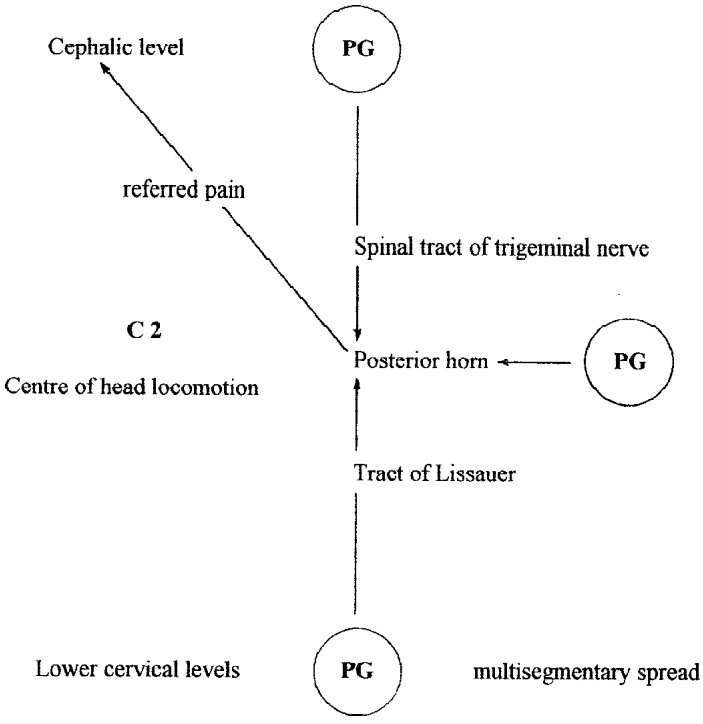

Fig. 1. The pain generator (PG) principle in headache.

vertebral artery. In both events transient neurological troubles or completed stroke can be the result. The architecture of the vertebro-basilar system in each individual patient will most likely be a decisive factor (18-21). The effect of repeated vertebro-basilar ischemia is, predictably, cumulative and may well account for a broad spectrum of nervous deficiencies, not originating in the cervical spine according to currently accepted concepts.

Headache is liable to cease when the responsible pain generator in a root sleeve becomes associated with deafferentiation. Most disc troubles are realized at that time, 10 to 20 or more years after incapacitating, recurrent headaches and proximal pain in the shoulder girdle from a PG in a posterior disc wall.

The atlanto-axial joint has apparently been neglected as a common potential PG to headache. Normally the anterior arch of C1 stays in close opposition to the tooth of C2 in all head positions (22), and the head is in neutral position carried by biconvex joint facets at a 5° backward inclination. Any degenerative disc condition with cervical rectitude will abolish the backward tilt and the head then tends to slide forward. The strain imposed on the transverse ligament is difficult to relieve, unless one lies down. Tear and relaxation of the transverse ligament from injury leads to the same problem, particularly among desk workers. In a personal study of African headache patients, I found a lack of parallelism of the facets in the atlanto-dental joint in 49% and cervical rectitude in 48% of them. In randomized autopsies only 1 of 300 had still intact cervical discs at the age of 20 (23).

Children are known to fail at school and students at university due to disabeling holocrania, very likely from atlanto-axial strain. This may be a cause for anxiety and not vice versa.

Consequently it may be recommended that the official diagnosis be supplemented with a personal note concerning the expected location of the pain generator. This will force the clinician to think in terms of physiology and make it easier to find the best way to pacify the pain generator. A long-term consequence might be to modify the IHS headache classification system.

References

1. International Headache Society: Diagnostic criteria. *In: Members Handbook*, Scandinavian University Press, 1999: 49-108.
2. WALTERS ET: Comparative and evolutionary aspects of nociceptor function. *In*: BELMONTE C and CERVERO F (Eds.): *Neurobiology of Nociceptors*, New York, Oxford University Press, 1996: 92-114.
3. MARGULIS L, SAGAN D: *Microcosmos*. University of California Press 1997: 137-54.
4. SARNAT HB, NETSKY MG: *Evolution of the Nervous System.* New York, Oxford University Press, 1974: 27-9, 137-219.
5. COLLINS FW, NULSEN FE: Studies on sensation interpreted as pain: Central nervous system pathways. *In: Clinical Neurosurgery*, Baltimore, Williams and Wilkins, 1962; 8: 271-81.
6. LA MOTTE RH: Secondary cutaneous dysaesthesia. *In*: BELMONTE C and CERVERO F: *Neurobiology of Nociceptors*, Oxford University Press, New York, 1996: 390-417.
7. SARNAT HB, NETSKY MG: *Evolution of the Nervous System*; New York, Oxford University Press, 1974: 6, 77-80.
8. KERR FWL, OLAFSON RA: Trigeminal and cervical volleys. *Arch Neurol* 1961; 5: 171-8.
9. GORDON V, LAMBERT G, MICHALIECK J: The pathway to C2 cervical spinal neurons responsive to superior sinus stimulation does not synapse in trigeminal subnucleus caudalis. *Cephalalgia* 1993; 13 (Suppl.13): 116.
10. SARNAT HB, NETSKY MG: *Evolution of the Nervous System*. New York, Oxford University Press, 1974: 64.
11. KELLGREN JH: On the distribution of pain arising from deep somatic structures with charts of segmental pain areas. *Clin Sci* 1939; 4: 35-46.
12. INMAN VT, SAUNDERS MB: Referred pain from skeletal structures. *J Nerv Ment Dis* 1944; 99: 660-7.
13. CAMPBELL DG, PARSONS CM: Referred head pain and its concomitants. *J Nerv Ment Dis* 1944; 99: 544-51.
14. FRYKHOLM R: The clinical picture. *In:* HIRSCH C and ZOTTERMAN Y (Eds.): *Cervical Pain.* Pergamon Press, 1972: 5-16.
15. FEINDEL WH, WEDDEL G, SINCLAIR DC: Pain sensibility in deep somatic structures. *J Neurol Neurosurg Psych* 1947; 10: 113-7.
16. LANCE JW: The pathophysiology of migraine: A tentative synthesis. *Path Biol* 1992; 40: 355-60.
17. LYSELL E: The pattern of motion in the cervical spine. *In*: HIRSCH C and ZOTTERMAN Y (Eds.): *Cervical Pain.* Pergamon Press, 1972: 53-8.
18. LOEB C: Clinical syndromes due to ischemia in the distribution of the vertebro-basilar arterial systtem. *In*: ZÜLCH KJ (Ed.): *Cerebral Circulation and Stroke*. Berlin/Heidelberg, Springer, 1971: 57-66.
19. RIGGS H, RUPP C: Variation in form of the circle of Willis. *Arch Neurol* 1963; 8: 24-30.
20. ALPERS BJ, BERRY RG: Circle of Willis in cerebral vascular disorders. *Arch Neurol* 1963; 8: 68-72.
21. TOOLE JF, TUCKER SH: Influence of head position upon cerebral circulation. *AMA Arch neurol* 1960; 2: 616-23.
22. DE SÈZE S. DIJAN A, MARTIN M: De l'utilité des épreuves radiologiques fonctionelles dans les traumatismes cervicaux atlas-axis. *Rev Rhum Mal Osteo-Artic* 1951; 18: 99-104.
23. TÖNDYRY G: The behaviour of the cervical discs during life. *In*: HIRSCH C and ZOTTERMAN Y (Eds.): *Cervical Pain.* Pergamon Press, 1972: 59-68.