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Acute Fractures and Dislocations of the Cervical Spine^{*†}

AN ANALYSIS OF THREE HUNDRED HOSPITALIZED PATIENTS
AND REVIEW OF THE LITERATURE

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ABSTRACT: Of 300 patients who were hospitalized for acute cervical injuries, 216 lived, fifty-one died within four months of injury, and thirty-three were lost to follow-up. The important findings in a retrospective review were that laminectomy resulted in a high mortality rate and loss of motor function and that steroids did not improve neural recovery in quadriplegics and their use was associated with gastrointestinal hemorrhage.

Closed or open reduction followed by posterior fusion for subluxations or dislocations, and anterior decompression and fusion for vertebral compression fractures, offered the best chance for recovery of neural function and restoration of stability.

Massive epidural hemorrhage was found only in patients with ankylosing spondylitis.

Acute injuries of the cervical spine are among the most common causes of severe disability⁵⁶ and death following trauma, yet the diagnosis of these injuries is often delayed and the treatment, inadequate. In an attempt to develop better guidelines for the care of these injuries, the records of 300 patients who were hospitalized for cervical spine injuries with or without paralysis and who were treated at the Johns Hopkins Hospital and Baltimore City Hospitals between 1950 and 1972 were analyzed. In addition, the literature was reviewed and the findings were correlated with those previously reported.

Very few authors have compared the results of

laminectomy^{80,128,132} with those of other methods of surgical treatment such as posterior stabilization^{94,95} and anterior fusion with or without decompression⁹².

The results of laminectomy reported by Holdsworth⁶³, Wharton¹²⁸, and others^{17,31,39,78,132} were uniformly poor, with increased neural deficit after the procedure. Cheshire²⁹, Bedbrook¹⁰, and Norton⁸³ stressed non-operative treatment and reported good results and a low incidence of late instability after such treatment²⁷.

Bailey and Badgley^{7,8}, Robinson and Southwick^{91,93}, and others^{9,15,32,50,65} have described the importance of surgical stabilization to prevent further damage when the spinal cord is injured. More recently, the feasibility and results of anterior decompression and fusion for compression fractures and disc protrusions have been described by various authors, but these results have not been compared with those of other forms of treatment^{19,20,30,52,82,84,87,89,120-122}.

The pathophysiology of cervical spinal-cord injuries has been studied experimentally, clinically, and at autopsy in humans^{2,6,18,24,25,33,36-38,40,45,51,53,55,66,70,71,124}, but there have been few attempts to correlate the pathological findings with the clinical picture¹⁰. Efforts by various authors to classify the neurological syndromes seen clinically with respect to the different pathological processes that follow injury^{16,21,41,74-78,98-104,106,113,129} have suggested that any correlation between the neurological syndrome seen clinically and the histological changes in the spinal cord found at autopsy is difficult.

The present study was undertaken in an attempt to correlate the pathological findings at autopsy with the various types of cervical spine and cord injury and to determine the best method of treatment for these injuries as judged from the results after long-term follow-up.

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Materials and Methods

The 300 cases, which were collected in Baltimore, Maryland, were not consecutive. The Baltimore City Hospitals did not have a computerized retrieval system and the cases were identified by reviewing operative notes, admission lists, and neuropathological reports at the Central Anatomic Laboratory in Baltimore for the years 1950 through 1972. Patients in the following categories were excluded from the study: (1) those who were pronounced dead on arrival at the emergency room, (2) those with a simple cervical sprain, and (3) those with inadequate records. The data gathered from each patient's chart were analyzed by computer. Follow-up information was obtained from the records except for some of the patients treated between 1966 and 1972, who were re-examined. All patients were treated by orthopaedic or neurosurgical services or, for those with more complicated lesions, by both.

The patients' ages ranged from birth (two patients) to eighty-seven years; the average age was forty-seven years. More than half (161 patients) were twenty-one to fifty years old. Of the 300 patients, 180 were paralyzed and 120 were not. Of the 180 paralyzed patients, forty-eight died, nineteen survived but were not followed sufficiently to determine the results of treatment, and 113 survived and had adequate follow-up for at least one year: twenty-seven for one to two years and eighty-six for two to sixteen years (average follow-up, five years). Of the 120 patients without paralysis, three died, fourteen survived but were not followed, and 103 survived and were followed adequately for six months or more: thirty-eight for six months to two years and sixty-five for two to fourteen years (average follow-up, three years and eight months). Of the seventy-four patients who died, fifty-three died within four months of injury and twenty-one died at some later date of unrelated causes.

Approximately one-third of the injuries were due to a motor-vehicle accident; one-third, to a fall; and the remaining one-third, to an athletic injury or a wound inflicted by a missile. The levels involved were the atlanto-occipital joint in two patients, the atlanto-axial articulation in sixty-nine, and between the third and seventh cervical vertebrae in 221. The other eight patients had no fracture visible on roentgenograms despite the presence of cervical paralysis at a specific level. Other authors also have described cases of spinal cord injury in which there was no roentgenographic evidence of fracture or dislocation^{26,77,114}.

Of the 300 patients in this series, 200 were diagnosed initially in an emergency room and 100 were not. The delay in diagnosis ranged from two days to six months in ninety patients, and from six to eighteen months in ten.

The causes for the delay in diagnosis (present singly or in combination) included brain injury with a decreased level of consciousness in twenty-five patients, head injury without loss of consciousness in thirty-three, alcohol intoxication in fourteen, and associated fractures or multiple



FIG. 1

A fourteen-year-old boy was comatose after he was struck by an automobile and received injuries to the head and neck. The atlanto-occipital dislocation was not discovered until an arteriogram was made. He subsequently died of respiratory arrest. This sagittal section of a specimen taken from the cervical spine, leaving the foramen magnum intact, shows complete transection of the spinal cord at the first cervical vertebra (arrow) with complete disruption of all ligaments between the foramen magnum and the atlanto-axial complex. (Courtesy of James Milgram, M.D., Department of Pathology, Johns Hopkins Hospital, Baltimore, Maryland.)

injuries in thirty-three. Three patients with hemiparesis were either admitted or discharged with the erroneous diagnosis of stroke. The injuries that were not diagnosed involved the occiput, atlas, and axis in thirty patients; the third and fourth cervical vertebrae in twenty-seven; and the fifth, sixth, and seventh cervical vertebrae in forty-three.

For purposes of analysis, the 300 patients were divided into four groups: the first was composed of two patients with an atlanto-occipital lesion; the second, of sixty-nine patients with an atlanto-axial defect; the third, of 221 patients with lesions between the third and seventh cervical vertebrae; and the fourth, of eight patients with ankylosing spondylitis. Each of these groups was subdivided into two subgroups, the patients with and those without neural deficit, and each subgroup was analyzed with respect to diagnosis, treatment, results, complications, and pathological changes to determine the most accurate means of diagnosis and the best treatment based on

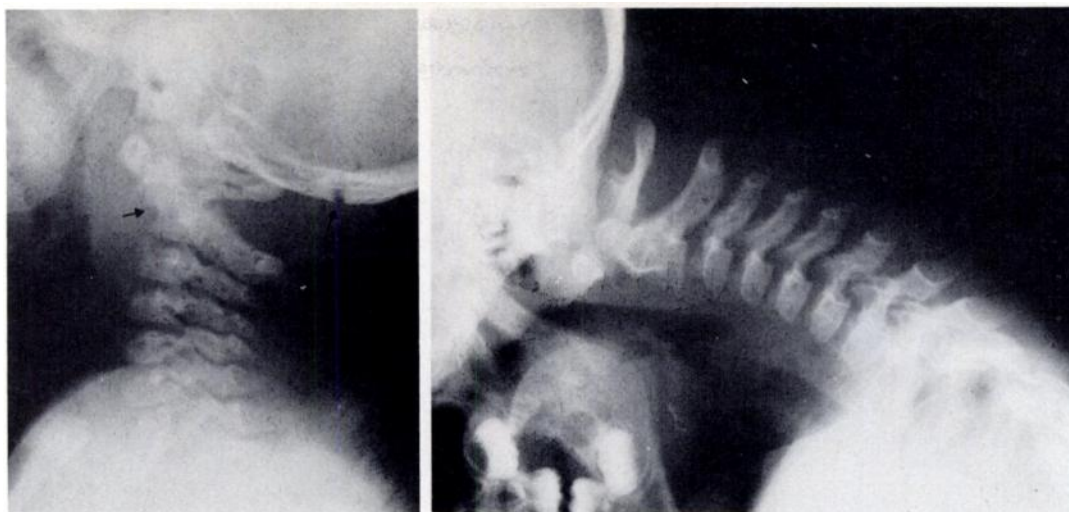


FIG. 2-A

FIG. 2-B

Fig. 2-A: An epiphyseal fracture of the odontoid process and a fracture of the occiput are seen in this two and one-half-year-old child whose level of consciousness was decreased. The diagnosis was not determined for two days.

Fig. 2-B: A flexion roentgenogram made six months after injury shows that the fractures have healed.

the recovery of spinal and neural function. Available pathological specimens were analyzed and correlated with the neurological syndromes seen clinically in each group.

Atlanto-Occipital Lesions

A previous study of fatal craniocervical injuries showed that in these patients the occipitocervical junction was commonly affected³⁶. However, cases of atlanto-occipital dislocation with survival have been recorded by several authors^{11,43,44,106}.

In the present series, two patients had an anterior atlanto-occipital dislocation associated with brain injury and were in coma at admission. One was a pedestrian who was struck by an automobile; the other was struck by a falling object. Roentgenograms in both patients showed that the skull was separated from the axis and displaced anteriorly. Both patients were treated with very light skeletal traction and died of the spinal cord injury within forty-eight hours of admission. At autopsy, both patients were found to have transection of the spinal cord at the atlanto-occipital level and total disruption of all ligaments connecting the occiput to the atlas and axis (Fig. 1).

Atlanto-Axial Lesions

Sixty-nine patients had a lesion involving the atlas and axis, fifty-eight without and eleven with a neural deficit. The types of fractures and dislocations of the atlanto-axial region as well as the indications for stabilization have been described previously^{13,14,28,35,42,46,58,68,69,96,105,107,130}. Considering all 300 patients in the present series, there were only fifteen fractures in patients younger than fifteen years old and of these fifteen fractures, eleven were at or above and four were below the third cervical level.

Diagnosis

Of the fifty-eight patients who had an atlanto-axial

lesion with no neural deficit, ten had a fracture of the atlas; three, an atlanto-axial dislocation without fracture; twenty-one, a fracture involving the pedicles of the axis; three, a fracture of the body of the axis; and twenty-one, a fracture of the odontoid process (seven without and fourteen with atlanto-axial dislocation). Two of the twenty-one fractures of the odontoid process were epiphyseal fractures in young children and were not associated with dislocation of the atlanto-axial joint (Figs. 2-A and 2-B).

Of the eleven patients who had atlanto-axial involvement and an associated spinal-cord injury, nine had a partial cord lesion, as described by Stratford¹¹¹ and by Wadia¹²³, and two were comatose on admission and died. Of the skeletal lesions in these eleven patients, five were atlanto-axial dislocations without fracture in patients with a dysplastic odontoid process (three in otherwise normal adults and two in children who had Morquio's disease [Fig. 3]); five were odontoid fractures (four with and one without dislocation of the atlas); and one was a fracture of the posterior arch of the atlas with associated quadriplegia secondary to a gunshot wound.

Treatment and Results in Patients without Neural Deficit

The fifty-eight patients who had an atlanto-axial lesion without paralysis had the following treatment and results. Ten patients had a fracture of the atlas that healed without operation but two of them, both with a fracture involving the articular surfaces of the lateral masses, had neck pain at follow-up.

Seven patients had a non-displaced fracture through the odontoid process extending into the body of the axis, which healed after treatment with a halo cast, Minerva jacket, or traction with Crutchfield tongs.

Fourteen patients had a displaced fracture of the odontoid process combined with atlanto-axial displacement. Of these fourteen, one died of a pulmonary embolus two months after injury; four with a fracture through the

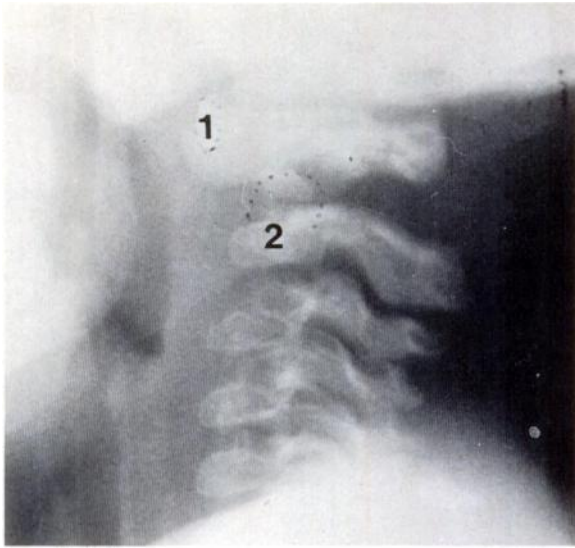


FIG. 3

A five-year-old child with Morquio's disease and a dysplastic odontoid process became quadriparetic after a minor injury. Note the increased distance between the anterior arch of the atlas (1) and the odontoid process (2).

waist of the odontoid process had a successful posterior atlanto-axial fusion; two had a successful result after an occipitocervical fusion (one of these two required a second fusion as described by Lipscomb⁷³); and seven were treated non-operatively and had satisfactory results.

Three patients had an atlanto-axial dislocation without fracture. Of these three, two were treated successfully with posterior atlanto-axial fusion. One with unilateral rotatory dislocation refused operation, and at follow-up he reported recurring syncopal episodes.

Twenty-one patients had a fracture of the pedicles of the second cervical vertebra, all of which healed after closed reduction and immobilization (Brashear and associates²³ reported similar results in their series). However, one of these twenty-one patients had a painful anterior subluxation of the second on the third cervical vertebra, which was successfully treated with anterior fusion.

The three remaining patients had a fracture of the body of the axis that healed without surgical treatment, a result similar to those described by Schatzker and associates⁹⁶.

Treatment and Results in Patients with Neural Deficit

Of the eleven patients with atlanto-axial injury and a neural deficit, two were comatose on admission and died, five had severe quadriparetic, and four had mild paralysis. Of the five with severe quadriparetic, two had a chronic dislocation and were treated with laminectomy of the atlas and occipitocervical fusion; two with atlanto-axial dislocation were treated with reduction and atlanto-axial fusion without decompression; and the fifth patient was treated non-operatively for a fracture of the odontoid process. The four fusions healed, and all five patients with quadriparetic had complete recovery. Of the four patients with mild paralysis, two had a Brown-Séquard syndrome and two, a

transient quadriparetic. The treatment for these four patients was occipitocervical fusion in one, atlanto-axial fusion in another, and a rigid cervical brace and Minerva jacket in one each. The two fusions healed; one of the other two atlanto-axial joints was stable while one remained unstable and dislocated. The two patients who were treated by fusion and one of the two treated without fusion recovered completely. The remaining patient, with a Brown-Séquard syndrome and a chronic atlanto-axial dislocation, had a mild hemiparesis, and he refused operation.

Complications

Sixteen of the sixty-nine patients with atlanto-axial dislocation had non-operative treatment, and of these sixteen, nine required arthrodesis for late instability. Of the fifty-three patients who were operated on shortly after injury, three had respiratory arrest during an occipitocervical fusion done two to four weeks after injury (one as a dural elevator was passed under the posterior arch of the atlas and the other two as they were being intubated and turned). No patient who had a halo cast applied preoperatively had this complication, a finding consistent with the experience of others^{16,114}. The four remaining patients with complications in this group died: one from an infected occipitocervical fusion, two from multiple head and other injuries, and one from a pulmonary embolus.

Pathological Findings

A lateral roentgenogram showing a normal odontoid process and a space more than four millimeters wide between the odontoid process and the anterior arch of the



FIG. 4-A

Figs. 4-A, 4-B, and 4-C: This five-year-old child was admitted to the emergency room in coma with circulatory collapse after he was struck by an automobile.

Fig. 4-A: The facial abrasions shown by this postmortem photograph suggest the presence of a cervical injury.

atlas implies rupture of the transverse ligament, as described by Fielding and co-workers⁴⁶. Three patients in this group had such an injury. Similar displacement may occur in otherwise normal adults as well as in dwarfs with ligament laxity associated with dysplasia of the odontoid process, and may be demonstrated by flexion-extension roentgenograms.

Autopsy was performed on two patients with an atlanto-axial lesion. In one, who had a fractured odontoid process and an atlanto-axial dislocation without neural deficit, there were parenchymal hemorrhages in the spinal cord. In the other, who had an atlanto-axial dislocation that was discovered thirty-six hours after burr holes were made and a laparotomy was performed, autopsy revealed a tear of the transverse ligament, separation of the atlanto-axial joint, and complete transection of the cervical cord (Figs. 4-A, 4-B, and 4-C).

spinous process; and four, one or more fractured transverse processes⁴¹.

The 167 patients with a neural deficit had 250 fractures, subluxations, or dislocations that also occurred singly or in combination at one level between the third and seventh cervical vertebrae. These spine lesions included 105 fractures, subluxations, or dislocations of one or both articular processes, eighty-five fractures of a vertebral body, five fractures of pedicles, twenty-one fractures of one or more spinous processes, and thirty-four fractures of a lamina.

Excluding the four patients who were comatose on admission and were quadriparetic, and the seven with spondylitis and a neural deficit (five with anterior cord syndrome and two with a complete cord lesion [Table III]), there were twenty-nine patients with a nerve-root lesion, fifty-five with a complete cord lesion (Table II), and

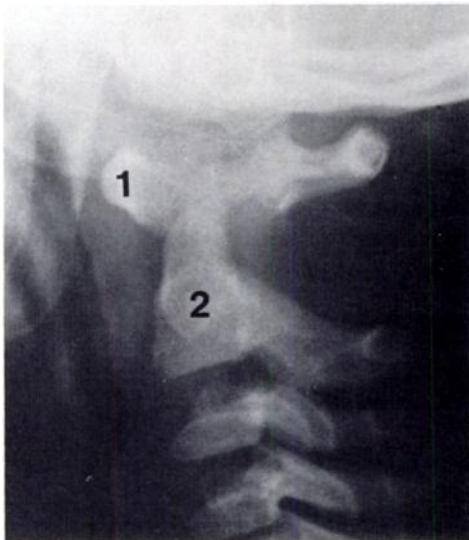


FIG. 4-B



FIG. 4-C

Fig. 4-B: This roentgenogram was made while the patient was in traction thirty-six hours after injury, after laparotomy had been performed and burr holes were made, when the nurse in the recovery room felt crepitus in the neck. Note the distraction of the occiput, atlas (1), and axis (2).

Fig. 4-C: A postmortem sagittal section of the cervical spine and odontoid process shows complete transection of the cord at the level of the odontoid process. Note the separation of the atlas (1) and axis (2), and local hemorrhage.

Lesions Between the Third and Seventh Cervical Vertebrae

Including the eight patients with ankylosing spondylitis, there were 229 patients with spinal lesions between the third and seventh cervical vertebrae: sixty-two without and 167 with a neural deficit.

Diagnosis

The sixty-two patients with no paralysis had 127 fractures or subluxations between the third and seventh cervical vertebrae that occurred singly or in combination at one level in each patient. Fifty-three had a subluxation (either unilateral or bilateral) or a fracture of one or more articular processes; thirty-four had a crushed vertebral body with or without displacement of fragments; nineteen, a fracture of a lamina; one, a pedicle fracture; sixteen, a fracture of a

seventy-one with an incomplete cord lesion. Included in the group with an incomplete cord lesion were four patients with an upper motor neuron lesion with one upper extremity paralyzed, six with a Brown-Séquard syndrome, fifteen with a central cord syndrome, and forty-six with an anterior cord syndrome. However, only thirty-three of the forty-six patients with an anterior cord syndrome were analyzed with respect to treatment (Table I), for reasons to be described later. Since many patients had more than one skeletal lesion (subluxation, dislocation, or fracture), it was difficult to relate a specific skeletal lesion to the neural deficit. For purposes of this analysis, the skeletal lesions were divided into five groups: lesions of an articular process, fractures of a vertebral body, fractures of a pedicle, fractures of a lamina, and fractures of a spinous process. Each group was analyzed with respect to the neural lesions

TABLE I
 ANTERIOR CORD SYNDROMES*: TYPES OF TREATMENT AND RESULTS IN THIRTY-THREE PATIENTS

Case	Osteoarticular Lesion			Lesion of the Cord		Results of Treatment	Length of Follow-up (Yrs. + Mos.)
	Vertebral Body and Inter-vertebral Disc	Articular Processes	Other Fractures	Level	Severity†		
No operation							
1	Compress. fract., C5	Intact		C4	+++	Slight recovery in lower limbs	1 + 0
2	Compress. fract., C5	Fract., C4		C4	++++	Slight recovery in arms; severe spasticity	3 + 2
3	Cervical spondylosis	Intact	Lamina, C5	C5	++++	Slight recovery in arms	1 + 0
4	Intact	Bilat. sublux., C5-C6		C5	+	Complete recovery	7 + 0
5	Cervical spondylosis	Bilat. sublux., C3-C4		C3	+++	Died 1 day after injury — cord damage	
6	Compress. fract., C5	Intact	Spinous process, C5	C5	++	Died 9 days after injury — pulmonary embolus	
7	Intact; birth injury	Intact		C6	++	Died 2 mos. after injury — cause unknown	
8	Cervical spondylosis	Intact		C4	++	Slight recovery in arms; died 9 mos. after injury — sepsis	
9	Cervical spondylosis	Bilat. sublux., C3-C4		C3	+	Complete recovery	2 + 0
Laminectomy							
10	Cervical spondylosis	Unilat. sublux., C4-C5		C4	+	Lost motor function	14 + 0
11	Cervical spondylosis	Bilat. sublux., C6-C7		C6	++	Lost motor function	2 + 9
12	Cervical spondylosis	Intact		C6	+	Lost motor function	6 + 0
13	Cervical spondylosis	Intact		C6	+	Slight recovery in arms	4 + 7
14	Cervical spondylosis	Intact	Lamina, C6	C6	++	Died 10 days after injury	
15	Cervical spondylosis	Intact		C6	++++	Died 2 mos. after injury	
Laminectomy and posterior fusion							
16	Herniated disc, C5-C6; cervical spondylosis			C5	+	Unchanged	3 + 0
17	Compress. fract., C6	Bilat. disloc., C5-C6	Lamina, C6	C5	++++	Steroids; gastrointest. hemorrhage; slight recovery in arms	3 + 0
18	Compress. fract., C7	Unilat. disloc.; fract., C6		C6	+	Steroids; complete recovery	1 + 4
19	Compress. fract., C7	Unilat. sublux., C6-C7	Lamina, C7	C6	+++	Lost motor function	2 + 9
20	Intact	Unilat. disloc.		C6	++	Steroids; died 5 days after injury — cord damage	
21	Compress. fract.; herniated disc	Bilat. sublux., C4-C5		C4	+	Died six days after injury — pulmonary embolus	
22	Compress. fract., C7	Bilat. sublux., C6-C7	Lamina, C7	C6	++++	Unchanged	10 + 0
23	Intact	Bilat. disloc., C5-C6		C5	+	Steroids; lost motor function	4 + 6
24	Cervical spondylosis	Bilat. sublux.; fract., C5-C6		C5	+	Steroids; died 19 days after injury — pulmonary embolus	
Laminectomy, posterior fusion, and anterior fusion							
25	Intact	Unilat. disloc., C5-C6	Lamina, C6	C5	+++	Complete recovery	8 + 0
26	Compress. fract., C7	Bilat. disloc., C6-C7		C6	++	Near-complete recovery	9 + 0
Posterior fusion							
27	Compress. fract., C6	Intact	Pedicle, C6	C5	+++	Slight recovery in arms	7 + 0
28	Intact	Bilat. disloc., C5-C6		C5	++++	Steroids; complete recovery	4 + 0
Anterior discectomy and fusion							
29	Compress. fract., C6	Intact		C5	++	Slight recovery in arms	9 + 0
Anterior and posterior fusion							
30	Compress. fract., C6	Bilat. sublux., C5-C6	Lamina, C6	C5	++	Steroids; slight recovery in arms; able to move toes	4 + 0
Anterior vertebral-body excision and fusion							
31	Compress. fract., C4	Intact		C3	+++	Steroids; gastrointest. hemorrhage; complete recovery	2 + 6
32	Compress. fract., C6	Bilat. sublux., C5-C6		C5	++++	Steroids; gastrointest. hemorrhage; slight recovery in arms	1 + 0
33	Compress. fract., C7	Intact	Lamina, C7	C6	+	Complete recovery	3 + 5

* Anterior cord syndrome is characterized by quadriplegia or paraplegia and hypalgesia below the level of the lesion in the presence of varying degrees of preservation of touch, position, motion, and vibration sense. The motor paralysis is more severe in the lower than it is in the upper extremities.

† + = can move limbs and feet, and good sparing of position and vibratory sense; ++ = slight distal motor function (toe wiggle), and good position and vibration sense; +++ = no distal motor function, touch sensation present, and good position and vibratory sense; and ++++ = no distal motor function, touch sensation present, and little or no position and vibratory sense.

associated with it. However, in patients with multiple lesions it was not always possible to determine which

skeletal defect or defects were responsible for the cord or root lesion.

Lesions of the articular processes: The 105 patients with lesions of the articular processes between the third and seventh cervical vertebrae included twenty with nerve-root involvement, forty-one with a partial cord lesion, and forty-four with complete transection of the cord. Of the twenty patients with nerve-root involvement, ten had subluxation; four, unilateral dislocation; and six, fracture of an articular process with subluxation of the process into the adjacent intervertebral foramen. Of the forty-one patients with a partial cord lesion and injury of one or more articular processes, four had unilateral and twelve, bilateral subluxation; four had unilateral and seven, bilateral dislocation; and fourteen had a fracture (seven of these fractures being associated with unilateral or bilateral subluxation and four with unilateral or bilateral dislocation of the articular processes). Of the forty-four patients with complete transection of the cord and injury to an articular process, twenty had a subluxation (bilateral in seventeen and unilateral in three), twenty had a dislocation (bilateral in fourteen and unilateral in six), and four had a fracture.

Fractures of a vertebral body: The eighty-five patients who had a fracture of a vertebral body between the third and seventh cervical segments and had associated paralysis included twelve with nerve-root paralysis caused by posteriorly displaced fragments of a crushed vertebral body, thirty-three with a partial cord lesion caused by a bulging, compressed vertebral body, and forty with a complete cord injury associated with gross posterior displacement of a fragment of a vertebral body. Twenty of these eighty-five patients also had a lesion of an articular process.

Pedicle fractures: The five patients who had fracture of one or both pedicles below the level of the axis and also had paralysis (an infrequent lesion) included: one with a displaced fracture of one pedicle and nerve-root paralysis; three with a fracture (two of them displaced) of both pedicles and a partial cord lesion; and one with a displaced fracture of one pedicle and a total cord lesion.

Laminar fractures: The thirty-four patients with a laminar fracture between the third and seventh cervical levels as well as paralysis included three with a nerve-root lesion and thirty-one with a lesion of the cord (fourteen with partial and seventeen with complete transection). In all thirty-four patients there was associated fracture of a vertebral body and of one or more articular processes. None of the laminar fragments were displaced into the spinal canal, and in no patient was the cord compression caused by the laminar injury in itself.

Fractures of a spinous process: In none of the twenty-one patients with a fracture of a spinous process and paralysis did the fracture of the spinous process itself cause compression of the neural structures.

Thus, the neural deficits were associated predominantly with a displaced fracture of an articular process, vertebral body, or pedicle; the degree of osseous displacement was related to the severity of the neural deficit. None of the 105 patients with a lesion of an articular pro-

cess and nerve-root involvement had bilateral displacement, while 46 per cent of the incomplete and 70 per cent of the complete cord lesions were associated with bilateral subluxation or dislocation.

Of the eighty-five patients with fracture of a vertebral body and paralysis, seventy-three with a lesion of the cord had significant protrusion of the vertebral fracture into the spinal canal. Similarly, three of the five pedicle fractures with a neural deficit were displaced.

Although fractures of the lamina and spinous process were associated with neural deficit, none were found to be directly compressing any neural structure.

Myelography

During the acute phase, a myelogram was made for sixty-eight patients who had a neural deficit between the third and seventh cervical segments. These sixty-eight patients included: forty with a partial block and an anterior defect, indicating compression of the anterior aspect of the cord; twenty-two with a total block caused by edema of the cord, massive displacement of bone, or hemorrhage into the spinal canal; and six with a normal myelogram. A small-to-moderate amount of blood was present in the spinal fluid in forty-three of these sixty-eight patients, but this was not considered a contraindication to myelography, and no ill effects were noted³⁰.

Treatment and Results in Patients without Neural Deficit

Of the sixty-two patients without a neural deficit, twenty-three were treated non-operatively with skeletal traction, a halo cast, or a brace, and thirty-nine were treated by fusion. Of the twenty-three patients treated non-operatively, seventeen were asymptomatic after an average follow-up of three years and eight months (range, six months to fourteen years) and six were lost to follow-up.

Thirty-nine of the sixty-two patients had an anterior or posterior fusion, or both. The indications for posterior fusion were: (1) failure to obtain a closed reduction with skeletal traction, (2) a major tear of the posterior ligaments, and (3) late instability and pain. The indications for anterior fusion included: (1) compression or bursting fracture of a vertebral body producing angulation or potential instability, (2) pain secondary to a disrupted disc, and (3) laxity of the longitudinal ligaments with resultant vertebral subluxation.

Of the thirty-nine patients treated by fusion, thirty-two with torn posterior ligaments and subluxation or dislocation were treated with posterior fusion and seven, seen after 1954, were treated with Robinson anterior discectomy and fusion (two of them having a posterior fusion as well).

Three of the thirty-nine patients who had fusion were lost to follow-up. The remaining thirty-six had a solid fusion, but one of them still complained of neck pain (believed to be psychogenic in origin) six years after fusion.

TABLE II
TOTAL CORD LESIONS*: TYPES OF TREATMENT AND RESULTS IN FIFTY-THREE PATIENTS

Case	Osteoarticular Lesion			Level of Cord Lesion	Results of Treatment	Length of Follow-up (Yrs. + Mos.)
	Vertebral Body and Inter-vertebral Disc	Articular Processes	Other Fractures			
No operation						
34	Intact	Bilat. sublux., C6-C7		C7	Died — head injury and pulmonary embolus	
35	Compress. fract., C6	Bilat. sublux. and fract., C6		C5	Died 12 days after injury — ascending cord necrosis	
36	Compress. fract., C6	Bilat. disloc., C5-C6		C5	Died 1 day after injury	
37	Compress. fract., C6	Bilat. sublux., C5-C6	Spinous process and lamina, C6	C5	Died 1 wk. after injury — acute hemorrhagic pulmonary edema	
38	Compress. fract., C5	Bilat. sublux., C4-C5	Spinous process and lamina, C5	C4	Died — multiple trauma	
39	Compress. fract., C4	Bilat. sublux., C3-C4		C3	Died — cord injury; bronchopneumonia	
40	Birth injury (spine intact)			C7	Died — cord damage	
41	Intact	Bilat. disloc., C3-C4	Spinous process, C4	C3	Slight recovery in arms	5 + 7
42	Compress. fract., C7	Bilat. disloc., C6-C7	Spinous process, C5	C6	Slight recovery in arms	3 + 0
43	Intact	Bilat. disloc., C4-C5		C4	Died 5 days after injury — ascending cord necrosis	
44	Compress. fract., C5	Intact		C4	Steroids; gastrointest. hemorrhage; died 6 days after injury	
Laminectomy						
45	Compress. fract., C6	Unilat. sublux., C5-C6	Lamina, C6	C5	Unchanged	5 + 0
46	Compress. fract., C6	Fract., C6	Lamina, C6	C5	Slight recovery in arms	10 + 5
47	Compress. fract., C7	Intact		C6	Unchanged	16 + 0
48	Compress. fract., C6	Bilat. sublux., C5-C6	Spinous process and lamina, C6	C5	Unchanged	1 + 0
49	Intact; gunshot wound, C6			C6	Steroids; unchanged	6 + 10
50	Compress. fract., C6	Bilat. sublux., C5-C6		C5	Gastrointest. hemorrhage; lost motor function in arms	8 + 0
51	Compress. fract., C5	Bilat. sublux., C4-C5		C4	Died 5 days after injury	
52	Intact	Unilat. disloc., C4-C5	Lamina, C5	C4	Died 1 day after injury	
53	Compress. fract., C6	Bilat. sublux., C5-C6	Lamina, C6	C5	Died 1 wk. after injury — gastrointest. hemorrhage; steroids	
54	Compress. fract., C7	Fract., C6	Lamina, C7	C6	Died 16 days after injury — gastrointest. hemorrhage; steroids	
55	Intact	Bilat. disloc., C6-C7		C6	Lost motor function in arms	1 + 0
56	Compress. fract., C6	Intact	Lamina, C6	C5	Lost motor function in arms	10 + 7
Laminectomy and posterior fusion						
57	Compress. fract., C6	Unilat. sublux., C5-C6		C5	Unchanged	10 + 0
58	Compress. fract., C7	Bilat. sublux., C6-C7	Lamina, C7	C6	Unchanged; steroids; gastrointest. hemorrhage	5 + 0
59	Compress. fract., C6	Unilat. disloc., C5-C6	Spinous process and lamina, C6	C5	Unchanged; steroids; gastrointest. hemorrhage	6 + 0
60	Intact	Bilat. sublux., C4-C5; fract., C5		C4	Unchanged; gastrointest. hemorrhage	6 + 9
61	Compress. fract., C6	Bilat. disloc., C5-C6		C5	Slight recovery in arms; gastrointest. hemorrhage	1 + 6
62	Intact	Bilat. sublux., C6-C7		C6	Lost motor function in arms	2 + 7
63	Compress. fract., C6	Bilat. disloc., C5-C6	Lamina, C6	C5	Unchanged	4 + 0
64	Compress. fract., C6			C5	Died 6 days after injury — gastrointest. hemorrhage	
65	Compress. fract., C6	Bilat. disloc., C5-C6	Lamina, C6	C5	Died 6 wks. after injury — gastrointest. hemorrhage; steroids	
66	Compress. fract., C6	Bilat. sublux., C5-C6		C5	Died 10 days after injury — gastrointest. hemorrhage; steroids	
67	Compress. fract., C5	Unilat. disloc., C4-C5	Lamina, C5	C4	Died 5 days after injury — gastrointest. hemorrhage; steroids	
68	Compress. fract., C5; cervical spondylosis			C4	Died 7 days after injury; steroids	
69	Compress. fract., C7			C6	Unchanged	1 + 4
70	Compress. fract., C6		Spinous process, C6	C5	Died 2 mos. after injury — pneumonia	
71	Compress. fract., C7	Unilat. disloc., C6-C7		C6	Unchanged	3 + 6
72	Compress. fract., C5		Spinous process and lamina, C5	C4	Unchanged	2 + 6

TABLE II (continued)
TOTAL CORD LESIONS*: TYPES OF TREATMENT AND RESULTS IN FIFTY-THREE PATIENTS

Case	Osteoarticular Lesion			Level of Cord Lesion	Results of Treatment	Length of Follow-up (Yrs. + Mos.)
	Vertebral Body and Inter-vertebral Disc	Articular Processes	Other Fractures			
Laminectomy, posterior fusion, and anterior fusion						
73	Compress. fract., C5	Bilat. sublux., C4-C5	Lamina, C5	C4	Unchanged	5 + 2
74	Intact	Unilat. disloc., C4-C5		C4	Unchanged	3 + 6
75	Compress. fract., C6		Lamina, C6	C5	Unchanged	5 + 8
76	Compress. fract., C7		Spinous process, C7	C6	Died 1 mo. after injury — gastrointest. hemorrhage	
Open reduction and posterior fusion						
77	Compress. fract., C7	Bilat. sublux., C6-C7		C6	Slight recovery in arms	18 + 0
78	Compress. fract., C7	Unilat. disloc., C6-C7		C6	Died 8 days after injury; steroids	
79	Intact	Bilat. sublux., C6-C7		C6	Slight recovery in arms	1 + 8
80	Intact	Bilat. disloc., C4-C5		C4	Slight recovery in arms	4 + 4
81	Intact	Bilat. disloc., C6-C7	Spinous process and lamina, C7	C6	Slight recovery in arms; steroids	2 + 0
Anterior discectomy and fusion						
82	Gunshot wound	C6	Spinous process, C6	C6	Slight recovery in arms	2 + 8
Anterior vertebral-body excision and fusion						
83	Compress. fract., C6	Bilat. sublux., C5-C6		C5	Unchanged; gastrointest. hemorrhage; steroids	5 + 7
84	Gunshot wound	C7		C6	Unchanged	5 + 0
85	Compress. fract., C7	Bilat. sublux., C6-C7		C6	Died 15 days after injury — sepsis, gastrointest. hemorrhage; steroids	
Anterior and posterior fusion						
86	Compress. fract., C6	Bilat. disloc., C5-C6	Spinous process, C6	C5	Unchanged	2 + 2

* Total cord syndrome is characterized by quadriplegia with complete loss of all motor and sensory functions below the level of injury, still present more than forty-eight hours after injury.

Treatment and Results in Patients with Neural Deficit

Of the 167 patients with a neural deficit, twenty-nine had a nerve-root lesion and 138 had a lesion of the cord. Of the 138 lesions of the cord, seventy-seven, including six (Cases 87, 90A, 90B, 91, 93, and 94) (Table III) in patients with spondylitis, caused incomplete transection; fifty-seven, including two (Cases 89 and 92) (Table III) in patients with spondylitis, caused complete transection; and four were in patients who were in coma and quadriparetic at the time of admission.

Nerve-Root Paralysis

The twenty-nine patients with nerve-root paralysis all had a lesion between the fifth and seventh cervical vertebrae. Five of these patients were lost to follow-up. In one of these five, signs of cord involvement developed after laminectomy and the patient was unable to walk when last seen, six months after laminectomy. Of the remaining twenty-four patients, nine were treated non-operatively and fifteen had surgical treatment. The nine who were treated non-operatively improved: three slightly and six completely. Of the fifteen who had surgical treatment, one patient with a fracture fragment compressing a nerve root had a foraminotomy and recovered partially; ten with dislocation had open reduction and posterior fusion, and they had complete motor and sensory recovery; and four with a herniated disc compressing a nerve root had anterior disc-

ectomy and fusion (one also having a posterior fusion), and all four had complete recovery of neural function.

Incomplete Cord Lesions

The seventy-six patients with seventy-seven incomplete cord lesions included four who had an upper motor neuron lesion with paralysis of one upper extremity¹²¹, six who had a Brown-Séquard syndrome, fifteen who had a central cord syndrome, and forty-six who had an anterior cord syndrome: a total of seventy-one patients. The remaining patients were five of the eight patients with ankylosing spondylitis who had an anterior cord syndrome. One of them had this syndrome on two occasions because of a second injury. These five patients (one with two separate lesions) brought the total number of incomplete cord lesions to seventy-seven, but, as previously indicated, these five patients were analyzed separately in the ankylosing spondylitis group (Table III).

The four patients with an upper motor neuron lesion of one upper extremity had a lesion located between the fifth and seventh cervical levels. Three recovered completely after open reduction of the dislocation and posterior fusion and one, referred for treatment five months after a compression fracture of the fifth cervical vertebra with resultant kyphosis, had complete recovery after removal of two discs and part of the body of the fifth cervical vertebra followed by fusion to correct the kyphosis (Figs. 5-A and 5-B).

TABLE III
ANKYLOSING SPONDYLITIS: TYPES OF TREATMENT AND RESULTS IN EIGHT PATIENTS

Case	Osteoarticular Lesion		Lesion of the Cord		Treatment and Results	Pathological Findings at Autopsy or Operation	Length of Follow-up (Yrs. + Mos.)
	Vertebral Body and Intervertebral Disc	Articular Processes	Neurological Syndrome	Level			
No operation							
87	Fract., disc	Bilat. disloc., C3-C4	Anterior cord syndrome (+++)*	C3	Skel. traction; died 1 day after injury	Large epidural hemorrhage	
88	Fract., disc	Bilat. disloc., C6-C7	None		Skel. traction, Minerva jacket; complete recovery		6 + 11
89	Fract., disc	Bilat. sublux., C6-C7	Complete quadriplegia	C6	Skel. traction, steroids; died 1 day after injury — resp. arrest while being turned	Bronchopneumonia; large epidural hemorrhage; necrosis of cord, C7	
90A†	Fract., disc	Bilat. sublux., C5-C6	Anterior cord syndrome (+)*	C5	Minerva jacket; complete recovery		7 + 0
Laminectomy							
90B†	Lytic lesion, disc space	2nd bilat. sublux., C6-C7, 7 yrs. later	Anterior cord syndrome (++)*	C6	Skel. traction; loss of motor function; barely able to walk	Scar tissue and brown pigmentation	10 + 0
91	Fract., disc	Bilat. disloc., C7-T1	Anterior cord syndrome (+)*	C7	Disloc. after laminectomy; skel. traction; loss of motor function, then completely recovered	Large epidural hemorrhage	4 + 0
92	Oblique fract., C3	Bilat. disloc., C3-C4	Complete quadriplegia	C3	Skel. traction; died 3 days after injury	Small epidural hemorrhage; central necrosis of entire cord, C1-C7	
93	Fract., disc	Bilat. disloc., C7-T1	Anterior cord syndrome (++++)*	C7	Skel. traction, steroids; died 18 days after injury — gastrointest. hemorrhage	Large epidural hemorrhage	
94	Fract., disc	Bilat. sublux., C2-C3	Anterior cord syndrome (+)*	C3	Skel. traction, steroids; died 3 mos. after injury — gastrointest. hemorrhage	No autopsy performed	

* See Table I.

† This patient had a third fracture, at the seventh cervical and first thoracic levels, in 1974 (after the completion of this study). It was treated with a rigid brace and it healed uneventfully. There was no change in neural function after this injury.

The six patients with a Brown-Séquard syndrome had a fracture or dislocation involving the sixth or seventh cervical segment. Two were treated non-operatively and recovered, but one of them had had only a concussion of the cord with transient paralysis. Two others were treated with open reduction and posterior fusion and they recovered completely, while the remaining two had laminectomy and posterior fusion and were lost to follow-up.

The fifteen patients with a central cord syndrome had a lesion of the spine in the following locations: six involved the third and fourth cervical vertebrae; four, the fourth and fifth; and five, the fifth, sixth, and seventh cervical levels. Eight of these fifteen patients were treated non-operatively and of these eight, four became ambulatory, two had only slight recovery, and two died during the acute phase. The other seven patients were treated surgically, four with and three without laminectomy. Of the four with laminectomy, two had laminectomy alone; one, laminectomy combined with posterior fusion; and one, laminectomy combined with posterior fusion and sub-

sequently a Robinson anterior discectomy and fusion. Three of the four patients who had a laminectomy died early in the postoperative period (two having lost motor function prior to death). The fourth patient, who had a herniated disc, recovered completely after anterior discectomy and fusion was done after laminectomy failed to decompress the cord (Figs. 6-A and 6-B). Of the three patients who were treated surgically without laminectomy, one had an open reduction of the dislocation with a posterior fusion; one, an anterior discectomy and fusion; and one had both anterior and posterior fusion. All three recovered completely.

In the forty-six patients with an anterior cord syndrome, the spinal lesions were distributed as follows: thirteen were between the third and fifth cervical vertebrae; sixteen, at the sixth cervical level; and seventeen, at the seventh cervical level. The cases of thirteen of these forty-six patients were not analyzed with respect to treatment because they had only concussion of the cord with transient quadriplegia for one to three days, they were lost

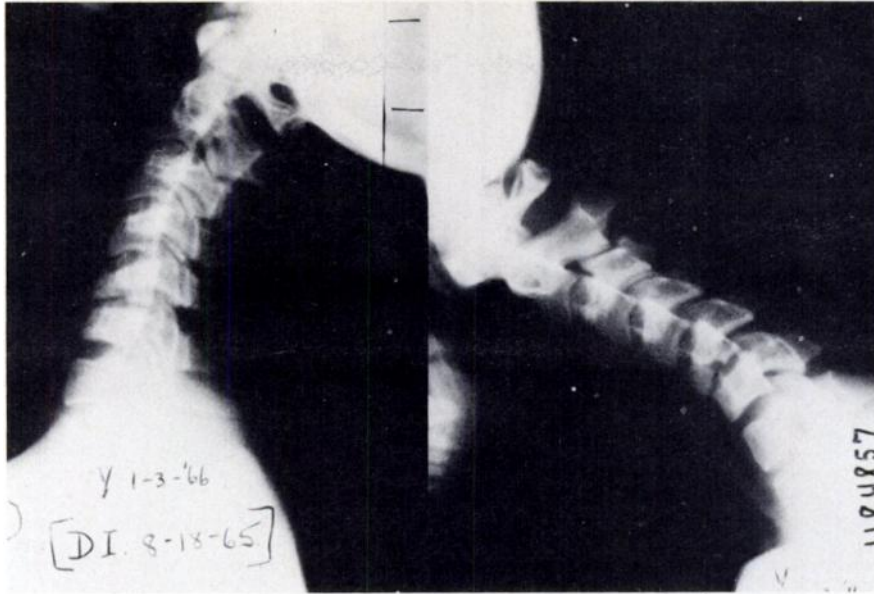


FIG. 5-A

Figs. 5-A and 5-B: An eighteen-year-old boy with a crush fracture of the fifth cervical vertebra due to an automobile accident still had a left upper monoparesis and neck pain five months later.

Fig. 5-A: Flexion-extension myelograms made five months after injury show kyphotic deformity and subluxation of the fourth on the fifth cervical vertebra. (Courtesy of Robert A. Robinson, M.D.)

to follow-up, or they had ankylosing spondylitis (five patients) and were analyzed in that group (Table III). The remaining thirty-three patients, all with severe anterior cord syndrome (quadripareisis), were treated as follows: nine, non-operatively; fifteen, by laminectomy with or without posterior fusion; four, by anterior decompression (one discectomy and three vertebral-body resections) and anterior fusion; two, by laminectomy and posterior and anterior fusion; two, by posterior fusion; and one, by anterior and posterior fusion only. Of the nine patients treated non-operatively, two recovered completely, four re-

covered partially, and three died during the acute stage. Of the fifteen patients who had a laminectomy (nine of them also with a posterior fusion), one recovered completely, two recovered partially, two remained unchanged, five had loss of motor function, and five died in the early post-operative period (Table I).

The other nine patients with severe anterior cord syndrome included seven who had anterior decompression with removal of bone or disc fragments and two who had only a posterior fusion. Of the seven who had anterior decompression, two had anterior decompression after

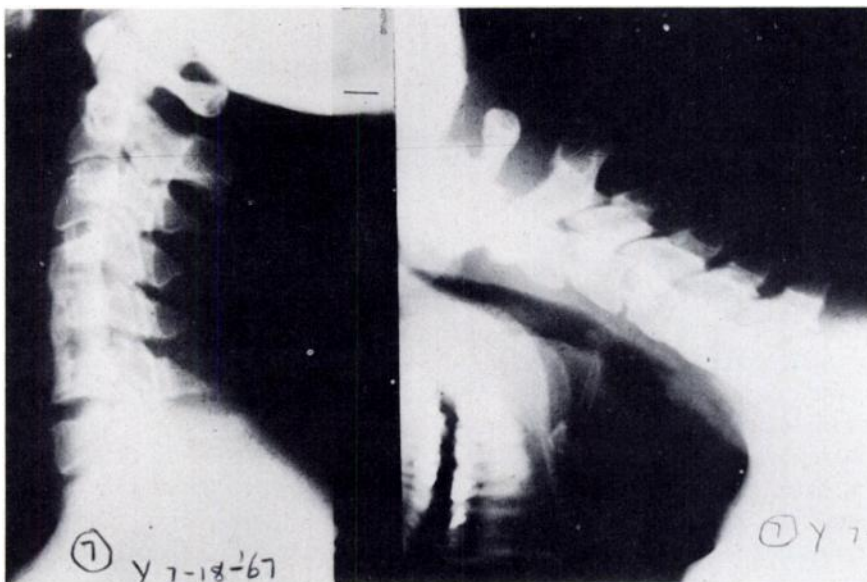


FIG. 5-B

Six months after the removal of two discs and part of the body of the fifth cervical vertebra, and a fusion between the fourth and sixth cervical vertebrae, the patient was pain-free and the fusion was solid. One year postoperatively the monoparesis had disappeared completely, demonstrating the value of late decompression under these circumstances. (Courtesy of Robert A. Robinson, M.D.)

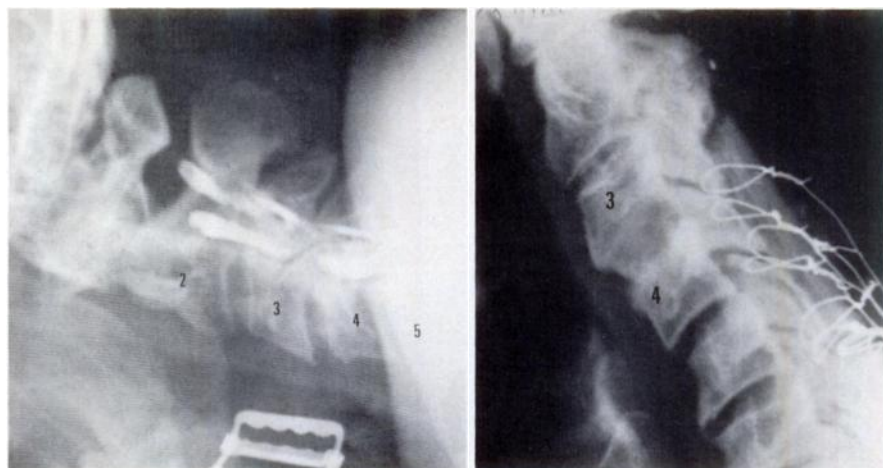


FIG. 6-A

FIG. 6-B

Figs. 6-A and 6-B: This patient showed no roentgenographic evidence of fracture or dislocation but was quadriplegic (central cord syndrome) at the fourth cervical level.

Fig. 6-A: This cervical myelogram, made on admission, reveals a filling defect anteriorly between the third and fourth cervical vertebrae and a partial block of the column of contrast medium. A laminectomy and posterior fusion was performed, resulting in slight improvement initially, but then the neural deficit remained unchanged. Eighteen days later, a second myelogram showed that the defect had persisted, so the anterior disc material was removed and fusion was performed at this level.

Fig. 6-B: Three months later, there was complete recovery of motor function.

laminectomy, one had a posterior fusion performed after the anterior procedure, and four had only an anterior decompression and fusion (Figs. 7-A, 7-B, and 7-C). Three of the seven recovered completely and four, partially. Of the two patients who had posterior fusion only, one had partial and one, complete recovery. None of the nine patients who underwent either anterior or posterior fusion lost motor function or died^{21,82,87} (Table I).

Complete Cord Transection

Of the fifty-seven patients who had complete transection of the cord, two were lost to follow-up so their cases were not analyzed, and two who had ankylosing spondylitis were considered separately with the other patients who had that lesion. Therefore, fifty-three completely quadriplegic patients were available for analysis after different forms of treatment (Table II). Of the fifty-three, eleven were treated non-operatively; thirty-two, by laminectomy alone or in combination with other procedures; and ten, by various surgical procedures not including laminectomy.

Of the eleven patients who were treated non-operatively, two showed recovery of nerve-root function and nine died during the acute phase. Of the thirty-two who had laminectomy alone or in combination with other procedures, the results were as follows: of the twelve who had only laminectomy, one showed slight recovery of root function, four remained unchanged, and seven lost motor function or died. Of the sixteen who had laminectomy and posterior fusion, one showed slight recovery of root function but the others remained unchanged, lost motor function, or died. Of the four who had laminectomy combined with posterior and anterior fusion, the condition of three remained unchanged and one died. Thus, of the thirty-two patients treated by laminectomy, two had slight recovery

of function of the nerve root, the condition of fifteen remained the same, four lost motor function, and eleven died during the acute phase (Table II).

Of the remaining ten patients, who were treated surgically without laminectomy, five had open reduction of the dislocation and a posterior fusion, and five had anterior decompression and fusion. After open reduction and posterior fusion, one patient died and four had slight recovery of function in the nerve root. After anterior decompression with removal of bone fragments and anterior fusion in the other five patients (one also having a posterior fusion), one had slight recovery of function of the nerve root, the condition of three remained the same, and one died (Table II).

Comatose on Admission

Of the four patients with injury between the third and seventh cervical levels who had quadriplegia and entered the hospital in coma, two died of the brain injury (one after laminectomy and the other after burr holes were made). The other two patients regained consciousness and one was treated by anterior fusion and the other, by posterior fusion; both recovered completely.

Complications

Gastrointestinal hemorrhage: This was a major complication in quadriplegic patients and usually occurred ten to fourteen days after injury, as other authors have noted^{62,127}. Thirty-seven patients (two with spondylitis) who had spinal cord injury between the third and seventh cervical vertebrae received steroid therapy within seventy-two hours of injury. Fifteen (41 per cent) of these patients had gastrointestinal hemorrhage. In contrast to this, only nine (9 per cent) of the 101 patients who had injury at the same levels but were not treated with steroids had gastrointestinal bleeding. Since the extent of neural

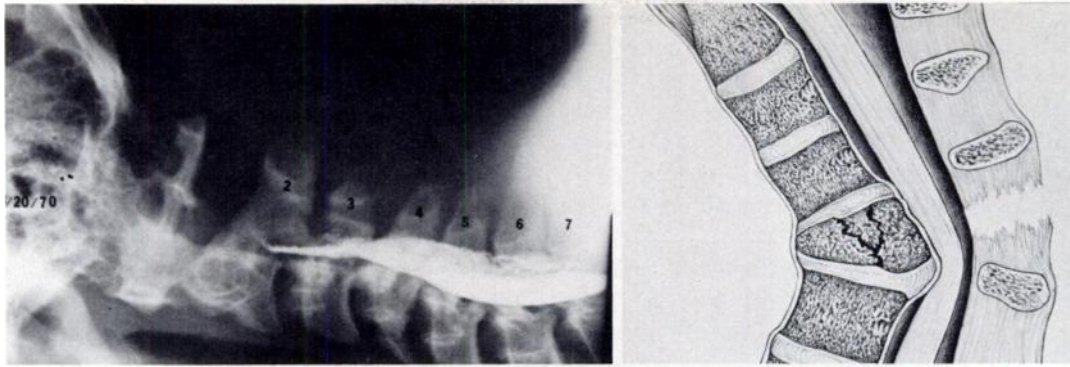


FIG. 7-A

FIG. 7-B

Figs. 7-A, 7-B, and 7-C: This sixteen-year-old boy, who sustained a crush fracture of the fourth cervical vertebra in a sports accident, entered the emergency room with an anterior cord syndrome. He was unable to move the extremities, but the function of the posterior part of the column was markedly spared.

Fig. 7-A: A lateral myelogram made one month after injury shows a filling defect anteriorly between the third and fourth cervical vertebrae while an anteroposterior myelogram showed partial obstruction at this level.

Fig. 7-B: This drawing shows a typical compression fracture with protrusion of disc material, bone fragments, and a kyphotic deformity causing compression of the anterior aspect of the cord.

recovery was the same in both groups of patients, there was no evidence in this series that steroid therapy was beneficial.

Pulmonary embolus: Of the 229 patients with a lesion between the third and seventh cervical vertebrae (including the eight with spondylitis), eleven (nine of whom were quadriplegic) had a pulmonary embolus. This incidence is similar to that described by Shull and Rose¹⁰⁸ and by Watson¹²⁶. In one patient with an anterior cord lesion causing quadriparesis, a superior mesenteric artery syndrome developed after the application of a halo cast.

Onset of a lesion in the cord after the beginning of

treatment: Signs of a partial or complete cord lesion developed in eleven patients after they had reached the hospital¹⁷, in three patients while they were in the emergency room, and in seven others after neck immobilization was not provided. In the eleventh patient, who was being treated with skeletal traction, an anterior cord syndrome developed after an episode of delirium tremens; laminectomy then revealed a herniated disc that could not be resected through the posterior approach.

Chronic instability: Thirty-three (42 per cent) of the seventy-eight patients who initially had non-operative treatment had chronic instability characterized by exces-

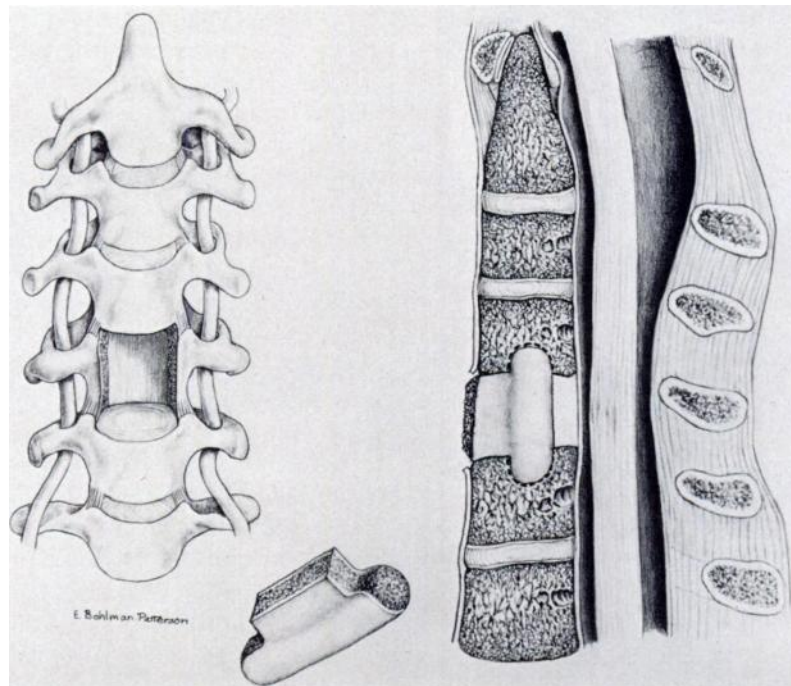


FIG. 7-C

At left, a drawing of the anterior part of the cervical spine shows the extent of a vertebral body resection in which the vertebral body was removed back to the posterior longitudinal ligament, sparing the lateral cortices to protect the vertebral arteries. The posterior longitudinal ligament is not violated, and no instruments enter the spinal canal. At right, a full-thickness iliac-crest graft is inserted to replace the resected vertebra, with the cortical surface of the crest placed posteriorly. This procedure corrects the kyphotic deformity and relieves the spinal cord compression.

sive motion at the level of the lesion and neck pain, with or without progressive neural deficit^{12,29}. Of these thirty-three patients, twenty-one had a flexion injury with disruption of the posterior ligaments (five of these twenty-one had redislocation while in a Minerva jacket) and the other twelve, all with osteoarthritis, had had a hyperextension injury with disruption of the anterior longitudinal ligament and a disc.

Pseudarthrosis: There were no non-unions after the seventy-two posterior fusions that were done at the interspace between the third and seventh cervical vertebrae. In two of the twenty-two patients who had an anterior fusion done after a flexion injury, the graft displaced because the posterior ligaments were torn. One of these two patients subsequently had an increased neural deficit.



FIG. 8

This specimen shows a large, extruded disc fragment lying anterior to the posterior longitudinal ligament and narrowing the anteroposterior diameter of the spinal canal. Most herniated discs caused by cervical trauma remain anterior to the posterior longitudinal ligament and can be excised through an anterior approach. In this specimen there was no gross evidence of necrosis of the spinal cord. (Courtesy of Richard Lindenberg, M.D., Central Anatomic Laboratory, Baltimore, Maryland.)

Additional permanent loss of neural function after treatment: Additional permanent loss of neural function was observed postoperatively in twelve (22 per cent) of the fifty-five patients who were treated by laminectomy: in four of the thirty-two who were admitted with clinical evidence of a complete cord lesion, in seven of the twenty-one who were admitted with an incomplete cord lesion, and in one of the two with a lesion of the nerve root. Permanent loss of neural function did not occur after anterior or posterior fusion or after adequate non-operative treat-

ment in any of the twenty-five patients who were admitted with a partial cord lesion.

Death after injury to the cord: Of the seventy-one patients with partial cord lesions, thirteen died: five after non-operative treatment and eight after laminectomy. The five who died after non-operative treatment included one who died because of a thrombosis of the superior mesenteric artery, three whose deaths were due to damage to the cord, and one who died of a pulmonary embolus. Of the eight who died after laminectomy, three had a pulmonary embolus, four died because of damage to the cord, and one had postoperative wound sepsis. Of the forty-eight patients who had complete transection lesions of the cord, twenty-two died: nine who were treated non-operatively; eleven who were treated by laminectomy; one, by anterior fusion; and one, by posterior fusion. In the nine patients (of the eleven) who were treated non-operatively, death was due to ascending necrosis of the cord or the initial damage to the cord in three⁵¹; pulmonary complications in two; a head injury and multiple injuries in one each; and an unknown cause in two. In the eleven (of the thirty-two) patients who were treated by laminectomy, death was due to massive gastrointestinal hemorrhage in five, cord damage in four, and pneumonia and multiple traumatic lesions in one each. In the one (of the five patients) treated by anterior fusion, death was caused by a gastrointestinal hemorrhage, and in the one (of the five patients) treated by posterior fusion, death was the result of a pulmonary embolus.

Pathological Findings

Rupture of the posterior ligaments without associated fracture was diagnosed roentgenographically in thirty-two patients with a lesion between the third and seventh cervical levels. This injury was associated with subluxation or dislocation of the articular processes as seen on roentgenograms. Torn interspinous ligaments were found at operation in twenty-nine other patients.

Disruption or protrusion of a disc at one of the interspaces between the third and seventh cervical vertebrae was found at operation in twenty-six of the sixty-four patients who had a laminectomy or anterior fusion. All of these disc lesions were associated with fracture or subluxation of a vertebral body (Fig. 8).

Roentgenographic evidence of osteoarthritis of the cervical spine was present in seventy-eight of the 221 patients with a lesion between the third and seventh cervical vertebrae (excluding the eight with ankylosing spondylitis), an association noted by others^{5,81}. Seventy-one of these seventy-eight were older than forty years. Fifty-nine (76 per cent) of the seventy-eight patients with osteoarthritis had some type of neural deficit clinically, while 116 (81 per cent) of the 143 patients with no osteoarthritis had such a deficit.

Assessment of damage either to the spinal cord or to nerve roots at operation was almost impossible unless there was complete severance. Damage to the spinal cord

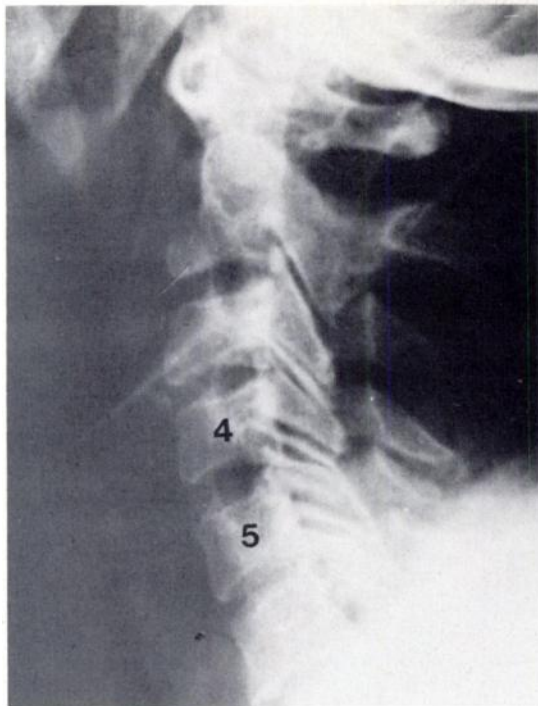


FIG. 9-A

Figs. 9-A and 9-B: This eighty-six-year-old woman had a laceration in the frontal area suggesting a hyperextension injury and had a central cord quadriplegia. Because roentgenograms of the cervical spine were normal except for severe spondylosis, traction with Crutchfield tongs was discontinued. The paralysis then became complete, and the patient died eighteen days after injury due to thrombosis of the superior mesenteric artery and pulmonary embolus. Autopsy showed a soft disc herniation between the fourth and fifth vertebrae as well as necrosis of the cord.

Fig. 9-A: A lateral roentgenogram of the cervical spine shows nothing abnormal except for spondylosis.

was identified in only eight of the fifty-five patients with a neural deficit who underwent laminectomy, but the dura was not opened in all patients. In no patient was severance of a nerve root identified at operation.

Autopsy was performed on thirty-five of the 167 patients with a cervical fracture and a neural lesion between the third and seventh cervical vertebrae. Of these thirty-five patients, twenty-eight had a brain lesion, thirteen had a partial cord lesion, twenty had total cord interruption, and two were infants who had birth injuries without associated skeletal lesions. The brain lesions in the twenty-eight brain-damaged patients included contusion with or without laceration at various sites in seven patients, swelling in twelve (one with herniation through the tentorium), and hemorrhage in nine (subdural in six and intracerebral in three).

In the thirteen patients who had a clinically diagnosed partial cord syndrome, the postmortem findings included contusion and necrotic softening of part of the cord substance in ten (Figs. 9-A and 9-B); parenchymal hemorrhage in the white and gray matter in two; and only small, peripheral hemorrhage in the white matter in the remaining patient (a quadriplegic) (Fig. 10).

In the twenty patients who had clinical evidence of total cord interruption, the pathological findings were: contusion of the cord in twelve (associated with a parenchymal hemorrhage in five, a small epidural hemorrhage in four, and an anatomical transection of the cord in three) and necrotic softening associated with parenchymal hemorrhage in the other eight.

Two infants had birth injuries without associated fracture or dislocation. The first patient, an infant injured during a breech delivery when the obstetrician reportedly heard a snap, was quadriplegic at the seventh cervical level and lived for four months. Autopsy revealed transection of the cord between the sixth and seventh cervical levels and necrotic softening of the entire distal part of the cord as the result of vascular impairment (Figs. 11-A and 11-B). The other patient, an infant born quadriplegic after prolonged



FIG. 9-B

A cross section of the cord shows central necrosis with destruction of the dorsal horns of the gray matter and demyelination of the anterior and lateral corticospinal tracts (myelin, $\times 6$). (Courtesy of John R. Wright, M.D., Department of Pathology, Baltimore City Hospitals, Baltimore, Maryland.)

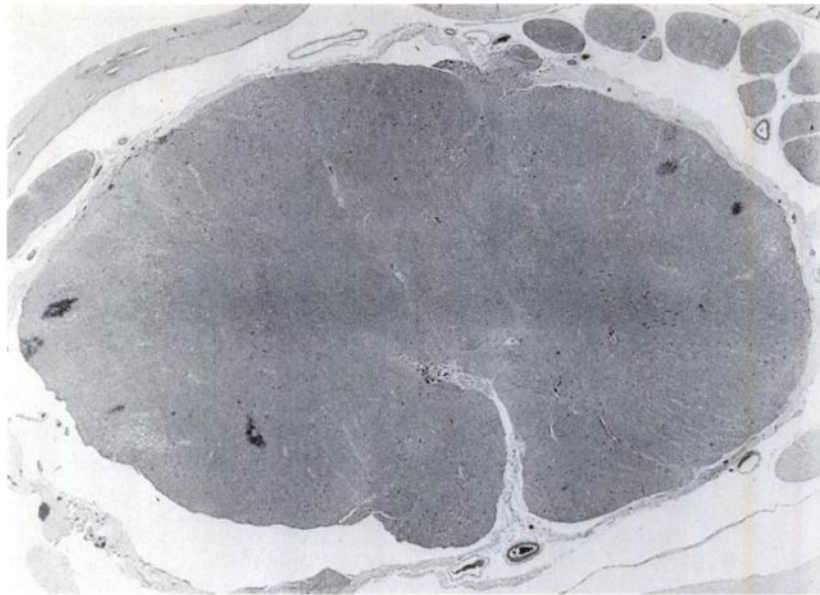


FIG. 10

A photomicrograph of the cervical spinal cord of a quadriplegic patient with anterior cord syndrome seen clinically, but no gross evidence of destruction of the cord. Laminectomy revealed an extruded disc between the fourth and fifth cervical vertebrae which could not be removed. The patient died six days after injury, four days postoperatively. At autopsy, focal hemorrhages were found on the anterior and lateral aspects of the cord, but no gross destruction of the cord was seen. The absence of cord destruction supports the concept that ischemia is the basis of this syndrome (hematoxylin and eosin, $\times 15$).

labor and a difficult delivery, had an anterior cord syndrome at the sixth cervical level, and two months later an autopsy revealed hemorrhage in the anterior gray and white matter. The mechanism of cervical injury during birth and the pathological findings characterized by a sparsity of osseous changes have been described by many authors^{1,3,22,34,48,49,59,60,61,85,88,110,115,125}.

Arterial injury was found in only five of the thirty-five

patients examined at autopsy. These injuries included one thrombosis of a vertebral artery and four compressed radicular arteries, all in patients who had necrosis of the cord associated with dislocations of articular processes.

Cervical Fractures in Patients with Ankylosing Spondylitis

The characteristic fractures that occur in ankylosed

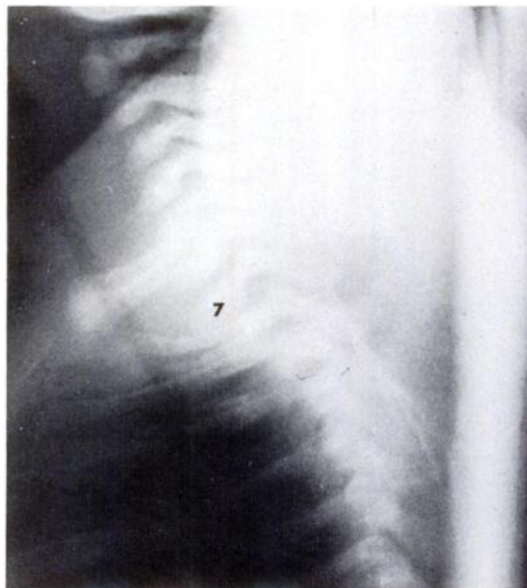


FIG. 11-A



FIG. 11-B

Figs. 11-A and 11-B: This infant, who was quadriplegic after a breech delivery, died four months after delivery. Fig. 11-A: A lateral roentgenogram made after delivery shows no abnormality. Fig. 11-B: Autopsy, four months after delivery, showed complete severance of the cord at the interspace between the sixth and seventh cervical vertebrae. No fractures or dislocations of the cervical spine were demonstrated by roentgenogram or at autopsy.



FIG. 12

A sixty-year-old man with ankylosing spondylitis fell and sustained a fracture of the seventh cervical vertebra without neural involvement. His facial lacerations were sutured on the day of injury and he was discharged. One week after injury he returned with progressive quadriplegia. Laminectomy revealed a massive epidural hemorrhage (arrows) which was confirmed at autopsy eighteen days later. (Courtesy of Richard Lindenberg, M.D.)

spines, and which are occasionally associated with epidural hemorrhage, have been described by several authors^{12,57,64,67,131}.

In this series, ankylosing spondylitis was diagnosed in eight patients who had a transverse fracture through an ossified intervertebral disc located between the third cervical and first thoracic vertebral segments (Table III). Seven of the eight patients had an associated neural deficit (five had a partial and two, a total cord lesion), and one (Cases 90A and 90B) had a second partial cord lesion after a second injury. In four of the eight patients, the diagnosis of cervical spine injury was not made initially in the emergency room. Three of these four subsequently returned to the hospital with increasing quadriplegia⁸⁶. The fourth patient fell in the emergency room after receiving a tranquilizer and became completely quadriplegic. Of the four fractures that were not diagnosed initially, three were between the sixth and seventh cervical segments and the fourth was between the seventh cervical and first thoracic segments. The injury was caused by a fall in five patients, an automobile accident in two, and a kick by a mule in one.

Treatment and Results

Of the eight patients with ankylosing spondylitis and a cervical fracture, only one (Case 88) did not have paralysis. This patient was treated successfully by closed reduction in traction and immobilization in a Minerva jacket. Of the seven patients with a neural deficit, five had an anterior cord syndrome (one, on two occasions after different injuries) and two had complete quadriplegia.

Considering the five patients with anterior cord syndrome, three died (one after non-operative treatment and two after laminectomy) and the other two had loss of

neural function (both after laminectomy) but survived.

The two patients with complete quadriplegia both died within three days of injury: one after laminectomy and posterior fusion and the other after non-operative treatment. Therefore, only three of the eight patients with spondylitis survived and had a healed fracture. One of the survivors (Cases 90A and 90B) had three separate fractures of the cervical spine with complete recovery after the first and severe loss of motor function after the second (Table III). The third fracture, which occurred at still another vertebral level, was sustained after the completion of this study and therefore it was not included. This fracture occurred at the interspace between the seventh cervical and first thoracic vertebrae; the patient was treated in a rigid brace and the fracture healed uneventfully. The patient had no further loss of neural function.

Complications

Massive epidural hemorrhage occurred in four of the eight patients with ankylosing spondylitis but was not recognized in any of the other 292 patients in this series. A gastrointestinal hemorrhage occurred in two of the three patients with spondylitis whose cord lesion was treated with steroids (Table III). One dislocation at the fracture site occurred after laminectomy in a patient who had loss of neural function postoperatively, but who eventually became ambulatory after the fracture healed spontaneously in skeletal traction. One patient died of respiratory arrest while he was being turned during the application of a Minerva jacket.

Pathological Findings

The autopsies performed on four of the five patients who died showed that the causes of death were ascending



FIG. 13-A

Fig. 13-A: A lateral roentgenogram shows moderate anterior subluxation (three to five millimeters) of the sixth on the seventh cervical vertebra secondary to a unilateral dislocation of an articular process.

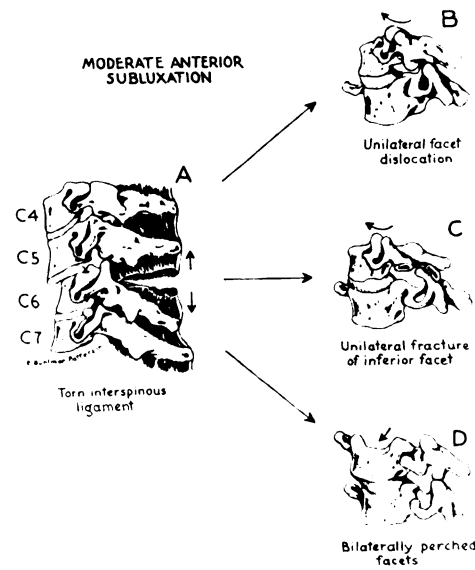


FIG. 13-B

Fig. 13-B: This drawing shows various types of fractures and dislocations (B, C, and D) of the articular processes that may cause moderate anterior subluxation.

necrosis of the cord in one, and massive epidural hemorrhage with cord compression in three.

The skeletal lesions in the eight patients with spondylitis, as seen on the roentgenograms, included a fracture through a disc space combined with either subluxation or dislocation of the articular processes. The patient who had three fractures had a fibrous lytic lesion at the site of the second fracture, one interspace below the level of the first fracture (Table III). This lesion was demonstrated at a laminectomy performed after the second fracture occurred.

Of the five patients who died, two had a complete and three, an incomplete cord lesion. The autopsies of four of these patients showed an epidural hemorrhage which was large in three (Fig. 12) and small in one. In addition, three of the four patients had contusion and necrotic softening of the cord and two patients had a hemorrhage into the cord substance.

Discussion

Head and neck injuries are found in about 61 per cent of persons who have had a fatal accident^{15,18,36}. Evidence of trauma to the head should immediately raise the question of whether neck injury has occurred.

A lateral roentgenogram of the cervical spine should be the first step in the diagnostic work-up of any patient in whom a cervical fracture-dislocation is suspected¹²⁹. If the lateral roentgenogram shows no vertebral displacement and the patient is quadriplegic, then myelography should be considered to rule out the possibility of a herniated, soft disc. If the lateral roentgenogram is normal, without evidence of vertebral displacement or paralysis, flexion-extension stress roentgenograms can be made with protective head-halter traction and 4.5 kilograms of weight,

provided the patient's complaints and history indicate a severe cervical injury.

A lateral roentgenogram showing anterior or posterior vertebral displacement indicates acute spinal injury, and anteroposterior and oblique roentgenograms should then be made without moving the patient. The lateral roentgenograms in this series revealed three major types of displacement of a vertebral body¹⁶: (1) minimum posterior subluxation (two to three millimeters) of the upper on the lower vertebra; (2) moderate anterior subluxation (three to five millimeters) with angulation of the upper vertebra; and (3) anterior dislocation, defined as displacement of the upper vertebra equivalent to one-half of the width of the vertebral body or more. The first type of displacement occurred in spondylitic spines and was associated with extension injuries that disrupted a disc and the related ligaments. The second type was observed in association with unilaterally dislocated articular processes, bilateral so-called perched processes, fractured articular processes, or a combination of these lesions (Figs. 13-A and 13-B). The third type, anterior displacement associated with bilaterally dislocated articular processes, was usually accompanied by severe damage to the spinal cord (Fig. 14). As in Marar's series⁷⁶, the higher incidence of cord injury associated with bilaterally subluxated or dislocated articular processes is not surprising in view of the greater amount of displacement involved. Therefore, the roentgenographic findings, especially the amount of narrowing of the spinal canal, may be quite important in predicting the degree of neural damage⁷⁹.

Myelography was used during the acute stage despite the inherent risk of arachnoiditis. The importance of demonstrating the presence and location of a lesion causing compression outweighs the risks associated with this pro-

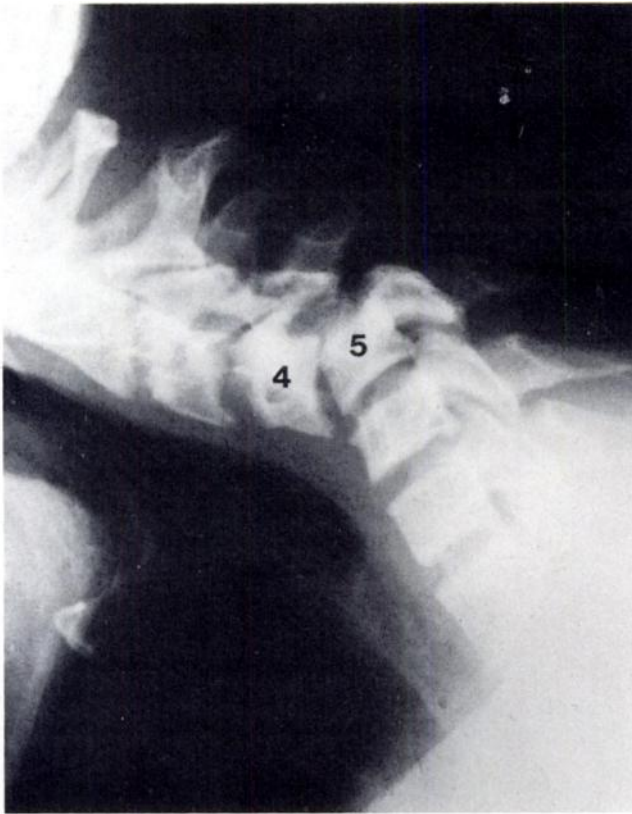


FIG. 14

Anterior dislocation of the fourth on the fifth cervical vertebra with bilateral dislocation of the articular processes is seen in a patient who was quadriplegic.

cedure. However, in the acute stage, excessive cord edema may make it impossible to identify the exact level of compression. Under these circumstances, myelography can be repeated at a later date to determine whether there is an operable lesion. In the sixty-eight patients examined by myelography, no ill effects secondary to this procedure were recognized. Raynor⁹⁰ described similar results.

Cases of occipitocervical dislocation without fatal compression of neural elements have been described^{11, 43, 44}, but it is a rare injury. Both patients in this series who had this dislocation were found to have total transection of the spinal cord and rupture of the surrounding ligaments, as has been described previously^{18, 36}.

Atlanto-axial dislocation, if it causes instability, may result in neural compression, paralysis, and death^{4, 13, 14, 35, 42, 46, 47, 54, 58, 73-75, 111, 123, 130}. In this series, a progressive neural deficit developed in three patients with atlanto-axial instability secondary to dislocation.

Although Schatzker and co-workers⁹⁶ found no relationship between the incidence of non-union and the type of fracture of the odontoid process (except in patients with a posteriorly displaced fracture), in the present series only the fractures through the waist of the odontoid process failed to unite.

For acute fractures and dislocations of the cervical spine between the third and seventh vertebrae, the goal of treatment is to achieve reduction and restore stability^{10, 32, 63, 64, 94}.

Halo or skeletal-tong traction reduces the fracture, restores the alignment of the spinal canal, and protects the neural structures until sufficient healing has occurred to provide stability. However, after torn posterior ligaments heal, they are frequently grossly lax, even after a Minerva or halo cast has been worn for three months^{15, 17}. Rogers^{94, 95}, Bailey⁸, and others^{91-93, 109} have emphasized the importance of stabilization by anterior or posterior fusion.

The roles of vascular compression and contusion and of compression of neural tissue in cervical spine injuries associated with paralysis are well known^{24, 38, 41, 84, 113, 116-119, 124}. The pathophysiology and treatment of spinal cord trauma² in experimental animals have been described¹¹⁷⁻¹¹⁹. These studies demonstrated that steroids control swelling of neural tissue in animals and, based on this evidence, steroids have been used in humans with spinal cord injuries. However, in the present series there was no evidence that the steroids that were given to thirty-seven patients with spinal cord injury improved the recovery of neural function.

No patient in this series with complete quadriplegia that was still present forty-eight hours after injury recovered function of the spinal cord. Similar findings were documented by Matson⁷⁸ and by Holdsworth⁶³. Nerve-root function in the upper extremity returned if the dislocations were reduced and stabilized. Early closed reduction, followed later by open reduction and posterior fusion, offered the best opportunity for nerve-root recovery. Verbiest's¹²⁰ finding that a high mortality rate is associated with early operative treatment of completely quadriplegic patients was reinforced by the results in this series. The highest mortality rate in the present series (80 per cent) was in the eleven completely quadriplegic patients who were treated non-operatively, but their deaths were attributable to other severe injuries and to pulmonary problems. A high mortality rate also was seen in quadriplegic patients who were treated with steroids and laminectomy. Of thirty-seven quadriplegic patients who were treated with steroids (seventeen also having a laminectomy), fifteen had associated gastrointestinal hemorrhage and seventeen died. Eleven patients who were treated with steroids and laminectomy died. None of the other 101 patients who were not treated with steroids died of gastrointestinal hemorrhage.

Based on the examination of pathological specimens and the results of operative treatment, it appeared that contusion of the cord and mechanical compression play important roles in many patients with incomplete cord lesions. Compression of the cord anteriorly probably produces a physiological block in neural function as a result of microvascular oligemia and distortion of neural tissue^{13, 118}. In this series, anterior decompression and fusion, performed one week to six months after injury in seven patients with an incomplete cord lesion, resulted in immediate recovery of neural function in six patients (Figs. 15-A, 15-B, and 15-C), results similar to those observed

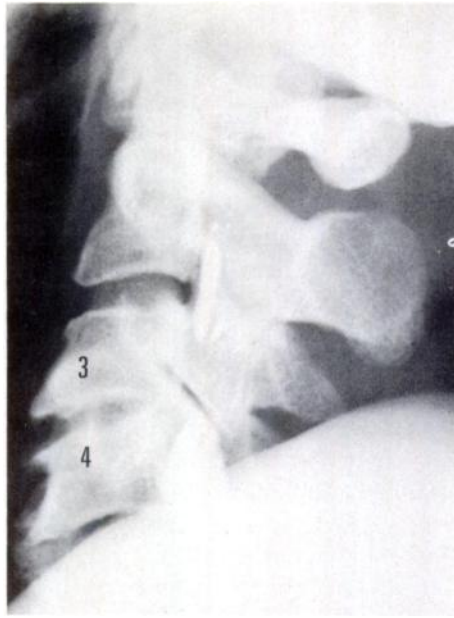


FIG. 15-A

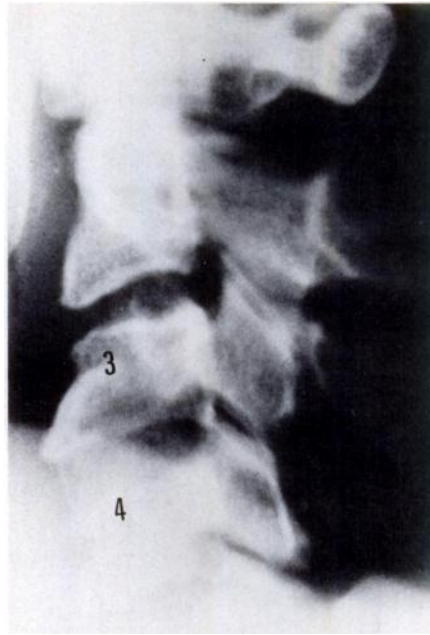


FIG. 15-B

Figs. 15-A, 15-B, and 15-C: A fifty-three-year-old man had a central cord quadriplegia at the third cervical level after he fell while intoxicated.

Fig. 15-A: A preoperative myelogram shows protrusion of disc material between the third and fourth cervical vertebrae.

Fig. 15-B: A postoperative myelogram made after a Robinson anterior cervical discectomy and fusion were performed, ten days after injury. There was a rapid recovery of function but the patient died of a pulmonary embolus ten months after injury.

by other authors^{19,30,52,82,89,121,122}. These authors noted difficulty in removing anterior fragments of bone and disc by posterior laminectomy in patients who became worse after laminectomy, only to recover following anterior decompression and fusion.

Anterior cord syndrome⁹⁸, the most common type of partial cord lesion in this series, was caused by a protruding disc, fragments of a fractured vertebra, or a dislocated vertebra pressing against the anterior surface of the spinal

cord. The most complete recovery in these patients occurred after reduction of the dislocation and fusion, or after anterior decompression by removal of either a protruding disc or bone fragments followed by fusion. The concept that laminectomy is contraindicated in these patients is supported by the high mortality rate and frequent loss of motor function that were observed after this procedure in the present series and in those reported by other authors^{41,80,129}.



FIG. 15-C

This histological section of the spinal cord at the level of the third and fourth cervical vertebrae shows little more than peripheral vacuolization (hematoxylin and eosin, $\times 10$).

When Schneider and co-workers¹⁰⁴ first described central cord syndrome (quadripareisis with more motor impairment in the upper than in the lower extremities and varying degrees of sensory loss), they recommended non-operative treatment, stating that laminectomy was contraindicated because manipulation of the spinal cord exacerbated the neural deficit of one of their patients and caused death in another. Cloward³⁰, who later reviewed the cases of eleven patients with central cord syndrome, reported that one who failed to recover after laminectomy did recover completely after anterior decompression. Verbiest's¹²⁰ findings in nine of his patients were similar. Of these nine patients, three who had anterior decompression by removal of disc fragments recovered fully, whereas all three patients who were treated by laminectomy without anterior decompression died — two of them after losing motor function postoperatively. Therefore, I believe that skeletal traction should be used in patients with central cord syndrome, even though the roentgenographic findings are normal; that anterior discectomy and fusion may be indicated; and that laminectomy is contraindicated.

Closed or open reduction of the dislocation and posterior fusion resulted in excellent recovery in eight of the ten patients with a Brown-Séquard syndrome or monoparesis. One patient with monoparesis did not improve after reduction and posterior fusion, but subsequently recovered completely after anterior excision of a herniated disc and fusion, done six months after injury.

Patients with atlanto-axial instability may have cardiorespiratory arrest as the result of manipulation during operation. Therefore, a halo cast should be applied preoperatively to ensure stability¹⁷. Generally, posterior fusions are successful and the only ones that fail are at the occipitocervical level, where tension forces predominate⁷³. As has been reported by others^{4,46,96}, an unstable atlanto-axial joint is most satisfactorily stabilized by atlanto-axial arthrodesis, which will prevent the insidious onset of myelopathy^{35,43,75,123,130}.

Late pain and instability are commonly the result of incomplete healing of ligaments and disruption of a disc. In young children the disc injury may be a separation of the cartilaginous and osseous end-plates^{6,36}, while in young adults axial-loading or flexion injuries may retropulse bone or disc fragments, causing anterior compression of the spinal cord. In older patients with spondylosis, on the other hand, an extension injury may cause separation at the level of a degenerated disc space, resulting in neural compression due to vertebral displacement¹⁶. Any congenital narrowing of the spinal canal (to an anterior-posterior diameter of less than thirteen millimeters) predisposes a patient to a spinal cord lesion after any injury to the spine⁷⁹.

Massive epidural hemorrhage caused compression of the cord in four of the eight patients with ankylosing spondylitis, three of whom died. In patients with ankylosing spondylitis, a cervical injury causes a fracture through

an ossified disc space. It would appear that when the rigid ankylosed spine fractures, the perivertebral or epidural veins which are fixed in scar tissue are torn, and profuse bleeding occurs from these vessels and from the fractured cancellous bone. In none of the other patients in this series was there evidence of a large epidural hemorrhage that compressed the spinal cord. Also, in a previous postmortem study of patients who had fatal injury to the neck, no large epidural hemorrhages were found¹⁸.

A force sufficient to sublunate, dislocate, or fracture the spinal column usually disrupts the intervertebral disc at the level of the injury. In this series, all protruding disc material that was found either at operation or at autopsy had remained anterior to the posterior longitudinal ligament. As has been noted previously¹⁹, this is a very important consideration during anterior decompression.

In this series, autopsy showed that a major radicular artery feeding the anterior spinal artery was compressed by a dislocated articular process in four patients with necrosis of the cord. Functional recovery of neurons and axons depends on preservation of the microcirculation of the cervical cord³⁸. One-half of the autopsies performed on thirty-five patients in this series revealed vascular impairment and necrosis of the cervical cord. The necrosis, which was partial in ten patients and complete in eight, extended above and below the level of the osseous lesion. Complete anatomical transection of the cord was rare.

The extent of the damage to the spinal cord, evident both grossly and histologically at autopsy, did not correlate well with the clinical patterns of loss of neural function. One patient, who had a clinical diagnosis of anterior cord quadripareisis and died of a pulmonary embolus ten months after injury, had only spotty demyelination of the white matter of the cord at autopsy. Conversely, a patient with marked destruction of the central gray matter could move the left lower extremity. There was no definite correlation between clinical findings that would be consistent with a central or anterior cord syndrome and the extent of destruction of the spinal cord found at autopsy. Indeed, anterior and central cord syndromes may represent different degrees of the same pathological process; that is, contusion of the cord initially followed by microvascular oligemia, edema, and subsequent necrosis of the cervical spinal cord.

Conclusions

The delay in the diagnosis of the cervical lesion in one-third of the patients in this series appeared to result either from error or from lack of suspicion on the part of the examining physician. Among the specific causes for delay were the presence of a head injury, a decreased level of consciousness, inadequate roentgenograms of the lower part of the cervical spine, alcoholic intoxication, and multiple injuries.

Non-operative treatment is recommended for isolated fractures of the atlas, of the pedicles of the axis, and through the body of the axis. However, posterior surgical

stabilization is indicated for an atlanto-axial dislocation associated with a fracture through the waist of the odontoid process, a dysplastic odontoid process, or a torn transverse ligament.

Patients with a severe flexion injury and torn posterior ligaments with or without a neural deficit should be considered as candidates for open reduction and stabilization by posterior fusion. Conversely, patients with a hyperextension injury that has torn the anterior and posterior longitudinal ligaments and caused disruption of the disc should have an anterior fusion if non-operative treatment fails to restore stability. For severe compression fractures secondary to axial-loading injuries, anterior vertebral-body resection and fusion may be required to prevent kyphosis and pain^{15,122}.

Fusion was not required for fractures of the cervical spine in patients with ankylosing spondylitis since adequate osseous union occurred when proper immobilization was provided. On the other hand, if the immobilization is inadequate, progressive paralysis may develop secondary to massive epidural hemorrhage.

In patients with a fracture-dislocation or a lesion of a nerve root between the third and seventh cervical levels, reduction and stabilization are required. A unilateral dislocation of an articular process usually necessitates open re-

duction and posterior fusion, while so-called perched articular processes can be reduced by skeletal traction. Limited posterior decompression (foraminotomy) and fusion are indicated for fractures of the articular processes causing posterior compression of nerve roots. If nerve-root compression is caused by a herniated disc or anterior bone fragment, anterior decompression is indicated¹⁵. Fourteen of the fifteen patients with this type of injury who were treated surgically recovered completely, whereas only five of the nine patients who were treated non-operatively recovered nerve-root function.

In patients with incomplete cord syndromes who are recovering neural function slowly and have bone and disc fragments compressing the spinal cord anteriorly, as demonstrated by myelography, anterior decompression and fusion should be carried out. Laminectomy is contraindicated under these circumstances.

Early surgical treatment is not indicated in patients with complete quadriplegia, but open reduction and posterior fusion may be carried out to stabilize a dislocation when the patient is medically able to undergo the operation.

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