A Mechanistic Classification of Closed, Indirect Fractures and Dislocations of the Lower Cervical Spine

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Closed, indirect fractures and dislocations of the lower cervical spine occur in families or groups within which there is a spectrum of anatomic damage to a cervical motion segment. This study of 165 cases demonstrates the various spectra of injury, called phylogenies, and develops a classification based on the mechanism of injury. The common groups are compressive flexion, vertical compression, distractive flexion, compressive extension, distractive extension, and lateral flexion. The probability of an associated neurologic lesion relates directly to the type and severity of cervical spine injury. With use of the classification, it is possible to formulate a rational treatment plan for injuries to the cervical spine. [Key words: cervical spine, indirect trauma, fracture, dislocation, classification]

A UNIVERSALLY ACCEPTED CLASSIFICATION for indirect, lower cervical spine fractures and dislocations does not exist.

Clinical reviews have categorized lower cervical injuries in many different ways. While some works focus on neurologic injury without analysis of the soft tissue or bone injury,[24,46,52] other papers divide cases according to the specific bone of soft-tissue abnormality without consideration for the pattern or the mechanism of injury.[14,53] Authors who have grouped cases according to the mechanism of injury have generally divided them into flexion, extension, or vertical compression categories.[23,27,36,46]

Inconsistency exists as to what constitutes a flexion or extension injury. There are clearly two distinct types of flexion injuries. One is a *compressive flexion* injury producing for example, the "tear drop" fractures described by Schneider and Kahn;[65] and the other is a *distractive flexion* injury such as the bilateral facet dislocation studied by Beatson.[9] Also, two types of extension injury are recognized. The *compressive extension* injury, characterized by fracture of the posterior elements and anterior displacement of the vertebral centra, described by Forsyth, is one variety[29,30] The *distractive extension* injury, characterized by rupture of the anterior longitudinal ligament and posterior displacement of the vertebra, described by Taylor and Blackwood, is another type.[68] The majority of previous works have in one way or another mixed traumatic lesions with clearly different mechanisms of injury.[9,28,34,46,57] The inconsistent reported incidences of flexion and extension injuries in clinical reviews of similar populations are most likely a function of the different ways of categorizing the injuries.[9,21,27,30,46,53]

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Commonly, upper and lower cervical spine injuries are considered collectively even though these regions are very dissimilar anatomically. The combination of upper and lower neck injury in a single patient is unusual.[33,76] The lack of a consistent and precise terminology for lower cervical spine injuries continues to hamper communication between investigators. After reviewing the literature, one is unable to make meaningful comparisons between clinical series.

In this work, we demonstrate that there are at least six common patterns of indirect injury to the lower cercical spine, that each pattern can be divided into stages according to the severity of the musculoskeletal damage, that there is good correlation between the neurologic and musculoskeletal injury in each pattern, that current biomechanical concepts can be used to deduce the mechanism of injury, and that, based on the foregoing, a precise classification of fractures and dislocations in the lower cervical spine can be formulated.

MATERIALS AND METHODS

The senior author generated the classification from a critical retrospective analysis of 165 closed, indirect fractures and dislocations of the lower cervical spine. A preliminary report has been given. The cases were collected from 471 codings of neck injury seen at the University of Texas Medical Branch from 1960 through 1974. Those with only upper cervical spine pathology, a direct injury mechanism, pathologic fracture, no radiographic pathology, incomplete records, or incomplete radiographic studies were excluded from this series.

To mitigate bias in the study, records and x-ray studies were analyzed independently with a number assigned to each record data sheet and to each x-ray data sheet. Upon completion of the review process, the numbers were matched and the data tabulated. From each record, in addition to demographic data, we collected a detailed description of any neurologic injury, craniofacial soft-tissue injury, craniofacial fracture, and when available, the specific circumstance of the injury. Each set of radiographs was studied for injury to the bone evidenced by fracture and injury to the supporting tissues, evidenced either by residual displacement or abnormal movement within a motion segment.

A detailed description of the neurologic injury was available for each case. Initially we tried to classify the incomplete myelopathies as specific cord syndromes such as central cord, anterior cord, lateral cord, and posterior cord.[13,63,64,70,71] Only the central cord syndrome was a sufficiently clear entity to stand without many qualifications among our cases. Therefore, central cord syndrome and partial cord lesion were the groupings which best categorized all of our incomplete myelopathies.[15,48] Complete myelopathies are termed total cord lesions or injuries. For patients with identifiable components of different neurologic syndromes, such as a combination radiculopathy and partial myelopathy, the neurologic lesion was classified acording to the most severe syndrome. Normal, radiculopathy, central cord syndrome, partial cord lesion, and total cord lesion are symbolized N, R, C, P, and T, respectively in all tables.

An unequivocal history of a specific injury mechanism was sought for each case. To be considered unequivocal, the posture of the head and neck at the time of injury, the location and direction of application of force, and the exact circumstance of the trauma had to be known. Radiographic evidence was not considered. Examples of specific injury mechanisms included such cases as a man who had recently cut a tree and was fleeing to avoid its falling on him when it struck the back of his head, a girl who fell onto her face when her ten-speed bicycle abruptly stopped, a worker struck on his posterior head by a falling bag of cement as he stood looking slightly downward, or a patient who was struck in the occipital area when a truck at a loading dock accidently backed into him. Although general mechanisms, such as automobile accident (AA), motorcycle accident (MA), shallow dive (SD), direct blow (DB), fall (F) and other (0) were recorded, they were not considered as specific injury mechanisms because the specific details of the injury were unknown. Both the general and specific injury mechanisms are indicated in the tables which summarize the clinical data.

We grouped cases which demonstrated similar radiographic pathology, and then arranged the groups into what appeared to be a continuous spectrum of anatomic damage. In instances of combined or associated fracture and/or dislocation, the most severe injury was considered the primary one and was used to classify the particular lesion. All other cervical trauma was listed as an associated injury. From study of the groups, we postulated a mechanism of injury for each spectrum and labeled each spectrum, which we call a phylogeny to emphasize the orderly sequence, according to the injury mechanism.[3] The terminology is consistent with that of Braakman and Penning and that previously presented.[2–4,15] Each phylogeny is named according to the presumed attitude of the cervical spine at the time of failure and the initial, dominant mode of failure. "Compressive" indicates that compression is the stress which accounts for the initial, most conspicuous damage in a motion segment, and "distractive" indicates that tension or shear is the stress which produces the initial, most evident structural failure. *Compressive flexion, vertical compression, distractive flexion, compressive extension, distractive extension* and *lateral flexion* were the six phylogenies identified. Their distribution is shown in Figure 1. Because we found no spectrum of injury attributable to rotation, we concluded that rotation is best considered a lateralizing force within the foregoing groups.

Although most cervical spine injuries involve a motion segment, accurate identification of the injured motion segment has not been done in any consistent

manner in the literature. There has been a tendency to label a fracture of a vertebra according to its number; for example, a fractured fifth cervical centrum might be called a "C5 fracture," and so on. On the other hand, injuries which involve vertebral displacement without severe bony injury are usually identified according to the motion segment in which the displacement occurs; a unilateral facet dislocation with displacement of the fifth on the sixth cervical vertebra, for example, is called a "C5, C6 unilateral facet dislocation." As will be demonstrated, most of the injuries to be considered are to a cervical motion segment, and there are typical patterns of fracture and ligamentous failure within each phylogeny. It is more accurate to identify a lesion according to the motion segment involved rather than simply according to the most conspicuous feature. We therefore adopted the convention of labeling each injury according to the motion segment in which it occurred. To facilitate the identification of a fracture, we underlined the number of the cervical vertebra which was fractured in a manner typical for the particular phylogeny.

Throughout the discussion, we have attempted to utilize consistent anatomic terminology In keeping with White's suggestions, we label the posterior longitudinal ligament and the cervical spine structures anterior to it as the anterior elements.[75] The soft-tissue component consisting of the intervertebral disc and the portion of the anterior and posterior longitudinal ligaments which lie between adjacent vertebral bodies is the anterior ligamentous complex. All structures of the cervical spine which lie dorsal to the posterior longitudinal ligament are posterior elements. The vertebral arch and the posterior ligamentous complex are the osseous and connective tissue components, respectively.

BIOMECHANICAL CONSIDERATIONS

From study of radiographic injury residuals in cervical motion segments, one cannot measure the force which produced the trauma, but it is possible to deduce the modes of failure for the various components of the spine. This analysis yields a rough idea of the direction of the forces producing failure. We call these injury-producing forces "injury vectors." The one which produces the initial failure within a phylogeny is the major injury vector, and any associated force with a different direction which produces associated tissue failure is the minor injury vector. Stress propagation of the major injury vector is through the major load path and of the minor injury vector through the minor load path. The major and minor vectors act on opposite sides of a neutral axis. Drawing an analogy to the stresses generated in a beam subjected to a bending moment may be helpful. The concave side, toward which the bend occurs, is stressed in compression, while the convex side is stressed in tension. The neutral axis separates the portion stressed in compression from that stressed in tension. The component of

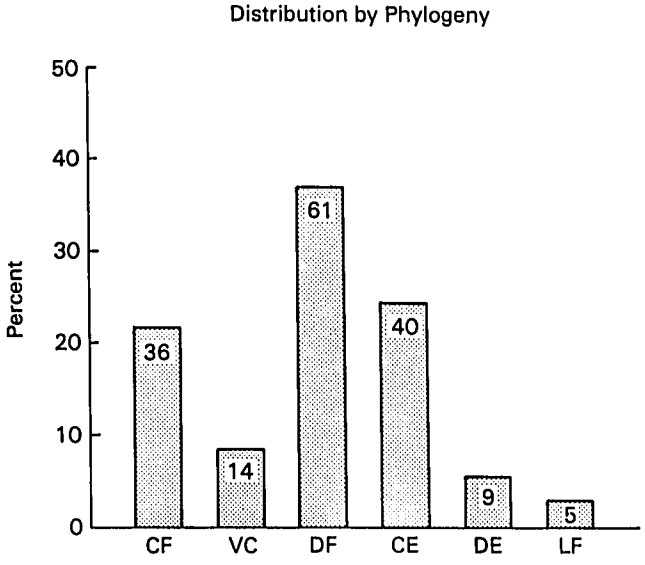

Fig 1.

the forces acting perpendicular to the neutral axis subjects the beam to shear stress.

Because we have analyzed tissue failure rather than measured stress, the term "transitional axis" will be used rather than neutral axis. Whereas the neutral axis separates stresses, the transitional axis separates tissue failures of different modes. If different modes of failure occur within the motion segment simultaneously, the neutral axis and the transitional axis are likely to be one and the same. But if they do not occur at the same instant, the neutral axis and transitional axis may not approximate each other.

Other factors, such as the stress rate and the effect of the cervical lever arm, are probably important in the genesis of cervical spine injury but are difficult to assess in a review of this sort.[31,54]

The characteristic patterns of failure for the tissues which comprise a cervical motion segment are basic to the analysis of the specific injury. Cancellous bone, cortical bone, and ligamentous tissue all have different failure characteristics. Cancellous bone, because a large fraction of its volume is marrow, can undergo impaction when strained to failure by a compressive stress. In such situations, it deforms in a manner which reflects the direction from which the force is applied.[25,44] When cancellous bone is strained to failure in tension or shear, the phenomenon of impaction is not observed, and a brittle fracture typically results.

Cortical bone, because of its homogeneous physical structure and brittleness, does not undergo obvious plastic deformation when it is strained to failure in compression, tension, or shear. Fracture lines do follow stress isoclines, and the degree of comminution is proportional to the energy absorbed prior to fail-

ure.[25,43,62] Low stress rates produce linear fractures along a stress line, and high stress rates cause comminution. With the exception of the axial, splitting fracture of a spinous process, cortical bone failure patterns are of little use in the deduction of the mechanism of closed, indirect fractures in the lower cervical spine.

Ligamentous failure, which is not radiographically visible, cannot be assessed as directly as bone failure. But because ligaments define and constrain a spacial relationship between bones, abnormal relationships between vertebrae infer ligament failure. From residual vertebral displacement, the *minimum* ligament failure present in a motion segment may be deduced. Appreciation of the full magnitude of the failure may require stress studies aimed at testing the particular ligaments in question.[9,56,74,75] In clinical situations, ligaments fail in tension and/or shear but not in compression.[31,32,40,42,50,54,72]

The hypotheses which are the basis of our biomechanical considerations can now be listed: (1) the forces producing either fracture or dislocation of the cervical spine can be considered as major and minor injury vectors; (2) the injury vectors can be deduced from the radiographic examination of the cervical spine; (3) the magnitude of the vectors determines the severity of an injury; (4) similar injuries result from similar injury vectors; and (5) within a given injury mechanism, there is a spectrum of injury, a phylogeny, which ranges from trivial to severe.

For the sake of clarity, the deduced biomechanics for each phylogeny immediately follow the particular clinical data. More complete data for cases are given in the tables.

COMPRESSIVE FLEXION

The compressive flexion phylogeny (CF) is shown in Figure 2. Each group of similar cases is considered a stage (S) of injury, a convention followed throughout this classification. Details of the clinical data are shown in Table 1.

CF Stage 1

The stage 1 lesion (CFS1) consists of blunting of the anterior-superior vertebral margin to a rounded contour. There is no evidence of failure of the posterior ligamentous complex (Figure 2A).

CF Stage 2

The compressive flexion stage 2 (CFS2) lesion, in addition to the changes seen in CFS1, shows obliquity of the anterior vertebral body and loss of some anterior height of the centrum. The result is a "beak" appearance of the anterior–inferior vertebral body. The concavity of the inferior end-plate may be increased, and there may be a vertical fracture of the centrum (Figure 2B).

CF Stage 3

The compressive flexion stage 3 lesion (CFS3) has, in addition to the features of the CFS2, a fracture line passing obliquely from the anterior surface of the vertebral body through the centrum and extending through the inferior subchondral plate. There is a fracture of the beak (Figure 2C).

CF Stage 4

In addition to the deformation of the centrum and the fracture of the beak, the compressive flexion stage 4 lesion (CFS4) demonstrates mild, less than 3-mm displacement of the inferior-posterior vertebral margin into the neural canal at the involved motion segment. There is no evidence for additional bone deformation as one proceeds from CFS3 to CFS4 (Figure 2D).

CF Stage 5

The compressive flexion stage 5 lesion (CFS5) has the features of the bone injury seen in the CFS3 and in addition has displacement of the posterior portion of the vertebral body fragment posteriorly into the neural canal. The vertebral arch characteristically remains intact. The articular facets are separated, and there is increased distance between the spinous processes at the injury level. The displacement indicates that both the posterior portion of the anterior ligamentous complex and the entire posterior ligamentous complex have failed. The beak fragment remains anterior. The posterioinferior margin of the upper vertebrae may approximate the lamina of the subjacent vertebra (Figure 2E).

GENERAL CONSIDERATIONS FOR CF

Thirty-six cases fit the CF phylogeny with six CFS1, seven CFS2, four CFS3, eight CFS4, and 11 CFS5. Thirty-two males and four females with an average of 24.6 years comprised the group.

None of the patients with a CFS1 had a neurologic deficit. One patient with a CFS2 had a central cord injury. One patient with a CFS3 had a central cord and another a total cord lesion. Two of the patients with CFS3s had a central lesion, one a partial lesion, and three total cord injury. One of the patients with a CFS5 had a central cord syndrome, and the remaining ten had total cord lesions. These data are displayed as percentages in Figure 3.

Seventeen patients were injured in automobile accidents, two in motorcycle accidents, 11 in shallow dives, four in falls, one by a direct blow, and one by another mechanism. In each of the eight cases in which the specific injury mechanism was known, the neck was postured in flexion, and the impact occurred near the vertex of the calvarium.

The deformation which the centrum undergoes in CFS1 and CFS2 indicates a compressive force directed obliquely downward and posterior in the sagittal plane, with stress concentration at the anterior-superi-

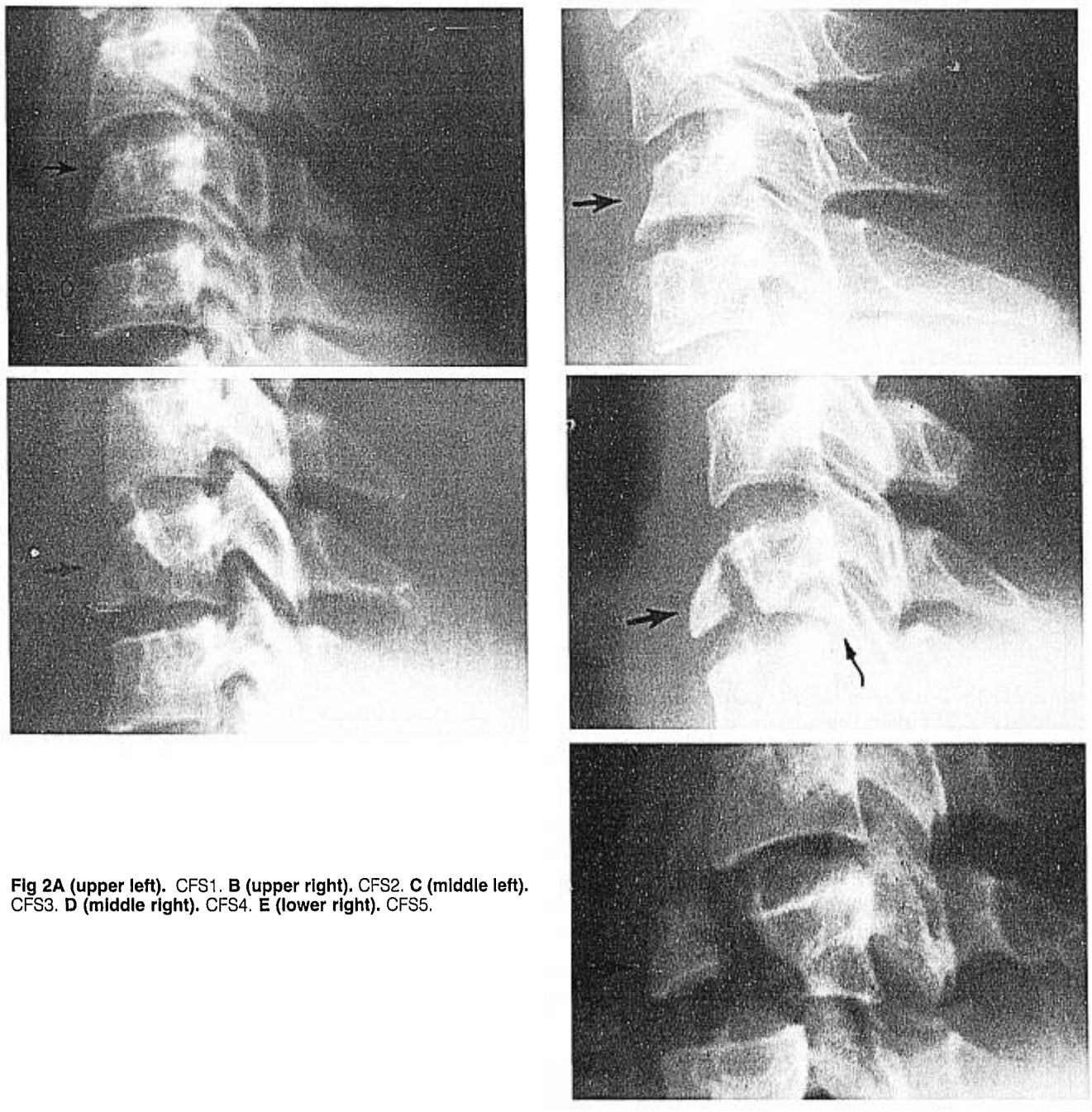

Fig 2A (upper left). CFS1. **B (upper right).** CFS2. **C (middle left).** CFS3. **D (middle right).** CFS4. **E (lower right).** CFS5.

or margin of the vertebral body. This force, because it produces the initial, most conspicuous failure in the phylogeny, is the major injury vector. The occurrence of multiple contiguous CF lesions supports the deduction of a major compressive stress propagated through the anterior portion of the anterior elements at a high stress rate (Table 8). Such a load path requires the cervical spine to be in flexion. The oblique fracture of the centrum observed in CFS3 is consistent with a shear stress generated by the bending moment across the motion segment. Because there appears to be no greater deformation of the centrum in CFS4 and CFS5 than in CFS3, it is reasonable to conclude that the compressive stress has been resolved by the oblique fracture of the centrum.

The additional injury residuals seen in CFS4 and CFS5, because they reflect ligament failure, infer a tension/shear minor injury vector through the posterior part of the anterior elements and the entirety of the posterior elements. The site at which the oblique fracture of the centrum crosses the inferior chondral plate marks the transition from compressive to tension/shear failure and thereby identifies the transitional axis.

The true extent of ligament failure in CFS4 is unclear. The posterior displacement reflects at the

Table 1. Compressive Flexion

Case no.	Sex	Age	Stage	Level	Associated lesion	Level	Vertical fracture centrum	Level	Spinous process fracture	Level	Bilaminar fracture	Level	General mechanism	Specific mechanism	Craniofacial lacerations	Craniofacial fractures	Neurologic injury
1	M	33	1	C6,7			–		–		–		AA	–	–	–	N
2	M	17	1	C6,7			–		–		–		AA	–	Right occiput	–	N
3	M	20	1	C5,6			–		–		–		AA	–	–	Nasal and facial	N
4*	M	28	1	C5,6			–		–		–		MA	(+)	–	Nasal	N
5	M	14	1	C4,5			–		+	C7	–		AA	–	–	–	N
			1	C5,6													
6†	M	24	1	C5,6			–		–		–		DB	(+)	vertex	–	N
			1	C6,7													
7	M	29	2	C4,5			–		–		–		MA	–	–	–	N
8	M	32	2	C5,6			+	C5	–		–		AA	–	–	–	C
9	M	51	2	C5,6	VCS2	C6,7	–		–		–		AA	–	–	–	N
10‡	F	13	2	C3,4													
			2	C4,5													
			2	C5,6			–		–		–		F	(+)	–	–	N
11§	M	36	2	C4,5			–		–		–		AA	(+)	vertex	–	N
12	F	17	2	C4,5			–		–		–		AA	–	Chin	–	N
13	M	21	2	C5,6			–		–		–		O	–	Facial	–	N
14	M	14	3	C4,5			+	C4	–		–		SD	–	–	–	T
15	M	19	3	C4,5	DFS2	C3,4	–		–		–		AA	–	–	–	C
16¶	M	23	3	C5,6			–		–		–		F	(+)	–	–	N
17	M	59	3	C5,6			–		–		–		AA	–	–	–	N
18**	M	24	4	C5,6			+	C5	–		+	C5	AA	(+)	Vertex occiput	–	C
19	M	22	4	C6,7			–		–		–		SD	–	–	–	N
20	M	19	4	C5,6	DFS4	C4,5	–		–		–		AA	–	–	–	T
21	M	17	4	C5,6	DFS1	C4,5	+	C5	–		–		SD	–	–	–	C
22	F	17	4	C5,6			+	C5	–		–		SD	–	–	–	T
23	F	48	4	C5,6			–		–		–		AA	–	Right temple and frontal	–	T
24††	M	20	4	C5,6			–		–		–		F	(+)	–	–	P
25	M	17	4	C7,D1			–		–		–		SD	–	Frontal	–	N
26	M	17	5	C4,5			+	C4	–		–		SD	–	–	–	C
27‡‡	M	17	5	C5,6			+	C5	–		–		F	(+)	–	–	T
28	M	23	5	C4,5			–		–		–		SD	–	Vertex and temporal	–	T
29	M	22	5	C5,6			–		–		–		SD	–	–	–	T
30	M	27	5	C5,6			+	C5	–		–		SD	–	–	–	T
31	M	18	5	C4,5			+	C4	–		–		SD	–	–	–	T
			5	C5,6			+	C5									
32	M	21	5	C5,6			–		+	C5*	–		AA	–	–	–	T
33	M	24	5	C4,5			–		–		–		SD	–	–	–	T
34	M	28	5	C5,6			+	C5	–		–		AA	–	–	–	T
35	M	20	5	C5,6			–		–		–		AA	–	–	–	T
36	M	34	5	C6,7			–		–		–		AA	–	–	–	T

* Bike flipped forward, throwing patient onto top of head, forcing extreme flexion at impact.
† Car hood fell onto top of head of patient who was working on the engine and looking downward at time of impact.
‡ Landed on head while she was jumping on trampoline; the spine was forced into acute flexion; the accident was witnessed.
¶ Patient was thrown from back seat of jeep into front windshield, with impact over vertex forcing neck into flexion.
¶ Patient fell eight feet from scaffold onto top of head, forcing neck into flexion.
** Patient was thrown into dashboard from back seat of car, striking top of head.
†† Patient dived onto top of head while tumbling, forcing neck into flexion. The accident was witnessed.
‡‡ Patient was thrown by a horse and landed on top of his head, forcing the neck into flexion. The accident was witnessed.
§§ Horizontal splitting fracture.
Key: AA = automobile accident; MA = motorcycle accident; SD = shallow dive; DB = direct blow; F = fall; O = other mechanism; N = normal; C = central cord syndrome; P = partial cord lesion; T = total cord lesion; R (not evident among these patients) = radiculopathy.

very least partial failure of the posterior portion of the anterior ligamentous complex and probably partial failure of the posterior ligamentous complex. In the two patients with this lesion who were subjected to flexion–extension studies (a procedure we do not advocate), no definite subluxation of the articular facets could be indentified. Nonetheless, the degree of integrity of the posterior ligamentous complex remains in question because it has been demonstrated that serial sectioning of the supporting structures does not produce a correspondingly graduated displacement within a motion segment.[55,75] The 3-mm limit for posterior displacement used to distinguish CFS4 from CFS5 is not rigidly documented by our cases; all CFS4s had 3-mm or less posterior displacement, and all CFS5s had gross displacement.

The displacement in CFS5 denotes complete failure through the motion segment. Although the usual tension failure of the posterior portion of the anterior elements is rupture of the annulus and posterior longitudinal ligament, a brittle fracture of the posterior inferior margin of the centrum may sometimes occur An axial splitting fracture of the spinous process or an occasional bilaminar fracture represent a less common variety of tension/shear failure through the posterior elements. However, bilaminar fracture is not specific for tension/shear failure.

In the CF phylogeny, the uppermost vertebra in the motion segment which fails sustains the typical osseous injury. Designation of CF motion segments will therefore have the number of the upper vertebra in the motion segment underlined to indicate that a fracture typical for the phylogeny has occurred. For example, a CFS4 with fracture of C5 would have a level identity of *C5*,6 because C5 sustains the typical fracture, and C5–C6 is the motion segment in which displacement occurs.

The 41 CF lesions were distributed as follows: one at *C3*,4, 11 at *C4*,5, 23 at *C5*,6, five at *C6*,7 and one at *C7*, D1. The general pattern of distribution seemed to be independent of the stage of the CF lesion.

Vertical fractures of the centrum, observable on plain anterior-posterior views, were frequent and could be correlated with the stage of injury, occurring in 0% CFS1 (0/8 lesions/six cases), 11% of CFS2 (1/9 lesions/seven cases), 25% of CFS3 (1/4 lesions/four cases), 37.5% of CFS4 (3/8 lesions/eight cases), and 50% of CFS5 (6/12 lesions/II cases). According to Roaf, this type of fracture results from the nucleus pulposus being forcefully driven into the centrum by a compressive stress.[60] The increasing frequency with progression up the phylogeny probably reflects the greater force required to produce the more severe injury.

In four patients with CF injuries, there were associated lesions. These are listed in Table 7.

VERTICAL COMPRESSION

The spectrum of injury within the vertical compression phylogeny (VC) is shown in Figure 4. Details of the clinical data are listed in Table 2.

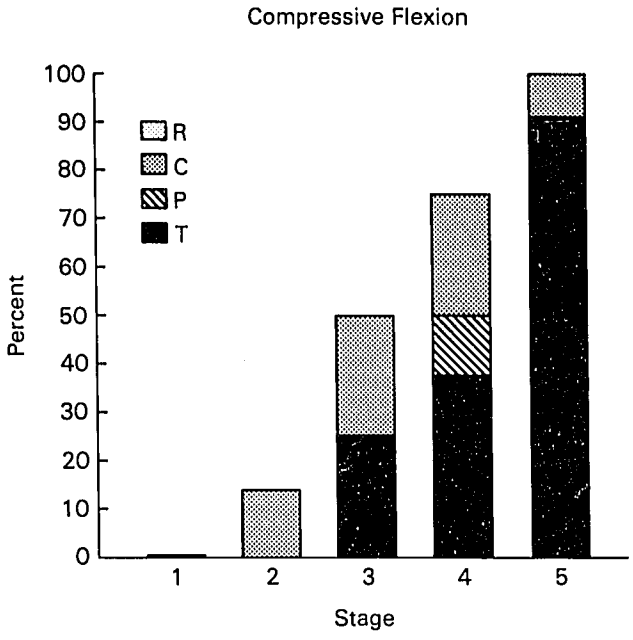

Fig 3.

VC Stage 1

The stage 1 lesion (VCS1) consists of a fracture of either a superior or inferior end-plate with a "cupping" deformity. The initial end plate failure is central rather than anterior, and there is no evidence of ligamentous failure (Figure 4A).

VC Stage 2

The vertical compression stage 2 (VCS2) lesion consists of fracture of both vertebral end-plates with cupping deformities. There may be fracture lines through the centrum, but displacement is minimal (Figure 4B).

VC Stage 3

The vertical compression stage 3 lesion (VCS3) shows a progression of vertebral body damage seen in VCS2. The centrum is fragmented, and its residual pieces are displaced peripherally in multiple directions. When there are only a few major fragments, a vertical fracture similar in appearance to those seen in CF may be seen, but more commonly the centrum fails with much impaction and fragmentation. The posterior portion of the vertebral body is fractured and may be displaced into the neural canal. In some cases, the vertebral arch is completely intact, and there is no evidence of ligamentous failure, while in others there is comminution of the vertebral arch with gross failure of the posterior ligamentous complex. In cases with vertebral arch comminution, the level of ligamentous disruption was between the fractured vertebra and the

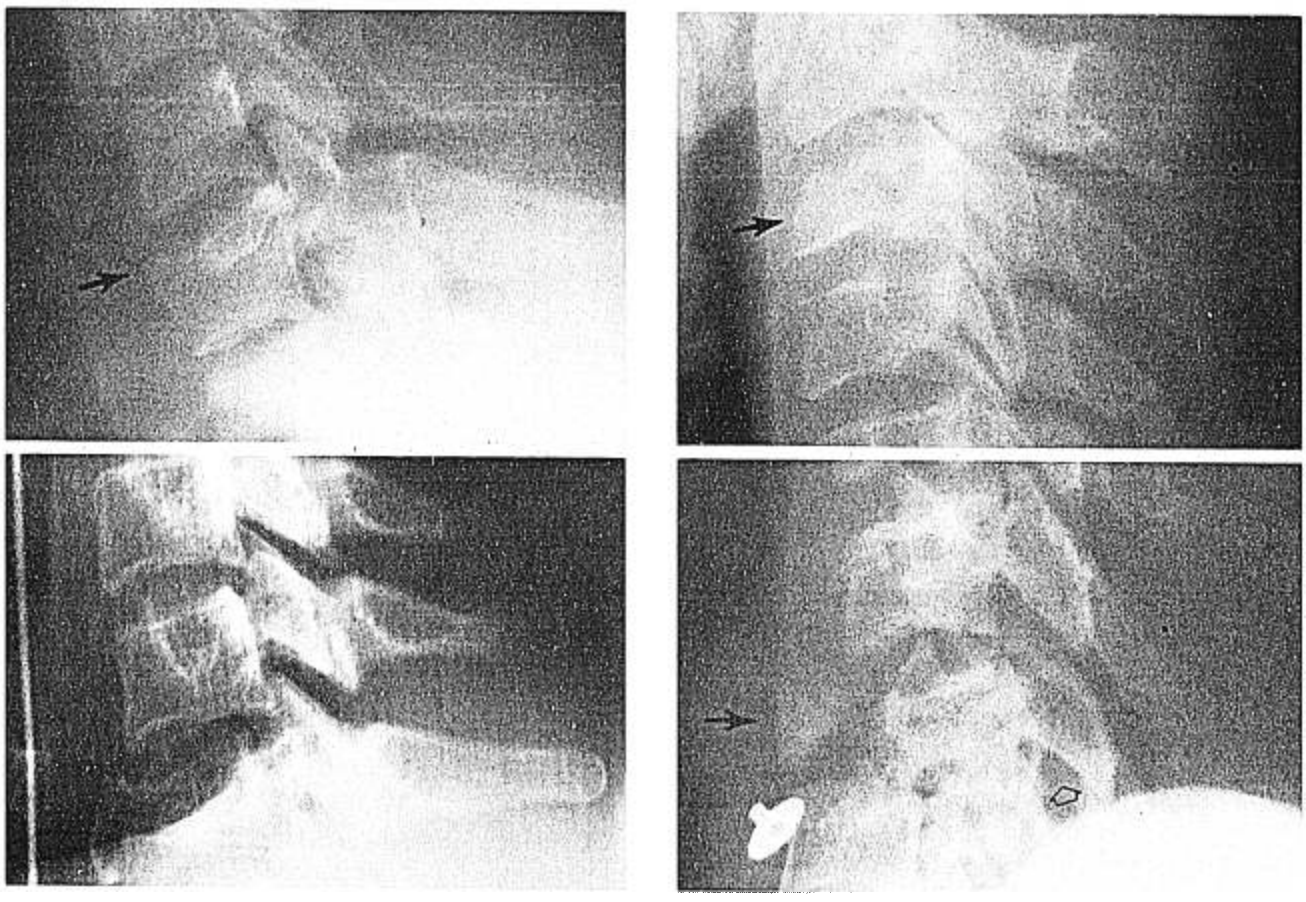

Fig 4A (upper left). VCS1. **B (upper right).** VCS2. **C (lower left).** VCS3, late flexion type. **D (lower right).** VCS3, late extension type.

subjacent one. Stage 3 lesions in which the vertebral arch is intact may have acute kyphotic angulation at the injury level (Figures 4C,D).

GENERAL CONSIDERATIONS FOR VC

Fourteen cases fit the VC phylogeny, with five VSC1, four VCS2, and five VCS3. All 14 patients were males, and the average age was 35.6 years.

Of the patients with a VCS1, one had a central cord syndrome. None of those with a VCS2 had a neurologic injury, and three with a VCS3 had a total cord lesion. These data, converted to percentages, are show in Figure 5.

Nine individuals were injured in automobile accidents, one in a motorcycle accident, three in shallow dives, and one by a fall. In both cases in which the specific injury mechanism was known, the history was consistent with a compressive force transmitted through the cervical spine with the neck in a neutral position. In one case, the impact was over the buttocks with the inertia of the head serving to injure a cervical motion segment.[38,41]

The pattern of deformation seen in VCS1 and VCS2 is consistent with compressive loading of the entire vertebral centrum and not with the obliquely downward and posterior force seen in the CF phylogeny. Because there is compressive failure of the entire centrum in VCS3, the transitional axis lies posterior to the anterior elements. There may be retropulsion of bone into the neural canal. This phenomenon is not seen in the compressive flexion phylogeny.

The fact that the vertebral arch is comminuted in some cases and not in others suggests that in the stage 3 injury one of two late events may occur. The cervical spine may go into flexion, shifting the neutral axis forward, thereby sparing the vertebral arch from compressive failure but subjecting the posterior elements to a tension/shear stress. The other possibility is that the cervical spine goes into late extension, shifting the neutral axis backward with loading of the posterior elements in compression. This may account for the comminuted fracture of the vertebral arch seen in some cases. In our stage 1 and stage 2 injuries, there was usually no injury residual to identify a minor injury vector.

None of the VCS3s without fracture of the vertebral arch had displacement suggestive of tension/shear failure through the posterior elements. The cases with comminution of the vertebral arch showed gross displacement between the vertebra sustaining the fracture and the subjacent one. We therefore feel that the VC motion segment should be identified in a manner similar to the CF phylogeny, with the fractured verte-

Table 2. Vertical Compression*

Case no.	Sex	Age	Stage	Level	Associated lesion	Level	Vertical fracture centrum	Level	Comminuted vertebral arch	Level	Bilaminar fracture	Level	General mechanism	Specific mechanism	Craniofacial lacerations	Craniofacial fractures	Neurologic injury
37	M	25	1	C5/6			–		–		–		AA	–	Left temple and face	–	N
38	M	25	1	C7,D1			–		–		–		SD	–	–	–	N
39	M	40	1	C7,D1			–		–		–		AA	–	–	–	N
40	M	18	1	C6/7			–		–		–		AA	–	–	Mandible	N
41	M	40	1	C6/7			–		–		–		MA	(+)	–	–	N
42†	M	40	2	C3/4			–		+	C6	–		AA	–	–	–	N
43	M	25	2	C6/7			–		–		–		AA	–	–	–	N
44	M	51	2	C4/5			–		–		–		AA	–	–	–	N
45	M	56	2	C6/7	CFS2	C5/6	–		–		–		AA	–	–	–	T
46	M	34	3	C7,D1	VCS1	C6/7	+	C6,C7	+	C7	+	C6	SD	–	–	–	T
47‡	M	68	3	C7,D1			–		–		–		F	(+)	–	–	T
48	M	28	3	C7,D1			–		–		–		AA	–	–	–	T
49	M	26	3	C6/7			–		–		–		SD	–	–	–	N
50	M	22	3	C5/6			+		+		–		AA	–	–	–	T
	C5							C5 (coronal plane)		C5							

* There were no spinous process fractures.
† Patient was thrown forward over motorcycle handlebars and landed on top of his head. The accident was witnessed
‡ Patient fell off lower rung of ladder onto buttocks, landing in a sitting position. The accident was witnessed.
§ See key to Table 1.

Fig 5.

bra considered to be the uppere one in the injured motion segment. By this convention, a VCS3 with fracture of the fifth cervical vertebra with gross displacement of the fifth and sixth vertebrae would be identified as *C5*,6.

Vertical compression injuries tended to be located low in the cervical spine. Of the 15 lesions, one occurred at *C3*,4, one at *C4*,5, two at *C5*,6, six at *C6*,7, and five at *C7*,D1. Three of the five VCS3s occurred at *C7*,D1.

As the severity of the vertebral body fracture increased, the incidence of vertebral arch fracture also incresed from 16% in stage 1 (1/6 lesions/five cases), to 25% in stage 2 (1/4 lesions/four cases), to 40% in stage 3 (2/5 lesions/five cases). The single VCS1 in which there was a vertebral arch fracture was associated with a stage 3 lesion of the subjacent motion segment in which there was comminution of the vertebral arch.

There was one instance of an associated lesion (Table 7) and one case with multiple lesions (Table 8) in the VC phylogeny.

DISTRACTIVE FLEXION

The distractive flexion (DF) phylogeny is shown in Figure 6. Details of the clinical data are listed in Table 3.

DF Stage 1

The stage 1 lesion (DFS1) consists of failure of the posterior ligamentous complex as evidenced by facet subluxation in flexion with abnormally great divergence of the spinous processes at the injury level, a condition which has been called "flexion sprain."[16,23,51,73] Frequently there is blunting of the

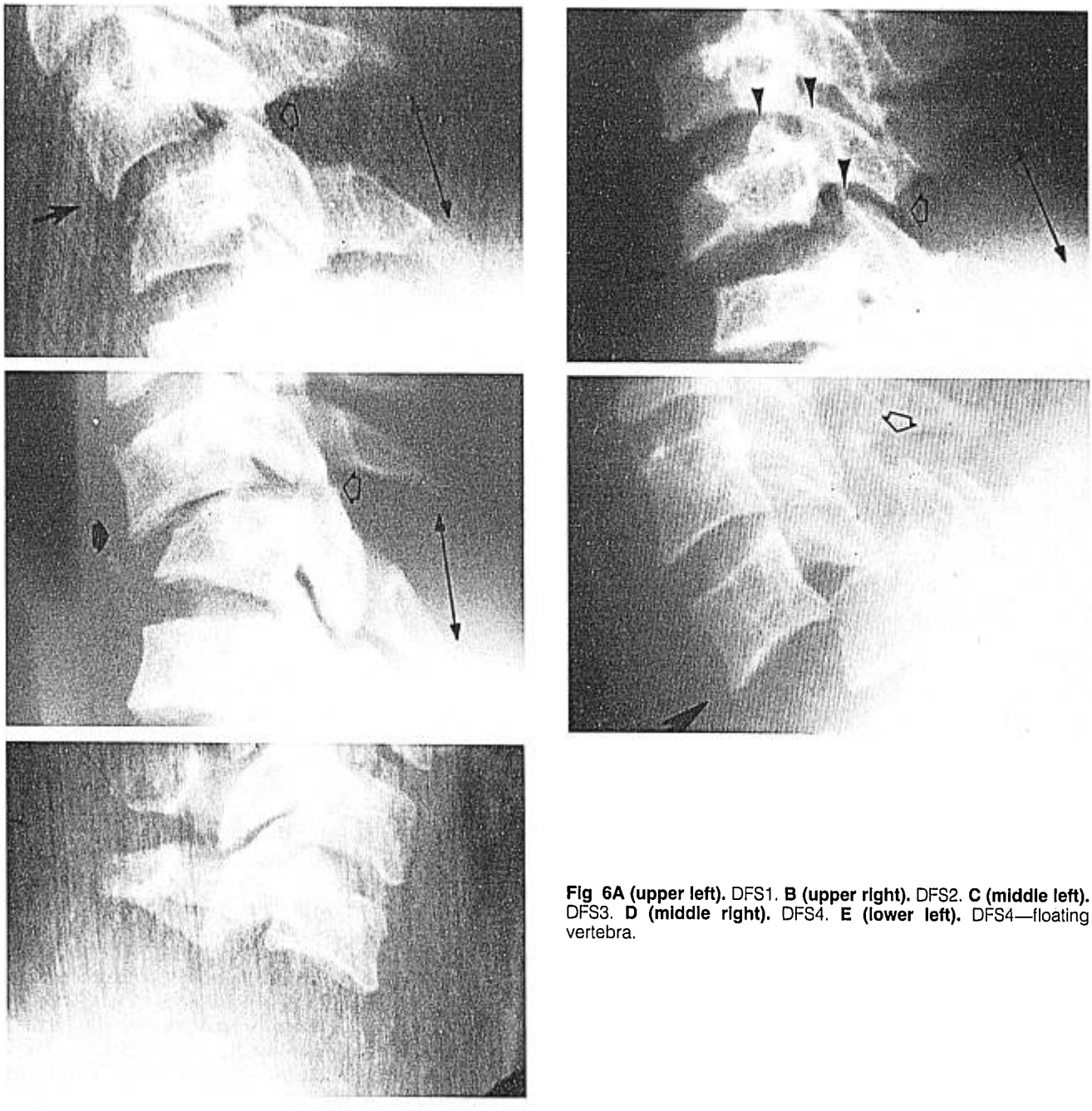

Fig 6A (upper left). DFS1. **B (upper right).** DFS2. **C (middle left).** DFS3. **D (middle right).** DFS4. **E (lower left).** DFS4—floating vertebra.

anterior superior vertebral margin to a rounded contour, reminiscent of the changes seen in CFS1. Occasionally there may be greater compressive failure of the lower vertebral centrum in the motion segment. This greater compressive failure is consistently one of the patterns seen in the early stages of the compressive flexion phylogeny (Figure 6A).

DF Stage 2

The distractive flexion stage 2 (DFS2) lesion is a unilateral facet dislocation (locked facet, interlocked facet, facet luxation). The degree of posterior ligamentous failure may not be obvious on the initial film as it may range from partial failure sufficient only to permit the abnormal displacement or, rarely, complete failure of both the anterior and posterior ligamentous complex.[9,17] Facet subluxation on the side opposite the dislocation suggests severe ligamentous injury. Dynamic studies may be necessary to fully define the full extent of ligament failure.[74,75]

There may be a small fleck of bone displaced from the posterior surface of the articular process which is

displaced forward. Rotary listhesis may be seen in the injured motion segment, with widening of the uncovertebral joint on the side of the dislocation and with displacement of the tip of the spinous process toward the side of dislocation (Figure 6B).

DF Stage 3

The distractive flexion stage 3 (DFS3) consists of bilateral facet dislocation with approximately 50% vertebral body with displacement anteriorly; the posterior surfaces of the superior vertebral articular processes lie either snugly against the anterior surfaces of the inferior vertebral articular processes or in a "perched" position. There may or may not be blunting of the anterior-superior margin of the inferior vertebra to a rounded contour (Figure 6C).

DF Stage 4

In the distractive flexion stage 4 lesion (DFS4), there is either full vertebral body width displacement anteriorly or a grossly unstable motion segment giving the appearance of a "floating vertera." (Figures 6D, E).

GENERAL CONSIDERATIONS FOR DF

Sixty-one cases fit the DF phylogeny, with 12 DFS1, 25 DFS2, 17 DFS3, and seven DFS4. Forty-nine males and 12 females with an average age of 33.3 years comprised the group.

Two patients with a DFS1 had a radiculopathy and two a central cord syndrome. Two DFS2 cases had a radiculopathy, three a central cord syndrome, three a partial cord syndrome, and five a total cord lesion. One of those with a total cord lesion had a five-hour symptom-free interval before the onset of his neurologic deficit. In the DFS3 category, two patients had a radiculopathy, one a central cord syndrome, two a partial cord lesion, and ten a total cord injury. In DFS4, there were six total cord lesions. These data are shown as percentages in Figure 7.

Thirty-five injuries were attributable to automobile accidents, six to shallow dives, 12 to falls, four to direct blows to the head, and four to other injury mechanisms. In each of the six cases in which the specific injury mechanism was known, the neck was postured in flexion and a force was transmitted to the occipital portion of the calvarium.

The characteristic tension/shear failure of the posterior ligamentous complex in the DF phylogeny indicates a major injury vector directed away from the trunk stressing the posterior elements in tension/shear. The degree of ligamentous failure is sequentially greater from DFS1 to DFS3. The frequently seen compressive lesion of the lowermost vertebral centrum in the motion segment sustaining DF trauma identifies a minor injury vector which is compressive. Because the compressive lesion is consistently of the same pattern as those seen in the CF phylogeny, the transitional axis lies somewhere in the middle third of the vertebral body as it did for CF. But not all DF lesions have a compressive injury to the lower vertebra; therefore, in many instances there is no significant minor injury vector. In such cases, the neutral axis must lie extremely anterior, or there is no neutral axis because the flexed spine is subjected to a pure tension/shear stress. When this is the case, the posterior elements and subsequently the anterior elements fail in tension/shear.

Compressive injury to the lowermost vertebra occurs in decreasing frequency with progression up the phylogeny; it occurred in 50% DFS1 (6/12 lesions/12 cases), 32% DFS2 (8/25 lesions/25 cases), 23.5% DFS3 (4/17 lesions/17 cases), and 14.3% DFS4 (1/7 lesions/seven cases). These data indicate that the transitional axis and probably the neutral axis lie more anterior in severe DF injuries.

Bauze and Ardran performed an experimental study which accurately reproduced clinical DF trauma.[8] They demonstrated that rotation was not necessary for cervical ligament failure at relatively low loads and pointed out how the engineering constraints of his experiments led Roaf to erroneous conclusions about the role of rotary stresses in the production of ligamentous injury.[60] Rotation appears to be a factor which may lateralize a DF ligament failure.

Fracture of a small fleck of bone from the posterior-inferior margin of an articular process was seen only in DFS2, in which it occurred in 20% of the cases (5/25 lesions/25 cases). Theoretically there is no reason for the fracture not to occur in any DF lesion. Always it had the appearance of a brittle fracture.

Although straightforward ligament rupture is the common type of tension/shear failure of the posterior elements, spinous process fracture, usually of the axial, splitting variety, and bilaminar fractures also occur in DF. These vertebral arch fractures were consistently at the uppermost vertebra of the injured motion segment and were usually isolated vertebral arch injuries. When a DFS3 or DFS4 was associated with a bilaminar fracture, there was in addition rupture of the ligaments between the vertebral arch fragment and the arch of the vertebra suprajacent to the motion segment involved in the DF injury. Consistently the fractured vertebral arch remained in place when its vertebra was displaced anteriorly. Although it seems logical that in this circumstance the arch fracture might have a cord-sparing effect, our data do not support this possibility; 40% DFS3s with bilaminar fracture (2/5 lesions/five cases) versus 41.6% DFS3s without bilaminar fracture (5/12 lesions/12 cases) did not have a total cord injury, and the only two DFS3s with no associated neurologic injury were cases with no bilaminar fracture. The sole DFS4 with no neurologic injury did have a bilaminar fracture.

Identification of the injured motion segment is easy because there is vertebral displacement in all stages of the DF phylogeny. If either vertebra in a motion segment is fractured, a line is placed under its number. For example, a DFS2 at C5,6 with a posterior marginal fracture of the articular process of C5 and compression

Table 3. Distractive Flexion

Case no.	Sex	Age	Stage	Level	Associated lesion	Level	Compressive lesion lower vertebra	Level	Spinous process fracture	Level	Bilaminar fracture	Level	General mechanism	Specific mechanism	Craniofacial lacerations	Craniofacial fractures	Neurologic injury
51	M	47	1	C5,6			–		–		–		AA	–	–	–	N
52	F	57	1	C4,5			+	C5	–		–		AA	–	–	–	R
53	F	43	1	C3,4			–		–		+	C3	F	–	–	–	N
54	M	28	1	C6,7			+	C7	–		–		F	–	–	–	N
55	F	16	1	C4,5			+	C5	–		–		AA	–	Posterior auricular and facial	–	N
56	M	19	1	C4,5	CFS2	C5,6	+	C5	–		–		SD	–	–	–	C
57	M	17	1	C4,5			+	C5	–		–		SD	–	–	–	N
58	F	61	1	C4,5			–	C5	–		–		O	–	–	–	N
59	M	53	2	C4,5			–		–		–		AA	–	–	–	N
60	M	60	1	C5,6			–		–		–		F	–	–	–	N
61*	M	32	1	C6,7			–		–		–		DB	(+)	–	–	R
62	M	32	1	C5,6			+	C6	–		–		AA	–	–	–	C
63	M	24	2	C5,6			+	C6	–		–		AA	–	–	–	P
64	M	50	2	C5,6	HNP	C5,6	–		–		–		F	–	–	–	P
65	F	33	2	C6,7			+	C7	–		–		AA	–	–	–	N
66	M	18	2	C7,D1			+	D1	–		–		AA	–	Vertex	–	N
67	M	18	2	C2,3			–		–		–		AA	–	–	–	N
68	F	18	2	C5,6			+	C6	–		–		AA	–	–	–	C
69	F	15	2	C6,7R	CES1	C6,7L	–		+	C6‡‡	–		AA	–	–	–	N
70†	M	20	2	C6,7			–		+	C6	–		DB	(+)	–	–	R
71	F	25	2	C6,7	FX.body	C2	–		–		–		AA	–	–	–	N
72	M	24	2	C4,5			–		–		–		AA	–	–	–	N
73	M	27	2	C6,7			–		–		–		AA	–	–	–	N
74	M	17	2	C5,6			–		+	C6‡‡	–		F	–	–	–	T
75	M	21	2	C3,4	CFS2	C4,5	+	C4	–		–		AA	–	Orbital	–	C
76	M	19	2	C4,5			–		–		–		AA	–	–	–	T+‡‡
77	M	19	2	C6,7			–		–		–		AA	–	–	–	T
78	M	27	2	C4,5	Jefferson fx.	C1	–		–		–		F	–	–	–	P
79	F	30	2	C4,5			+	C5	–		–		O	–	–	–	N
80	M	43	2	C5,6			+	C6	–		–		AA	–	–	–	N
81	M	25	2	C4,5			–		–		–		AA	–	–	–	T
82	M	23	2	C3,4	Fx. margin articular process	C2	–		–		–		SD	–	–	–	N
83	M	52	2	C6,7			–		–		–		F	–	–	–	N
84	M	30	2	C3,4			–		–		–		O	–	–	–	N
85	M	66	2	C3,4	CFS2	C4,5	+	C4	+	C3	–		AA	–	–	–	R
86‡	M	14	2	C3,4			–		–		–		O	(+)	–	–	N
87§	M	15	2	C4,5	CES2	C2,3,4	–		–		–		SD	–	–	–	T
88	M	16	3	C6,7			–		–		–		F	–	Parietal	–	N
89	M	19	3	C5,6			–		–		–		AA	–	Forehead	–	T
90	M	73	3	C5,6			–		–		–		AA	–	–	–	P
91	M	48	3	C4,5			+	C5	–		–		AA	–	–	–	T
92	M	25	3	C5,6			–		–		–		AA	–	–	–	T
93	M	17	3	C4,5			+	C5	–		–		SD	–	–	–	T
94	F	54	3	C6,7			–		+	C5‡‡	+	C6	AA	–	Orbital	–	T
95	M	55	3	C6,7			–		–		+	C6	F	–	–	–	T

Table 3. (*Continued*)

Case no.	Sex	Age	Stage	Level	Associated lesion	Level	Compressive lesion lower vertebra	Level	Spinous process fracture	Level	Bilaminar fracture	Level	General mechanism	Specific mechanism	Craniofacial lacerations	Craniofacial fractures	Neurologic injury
96[¶]	M	49	3	C4,5			+	C5	–		–		F	(+)	–	–	R
97	M	16	3	C4,5			+	C5	–		–		SD	–	–	–	T
98	M	28	3	C6,7			–		–		+	C6	AA	–	Forehead	–	T
99	M	59	3	C5,6			–		–		–		F	–	–	–	N
100	M	45	3	C6,7			–		+	C6[‡‡]	–		AA	–	–	–	T
101	M	56	3	C6,7			–		–		+	C7	AA	–	–	–	R
102[**]	M	57	3	C7,D1			–		–		+	C7	DB	(+)	–	–	C
103	M	49	3	C6,7			–		+	C6[‡‡]	–		F	–	–	–	T
104	F	20	3	C6,7			–		–		–		AA	–	–	–	P
105	F	25	4	C5,6			–		–		+	C5	AA	–	–	–	T
106	M	26	4	C7,D1			–		–		–		AA	-	–	–	T
107[††]	M	40	4	C6,7			–		–		+	C6	DB	(+)	–	–	N
108	M	41	4	C6,7			–		–		+	C6	AA	–	Multiple scalp and face	–	T
109	M	18	4	C4,5	CFS3	C5,6	+	C5	–		–		AA	–	–	–	T
110	M	40	4	C6,7			–		+	C6[‡‡]	–		AA	–	–	–	T
111	M	22	4	C6,7			–		–		–		AA	–	Auricular and orbital	–	T

[*] A fellow worker dropped a bag of cement onto the back of the patient's head while he was looking downward.
[†] Patient was struck in the back of the head by a medicine ball.
[‡] Patient was rolled over by a car so that the neck was slowly forced into flexion.
[§] Reportedly patient struck a submerged pipe.
[¶] Patient fell backward off a ladder onto the back of the head. The accident was witnessed.
[**] Patient was struck in occipital area when a truck backed into him at the loading dock.
[††] Patient was struck on back of head by falling tree.
[‡‡] Horizontal splitting fracture.
+ = five-hour symptom-free interval before onset.
See key to Table 1.

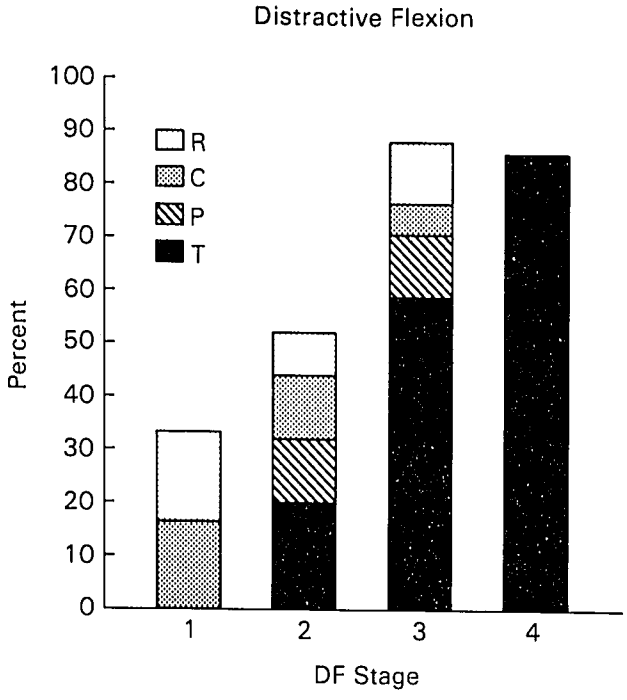

Fig 7.

of the anterior-superior margin of C6 would be designated *C5,6*. When the compressive injury of the lower vertebra consisted of more than blunting of the anterior-superior margin of the inferior centrum, that lesion was classified in the compressive flexion phylogeny and listed as an associated injury (Table 7).

When neurologic injury occurred in the DFS2, the x-ray studies usually showed evidence of a degree of ligamentous failure more severe than usual; most commonly this was subluxation of the opposite articular facet or distraction at the injury level with the application of skull traction. Three of our four patients with total cord injuries associated with DFS2 demonstrated facet subluxation on the contralateral side on initial radiographs. Additionally in each of these three, the motion segment was observed to be easily distracted when the patient was placed in traction. The fourth patient had a combined lesion. This suggests that DFS2s with severe neurologic involvement have greater ligamentous injury than those with no neurologic injury.

The 61 distractive flexion lesions were distributed as follows: one at C2,3, six at C3,4, 17 at C4,5, 13 at C5,6, 21 at C6,7, and three at C7,D1. As one moves up the phylogeny, there is a distinct tendency for the injury to be localized at a lower level in the cervical spine. Fifty percent of DFS1 occurred at C4,5 (6/12 lesions/12 cases), while only 14.3% DFS4 occurred at C4,5 (1/7 lesions/seven cases); 57.1% DFS4 occurred at C6,7 (4/7 lesions/seven cases), as compared with 16.7% DFS1 at C6,7 (2/12 lesions/12 cases). Stage 2 and 3 lesions were intermediate, with 24% DFS2s occurring at C4,5 (6/25 lesions/25 cases) and 23.5% DFS3s occurring at C4,5 (4/17 lesions/17 cases); 28% DFS2s occurred at C6,7 (7/25 lesions/25 cases), and 47% DFS3s occurred at C6,7 (8/17 lesions/17 cases). The anterior shift of the transitional axis as one moves up the phylogeny and the change in distribution to lower in the neck suggests a major role for the cervical lever arm and for shear stress in producing severe distractive injuries.

There were no multiple DF injuries; the ten cases with associated lesions are shown in Table 7.

COMPRESSIVE EXTENSION

The comparessive extension (CE) phylogeny is shown in Figure 8. Details of the clinical data are listed in Table 4.

CE Stage 1

The stage 1 lesion (CES1) consists of a unilateral vertebral arch fracture with or without anterorotary vertebral body displacement. Arch failure may consist of a linear fracture through the articular process, a compression of the articular process, an ipsilateral pedicle and laminar fracture resulting in the so-called "transverse facet" appearance, or a combination of ipsilateral pedicular and articular process fracture. Rotary listhesis of the centrum may occur with any of these fractures but is not an essential feature. When present, it is generally less than that seen in DFS2 (unilateral dislocated facet) (Figure 8A, B, C).

CE Stage 2

Bilaminar fractures without evidence of other tissue failure in the cervical motion segments constitute the compressive extension stage 2 lesion (CES2). Typically the laminar fractures occur at contiguous multiple levels (Figure 8D).

CE Stage 3 and CE Stage 4

These stages are hypothetical at this time, not having been encountered in our review. However, because they are a logical link between the early and late stages in the CE phylogeny, a description is given. The CES3 consists of bilateral vertebral arch "corner" fractures-articular processes, pedicles, lamina, or some bilateral combination without vertebral body displacement. The CES4 consists of bilateral vertebral arch fractures with partial vertebral body width displacement anteriorly.

CE Stage 5

Compressive extension stage 5 lesion (CES5) consists of bilateral vertebral arch fracture with full vertebral body width displacement anteriorly. The posterior portion of the vertebral arch of the fractured vertebra does not displace, while the anterior portion of the arch remains with the centrum. Ligamentous failure occurs at two different levels, posteriorly between the

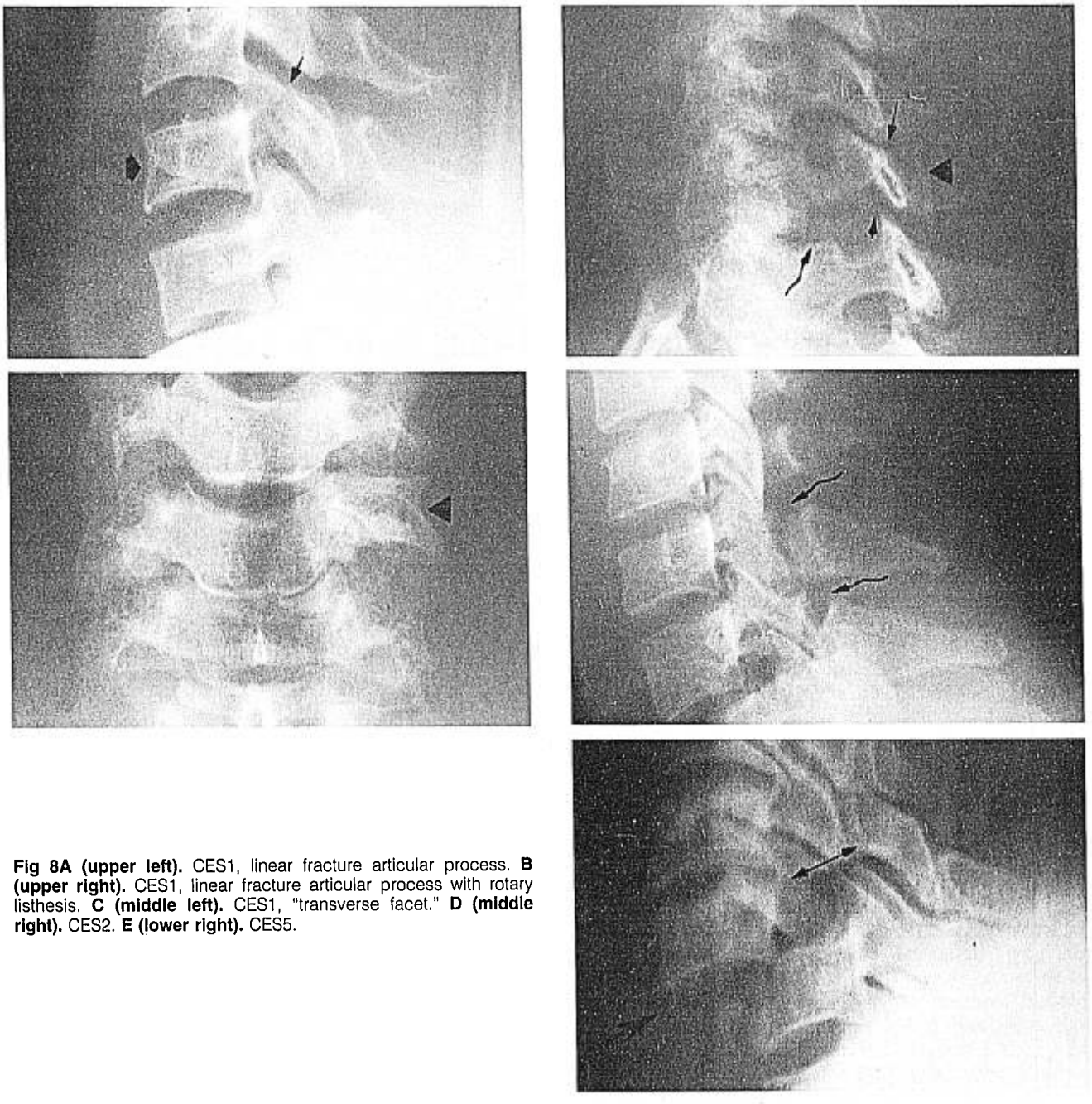

Fig 8A (upper left). CES1, linear fracture articular process. **B (upper right).** CES1, linear fracture articular process with rotary listhesis. **C (middle left).** CES1, "transverse facet." **D (middle right).** CES2. **E (lower right).** CES5.

suprajacent and the fractured vertebra and anteriorly between the fractured vertebra and the subjacent one. The anterior-superior portion of the subjacent vertebral centrum is characteristically sheared off by the anteriorly displaced centrum (Figure 8E).

GENERAL CONSIDERATIONS FOR CE

Forty cases fit the CE phylogeny, with 32 CES1, five CES2, and three CES5. Twenty-nine males and 11 females with an average age of 30.6 years comprised the group.

In the CES1 category, eight patients had a radiculopathy, four a central cord lesion, one a partial cord lesion, and one a total cord lesion. Two of the CES2s were associated with a total cord lesion. In CES3, one individual had a radiculopathy, one a central cord syndrome, and one a partial cord lesion. These data are displayed as percentages in Figure 9.

Twenty-two injuries occurred in automobile accients, two in shallow dives, nine from falls, five from direct blows to the head, and two by other injury mechanisms. In the three cases in which the specific injury mechanism was known, there was a blow to the forehead or face which forced the neck into extension and thrust the head toward the trunk.

Table 4. Compressive Extension*

Case no.†	Sex	Age	Stage	Level	Associated lesion	Level	Articular process fracture	Ipsilateral pedicle and lamina fracture	Spinous process fracture	Level	Bilaminar fracture	Level	General mechanism	Specific mechanism	Craniofacial lacerations	Craniofacial fractures	Neurologic injury
112*	M	25	1	C6,7			+	–	–		–		AA	–	–	–	R
113	M	28	1	C6,7			+	–	–		–		AA	–	–	Facial	R
114	F	30	1	C6,7			+	–	–		–		AA	–	–	Mandible	N
115	M	38	1	C6,7			+	–	–		–		F	–	–	–	N
116	M	37	1	C6,7			+ (also lamina)	–	–		–		AA	–	–	–	R
117	F	15	1	C6,7			+	–	–		–		AA	–	–	–	N
118	M	25	1	C7,D1			–	+	–		–		AA	–	–	–	N
119	M	36	1	C6,7			–	+	–		–		AA	–	–	–	R
120	M	22	1	C7,D1			+	–	–		–		AA	–	Right ear	–	T
121	F	16	1	C3,4	Fx. margin articular process	C2	–	+	–		–		AA	–	Facial	–	N
–	+																
122	M	51	1	C3,4	Fx. pedicles, bilateral	C2	+	–	–		–		F	–	–	–	N
123	F	13	1	C4,5			+	–	–		–		F	–	–	–	N
124	F	25	1	C6,7			+	–	–		–		AA	–	–	–	R
125	F	25	1	C3,4			+	–	–		–		AA	–	–	–	N
126	M	29	1	C7,D1			+	–	–		–		F	–	–	–	P
127	M	23	1	C6,7			+	–	–		–		AA	–	Parietal	–	N
128	F	35	1	C4,5			+	–	–		–		DB	–	–	–	C
129†	M	28	1	C4,5			–	+	–		–		AA	–	–	–	C
			1	C5,6			+	–									
130	M	37	1	C5,6			–	+	–		–		AA	–	–	–	C
131	M	19	1	C4,5			–	+	–		+	C4	AA	–	–	–	N
132	M	63	1	C6,7			+	–	+	C6‡	–		F	–	–	–	N
133	F	23	1	C6,7			–	+	–		–		AA	–	–	–	C
134	M	41	1	C6,7			+	–	–		+	C5	DB	–	–	–	N
135	M	60	1	C7,D1			+	–	–		+	C6	DB	–	Frontal occipital	–	N
136	F	16	1	C5,6			–	+	–		–		AA	–	–	–	N
137	M	21	1	C5,6			–	+	–		–		AA	–	–	–	R
138	M	27	1	C4,5			–	+	–		–		AA	–	–	Orbital	N
139	F	77	1	C4,5			–	+	–		–		O	–	–	–	N
140	M	18	1	C6,7			+	–	–		–		AA	–	–	–	N
141§	M	30	1	C4,5			–	+	–		–		DB	(+)	–	–	N
142	M	25	1	C6,7			+	–	–		–		SD	–	–	–	R
143	M	23	1	C7,D1			+	–	–		–		F	–	–	–	R
144	M	25	2	C2,3,4			–	–	–		–		AA	–	–	–	N
145¶	F	17	2	C6,7			–	–	–		–		F	(+)	–	Facial	N
146	M	16	2	C3,4,5			–	–	–		–		SD	–	–	–	T
147	M	60	2	C5			–	–	–		–		F	–	–	–	N
148	M	20	2	C6,7			–	–	–		–		O	–	–	–	T
149	M	56	5	C7,D1			+	–	–		–		AA	–	–	–	R
150**	M	22	5	C6,7			+	–	–		–		F	(+)	–	–	C
151	M	25	5	C6,7			+	–	+	C5,C6	–		DB	–	–	–	P

* No vertical fractures of the centrum occurred in the compressive extension phylogeny.
† Cases 112–128 had no rotary listhesis; cases 129–143 demonstrated a mild degree of rotary listhesis.
‡ Horizontal splitting fracture.
§ Patient struck forehead on overhang while going down stairs.
¶ Patient fell forward over handlebars of bicycle onto face.
** Patient fell headlong from branch of tree and landed on face.
See key to Table 1.

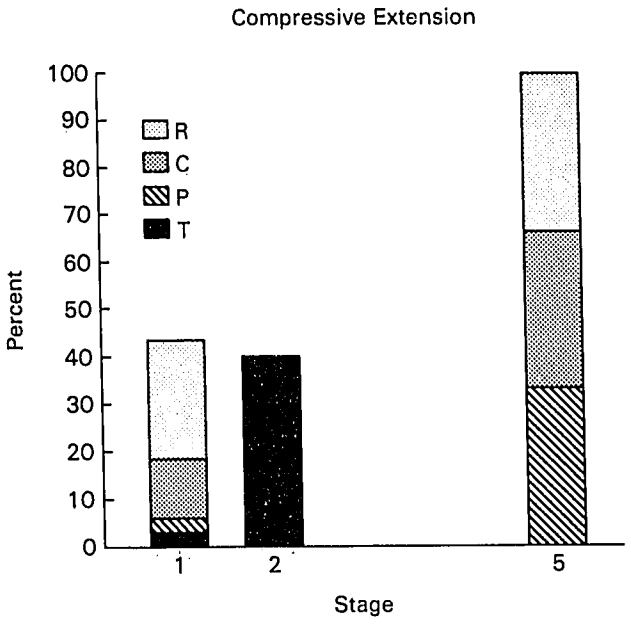

Fig 9.

The vertebral arch fractures, characteristic of CES1 and CES2, without evidence of damage to the posterior ligamentous complex, indicate a major injury vector directed toward the trunk, stressing the posterior elements in compression. The high frequency of CES1 indicates that there is frequent lateralization of the compressive stress by associated rotational forces. The patterns of injury in the CE phylogeny are not so definitely sequential as in the previous ones.

Early in this study, the stage 1 lesions were divided into two groups, one with anterorotary vertebral displacement and one without vertebral body displacement. However, when divided in this manner, there was no significant difference in the incidence of neurologic injury or in the distribution of the type or pattern of osseous failure. Cases 112 through 128 had no rotary listhesis, while cases 129 through 143 had a mild degree of rotary listhesis. It is evident from Table 4 that each of these sets contains a mixture of articular process and ipsilateral pedicle and laminar fractures. Because of this, we elected to include both the nondisplaced and displaced unilateral vertebral arch fractures in the CES1.

The articular process fractures in the CE phylogeny should not be confused with the ones seen in the DFS2. In CE mechanisms, the articular process may undergo an impacting type of fracture in which the shape is altered, a linear fracture through the body of the articular process, or some permutation of these two. No fracture was seen which resembled the brittle, marginal fracture seen in DF.

The CES5 has a consistent, complex pattern of failure involving two contiguous motion segments. For purposes of description, consider three cervical vertebrae A, B, and C with A being the most cephalad. Linear fractures through the articular processes of vertebra B permit anterior displacement of the body of B on C, while posterior element displacement occurs between the vertebral arch of A and the posterior vertebral arch fragment of B. There has been a shear failure of the midline portion of the posterior ligamentous complex between A and B, the lateral portion of the posterior ligamentous complex between B and C, and a tension/shear failure through the anterior elements between B and C. In all three of our cases, there was a "gouging" fracture of the anterior-superior surface of C. The fact that vertebra C appears undeformed, save the anterior-superior centrum fracture, suggests that a shear force accounts for the brittle nature of the fracture. There may be some impaction of the minor fragment. The reports of Pittman et al and of Bailey show typical CES5 without fracture of vertebra C.[5,58] The overall pattern in the CES5 is consistent with late unconstrained rotation in the motion segment, as suggested by Forsyth.[29]

In this phylogeny the severity of the anatomic damage to the cervical motion segment did not correlate well with the severity of the spinal cord lesion. The absence of total cord injury in the three CES5s is interesting and has been noted by other authors.[5,58]

We have found no discussion of cord injuries associated with the isolated laminar fractures by other authors. Bohlman, in his discussion of laminar fractures, found all asociated with paralysis to also be associated with a fracture of a vertebral body and of one or more articular processes.[14] We found no evidence to support a Taylor-Blackwood mechanism of injury in these cases; it is possible that the Taylor mechanism may be operative in young patients if the compression stress through the posterior elements is sufficiently large in magnitude.[47,68,69]

Since displacement in the CES1 is between the vertebra, with fracture of the vertebral arch and the subjacent one, labeling of the involved motion segment can be done as for previous phylogenies. For example, a CES1 with unilateral vertebral arch fracture at C5 with rotary listhesis of C5 on C6 would be designated *C5*,6. It is more difficult to apply this convention to CES2 and CES3. We have elected to label the CES2 according to the fractured laminae since there were no injuries with displacement. If a CES2 involved fracture of the lamina of C4, C5, and C6, it would be designated *C4, 5, 6*. In stage 5, because three vertebra are involved in the pattern of tissue failure, a method of designation had to be arbitrarily decided. We chose to label the CES5 according to the motion segment in which the anterior elements were displaced. For example, a CES5 with fracture through the articular processes of C6, shear failue through the posterior ligamentous complex at C5,6, and displacement of the C6 centrum anterior to the centrum of C7 would be designated *C6,7*.

CES1 and CES2 injuries occurred at all levels in the lower cervical spine, with the majority being concentrated at C6,7. Two CES5s occurred at C6,7 and one at C7,D1.

There was one multiple CE lesion (Table 8) and two CES1s with associated lesions (Table 7).

DISTRACTIVE EXTENSION

The distractive extension phylogeny is depicted in Figure 10. Details of the clinical data are given in Table 5.

DE Stage 1

The stage 1 lesion (DES1) consists of either failure of the anterior ligamentous complex or a transverse nondeforming fracture of the centrum. When the injury is primarily ligamentous, as it usually is, there may or may not be a brittle fracture of an adjacent anterior vertebral body margin. The radiographic tipoff to the injury is usually abnormal widening of the disc space. In the stage 1 injury, there is no posterior displacement (Figure 10A).

DE Stage 2

The distractive extension stage 2 (DES2) lesion, in addition to the changs seen in DES1, shows evidence of failure of the posterior ligamentous complex with displacement of the upper vertebral body posteriorly into the neural canal. Because displacement of this type tends to spontaneously reduce when the head is postured at neutral or in flexion, radiographic evidence of the displacement may be subtle, rarely greater than 3 mm on initial films with the patient supine (Figure 10B).

GENERAL CONSIDERATIONS FOR DE

Nine cases fit the DE phylogeny, with two DES1 and seven DES2. All nine patients were males with an average age of 47.2 years.

There were no neurologic abnormalities in DES1. Three of the DES2 individuals had a central cord lesion, two a partial cord lesion, and one a total cord injury.

Four lesions were sustained in automobile accidents and five in falls. The specific circumstance of injury was known in two cases. In each there was a fall onto the face from a height. These data are displayed in Figure 11.

The initial failure of the anterior ligamentous complex in the DE phylogeny implicates a major injury vector directed away from the trunk and stressing the anterior elements in tension. The tension/shar failure in DES2 indicates that this stress is transmited to the posterior elements following failure of the anterior elements. There is no evidence for a significant minor injury vector. These findings indicate that the neutral axis may lie extremely dorsal in the cervical spine. The data suggest that in the DE phylogeny the neck is extended and the force applied over the anterior calvarium or face. Whitley and Forsyth, in their classification of cervical injuries, identified an extension injury with compression of the articular processes and failure through the anterior ligamentous complex. These patterns were labeled "extension injury with compession, bilateral with ligament break" or "extension injury with compression, unilateral with ligament

Table 5. Distractive Extension

Case no.	Sex	Age	Stage	Level	Associated lesion	Level	Vertical fracture centrum	Level	Spinous process fracture	Level	Bilaminar fracture	Level	General mechanism	Specific mechanism	Craniofacial lacerations	Craniofacial fractures	Neurologic injury
152*	M	56	1	C7,D1			–		–		–		F	(+)	–	–	N
153	M	21	1	C5,6			–		–		–		AA	–	Occipital	–	N
154	M	49	2	C5,6			–		+	C6	–		F	–	Orbital	–	P
155	M	78	2	C4,5			–		–		–		F	–	–	–	N
156†	M	21	2	C5,6,7			–		–		–		F	(+)	–	–	C
157	M	49	2	C6,7			–		–		–		F	–	Forehead	–	C
158	M	53	2	C5,6			–		–		–		AA	–	–	–	P
159	M	53	2	C4,5			–		–		–		AA	–	–	–	C
160	M	45	2	C5,6			–		–		–		AA	–	–	Facial	T

* Patient fell off seawall, landing on chest and face.
† Patient fell from second story onto right shoulder and face.
See key to Table 1.

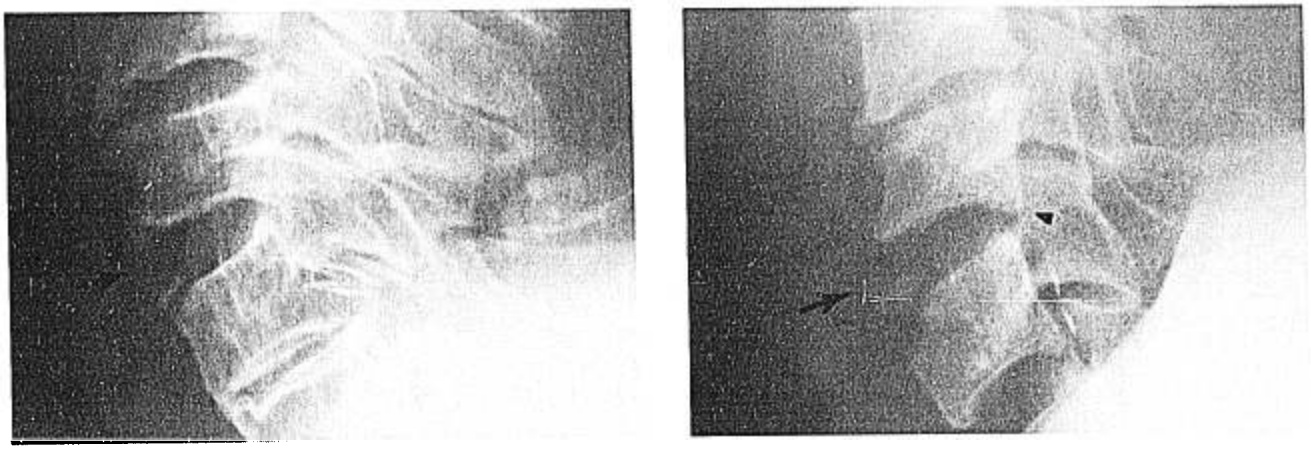

Fig 10A (left). DES1. **B (right).** DES2.

break." While it is certainly theoretically probable that this injury pattern might occur, we found no such cases, and the case illustrations of Whitley and Forsyth are unconvincing.[76]

Taylor and Blackwood discussed the mechanism of spinal cord injury in DES2; the cord is scissored between the posterior-inferior margin of the cephalad vertebra in the motion segment which fails and the ligamentum flavum and lamina of the caudal vertebra.[69] Additionally, they call attention to the possibility of normal radiographic appearance with this type of ligamentous failure. It is probable that many lesions go undetected because of the subtle evidence of injury on initial radiographics. Cases which might have been identified by the presence of reactive changes several weeks after injury were not available to us because the coding system we used was keyed to abnormality on initial x-ray studies. Therefore it seems probable that the frequency of DE lesions in this series is lower than in reality. Dynamic studies have generally not been done for detection of the Taylor-Blackwood injury.

Although the number of cases in this phylogeny was too small for a satisfactory analysis, two trends were apparent which are consistent with findings of other authors: the DE injury frequently resulted from a fall (five of nine cases) and occurred in an older age group than other cervical fractures and dislocations.[47,68]

Since there is ligamentous failure with displacement between two vertebrae, identification of the involved motion segment is obvious. In the event of a fracture, such as a marginal avulsion fracture, the number of that cervical vertebra is underlined. For example, a DES1 at CF,6 with avulsion of the anterior-inferior margin of C5 would be labeled *C5*,6.

The cases in this series were distributed from C4,5 to C7,D1, with the majority involving C5,6.

An anterior marginal avulsion fracture of the centrum might originate from either the cephalad or the caudal vertebra in DE injuries but most frequently was from the caudal (five caudal/one cephalad).

There were no associated and no multiple lesions in the DE phyolgeny.

LATERAL FLEXION

The lateral flexion phylogeny (LF) is shown in Figure 13. The clinical data for the few available cases are shown in Table 6.

LF Stage 1

The stage 1 lesion (LFS1) consists of asymmetric compression fracture of the centrum plus vertebral arch fracture on the ipsilateral side without displacement of the arch on the anterior-posterior view. Special views or tomography may show compression of the articular process or communition of the corner of the vetebral arch. The asymmetric compression of the vertebral body may appear as an uncovertebral fracture with some internal collapse of the cephalad verte-

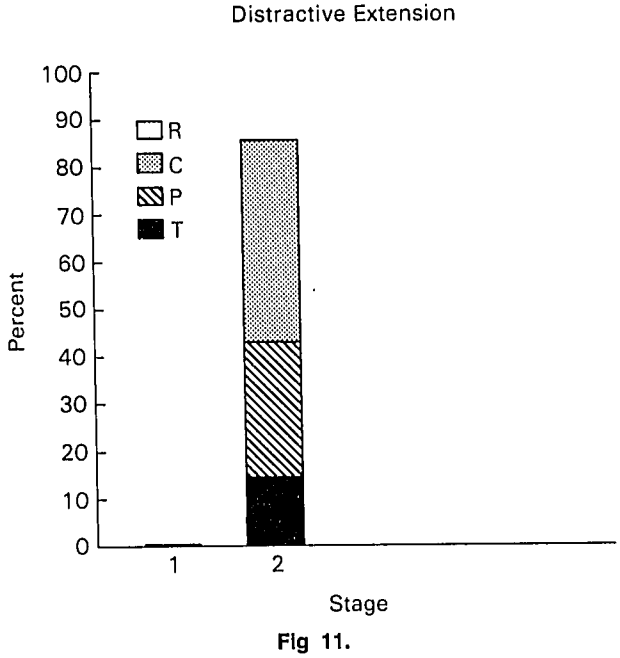

Fig 11.

Table 6. Lateral Flexion

Case no.	Sex	Age	Stage	Level	Associated lesion	Level	Vertical fracture centrum	Level	Spinous process fracture	Level	Bilaminar fracture	Level	General mechanism	Specific mechanism	Craniofacial lacerations	Craniofacial fractures	Neurologic injury
161	M	30	1	C3,4			–		–		–		AA	–	Temporal	–	N
162	F	22	1	C5,6			–		–		–		AA	–	–	–	N
163*	M	14	1	C2,3			–		–		–		O	(+)	–	–	N
164	M	27	2	C2,3			–		–		–		AA	–	–	–	N
165	M	21	2	C6,7			+	C7	–		–		AA	–	–	–	T

* Patient was trapped beneath a slowly moving car with his head pinned "ear against shoulder."
See key to Table 1.

bral body bone in the region of the uncovertebral joint. There may be a vertical fracture of the centrum (Figures 12 A, B).

LF Stage 2

The lateral flexion stage 2 lesion (LFS2) has both lateral asymmetric compression of the centrum and either ipsilateral vertebral arch fracture with displacement on the anterior-posterior view or ligamentous failure on the contralateral side with separation of the articular processes. In some cases, both the ipsilateral compressive and contralateral disruptive vertebral arch injury may be present (Figure 12C).

GENERAL CONSIDERATIONS FOR LF

Five cases fit the LF phylogeny, with three LFS1 and two LFS2. There were four males and one female with an average age of 22.8 years. No neurologic injury was associated with a LFS1, while one of the LFS2s was associated with a total cord lesion. Roaf, in a report of five cases of lateral flexion injury, emphasized the high incidence of neurologic damage with all of his cases having evidence of brachial plexus injury and three of the five having a total cord injury.[61] All of his cases showed evidence of tension failure on one side of the vertebral arch, and therefore we would classify them as LFS2s. None of our cases had evidence of a brachial plexus injury.

Four injuries were sustained in automobile accidents and one by another mechanism. In the one case in which the specific circumstance of injury was known, the head was slowly forced toward the shoulder so that the patient's ear was against the shoulder (Figure 13).

It is conceivable that compressive lateral flexion and distractive lateral flexion mechanisms may exist, but our case material is too limited to elucidate this probability. The asymmetric compression of the vertebral centrum seenin both LFS1 and LFS2, with fracture of the vertebral arch on the side of compression, implicates a compressive injury vector following a load path long the side of the spine to which the lateral flexion occurs. The occurrence of tension failure on one side of the vertebral arch opposite the compression of the centrum implicates a distractive injury vector following a load path through the side of the cervical spine away from the direction of lateral flexion. Judging from our case material, we suspect that usually the compressive injury vector is the major one and the distractive injury vector the minor one. Our data are too limited to predict any consistent placement of the transitional axis.

There may be "kissing" compressive lesions of the vertebral centra in the motion segment, with impaction of adjacent portions of the cancellous bone. This pattern of injury has been termed an uncovertebral fracture and was popularized by Abell as one of the occult fractures occurring in the cervical spine.[1]

Because of the paucity of available material, our recommendation for identification of the involved mo-

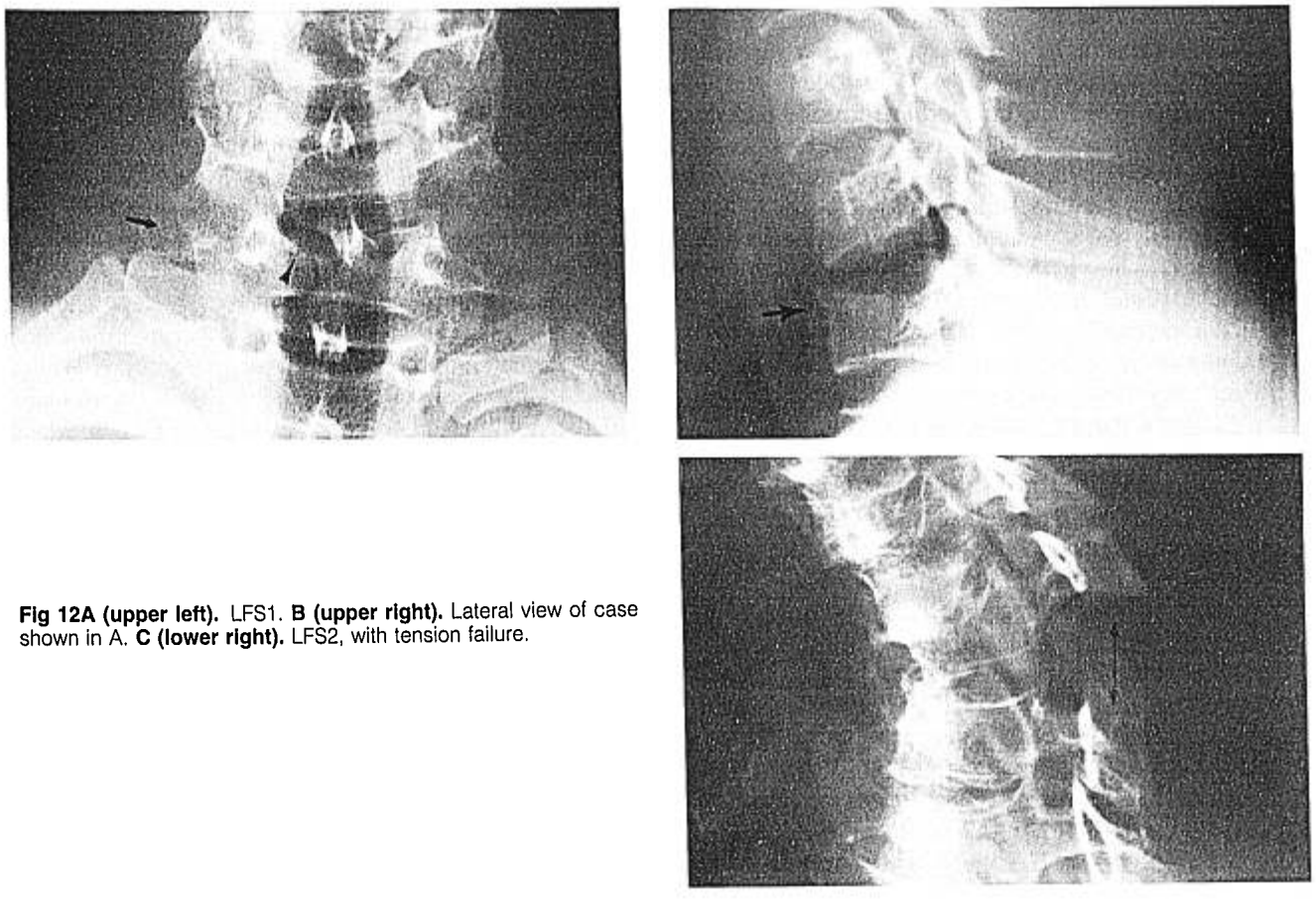

Fig 12A (upper left). LFS1. **B (upper right).** Lateral view of case shown in A. **C (lower right).** LFS2, with tension failure.

tion segment is tentative. Because the cases we have seen frequently involved fracture of adjacent centra, with the unilateral vertebral arch fracture lying caudal in the motion segment, and because when there is tension failure it is between the vertebra with fracture of the vertebral arch and the one lying superior, we identified the motion segment with the vertebra having the vertebral arch fracture being lowermost. For example, a LFS2 with asymmetric compression of the C5 vertebral body and ipsilateral fracture of the C5 vertebral arch would be identified C4, 5. The cases in the LF phylogeny were scattered from C2,3 to C6,7. In this phylogeny, there were no associated or multiple lesions.

DISCUSSION

All of the injury mechanisms identified in the present classification have been well described by other authors,[15,30,36,53,61,65] some have been validated by laboratory investigations on cadavers.[8,9,47,60]

This study is the first to categorize comprehensively all indirect, lower cervical spine injuries by mechanism of injury. Previous clinical reports have either focused on one mechanism of injury, excluded the minor injuries within a given injury mechanism, or mistakenly classified patients with clearly different mechanism of injury into a single category. The present classification avoids these shortcomings by including all lower cervical spine fractures and dislocations regardless of severity. This does make the classification significantly more detailed than those in the

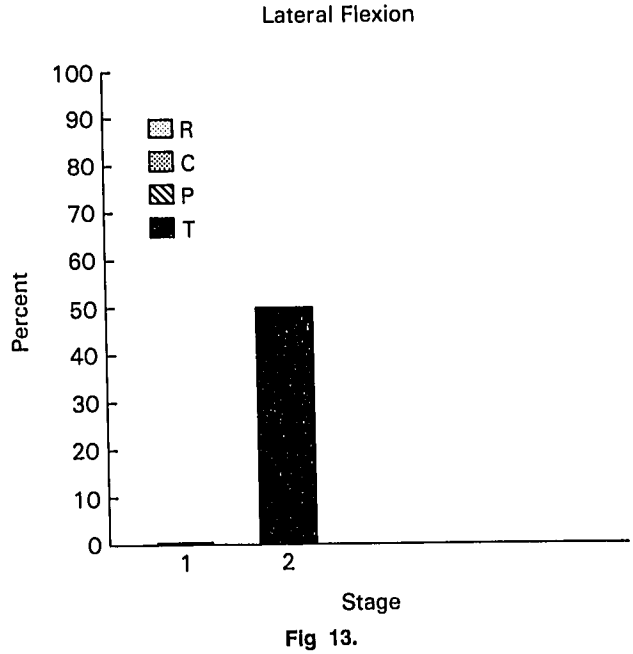

Fig 13.

literature. We think this additional complexity to be justified because each stage in a phylogeny represents a specific type and degree of injury to a cervical motion segment; because the probability and degree of neurologic damage correlates with the skeletal lesion; because precise assessment of diagnostic and treatment modalities is impossible without accurate identification of the particular injury; and because no other classification has provided a basis for unconfused communication.

An understanding of the clinical biomechanics for each phylogeny enables the orthopaedic surgeon to analyze the injury vectors in a given case and deduce its phylogeny and stage of injury. Direct injury mechanisms such as blows to the neck, missile wounds, and pathologic fractures are biomechanically different from indirect injury mechanisms. Because of this, the anatomic damage to the cervical motion segment is different, and lumping all of these together for clinical analysis, as is frequently done, makes it impossible to recognize significant correlates.

Our simplistic one-plane analysis of injury vectors, while workable for clinical radiographs, is clearly deficient from a stringent biomechanical point of view. Normal motion cannot occur in one plane of the three-dimensional spine without a coupled movement in other planes.[45,74] Although coupling has not been demonstrated to occur with injury, it may be one factor which lateralizes stress, thereby causing more damage on the left or right side of the spine. Another lateralizing factor is rotation about the vertical axis. Thus both coupling and rotation may account for the asymmetric injuries. On the basis of clinical evidence, we think each of these factors serves to focus the major stresses rather than to represent the major stress in their own right. Injuries previously described as being primarily secondary to torsion are assigned to one of the six injury mechanisms in the present classification.

If one accepts the validity of this mechanistic classification, the awesome state of vagueness in the cervical spine literature becomes apparent. While it is not our intention to burden the reader with a recitation of published inconsistencies, a few examples to emphasize the point are appropriate. "Teardrop fracture," "burst fracture," and "wedge fracture" are typical, frequently used terms devoid of precise meaning. Schneider and Kahn's written description of teardrop fracture closely approximates the CFS5;[65] compression of the anterior portion of the centrum, fracture of the anterior centrum, and displacement of the inferior vertebral body posteriorly into the spinal canal are all mentioned. But their case illustrations displayed a mixture of CFS3, CFS4, CFS5, VCS3, and combined lesions. Other authors further degraded the term teardrop by expanding it to include the brittle fracture of the centrum seen in disruptive extension lesions.[28] What is a teardrop fracture?

Burst fracture usually lumps together a mixture of CF and VC lesions.[34] Holdsworth illustrates a vertical compression lesion of the lumbar spine without ligamentous disruption as a prototype and states that these are "stable" fractures, but he uses a CFS5 as an example of a burst fracture in the cervical spine.[36,37] Marar and Gehweiler et al did not discern CF and VC lesions.[33,46] Kerwalramani and Taylor show a CFS4 with a large anterior fragment as a burst and a CFS3 with a small anterior fragment as a teardrop.[39] Wedge fracture has sometimes referred to CFS2, occasionally to the VCS2, and frequently to the DFS1 in which there is a compressive lesion of the lower vertebra in the injured motion segment.

It would be naive to believe that all lower cervical spine injuries would fit cleanly into the classification because our data base is too small to be definitive. The lack of a reasonable number of cases is especially a problem for the VC, DE, and LF phylogenies. It seems unlikely that any single center can collect a meaningful volume of material, and good understanding of these lesions may await a multicenter collaborative effort. To illustrate, we found two LFS2s among 471 cases of neck injury. If we wanted 20, which is still a small group, and if the distribution remained constant, 4710 cases would have to be reviewed. The most common injuries easily fit the classification, and probably all indirect fractures and dislocations can be analyzed by our biomechanical approach. Some uncommon lesions, such as the CES3, CES4, or one which is transitional between DF and CF can be anticipated because their occurrence may require only a specific balance between the stresses known to be operative in more common injuries. The stages for lateral flexion may need to be expanded when larger data base is available, and perhaps the VCS3 should be separated into late flexion and late extension types. We consider it best at this time to keep the classification as short as possible, consistent with available information.

The associated lesions which occurred in the compressive flexion, vertical compression, and disruptive flexion phylogenies (see Table 7) displayed several interesting trends. Any combination of lesions from these phylogenies always occurred at contiguous motion segments, suggesting stress concentration in a particular short segment of the spine. When compressive failure of the centrum occurred at two levels, the transitional axis consistently lay more posterior at the more caudal level. For example, when CF and VC lesions were associated, the VC injury was consistently inferior to the CF, and when DF and CF lesions occurred concomitantly, the CF was consistently inferior to the DF. There were no DF–VC combinations.

Table 7. Associated Lesions

Case no.	Primary lesion	Level	Associated lesion	Level
9	CFS2	C5,6	VCS2	C6,7
15	CFS3	C4,5	DFS2	C3,4
20*	CFS4	C5,6	DFS4†	C4,5
21	CFS4	C5,6	DFS1	C4,5
45	VCS2	C6,7	CFS2	C5,6
56	DFS1	C4,5	CFS2	C5,6
64	DFS2	C5,6	HNP	C5,6
69	DFS2	C6,7R	CES1	C6,7L
71	DFS2	C6,7	Fx. body	C2
75	DFS2	C3,4	CFS2	C4,5
78	DFS2	C4,5	Fx. ring (anterior and posterior)	C1
82	DFS2	C3,4	Fx. margin articular process	C2
85	DFS2	C3,4	CFS2	C4,5
87*	DFS2	C4,5	CES2	C2,3,4
109	DFS4	C4,5	CFS3	C5,6
121	CES1	C3,4	Fx. margin articular process	C2
122	CES1	C3,4	Fx. pedicles	C2

* C-5 quadriplegia.
† "Floating" vertebra.

Summarily, we can say that DF lesions occurred one level cephalad to CF lesions, which occurred one level cephalad to VC injuries. Although all of the operative factors cannot be deduced, it is highly probable that for these three phylogenies the neutral axis lies progressively more dorsal in the motion segments at sequentially lower levels in the cervical spine and that the higher the level, the greater the tension/shear stress across the motion segment.

If the preceding deductions are valid, one should expect isolated DF injuries to tend to occur at higher levels than CF injuries, which in turn would occur at higher levels than VC lesions. The median level for DF lesions was C5,6, for CF C5,6, and for VC C6,7. The mean level, calculated by assigning a number to each motion segment in the lower cervical spine with C2,3 having a numerical value of 1, was 3.92 for DF, 4.28 for CF, and 5.21 for VC. Thus, the stress patterns apparent for associated injuries appear to be true for isolated lesions as well.

As was the case with associated lesions, multiple similar lesions consistently occurred at contiguous motion segments and only in the phylogenies characterized by compressive failure (see Table 8). If the lesions occur simultaneously, the stress pattern is similar over several motion segments at the time of failure, and the stress rate is high enough to strain several vertebral centra to their yield point at one time. This suggests that the phylogenies in which multiple lesions commonly occur are higher-stress-rate injuries than those phylogenies in which lesions tend not to occur multiply. Furthermore, the caudal shift in localization of a lesion with progression up the DF phylogeny and the diminishing frequency of a compressive lesion of the anterior centrum strongly supports changing posture at low stress rates as being a major factor in the genesis of severe DF lesions, again supporting a difference in stress rate. If these stress rate deductions are valid, the cervical motion segment has higher yield strength in compression than in tension and than in shear.

The cervical lever arm may be more important in some injuries than in others. A high stress rate which quickly strains a motion segment to failure would tend to negate the effect of the cervical lever arm. Areas of maximum stiffness, such as those in the lower cervical region where there is normally a changing contour, might be more vulnerable in the rapid-loading situation. Low stress rates in DF might initially produce a change of posture, thereby maximizing the effect of the cervical lever arm, moving the neutral axis forward, and subjecting the cervical spine to predominantly a tension/shear stress, all by virtue of a change in cervical posture.

It has been generally conceded that the specific history of an injury can so rarely be elicited that it is impossible to be certain of the injury mechanism for the majority of patients.[15] Soft-tissue injuries are not a good predictor of the mechanism of injury. Fractures of the face and mandible correlate well with an extension injury, being present in 14.3% of extension cases and only 2% of flexion cases; obviously, these are sufficiently infrequent to be of great value. However, if one accepts that within a given injury mechanism the minor injuries on one end of the spectrum are caused by the same loading pattern as the more severe injuries on the other end and can corroborate the proposed mechanism from the clinical history for the minor injuries, then the proposed mechanism for the more severe injury is likewise corroborated. It is significant that in this study there were no historical mechanisms which were inconsistent with the deduced biomechanical ones (Table 9).

"Instability" in the lower cervical spine is an ethereal subject badly in need of rigorous definition.[6,49,56] It

Table 8. Multiple Lesions in Same Phylogeny

Case no.	Lesion	Level
5	CFS1	C4,5
	CFS1	C5,6
6	CFS1	C5,6
	CFS1	C6,7
10	CFS2	C3,4
	CFS2	C4,5
	CFS2	C5,6
31	CFS5	C4,5
	CFS5	C5,6
46	VCS3	C7,D1
	VCS1	C6,7
129	CES1	C4,5
	CES1	C5,6

Table 9. Unequivocal History of Injury Mechanism

Percent	Phylogeny	Stage 1*	Stage 2	Stage 3	Stage 4	Stage 5
22.2	CF	2/6	2/7	1/4	2/8	1/11
14.3	VC	0/5	1/4	1/5		
8.2	DF	1/12	2/25	2/17	1/7	
8.0	CE	1/32	1/5	—	—	1/3
22.2	DE	1/2	1/7			
20.0	LF	1/3	0/2			

* Numerator = definite history; denominator = total cases in that stage.

has sometimes been reduced to empiric variables in the literature.[28,75] We propose that much confusion would be avoided if the presence of neurologic injury were not considered a factor in instability. Since 49.3% of cases of cord injury in Braakman and Penning's series had negative radiology, and many cord-injured children have no musculoskeletal injury, it does not seem irrational to do this.[15,20] Instability should be defined as greater than normal range of motion within a motion segment. Just as is the case in other articulations, there are degrees of instability and different patterns of instability. These can be correlated with specific anatomic deficiencies, as demonstrated by the phylogenies in this series. When instability is a factor in treatment decisions, several questions should be posed. Is the degree of instability a threat to neural function? What is the natural history of the particular type of instability present? What anatomic structures must be deficient to permit the abnormal movement? As an example of the relevance of these questions, one might compare the flexion instability of the CFS3 with that of the DFS1. Both permit abnormal range of flexion in the injured motion segment. Neither presents a high probability of mechanical damage to the spinal cord. In the CFS3, increased flexion is secondary to the vertebral body deformation and fracture; the natural history is reliable healing of the fracture with restoration of stability, oftentimes with spontaneous vertebral body fusion.[12] In the DFS1, posterior ligament complex rupture permits increased flexion; the natural history is unreliable healing with a risk of late angular deformity and increased instability.[23,51,73] While there are insufficient clinical data to make an incontrovertible treatment decision, it is clearly rational to treat a CFS3 conservatively and the DFS1 by spine fusion. A similar pattern of analysis can be done for each stage of injury in the various phylogenies. It is accurate to say at this time that while rational treatment decisions can be made, a firm data base to validate the treatment decision has not yet been collected.

Another factor to be considered in cervical spine injuries is residual malalignment. In contrast to instability which indicates abnormal movement, malalignment implies a fixed abnormal relationship. Each phylogeny has its unique malalignment possibilities. It is difficult to do anything more than recognize the fact at present because data on the probability of late neural damage secondary to malalignment have not been compiled. There are, however, a number of reports which indicate the possibility of delayed cord damage.[65,66] In some, the interval has been 25 to 30 years. The pathophysiology is unclear. Our belief is that no malalignment which can be corrected without undue risk should be accepted; the ordinary risks of open reduction are reasonable. We therefore hold that all acute DFS2s, to cite one controversial example, should be reduced. Treatment should not produce malalignment, a classic example of which is the angular deformity produced by anterior arthrodesis in cases of incompetent posterior elements.[67] In those rare cases in which anterior decompression of the cord is indicated, alignment can be maintained by postural means.[78]

From this discussion, it should be evident that there is no "checklist" which provides a rational analysis of an injury or data-base approach to treatment. The skeletal injury, the neurologic injury, associated injuries, medical disorders, and unique individual factors should be analyzed in each case. Oftentimes, there will be questions which at this time are unanswerable. It is our hope that the mechanistic classification will facilitate a keener analysis of lower neck injuries so that eventually we will be confronted by fewer unknowns as we cope with the clinical problems.

The spectrum of injury to the spinal cord seen in the various phylogenies clearly demonstrates that more than one mechanism of cord injury is probably operative in clinical cases. The CF phylogeny is a good example. It is clear that in the CFS5 lesion, the cord may be pinched or scissored between the posterior inferior margin of the displaced body and the vertebral arch of the subjacent vertebra, but it is difficult to indict this "pincer" mechanism in the CFS2, CFS3, and CFS4 cases with total cord lesions. The possibility of vascular injury, traumatic edema, intraspinal hemorrhage, contact pressure, and "overstretching" cannot be put into perspective.[10,11,18–20,35,59,77] Well-correlated autopsy studies are needed. There is the possibility that some types of cord injury could be more effectively treated if we better understood the pathology.[26]

Our data do not support a correlation between the type of skeletal lesion and the pattern of incomplete myelopathy, which many studies have indicated.[14,46,65] Only one of the patients in the combined compressive flexion–vertical compression phylogenies had a partial cord syndrome, and it was not the anterior type but more like a mixed-central-cord, Brown-Sequard variety. We cannot reconcile our observations with the high incidence of anterior cord syndromes reported

with burst, teardrop, and wedge fractures. Kewalramani and Taylor, as did we, found predominantly central cord and total cord injuries in patients with a myelopathy who were injured in a shallow dive mechanism.[39]

In the other phylogenies, except for lateral flexion in which there were no central or partial cord injuries, the central cord lesion was more frequent than partial cord lesion and tended to occur in the earlier stages of a phylogeny. With increasing severity of the musculoskeletal lesion, there was an increase in the incidence of partial and total cord injuries. Excepting one patient with a DES2 and a Brown-Sequard syndrome, the partial cord cases could not be cleanly designated to classic categories. Our belief is that acute traumatic myelopathies fall into three consitent groups: central, partial, and total spinal cord injuries. Central cord injury represents the least degree of neurologic damage, and total the most severe. Partial covers an intermediate spectrum. Inferred is that the gray matter is more vulnerable to injury than the white matter. The topography of the cord lesions seems more consistent with damage to the blood supply of the cord than with any proposed mechanical mechanism of cord trauma.

Unless one classifies lower cervical injuries in a mechanistic manner, the correlation between musculoskeletal and nervous system lesions is not recognized. Barnes noted the lack of correlation between the degree of vertebral displacement and the severity of the spinal cord lesion.[7] Durbin agreed.[27] Castellano and Bocconi state, "There is not reasonable relationship between the gravity of the morphological lesion and the incidence of the neurological complication."[22] White et al conclude that "with the possible exception of bursting fracture of the vertebral body and the contrast between unilateral and bilateral facet dislocation, there is little correlation between observation of postraumatic x-rays and the degree of medullary damage."[74] All of these authors came to erroneous conclusions because their data could not be ordered in a manner which enabled a significant correlate to be recognized. It is evident that the higher stages of injury within each phylogeny are more likely to show a severe cord injury than are the lower stages. Higher stages are reflective of a more severe injury to the spine and predictably show a more severe cord involvement.

There are biases and limitations in this study which should be noted. A significant fraction of patients seen at the University of Texas Medical Branch hospitals are tertiary referrals; this series is therefore probably weighted toward more severe injuries. The coding system from which we have retrieved cases is keyed to initial diagnosis, and individuals who might have developed late signs of injury, ossification of the anterior longitudinal ligament following a DE injury, for example, were not identified. Additionally, patients who were not admitted to the hospital were not entered into the coding system because outpatient diagnoses are not coded at this institution. The neurologic injuries were catalogued according to clinical signs on the date of injury; total and central cord lesions were clear, but partial cord lesions might have been subdivided if later signs were used as a basis. For the bulk of our cases, tomograms were not available; some fractures, such as vertical fractures of the centrum, marginal fractures of articular processes, and undispalced laminar fractures which may not be apparent on plane films, were probably underrecognized. Myelography was seldom done in these cases; consequently we could not determine the probability of soft tissue being displaced into the neural canal for each of the phylogenies. The treatment provided in each case was so variable that no firmly documented recommendations for managment could be generated.

The unique character of the injury victims in our series deserves mention. By a large majority, young men who were driving an automobile constituted the patient population. It was our impression that they usually were engaged in a risk-taking or thrill-seeking activity at the time of injury and that more often than not they had been consuming alcohol or using mind-altering drugs. As a group, they tended to be either unemployed or worked at a manual "macho" job; most had no more than a highschool education. We cannot be certain to what extent these observations are biased by referral to a state hospital.

On the basis of this study, we are convinced that indirect fractures and dislocations of the lower cervical spine can be classified in a rational way which provides meaningful categorization of injuries. Whether or not the categories we have suggested are the final ones remains to be established. There is the obvious need for other investigators to study their cases and test this classification for ease of applicability and consistency. At this time, the biomechanical concepts voiced are deduced rather than demonstrated, and there is need for careful testing of these in the laboratory situation. Much remains to be done.

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