Biomechanics of the CVJ

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4.1 CVJ Motion

Spinal movements are characterized by two types of motion: angular (rotation) and linear (translations). Each type of motion is described relative to each of the three axes of motion on a three-dimensional Cartesian coordinate system (*x*, *y*, and *z*) [1]. Rotation about the *x*-axis is referred to as flexion/extension, *y*-axis rotation is referred to as axial rotation, and *z*-axis rotation is referred to as lateral bending (Fig. 4.1). Clinically, most translations are referred to as subluxations.

Both types of movement, rotations and translations, are important for understanding normal and pathological spinal behaviors.

Different spinal movements are coupled together. Coupling refers to simultaneous motions (rotation and\or translation) that occur secondarily and in combination to a main motion (rotation and\or translation) [2, 3].

4.2 Biomechanics Flexibility Testing

Most of biomechanical information concerning the CVJ has been derived from the experimental method known as *Flexibility Testing*. It uses, in vitro, cadaveric spinal segments of two or more vertebrae that have been deprived of muscle tissue, leaving the ligaments and the bone structures intact. Torsional forces (bending moments), lateral forces, or combined loads are applied to the spinal segments, and the spinal movement thus obtained are then misurated [4]. Analysis of load-deformation responses reveals parameters such as stiffness, flexibility, range of motion, rotation,

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E. Tessitore et al. (eds.), *Surgery of the Cranio-Vertebral Junction*,
https://doi.org/10.1007/978-3-030-18700-2_4

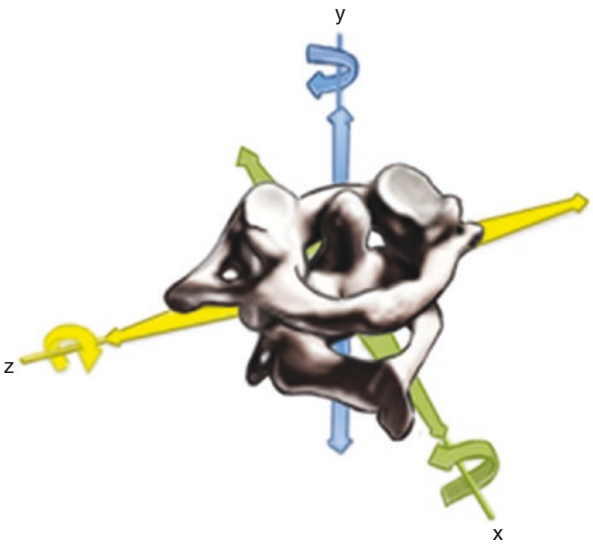

Fig. 4.1 Cartesian coordinate system for analyzing CVJ motions

translation, neutral zones, elastic zones, and axes of rotation. These biomechanical parameters are different and unique for each single spinal segment and are sensitive indices of spinal instability. The information obtained through the flexibility test "in vitro" represents the total contributions provided by bone joints and the ligamentous apparatus between each motion segment.

4.3 Load-Deformation Response of the CVJ

Several parameters can be calculated from the load-deformation curve.

Flexibility represents the amount of deformation in response to a unit load [5, 6]. *Stiffness* is the opposite of flexibility. It is the amount of resistance to a unit increment of displacement in the specimen.

Range of motion (ROM) is defined as the displacement between the neutral or resting position of the motion segment and the limit of its physiological motion. The *neutral position* is defined as the posture where minimal joint stresses occur and where minimal muscular effort is required to maintain the spatial orientation. This neutral position is best approximated by the midpoint of the bilateral *neutral zone* (NZ) [7].

The NZ is the portion of the ROM where the ligaments are lax and small forces produce large vertebral displacements. The *elastic zones* (EZ) is the steep portion of the load-deformation curve where the ligaments become stretched and stiffness increases, causing resistance to any further movement.

The motion characteristics of the different levels of the CVJ are due to the geometry of the vertebrae and skull base, the shapes of the joints, and the arrangements of the ligaments.

Neither the C0-C1 nor the C1-C2 joints have an intervertebral disc. The spherical shape (concave-convex) of C0-C1 joint allows slightly more flexion and extension than the other levels of the cervical spine, although these are quite rigid in axial rotation and lateral bending. The biconvex articular surfaces of the C1-C2 joints allow wide rotation of C1 around the dens. The atlantoaxial motion segment is the most flexible of the entire spine with respect to axial rotation, allowing a bilateral ROM of 80° or more. More than half of all cervical axial rotations occur at the atlantoaxial motion segment. Both C0-C1 and C1-C2 allow less lateral bending than the subaxial cervical motion segments, which average approximately 8° unilaterally [3] (Figs. 4.2 and 4.3).

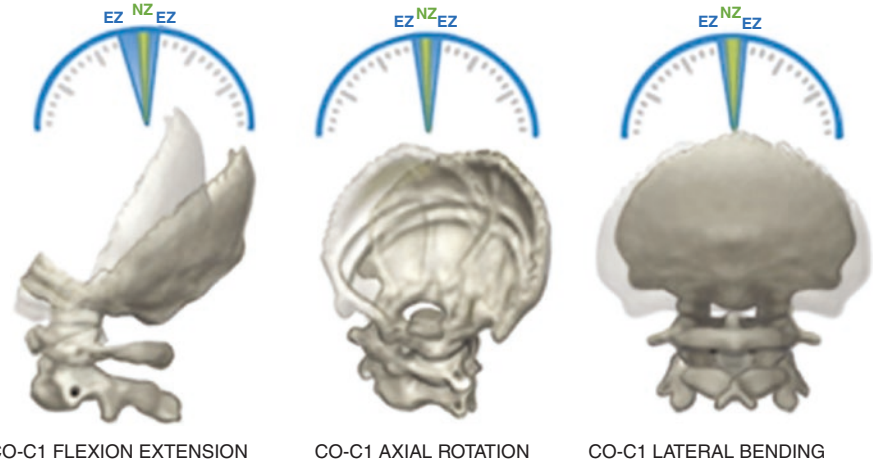

Fig. 4.2 Representation of the normal angular motions at the occipitoatlantal motion segments

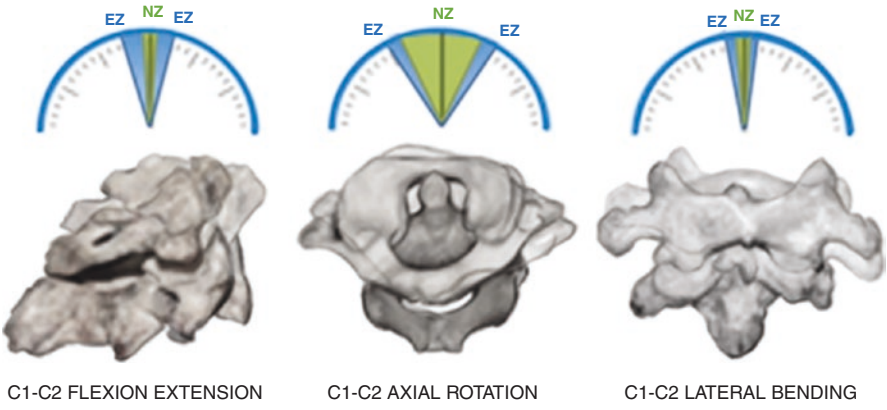

Fig. 4.3 Representation of the normal angular motions at the atlantoaxial motion segments

4.4 Physiological Biomechanics

The cervical spine's range of motion is approximately 80°–90° of flexion, 70° of extension, 20°–45° of lateral flexion, and up to 90° of rotation on both sides. However, movement of the cervical spine is complex and movement into any range is not the simple sum of equal motion from one vertebra to the next.

The occipitoatlantal junction contributes to 23°–24.5° of flexion/extension of the skull and the atlantoaxial joint provides an additional 10.1°–22.4° [6]. At the occipitoatlantal junction, the abutment of the dens against the foramen magnum prevents supraphysiologic flexion, whereas odontoid contact with the tectorial membrane has been proposed to limit extension. The transverse ligament prevents pathological flexion of the atlantoaxial segment while extension is inhibited by the bony elements of the atlantoaxial joint facets [8–10]. Physiological motion of the cervical spine can accomplish 90° of rotation from the midline. The atlantoaxial junction contributes to 25°–30°, at which point the motion occurs through subaxial segments. The bone facets of the atlantoaxial junction will permit up to 40° rotation before locking, contributing a major restriction to over rotation. The contralateral alar ligament and the ipsilateral transverse ligament also resist pathological rotation with support from the joint capsules of the occipitoatlantal and atlantoaxial junctions [11, 12]. The occipital condyles restrict lateral bending of the occipitoatlantal junction to 3.4°–5.5° in either direction.

The atlantoaxial segment reaches 6.7° before the alar ligaments discourage further motion [6]. Movement in the other planes of motion is minimal at the CVJ, including translation, distraction, and compression.

Ligamentous as well as osseous structures are responsible for this stability. The transverse ligament, alar ligaments, and capsular joints resist anterior translation in the sagittal plane while the occipital condyles and the contact of the dens with the atlas and foramen magnum constitute bone barriers against posterior translation [10, 11, 13, 14].

The capsular joints of the occipital condyles and the atlantoaxial zygapophyseal joints resist compression. Distraction is not a physiological motion of the CVJ [14].

4.5 Biomechanics of Simple CVJ Pathology: Overview

In response to trauma, the CVJ exhibits predictable patterns of failure based on the mechanism of injury [15]. The most commonly encountered traumas occur during motor vehicle accidents, falls, diving accidents, and gunshot wounds [16]. Fracture-dislocation or occipitocervical dissociation at the CVJ is a leading cause of death in motor vehicle accidents [16]. Multiple mechanisms have been proposed to explain these patterns, such as whiplash and flexion-distraction injury.

Pathological flexion increases tension on the transverse ligament, resulting in failure of either the cruciate ligament or the odontoid waist [16]. Failure of the tectorial membrane has also been associated with flexion in front-end motor vehicle

collisions [17] and may lead to dural tears. Isolated tectorial membrane failure contributes minor instability in flexion and extension [18–20].

Hyperextension may lead to fracture of the atlas at the posterior ring or fracture of the axis at the pars interarticularis or the odontoid. Shearing injury may occur to the ligaments of the anterior CVJ, including the alar ligaments, accessory atlantoaxial ligaments, cruciform ligament, and tectorial membranes. Supraphysiologic rotation at the atlantoaxial junction can predict, or even diagnose, alar ligament disruption.

Traumatic compression of the CVJ commonly causes osseous pathology at the occipitoatlantal junction. Axial loading has been associated with burst fractures of the atlas and with occipital condyle fractures. When evaluating trauma to the CVJ, current guidelines recommend initial evaluation by CT followed by MRI to assess ligamentous injury [21]. T2-weighted MRI obtained within 72 h of injury is the preferred modality for diagnosing soft-tissue injury. After 72 h, decreased tissue edema may lead to overlooked ligamentous damage [22]. Disruption of the ligamentous structures is sufficient to cause instability at the CVJ; additionally, these ligaments are irreparable once torn [23]. The most critical ligaments to evaluate for stability in the CVJ are the transverse ligament of the cruciform complex, the alar ligaments, and the tectorial membrane [9, 17, 24, 25].

4.6 Alar Ligament Failure

Failure of one alar ligament results in modest rotatory atlantoaxial instability. This instability is manifested as an increase in the C1-C2 ROM during the axial rotation, mostly through an increase in the NZ [26, 27]. The EZ and the flexibility, however, do not change significantly.

The bilateral transection of the alar ligaments determines considerably more extensive alterations of C0-C1-C2 motion than unilateral alar ligament disruption. The NZ and ROM during axial rotation, lateral bending, and flexion-extension are all increased significantly. The pivotal function of the alar ligaments is to stabilize the spine during flexion and extension and to limit axial rotation and lateral bending [3]. Failure of an alar ligament most likely occurs near the condylar insertion [25] and introduces instability in rotation and an increase in flexion, extension, and lateral bending [9]. Isolated rupture of the alar ligament is rare but has been associated with hyperflexion paired with rotation in all reported cases. Unfortunately, evaluation of the alar ligaments by MRI is complicated by their size and anatomical conformation [28, 29].

4.7 Transverse Ligament Failure

The transverse atlantal ligament is the thickest (1 cm) and strongest ligament of the entire spine. It is the most important stabilizer of the atlas, constraining C1 around the dens. Extremely high loads with anteriorly directed vectors at C1 are required to

disrupt the transverse ligament. The mechanisms leading to failure of the transverse ligament have been extensively evaluated in vitro [30]. The accessory ligaments at C1-C2 are relatively weak and stretch with ease after the transverse ligament is rendered incompetent. This feature has important clinical consequences. The transverse ligament tears suddenly with a principle of "all or nothing" because it is stiff and inelastic. This ligament does not tear partially or gradually. When torn, the transverse ligament is incapable of repair. Because the transverse ligament injury renders the articulation of C1 enormously unstable, stabilization (fusion) of the C1-C2 complex must be performed.

4.8 Capsular Ligament Failure

Failure of the C1-C2 joint capsular ligament primarily slightly increases the ROM during axial rotation but has a minimal effect on lateral bending or flexion and extension [31]. Most of the increase in ROM is due to an increase in the EZ. Injury to the capsular ligaments is an important mechanism associated with rotatory C1-C2 subluxation.

Avulsion of a synovial joint capsule causes only a mild increase in rotatory motion; however, a rupture of the joint capsule warrants investigation of the more critical ligaments as it has been associated with the disruption of the transverse atlantal and alar ligaments.

4.9 Biomechanics Effects of Atlas Fractures

Experimental laboratory studies regarding atlas injuries indicate that burst fractures of the atlas outbreak derive from compressive injuries [32, 33]. Atlas burst fractures result in instability that is manifested as increases in the NZ and the ROM during flexion, extension, and lateral bending. In these studies, "in vitro" compressive injury caused a 90% increase in the flexion\extension NZ, a 44% increase in the flexion\extension ROM, and a 20% increase in lateral bending NZ and ROM. However, no significant changes were seen in axial rotation NZ or ROM.

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