Penetrating gunshot wounds of the cervical spine in civilians

Review of 38 cases

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The authors present a series of 38 civilian patients with cervical gunshot injuries, and compare neurological recovery in patients with complete lesions and patients with incomplete lesions according to whether therapy was surgical or nonsurgical. In patients with incomplete injury, ultimate recovery was a function of the initial injury more than surgical or nonsurgical therapy; nor did patients with complete lesions show significant change in outcome with either mode of therapy. Cord pathology at laminectomy rarely provided a clue about neurological recovery, and dural decompression did not alter neurological outcome. The authors conclude that the sole indication for routine surgical intervention appears to be progressive neurological deficit.

KEY WORDS: spinal cord injury • missile injury in civilians • therapeutic indices

The present treatment of patients with penetrating spinal cord injury is chiefly based on experience with such wounds in wartime. The classical surgical objectives have been: 1) Debridement of all contaminated tissue along the missile tracts, including removal of all bone fragments and missile material from the spinal canal; 2) Dural closure with grafting as necessary to eliminate CSF fistulas; 3) Identification of gross cord pathology in an effort to determine ultimate prognosis; and 4) Surgical decompression for the injured spinal cord. The evolution of these principles has caused marked improvement in mortality and morbidity among military casualties. However, there are few reports of penetrating spinal cord injuries involving civilians. This paper reviews our experience with 38 penetrating missile injuries to the cervical spine seen during the 10-year period, 1963 to 1972.

Materials and Methods

Twenty patients received initial treatment at the Los Angeles County-University of Southern California Medical Center; 18 were cared for in the community hospitals of Southern California and subsequently

J. Neurosurg. / Volume 42 / May, 1975
transferred to our Rancho Los Amigos Hospital for rehabilitation. The injuries were caused by low velocity, .22, .25, and .38 caliber pistols. For the purpose of analysis, patients with penetrating cervical spine injuries were divided into three categories: Group A, with immediate complete sensorimotor paralysis; Group B, with incomplete sensorimotor paralysis; and Group C, with no neurological deficit.

**Group A**

The largest group included 25 patients with complete sensorimotor paralysis dating from the time of injury. The lowest levels of intact motor function in this group are shown in Table 1. Lumbar puncture and myelography were not generally performed on these patients; 16 patients were treated by laminectomy, decompression, and debridement, and nine medically, receiving supportive care as needed.

**Group B**

This group included nine patients, eight with Brown-Séquard syndrome and one with anterior cord paralysis. Table 2 shows the levels of spinal cord involvement for this group. Of these, eight patients had a static neurological deficit, unchanged between the accident and their initial evaluation. However, the patient with anterior cord syndrome secondary to a gunshot wound at C-6 had documented motor deterioration from C-8 to C-6 during the course of his initial evaluation. Six of the patients in Group B were treated by laminectomy and decompression, three were treated conservatively.

**Group C**

The smallest group included four patients; two had C1–2 fractures involving the odontoid vertebra, and two had C-5 vertebral body fractures. All were treated conservatively by external immobilization in a Minerva jacket and cervical brace for 4 months. One patient with a C-5 fracture required emergency neck exploration for a vertebral arteriovenous fistula.

Patients in all three groups were treated with tetanus prophylaxis and massive systemic antibiotics; some were treated with skeletal fixation when cervical stability was in doubt. Surgical exploration of penetrating missile injuries to the neck was performed only when esophageal lacerations were verified by hypaque esophagrams or endoscopy, and in cases with angiographic evidence of contrast extravasation from carotid or vertebral artery injury or arteriovenous fistula.

**Results**

**Group A**

The operative findings revealed dural lacerations in 77% of the cases in Group A. The cervical cord was anatomically intact in two-thirds of the patients; some degree of cord laceration was present in one-third. The dura was deliberately left open in three cases with intact, but swollen cord; however, no neurological improvement occurred in these patients even after dural decompression.

Neurological follow-up of all Group A patients averaged 9 months after injury with a range of 3 to 24 months. The only neurological change, if any, was the return of function in one or occasionally two cervical roots; this was seen in both operative and non-operative groups. The failure or lack of change in neurological function is very similar in patients with closed cervical frac-
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ture dislocation and initial complete sensorimotor paralysis.\(^\text{10}\)

Two patients operated on within 24 hours of injury subsequently developed febrile episodes as a result of their wounds. One of these had a local wound infection secondary to a coagulase-positive *Staphylococcus aureus* and *Neisseria*. The second of these patients developed meningitis along with a cerebrospinal fluid (CSF) fistula that developed through the operative wound on the fifth postoperative day, despite attempted use of a dural patch for closing a posterolateral dural avulsion. No organisms were recovered, but the CSF showed 2500 WBC/mm\(^3\), and hypoglycorrhachia. This patient was on massive antibiotic therapy using Keflin and gentamicin at the time meningitis was diagnosed. The meningitis was successfully treated with intravenous methicillin 150 mg/kg, gentamicin 4 mg/kg, and intrathecal gentamicin 2 mg daily for 5 days. The CSF fistula was successfully obliterated by placement of additional wire sutures in the skin and performance of daily lumbar punctures. There were no instances of wound infection or meningitis during the hospital stay of the nine patients without operation.

Four Group A patients died. Two patients with C-6 quadriplegia died during the early acute postoperative period; one of these, not receiving steroid therapy, died of massive gastrointestinal hemorrhage 6 weeks after his injury occurred. The second patient died of bronchopneumonia and sepsis at 5 weeks after injury. Two late deaths occurred; a patient with C-5 quadriplegia died of pneumonia 29 months after injury, and one with C-7 quadriplegia died of pyelonephritis 7 years after his injury.

**Group B**

Of the six patients undergoing laminectomy in Group B, dural laceration was observed in three. In these six patients, the operative appearance of the cervical cord was described as normal in one, contused in two, and partially lacerated in three. The dura was left open in the two with contused cords to effect cord decompression. After 1 year, two patients could walk unaided, two required braces or a cane, and two were confined to wheelchairs. No correlation existed between whether the cervical cord was normal, contused, or partially lacerated immediately after injury, and the final neurological recovery in these patients; all three of our nonoperated patients with Brown-Séquard syndrome were improved and ambulatory with walker aids or braces and crutches at follow-up 1 year later. Nor was there any apparent difference in the extent of neurological recovery between operated and nonoperated patients; recovery related solely to the extent of initial deficit. The patient with an anterior cord syndrome, treated by early laminectomy, remained unable to walk.

Two of the six surgically treated patients suffered postoperative complications. One patient had cervical instability at C3–4 after a C3–T1 laminectomy, requiring a late posterior cervical fusion. The other had had a gunshot wound at C-5 with an initial Brown-Séquard syndrome; this patient subsequently developed an arachnoiditis, documented at re-exploration, that caused a neurological deficit of the arm. The single complication in the three unoperated cases was in one patient who had a pulmonary embolus requiring anticoagulation. No tracheostomies were required in these nine patients and no deaths occurred. All of this group required active rehabilitation at Rancho Los Amigos Hospital for a period of 1 to 5 months, with an average of 3 months’ hospitalization.

**Group C**

In Group C, all fractures healed without instability after Minerva jacket immobilization for 3 months and cervical brace for 1 month. No wound infections or osteomyelitis occurred.

**Discussion**

The neurological morbidity of penetrating cervical injuries with low velocity missiles appears unaffected by surgical therapy. Of 25 cases with complete sensorimotor paralysis (Group A) no significant recovery occurred in the legs, whether the patient was treated by decompressive laminectomy or by supportive medical care. Although the recovery of one, or occasionally two, cervical nerve roots was observed, there was no difference in such recovery between the operated and nonoperated groups. Of the patients with incomplete neurological lesions (Group B), those with the Brown-Séquard syndrome showed similar improvement in neurological function regardless of the mode of treatment.
Patients with an initially more severe motor injury (hemiplegia) had greater residual motor disability than patients with hemiparesis; the ultimate extent of neurological recovery was a function of the extent of the initial injury rather than surgical or nonsurgical therapy.

Progressive neurological deficit was uncommon; we encountered this in only one case. In this case a documented ascending neurological deficit occurred in a patient with an anterior cord syndrome. In spite of decompressive laminectomy, at which time the dura was left open because of an edematous cord, the patient did not regain any motor function in the legs after 18 months of follow-up. This experience is in accord with the report by Bosch, et al., on long-term follow-up of patients with anterior cord syndromes; 20% of their patients with incomplete lesions had anterior cord deficits, and none of these patients were able to walk at the completion of rehabilitation therapy.

The evaluation of gross cord pathology, unless complete transection exists, does not provide an index of neurological improvement in these patients. Complete and incomplete sensorimotor deficits persist with apparently normal, as well as swollen and/or partially lacerated spinal cords. We feel that cord inspection as a prognostic tool is not a justified criterion for operative intervention. As a corollary, the decision to open intraoperatively a nonlacerated dura in a contaminated field to evaluate cord pathology or for decompression is unwarranted. A decompressed, as opposed to a closed or intact dura, did not alter the final neurological outcome in these patients.

The amount of wound contamination and tissue destruction is obviously less with a low velocity missile than with the high velocity missile usually encountered in wartime injuries. There were no cases of wound infection, meningitis, or spinal abscess in the 16 patients (42% of total series) treated nonoperatively. On the other hand, one case of meningitis and one local wound infection occurred in 22 operated cases. The case of postoperative meningitis and CSF fistula ironically occurred in spite of an attempted dural repair and points up the need for meticulous wound closure as opposed to repair of a macerated dura. Without surgical intervention, the incidence of infection occurring as the result of low velocity missiles appears to differ significantly from that of high velocity gunshot wounds and fragmentation injury similarly managed.

In their review of bullet wounds to cord and cauda equina, Yashon, et al., conclude: 1) Patients with complete or very severe lesions are to be treated nonoperatively unless gross contamination exists; 2) Patients treated within 5 to 6 hours of injury should have laminectomy and spinal cord cooling; 3) Patients with less severe injury are to be treated nonoperatively unless progression of neurological deficit occurs. We concur wholeheartedly with their call for expectant management. In both reviews, progression of the neurological deficit was an unusual occurrence; and, tragically, patients in both series had no neurological improvement in spite of surgical intervention. However, we still consider progression of deficit to be the indication for routine surgical intervention.

Certain similarities in spinal cord pathophysiology must exist between patients with closed spinal cord injury and patients with penetrating injury in which the spinal cord at the time of surgery is grossly intact or mildly contused. In these circumstances, where complete sensorimotor paralysis exists, the grave prognosis demands the development of new approaches to this problem in the appropriate setting. Current investigation directed at the management of such injuries includes normothermic or hypothermic cord perfusion, the use of pharmacological agents antagonistic to the biogenic amines and their release, and the use of early dorsal myelotomy and/or rhizotomy to inhibit possible progressive hemorrhagic cord necrosis. Such endeavors must be supported, since the outlook with conventional surgical approaches to this problem is bleak.

References

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