Article *NIMHANS Journal*

Pathogenesis of Cervical Spondylosis

Volume: 06 Issue: 2S December 1988 Page: 101-105

Phillip Harris, - *Edinburgh*

"Pathogenesis" is defined as "the production or development of a disease". Cervical spondylosis is not a disease, although it may give rise to clinical symptoms and signs. It is a degenerative condition of one or more parts of the cervical spine, involving one or more intervertebral discs, and often other joints in the cervical spine. Cervical spondylosis is probably as old as man himself, and has been found in man from the most ancient civilizations, and certainly discovered in the Egyptian and Inca peoples. It can also develop in the animal kingdom, and a frightening dream would be to require to investigate and then operate on the neck of a giraffe with cervical spondylosis, especially when I remind you that the giraffe, like ourselves, only has 7 cervical vertebrae!.

Cervical spondylosis is usually symptomless, and manifest either spontaneously, or due to an acute, or more often minor repeated trauma to cervical spine. Fortunately only a small proportion of people develop neurological abnormalities. However, cervical spondylosis is the commonest cause of spinal cord disorder in people over the age of 50 years.

Anatomy

It should be remembered that the cervical portion of the spine has very special anatomical features for mobility, and there are 5 articulations. These joints are:
(a) Intervertebral discs
(b) The neurocentral joints (joints of Luschka); two on either side of each vertebra
(c) The zygapophyseal joints (facet joints); two on each side.

Between each pair of cervical vertebrae from C2, and indeed including T1, there are 5 joints, an intervertebral disc, two neurocentral joints and two zygapophyseal joints. The facet joints are situated dorsally; the neurocentral joints lie between the intervertebral disc, dorsalateral to this and ventral to the intervertebral canal for the cervical nerve root.

Pathophysiology of intervertebral disc degeneration and pathology of the neurocentral and zygapophyseal joints

Microscopic and biochemical studies show that degenerative changes can occur in one or more cervical intervertebral discs, especially as a person grows older, with involvement of the collagen in the annulus fibrosus in particular (Eyre, 1982); also proteoglycans are especially involved in the degenerative process, and in addition non-collagen proteins, cells and enzymes. Another important factor is disturbance of the nutrition of the disc. The nucleus pulposus becomes dehydrated, more fibrotic and less distinct than the annulus fibrosus, and develops fissures and clefts. Simultaneously, there are changes in the annulus fibrosus, and the outermost fibres incite development of traction

spurs. Also the cartilaginous plates undergo excess wear and becomes thin. Trauma most likely plays a secondary role, disturbing an already biochemically deranged intervertebral disc. Aging is another obvious and important factor. There would appear to be a similarity between intervertebral disc degeneration and synovial joint degeneration.

The neurocentral and the zygapophyseal joints also tend to undergo degenerative changes. Intervertebral disc degeneration with narrowing of the disc, places excess strain on the neurocentral and zygapophyseal joints, and the changes occurring at the edges of the vertebral bodies and in the neurocentral and zygapophyseal joints will eventually give rise to the development of osteophytes.

These various, usually progressive, degenerative changes in the cervical spine will tend to disturb the normal biomechanics of this part of the spine, and this will be discussed shortly. Although many of these changes can be demonstrated in plain radiographs of the cervical spine, and also by computerised tomography, it is clearly seen that osteophytes per se are usually not the only neural and/or vascular compressing agent, because an intervertebral disc protrusion and / or soft tissue hypertrophy also play major roles.

Biomechanics

In cervical spondylosis, and depending on the extent of the disease and its severity, the cervical spine becomes shorter and will loss its normal lordotic curve and will tend to become straighter and indeed may become slightly kyphotic. The cervical spinal canal becomes smaller in diameter. The patient indeed tends to compensate for these changes, and with his somewhat stiff neck he tends, at rest, to hold his chin thrust forward. As has been well-demonstrated and described by Breig (1960) in the normal cervical spine, the spinal cord and cervical nerve roots and the vessels and theca become taut in flexion and lax in extension. Extension may result in 9 to 17% or more reduction in the diameter of the spinal canal, and about 15% reduction in the diameter of the spinal cord (Waltz, 1967)

The significance of spinal cord traction and of mechanical deformation by osteophytes is stressed by Adams and Logue (1971); and Nurick (1972) has emphasized the importance of the spinal cord diameter, and of cervical spine subluxation (28.7 % of his patients) in patients with cervical spondylosis, who developed a myelopathic syndrome. My own experience over many years and many hundreds of patients, is that of cervical spine subluxation in patients with cervical spondylosis is not common.

Ventral compression of the cervical spinal cord in patients with spondylosis is present in about 3/4 of such patients, the compression is dorsal in less than 1/4 and in the remainder of the patients there is both ventral and dorsal cord compression.

Congenital abnormalities of the cervical spine, for example the Klipper-Feil deformity, can have a bearing on the development of cervical spondylosis. Very occasionally, following anterior cervical spine operations for cervical degenerative disease, whether or not bony fusion techniques have been used, secondary biomechanical, pathological and indeed clinical effects may ensue, usually at the level cranial to the site of the highest level in the cervical spine that has been operated on.

The real significance of
(a) mechanical compression factors, and
(b) ischaemic factors

is not known. The pathogenesis of the myelopathy is a complex problem, in part due to many variables, patchy cord changes, variable radiological appearances and a variety of clinical effects. Mechanical factors include the shape and diameter of the spinal canal, osteophytes, the ligamentum flavum, movement, trauma. Neck extension caused increased narrowing of the spinal canal and simultaneously increased diameter of the spinal cord. In flexion, the spinal canal becomes longer as does the spinal cord, nerve roots, vessels and theca. The intervertebral formina are reduced in diameter by about 20% in cervical spine extension (Waltz, 1967). As Frykholm (1951) showed, "root sleeve fibrosis" can be an important factor regarding nerve root involvement.

It is known that some people have small diameter cervical spinal canals, on a constitutional basis, and this will further add to neural and vascular pathology and clinical abnormalities in patients with cervical spondylosis; and yet another factor would appear to be the ventral buckling of the ligamentum flavum, which has also undergone degenerative changes, and also ventral buckling of the dura mater. Thus a pincer effect will occur, especially when the neck is extended. A factor concerning constitutional narrowing of the spinal canal, could be excess secretion of the growth hormone, causing premature closure of ossification centres. Ossification occurs about the ninth week of gestation, in each half of the yet unclosed neural arch and in the centrum of the vertebral body. Cervical and lumbar spinal stenosis may not uncommonly be seen in the same patient and I now have several such patients, who indeed have required both, estaged operations, cervical and lumbar spine decompressive procedures, as staged operations.

Vascular factors are not well understood concerning cervical spondylosis, and occlusion of the anterior spinal artery mest be very rare (Hughes, 1964 and 1978). According to Nurick (1972), implication of a vital radicular artery is unlikely to be a factor in the development of myelopathy and cervical spondylosis, his reasoning being a factor in the development of myelopathy and cervical spondylosis, his reasoning being that so few patients with radicular lesions develop myelopathy. However, although the mechanical factor may be more important, an associated vascular factor with ischaemia of the neural elements, most probably occurs. Thus perpendicular perforating vessels supplying the spinal cord may be occluded in cervical spondylosis, with ischaemia of the cortico-spinal fibres and the spino-thalamic tracts in the lateral white columns. Such fibres and tracts appear to be particularly vulnerable to ischaemia. Occlusion of venous drainage would tend to cause oedema of the neural elements.

Again one would return to the great importance of the intervertebral disc in the pathology and biomechanics of cervical spondylosis; and I have personally carried out a large number of intravertebral disc studies in the cervical spine using special lumbar puncture needles with a micro-pressure transducer in the tip of the needle. The technique and the results of the studies have not yet been reported, but the findings would appear to be different from many of the findings noted by Nachemson in his studies in the lumbar intravertebral discs.

The importance of neck movements as a factor in cervical spondylosis when clinical symptoms and signs develop was clearly shown by the work of Brain, Northfield and Wilkinson (1952). The role of acute and of chronic recurring neck trauma was reported by Symonds; and of the narrowed cervical spinal canal by Payne and Spillaine (1957). There has not, as far as I can ascertain, been much in the way of animal experimental studies in cervical spondylosis, but Gooding et al. (1975) reported on the effects of ischaemia and compression of the canine cervical spinal cord, resulting in cervical myelopathy.

Clinical

Concerning the clinical features of cervical spondylosis, as I mentioned, there may be no symptoms whatsoever, indeed for the whole of the person's life, although it may have been found, incidentally, that a person may have quite a severe degree of cervical spondylosis. This of course is not unique in the body; there may be quite gross pathological changes without any obvious coincidental symptoms, and a very common example of this is someone who may have severe varicose veins and yet have no symptoms whatsoever from them. As already mentioned, some form of trauma may be the factor to produce symptoms in the person with cervical spondylosis, but not necessarily so. We are quite familiar now-a-days with the large variety of symptoms and the several syndromes that may occur as a result of cervical spondylosis, including neck pain and stiffness, referred pain to the occipital region and to the shoulder or shoulders, the interscapular region down one or both upper limbs and occasionally over the chest. Indeed, discography, or even after injection of the supraspinous or interspinous ligaments can produce many of these symptoms, and some of the symptoms may indeed arise from disturbance of the neurocentral and zygapophyseal joints. The commonest site for cervical spondylosis is the C5/6 level and then C6/7 and C4/5, but changes can occur at one or more levels and there may indeed also be an associated intravertebral disc protrusion.

It is known that such patients are at risk from a hyperextension neck injury with a possible production of the central cord syndrome. Manipulation of the neck of the patient with cervical spondylosis can, of course, be hazardous and is to be condemned. Particular care is required when an anaesthetist intubates a patient who has cervical spondylosis.

Claudication from neck movements may occur, and indeed as long ago as 1911, Dejering described what he termed "intermittent claudication of the spinal cord".

Presumably, because of the variability of the blood supply to the cervical spinal cord, cervical spondylosis can cause neurological disturbances at a different level, usually a more cranial level, thus the vertebral level of the spondylosis.

A postero-lateral osteophyte arising from the vertebral body from intervertebral disc degeneration or from an arthritic neurocentral joint, can impinge on a vertebral artery in its canal, and then especially with certain head and neck movements, and in particular extension, a vertebral-basilar insufficiency syndrome may develop, with such features as drop attacks, postural ataxia, positional nystagmus, dizziness, tinnitus, attacks of unconsciousness, cardiac bundle branch block; and very occasionally a vertebral artery becomes completely occluded, relating in the development of the Wallenburg Syndrome. The radiological and clinical neuro-electrophysiological changes seen in patients with cervical spondylosis may or may not have a direct bearing on the clinical neurological symptoms and signs found in any particular patient. The neurosurgeon requires to consider all of these aspects of the condition most carefully before deciding on possible treatment; but this is another subject altogether really, and outwith my remit concerning this paper.