

Biomechanics of Cervical Spine

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Any object if stretched over a period of time the length keeps on increasing and the resistance offered to it will gradually decrease. This has got a clinical implication. The fibres in different layers of annulus fibrosus of disc are arranged in different directions. The outer and inner layers are at right angles to each other. Therefore the nucleus pulposus cannot be totally compressed. As per Pascal's law it would dissipate pressure in various directions and at the peripheral level the force is either elevating force or a destructing force. So in case of an injury to a disc or to a vertebra with a normal nucleus pulposus there will be a deformation of the end plate. This damage to the endplate may not be detected radiologically and may not be associated with any compression fracture.

On the other hand if the disc at the site of injury is degenerated no deformation of the end plate will occur, and instead there will be load related compression stress. Peripherally this will result in disc rupture and prolapse or a compression injury to the vertebra.

Lateral or a forward flexion produce various compressive stresses on the annulus resulting in deformities. The deformity of these structures would vary, depending on the quality of elasticity. Initially deformity will occur as long as the compressing force exists and once the force is released or withdrawn it would go back to its original shape. This is called as elastic deformation. Once this range is exceeded the deformation remain, even in the absence of load, and is called block deformity. Beyond this there will be a total mechanical failure. This response to any kind of a stress is related to the load and its duration of application. If the load is applied gradually, over a period of time, the resultant damage may not be so bad as it is with quick short duration of high magnitude load.

Consecutive layers in annulus are arranged such that when one layer is subjected to compression or rotatory loading the other layer relaxes, therefore only 50% fibres will be protecting the disc. Normal rotational tolerance of healthy human is 16% of the total additional rotational movement available. A degenerated disc can only tolerate upto 12% and beyond that at 20% rotational stress the disc might rupture.

Hydrodynamics of the disc and the nuclear colloids are important in the understanding of the degenerative process. They undergo depolymerisation with the process of degeneration. In recumbancy the spine is unloaded and the water is reabsorbed and when a person stands erect the water is expressed out. This is a commonly observed phenomenon, with a person being taller at morning and becoming shorter towards evening. Once the degeneration sets in the depolymerised colloids absorb larger quantities of water, since there is loss of turgor, the water logging itself can cause increased tension resulting in pain. At the same time the rate of water movement is slowed down which is reflected as stiff movement and pain following rest. Patients do complain pain and stiffness as they get up and stiffness decrease with morning exercises.

Water content in nucleus pulposus is not constant. Whenever there is stress on one part of the nucleus the water gets shifted and gets reallocated to other areas. If a person maintains a constant position for eg. keeping the neck extended or flexed or sleeping in lateral position without a pillow, will shift the water from the stressed segment. This will offer resistance for change into different position at the end. If the disc is degenerated at one level there will be water logging in other segments resulting in stiffness or spasm of posterior muscles.

The ability of vertebral segments to absorb energy of the impact due to repetitive loading is called hysteresis. The impact

of the load gradually comes down after the initial one of similar kind. This is a protective phenomena. When a person sustains an injury in shallow water it is implied that the impact has already exceeded the capacity of absorbable energy in that cervical spine. The capacity is greater in young people. After middle age for various reasons the capacity decreases may be due to degenerative changes.

It is commonly believed that ligamentum flavum would buckle inward into the spinal canal in extension producing compression. But experiments prove otherwise. Normally healthy ligament is stretched by 15% in neutral position. In full extension it is not fully relaxed, still stretched by 5%. If the legament is buckled probably it has lost its tensile phenomenon due to degeneration. So degenerated ligament can buckle, on further stretching it might even rupture.

Lot of controversies about the range of movements at various cervical segments exist; partly it is because all the studies are not uniformly carried out. At atlanto occipital junction the movement is negligible, it is upto 4° or so. Anteroposterior movements is negligible. At C1 - C2 the lateral flexion is almost nil. Though there is mild flexion, the actual rotation is maximum. Normally 2- 2.5 mm of translocation is possible due to elasticity of the transverse ligament. In a child it would be 4 - 4.5 mm. At lower levels flexion, lateral flexion and rotation are almost of identical degree. The translocation is usually upto 2 - 2.7 mm. In all these measurements radiological magnification should be taken into consideration or a specific pattern should be used in taking X-rays. Rotational movement of about 130° to 170° is possible in cervical spine. 40 - 50% is contributed by C1 and C2 and remaining segment would contribute rest 50 - 60%. Initial 45 - 50% take place at C1 - C2. Lower spines usually join later. C1 always rotate with skull and if C1 is not rotated it denotes some problem between occipit and C1. Common mode of injury is fall on face, if chin gets rotated it results in severe rotational stress at these joints.

The lateral flexion is not a pure lateral flexion process. It is usually complicated with certain degree of rotation. During lateral flexion spinus processes go to the side of convexity. For every 3° of lateral flexion there is 2° of actual rotation at C2. While lower down every 7° of lateral flexion is coupled with rotation of 1° . This is because of the oblique orientation of facet joints. On the contrary in lumbar spine on lateral flexion the spinous process go to the same side.

In upper cervical region the lateral flexion is so much coupled with rotation, lateral flexion injury can lead to unilateral facet dislocation or forced rotational injury of C1 - C2, there can be compression fracture of lateral mass.

Facet joints normally carry 8 - 10% of load. In extension the load on these joints is more than in flexion. Whenever there is capsular laxity an articular derangement whole kinetics is completely out of gear, and hence further deterioration can come rather rapidly. The design and orientation of facets is normally responsible for the control over the degree of rotation. Whenever there is involvement of facet joints at C1 and C2 the entire stability depends upon the ligamental complex. During lateral flexion facets acts as a fulcrum, the other facet would translate and rotate.

It is commonly believed that whenever there is a degeneration, the range of movement goes down. The commonest area is C 5-6. If the range is reduced the adjacent segments usually have a hypermotility. Rotational resistance to rotational derangement is highest in lumbar spine. Various structures contribute to stability. Disc contributes 45 %, facet 45 %, and posterior segments 10 %. After laminectomy the loss of stability could be about 18 % and if facetectomy is added it raises to about 60 %.

Normal spine has anteroposterior, side to side, translational and rotational movements. When the physiological movement is increased it suggests instability. Clinical instability is the loss of ability to maintain normal relationship between vertebrae under physiological loads. Abnormal loads can produce varying degrees of derangement leading to pain and deformity. It could be of 2 types. Dynamic instability which means a gravitational loading like forward flexion. This would come into picture when people get pain while using high pillow.

We generally presume that in hyper extension there is reduction in the size of cervical spinal canal. This is probably because of infolding of the ligamentum flavum especially if it is degenerated. Whenever there is a translatory instability the canal would be reduced if the initial diameter is smaller.

Vertebral artery at C1 - C2, C3 remains comfortable even when a person rotates the head to s side as the facets or lateral masses would rotate backwards. If the lateral mass translocate anteriorly in relation to anterior mass of C2 kinking of vertebral artery may occur. Upto 30° rotation there is no effect on vertebral artery. But at 45° there is considerable

kinking. If opposite artery is insufficient or rudimentary it will lead to vertebrobasilar insufficiency.

A certain amount of preload always exists on vertebra while the person sits or stands, for example at C6 vertebra which acts as fulcrum the whole load of the top will be acting so that the center of gravity of the entire superstructure is lined anterior to it. Therefore there is continuous forward bending movements which tries to flex the neck. This is commonly seen whenever a person dozes, there is a loss of posterior muscle tone and head flexes forward. That means the erect position of the head is held by the muscular force from behind. So muscle tension depends upon the neck position. Electromyographic studies have shown that forward flexion of neck have high muscle tension. When a person stands the resultant of gravitational force and muscle force on a disc space or facet joint can be divided into 2 vectors. One is a compressive force going right to the end plate and a translator force which goes right angle to the compressive force. So whenever there is a deformity or kyphosis the resultant stress becomes more.

To see the proper alignment of the spine X-rays should be taken as it is without correction in neck position.

Biomechanically normal posture is one where there is no undue stretching of ligaments, annulus, capsules or of the soft tissues, and no undue demand on muscle activity, no undue load bearing by the disc. The posture is assumed by the person normally either as a habit or as an aesthetic demand, functional necessity, or as a result of pain. The pathological posture often biomechanically do not coincide with the normal one. In the treatment this should be remembered.