Spinal canal capacity in simulated displacements of the atlantoaxial segment

A SKELETAL STUDY

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In normal, physiological circumstances there is ample room in the spinal canal to accommodate the spinal cord. Our study aimed to identify the degree of compromise of the spinal canal which could be anticipated in various atlantoaxial pathological states. We examined paired atlas and axis vertebrae using high-definition radiography and simultaneous photography in both normal and simulated pathological orientations in order to measure the resultant dimension of the spinal canal and its percentage occlusion.

At the extreme of physiological axial rotation (47°) the spinal canal is reduced to 61% of its cross-sectional area in neutral rotation. The spinal cord is thus safe from compromise.

Atlantoaxial subluxation of up to 9 mm reduces the area of the spinal canal, in neutral rotation, to 60% with no cord compromise. Any rotation is, however, likely to cause cord compression.

The mechanism of fixation in atlantoaxial rotatory subluxation could be explained by bony interlocking of the facet joint, reproducible in dry bones.

Materials and Methods

We used a simple technique with matching dry atlas and axis bones. Six pairs were orientated in the following physiological and simulated displacements:

1) Normal rotation.
2) Anterior atlantoaxial subluxation.
3) Posterior atlantoaxial dislocation. This usually accompanies a fracture or a congenital anomaly. Two cases have been reported without fracture of the odontoid in which the mechanism of injury was believed to be extreme hyperextension, leading to rupture of the anterior ligaments and soft tissues joining the atlas to the axis, but preserving the transverse ligament.
4) Anterior rotatory fixation.
5) Posterior rotatory fixation.

The positions were held by Kirschner wires and Blue Tac. The vertebrae were suspended from a retort stand using external fixator pins (Fig. 1).

We used a fixed anterior focal distance of 100 cm with the body of the axis taken as the point of reference to overcome the problem of angulation and magnification. The directional beam of the X-ray machine was centred by the cross-hairs through the tip of the odontoid peg. Absence of the shadow cast by the odontoid ensured reproducibility. We used high-definition film and a lead scale of 1 to 5 cm applied to it to facilitate calculation of the cross-sectional area of the spinal canal (Fig. 2).

The most difficult technical problem was the measurement of the cross-sectional area of the functional spinal canal. We overcame this by using a computerised digital area analyser (Freelance Rev.4.25 from Sight Systems) on...
an IBM computer. The radiographic circumference of the spinal canal was mapped out and displayed on the computer screen. Input of the scale on the radiograph allowed calculation of the degree of magnification and thus the correct calculation of the cross-sectional area.

The residual area of the spinal canal was then calculated by subtracting the area of the spinal cord from the cross-sectional area of the spinal canal.

**Results**

**Normal physiological rotation.** We obtained measurements at 0, 15, 30 and 47° of rotation of the atlas on the axis. The last figure represents the accepted value of maximal atlantoaxial rotation as postulated by White and Panjabi³ (Fig. 3).

Details of the results are shown in Table I. At maximal atlantoaxial rotation, the cross-sectional area of the vertebral canal is reduced to 61% of the maximal value.

**Atlantoaxial subluxation.** We simulated atlantoaxial subluxation by aligning the axis and atlas at progressive intervals, measured by a simple ruler, and maintaining the position with Blue Tac (Fig. 4). No kyphotic deformity was incorporated into this model. The results are shown in Table II.

<table>
<thead>
<tr>
<th>Table I. Percentage change in area of the vertebral canal with rotation (degrees) of the atlas and axis</th>
</tr>
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<tbody>
<tr>
<td>Rotation</td>
</tr>
<tr>
<td>----------</td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>15</td>
</tr>
<tr>
<td>30</td>
</tr>
<tr>
<td>47</td>
</tr>
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</table>

<table>
<thead>
<tr>
<th>Table II. Percentage change in area (range) of the vertebral canal with increase in atlantoaxial interval (mm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atlantoaxial interval</td>
</tr>
<tr>
<td>-----------------------</td>
</tr>
<tr>
<td>0</td>
</tr>
<tr>
<td>3</td>
</tr>
<tr>
<td>6</td>
</tr>
<tr>
<td>9</td>
</tr>
<tr>
<td>12</td>
</tr>
<tr>
<td>15</td>
</tr>
</tbody>
</table>

**Fig. 1**

Photograph of a type-2 fixed atlantoaxial rotatory subluxation of the atlantoaxial complex showing the method used to support the dry vertebrae.

**Fig. 2a**

Lateral (a) and vertical (b) radiographs of the same specimen seen in Figure 1.
Bilateral posterior dislocation. This was simulated using Blue Tac to maintain the position. The odontoid peg was left intact. Table III gives the results.

Rotatory fixation. Fielding\(^4,5\) highlighted the difficulty in differentiating patients with atlantoaxial rotatory displacement, which usually resolves spontaneously and conservatively, from those with fixed atlantoaxial rotatory subluxation with the potential for neural damage and death. He classified rotatory fixation into four types\(^6\): type I, without anterior displacement of the atlas (displacement of 3 mm or less); type II, with anterior displacement of the atlas of 3 to 5 mm; type III, with anterior displacement of more than 5 mm; and type IV, with posterior displacement.

Measurements obtained with our model confirm Fielding’s clinical observations\(^4-6\) showing that the area of the canal is greatly compromised by anterior rotatory fixations, but even more so by posterior displacement.

We reproduced fixed rotatory subluxation by using a Kirschner wire to hold the position of the atlas on the axis. It was possible to produce the fixed rotatory deformity simply by interlocking the lateral masses of the paired vertebrae (Fig. 5). Table IV gives the results.

Figures 6 and 7 summarise the results for normal rotation and simulated pathological states.

**Discussion**

We aimed to identify the degree of compromise of the spinal canal which could be anticipated in various atlantoaxial positions of the atlas on the axis. We needed to understand the anatomy of the spinal canal in physiological situations and to estimate the percentage which is occupied by the cord at this level.

Steele\(^7\) described the ‘rule of thirds’. One-third of the diameter of the neural canal at the C1-C2 level is occupied by the odontoid and one-third by the cord; the remainder is ‘empty’ and provides space for the cord during movements of the head on the neck. This, however, does not represent...
the area occupied by the cord, simply the anteroposterior
diameter.

Table IV. Percentage change in the area of the vertebral
canal in fixed rotatory subluxation

<table>
<thead>
<tr>
<th>Type of rotatory fixation</th>
<th>Area of vertebral canal (%)</th>
<th>Residual area (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior (Fielding I)</td>
<td>42</td>
<td>15</td>
</tr>
<tr>
<td>Posterior (Fielding IV)</td>
<td>35</td>
<td>8</td>
</tr>
</tbody>
</table>

Table V. Actual area (percentage) occupied by the
cord as calculated from CT studies

<table>
<thead>
<tr>
<th>Type</th>
<th>Diameter (mm)</th>
<th>Area (mm²)</th>
<th>% Canal occupied by cord</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1 canal</td>
<td>21.3</td>
<td>356</td>
<td></td>
</tr>
<tr>
<td>C1 cord</td>
<td>19.2</td>
<td>290</td>
<td></td>
</tr>
<tr>
<td>C2 canal</td>
<td>10.0</td>
<td>79</td>
<td>27</td>
</tr>
<tr>
<td>C2 cord</td>
<td>19.2</td>
<td>290</td>
<td></td>
</tr>
</tbody>
</table>

![Photograph of four pairs of vertebrae showing type-A fixed atlantoaxial rotatory subluxation (transverse liga-
ment intact; odontoid peg as the axis of rotation).

Fig. 5

Summary of the results in normal rotation (C1/C2).

Fig. 6

Summary of the results in simulated pathological states.

Fig. 7

The actual area occupied by the cord has been calculated from CT studies as shown in Table V. At the level of the second cervical vertebra, the spinal cord occupies 27% of the total cross-sectional area of the vertebral canal.

The normal values for degrees of movement occurring at the atlantoaxial joint have been studied and well documented by Werne and by White and Panjabi. They are flexion/extension 10°, lateral tilt 0° and axial rotation 47°.

The first 45° of axial rotation take place at C1-C2 before the lower cervical spine begins to participate. The kinematic studies of Werne identified the instantaneous axis of rotation for flexion/extension to be in the region of the middle third of the dens. For axial rotation the instantan-

THE JOURNAL OF BONE AND JOINT SURGERY
neous axis of rotation lies in the centre of the axis. At the extreme of axial rotation (47°) the spinal canal is reduced to 61% of its cross-sectional area in neutral rotation.

Anterior atlantoaxial subluxation implies a widening of the joint space between the anterior arch of the atlas and the odontoid process. Coutts estimated the normal width of this joint to be 1 to 2 mm, but did not consider the effect of movement on this value. Jackson examined the cervical spines of 50 adults and 20 children in flexion and extension. He concluded that the atlantodens interval (ADI), as measured between the posteroinferior margin of the anterior arch of the atlas and the anterior surface of the odontoid process, was constant in the adult and did not exceed 2.5 mm.

In children, the ADI may be as much as 4.5 mm and can show an increase of 0.5 mm in flexion. Locke, Gardner and Epps reviewed 200 normal children and concluded that the upper limit of normal was 4 mm but reported an ADI of 5 mm in a normal 13-year-old, thus reinforcing Jackson's findings.

Atlantoaxial subluxation is seen most commonly in rheumatoid arthritis as a result of the destruction of joints, ligaments and bone caused by erosive synovitis. Traumatic atlantoaxial subluxation is relatively rare. Weissman et al. examined the radiographs of 194 rheumatoid patients with atlantoaxial subluxation and/or atlantoaxial impaction. No patient with less than 9 mm of subluxation had compression of the cord unless atlantoaxial impaction was also present. This is confirmed by our findings.

Atlantoaxial subluxation of 6 mm reduces the area of the spinal canal to 87% of normal. With full axial rotation of 47°, which is very unlikely in a rheumatoid patient, a further reduction by 39% could be anticipated. This would leave a canal area of 48% which will still comfortably accommodate the spinal cord (27%). Atlantoaxial subluxation of 9 mm reduces the area of the spinal canal to 60%. Theoretically, full rotation of 47° would further reduce the spinal canal to 21% which must cause compression of the cord. Our study does not allow for the compressive effect of rheumatoid granulation and oedema around the odontoid process as described by Crockard et al. Using myeloCT with sagittal reconstruction they showed clearly the contribution of rheumatoid pannus to medullary compression in cases of atlantoaxial subluxation.

Posterior atlantoaxial dislocation, without fracture of the odontoid, reduces the canal area to 36%, leaving sufficient space to avoid compression of the cord; there was no permanent neurological injury in either of the reported cases.

Rotatory dislocation of the atlas was reported by Corner, and spontaneous hyperaemic dislocation of the atlas in a patient with acute mastoid disease, by Watson-Jones although the first description of this is attributed to Bell.

Measurements obtained with our model indicate that the area of the canal is greatly compromised by anterior rotatory malalignment and even more by posterior displacement.

The mechanism of fixation remains a mystery, especially in type 1 which represents the physiological situation. With our dry bone specimens it is possible to achieve rigid bony interlocking of the lateral facets in both anterior and posterior rotatory subluxation. In the former a marked kyphotic tilt is an associated feature and in the latter there is slight hyperextension. To date pure bony interlocking has not been demonstrated clinically but this could be a cause of acute irreducible fixation.

Certain pitfalls are encountered in interpreting the results achieved with our simple model. Attempts were made to minimise error by using the axis as a fixed reference point, with the odontoid used to direct the X-ray beam in each simulated situation. Use of a scale on each X-ray film sought to minimise the error introduced by magnification. Only six pairs of matching vertebrae were studied but there was little deviation from the mean values recorded.

The use of dry bones makes no allowance for the resistance to compression of epidural fat, nor for the presence of granulation tissue and erosive bony destruction seen in rheumatoid arthritis. We also made no allowance for the presence of haematoma and soft-tissue injury accompanying trauma.

Conclusions. The anatomy of the atlantoaxial region is highly complex to facilitate function while protecting vital structures, and in normal physiological circumstances there is ample room in the spinal canal to accommodate the spinal cord. Nevertheless there is a considerable range of traumatic and pathological conditions which render this vulnerable area susceptible to potentially lethal disturbance.

An ADI of up to 3 mm in adults and 5 mm in children is the accepted normal value, but an anterior atlantoaxial subluxation of up to 9 mm is unlikely to cause significant neurological impairment, unless combined with atlantoaxial impaction in rheumatoid arthritis.

Caution must be taken with patients who have pre-existing narrowing of the spinal canal such as in congenital spinal stenosis, disc protrusion and hypertrophy of the facet joint. Eismont et al. showed that patients who have damage to the spinal cord, with a permanent neurological deficit, had narrower sagittal diameters than those who had sustained no damage to the cord, and those with a complete lesion of the cord had a narrower canal than patients with an incomplete lesion.

Children represent a further unique subgroup in whom it is difficult to predict neurological injury, even despite gross radiological disturbance. Because of its inherent ligamentous laxity, the atlantoaxial interval may be normal despite a rotatory dislocation of 90°.

Atlantoaxial rotatory fixation has both acute and chronic phases for which the mechanisms of locking are quite different. In the acute phase, muscle spasm, capsular or ligamentous interposition, or bony interlocking, are all
possible causes. In the chronic phase ankylosis of the lateral masses would appear to be a contributing factor.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References

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