

Surgical management of spinal epidural hematoma: relationship between surgical timing and neurological outcome

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✓ Thirty patients were treated surgically for spinal epidural hematoma (SEH). Twelve of these cases resulted from spinal surgery, seven from epidural catheters, four from vascular lesions, three from anticoagulation medications, two from trauma, and two from spontaneous causes. Pain was the predominant initial symptom, and all patients developed neurological deficits. Eight patients had complete motor and sensory loss (Frankel Grade A); six had complete motor loss but some sensation preserved (Frankel Grade B); and 16 had incomplete loss of motor function (10 patients Frankel Grade C and six patients Frankel Grade D). The average interval from onset of initial symptom to maximum neurological deficit was 13 hours, and the average interval from onset of symptom to surgery was 23 hours. Surgical evacuation of the hematoma was performed in all patients; 26 of these improved; four remained unchanged, and no patients worsened (mean follow up 11 months). Complete recovery (Frankel Grade E) was observed in 43% of the patients and functional recovery (Frankel Grades D or E) was observed in 87%. One postoperative death occurred from a pulmonary embolus (surgical mortality 3%). Preoperative neurological status correlated with outcome; 83% of Frankel Grade D patients recovered completely compared to 25% of Frankel Grade A patients. The rapidity of surgical intervention also correlated with outcome; greater neurological recovery occurred as the interval from symptom onset to surgery decreased. Patients taken to surgery within 12 hours had better neurological outcomes than patients with identical preoperative Frankel grades whose surgery was delayed beyond 12 hours. This large series of SEH demonstrates that rapid diagnosis and emergency surgical treatment maximize neurological recovery. However, patients with complete neurological lesions or long-standing compression can improve substantially with surgery.

KEY WORDS • neurological outcome • spinal cord compression • spinal epidural hematoma • surgical timing • surgical management

SPINAL epidural hematoma (SEH) is an uncommon clinical entity. Jackson¹⁵ was the first to describe it in 1869 and since then approximately 275 cases have been reported in the literature.^{9,16,20} There have been few large series of patients with SEH treated surgically, the largest of which consisted of 18 patients.¹³ The current report reviews our experience with the surgical management of 30 patients with SEH.

Patients with SEH are treated at this institution with rapid evacuation of the hematoma and decompression of the spinal cord. Controversy exists between those who advocate emergency surgery and those who operate on an urgent rather than emergency basis. Tarlov and colleagues²⁹⁻³² demonstrated experimentally in dogs that recovery of neurological function after spinal cord compression with an epidural balloon depends on the force and duration of compression and the rapidity of onset of motor deficits. McQuarrie²¹ confirmed these observations

in the clinical setting by showing that delay before surgery reduced the probability of recovery. Foo and Rossier⁹ reviewed the clinical literature and concluded that recovery did not depend on the timing of surgery but rather on the preoperative neurological condition of the patient, with better results in those with incomplete motor and sensory loss. We reviewed our experience with patients with SEH who were surgically treated and examined the relationship between surgical timing and neurological outcome.

Clinical Material and Methods

Thirty patients with SEH treated at Barrow Neurological Institute over a 14-year period (1979-1993) were identified and their medical records, histories, physical examinations, radiographs, operative reports, and pathology reports were reviewed. The diagnosis of SEH

was confirmed intraoperatively by the surgeon and histologically by the pathologist. Neurological function was evaluated pre- and postoperatively using the five-tier Frankel grading scale.¹⁰ For the purposes of statistical analysis, the alphabetical Frankel grades were assigned numerical scores, with Frankel Grade A being equal to 1, Grade B being equal to 2, and so on. Correlation of variables was analyzed statistically using Spearman rank correlation coefficients (one-tailed test). Patients were evaluated during office visits or by telephone interview or correspondence with primary care physicians.

Results

Patient Population and Presentation

Thirty patients with SEH were identified. The average age of the patients was 49.8 years (range 0.5 to 81 years), and there was a 2:1 male/female ratio. Spine surgery was the most common cause of SEH (12 patients). The initial operations consisted of five anterior cervical discectomies, one cervical vertebrectomy, one thoracic laminectomy for a spinal metastasis, two thoracolumbar instrumentations, and three lumbar laminectomies (two for stenosis and one for abscess evacuation). Postoperatively these patients developed new neurological deficits, which were present immediately after recovery from anesthesia in six patients and after a delay in another six patients.

Catheterization of the epidural space was the second leading cause of SEH (seven patients). One patient developed SEH after epidural anesthesia for childbirth and one after an epidural steroid injection for low-back pain. Five patients received anticoagulation medication with epidural catheters in place. All five patients had peripheral vascular disease: three were receiving anticoagulation therapy after thromboendarterectomy and two were receiving urokinase plus heparin for femoral artery occlusion.

Spinal epidural hematoma in the remaining patients was caused by vascular lesions (spinal arteriovenous malformation (AVM) in two patients, hemangioma in one, and epidural varix in one); anticoagulation medicine for medical disease (coumadin for phlebitis (one patient) and atrial fibrillation (one patient)), and heparin for transient ischemic attacks (one patient)); trauma (two patients); and spontaneous causes (two patients).

In 18 patients pain at the level of the lesion was the initial symptom. Neurological symptoms progressed to maximum deficits over variable periods of time: minutes (0 to 60) in eight patients, hours (1 to 24) in 13 patients, and days (1 to 4) in eight patients. In one patient timing intervals could not be accurately determined. The average interval from symptom onset to maximum deficit was 12.8 hours. Eight patients presented with complete motor and sensory loss (Frankel Grade A), six patients had complete motor loss but some sensation (Frankel Grade B), and 16 patients had incomplete loss of motor function (10 patients designated Frankel Grade C and six patients Grade D).

Diagnostic Evaluation

Plain radiographs and/or computerized tomography (CT) scans were obtained from trauma patients to identify fractures and dislocations and from postoperative patients

TABLE 1
Percentage of posterior nerve roots cut

Study Group	Posterior Nerve Root Transection (%)		Statistical Analysis (t-test)
	Spastic Quadriplegic Patients	Spastic Diplegic Patients	
Phase 1	68.1	65.0	t = 1.75, p = 0.09
Phase 2	68.5	64.7	t = 1.75, p = 0.09
Phase 3	56.6	47.4	t = 2.84, p = 0.008

to ensure proper placement of bone grafts or instrumentation. One trauma patient had fractures involving the posterior vertebral elements at the level of the SEH; none of the postoperative patients had graft or hardware malposition. The diagnosis of SEH was made using magnetic resonance (MR) imaging in 13 patients and myelography with postmyelogram CT scan in 14 patients. Three patients with new postoperative deficits underwent surgical reexploration without having had a diagnostic study.

The lesion was located in the cervical region in eight patients, cervicothoracic in three patients, thoracic in 10 patients, thoracolumbar in four patients, and lumbar in five patients. The average number of segments was 4.5 (range one to 14). The hematoma was located ventrally in the canal in 36% and dorsally in 64% of patients. In all patients the clinical level of the lesion matched the radiographic level.

Surgical Treatment

All patients were treated with surgical evacuation of the hematoma. In patients who had previously undergone surgery the original site was reexplored. In other patients the hematoma was approached through a laminectomy. The average interval from symptom onset to surgery was 23.4 hours (range 1.7 hours to 5.5 days). The average time interval from maximum deficit to surgery or the duration of maximum deficit was 10.7 hours (range 0 hours to 4.5 days).

Four patients developed a recurrent SEH that required another operation to relieve neural compression. In three of these patients an underlying vascular lesion was subsequently discovered (two patients had spinal AVMs and one had a hemangioma). The fourth patient had a spontaneous hematoma.

Patient Outcome

Surgical evacuation of SEH resulted in neurological improvement in 26 patients (mean follow up 11 months; range 6 weeks to 4.7 years) (Table 1). Twenty-six patients (87%; with a 95% confidence interval, 75% to 99%) had functional neurological recovery (Frankel Grade D or E), and 13 patients (43%; with 95% confidence interval, 26% to 61%) had complete neurological recovery (Frankel Grade E).

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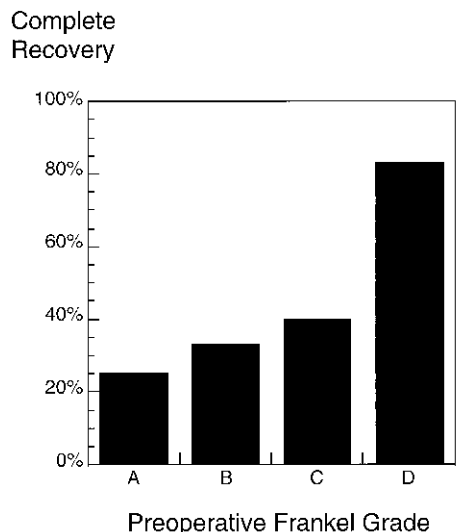


FIG. 1. Bar graph showing the relationship between preoperative Frankel grade¹⁰ and complete recovery rate (percent of patients who improved to Frankel Grade E).

Four patients had no improvement in neurological function after surgery. The first patient presented with complete motor and sensory loss (Frankel Grade A) after heparin-induced SEH and died 9 days after surgery from a pulmonary embolus. The second patient was involved in a motor-vehicle accident and presented with a complete spinal cord injury and an intracranial subdural hematoma. Craniotomy for this lesion delayed diagnosis of the SEH, and there were multiple associated posterior vertebral fractures at the site of the SEH to suggest an underlying primary spinal cord injury. The third patient had sepsis and coagulopathy and developed SEH after laminectomies for an epidural abscess. Failure to improve may have been due to spinal cord compression from the abscess rather than from the SEH. The fourth patient had failed back syndrome and developed SEH after his fourth back operation. The diagnosis of SEH was not made until 5 days after surgery and the chronic nature of his complaints, together with his stable neurological condition (Frankel Grade D), may have delayed diagnosis.

Neurological outcome correlated with the severity of the preoperative neurological deficit (Spearman rank correlation coefficient = 0.43). A larger proportion of patients with good preoperative Frankel grades recovered completely, ranging from 25% of Grade A patients to 83% of Grade D patients (Fig. 1).

Relationship of Surgical Timing and Neurological Outcome

Three time intervals were examined: 1) interval from initial symptom onset to maximum deficit; 2) interval from symptom onset to surgery; and 3) duration of maximum deficit. The interval from symptom onset to surgery correlated inversely with neurological outcome (Spearman rank correlation coefficient = -0.39). The average postoperative Frankel grade (in numerical equivalents) decreased from 4.7 in patients operated on in less than 6 hours to 3.7 in patients operated on after more than 24 hours (Fig. 2 upper), and there was a corresponding

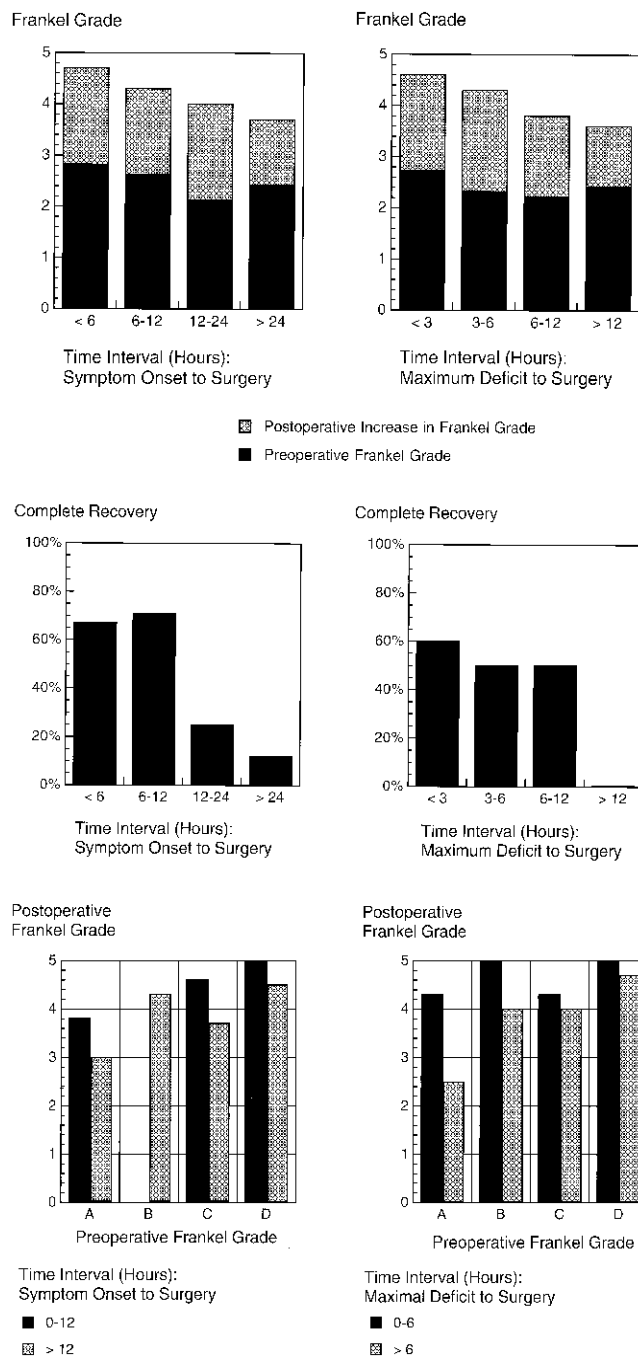


FIG. 2. Graphs showing the relationship between surgical timing and neurological outcome. Surgical timing is expressed as the time interval from symptom onset to surgery in the graphs on the left and from onset of maximum deficit to surgery in the graphs on the right. For the purposes of statistical analysis, the alphabetical Frankel grades¹⁰ were assigned numerical scores (A = 1; B = 2; C = 3; D = 4, E = 5), which appear on the Y axes in the upper and lower graphs of this figure. Upper: Neurological outcome is expressed as the average Frankel grade at late follow up. Center: Neurological outcome is expressed as the percent of patients who recovered completely (Frankel Grade E). Lower: Relationship of preoperative to postoperative neurological condition is presented. Neurological outcome is expressed as the average Frankel grade at late follow up. Note: all Frankel Grade B patients had intervals from symptom onset to surgery greater than 12 hours.

decrease in complete recovery rates from 67% to 12% (Fig. 2 *center*). A similar inverse correlation was observed between duration of maximum deficit and neurological outcome (Spearman rank correlation coefficient = -0.46) (Fig. 2 *upper and center*).

There was no correlation between the rate of symptom progression and neurological outcome; average postoperative Frankel grades were similar regardless of the rate of symptom progression, and no trends emerged from analyzing rates of complete recovery.

The relationship between surgical timing and neurological outcome was analyzed in groups of patients with the same preoperative Frankel grades. Patients with the same preoperative neurological status taken to surgery less than 12 hours from the onset of symptoms had higher average postoperative Frankel grades and higher complete recovery rates than those taken to surgery after 12 hours (Fig. 2 *lower*). Similarly, patients whose maximum deficit lasted less than 6 hours had increased average Frankel grades postoperatively and higher complete recovery rates.

Discussion

Etiology of Spinal Epidural Hematoma

There are many causes for SEH, including coagulopathy, trauma, vascular lesions, and spontaneous causes.^{5,6,11,19,22,33,35} The iatrogenic nature of SEH in this series of patients is striking. Spine surgery, epidural catheterization, and anticoagulation therapy accounted for 73% of the hematomas. The exact incidence of this complication with these various therapeutic interventions is difficult to ascertain. However, the incidence of postoperative SEH is exceedingly low considering that during a 14-year study period at this institution, which has a high volume of spine surgery (estimated 10,500 cases), it was seen in only 12 patients (0.1%). The risk of SEH with epidural catheterization and anticoagulation therapy has been reviewed and it is also low.^{2,7,23,25} Our experience with SEH demonstrates that procedures in the epidural space can cause SEH and that the risk is increased when anticoagulation medications are involved.

Diagnostic Evaluation

The time interval between onset of symptoms and surgery is determined by how long it takes a patient to recognize symptoms and enter into the medical system, and how long it takes physicians to recognize clinical signs and obtain radiological studies. Although physicians can do nothing about the patient's response time, they can accelerate the diagnostic evaluation, thereby minimizing surgical delay.

The differential diagnosis of patients with intense neck or back pain and progressive neurological deficits is extensive and includes other intradural hemorrhages, spinal cord compression from tumors, disc herniations, infectious masses, spine fractures, inflammatory conditions, spinal cord infarction, aortic dissection, and others.⁵ Despite this long list, the radiographic evaluation is straightforward: the spinal cord corresponding to the clinical level of the lesion must be imaged with either MR or myelography to determine whether it is compressed and whether surgery is indicated.

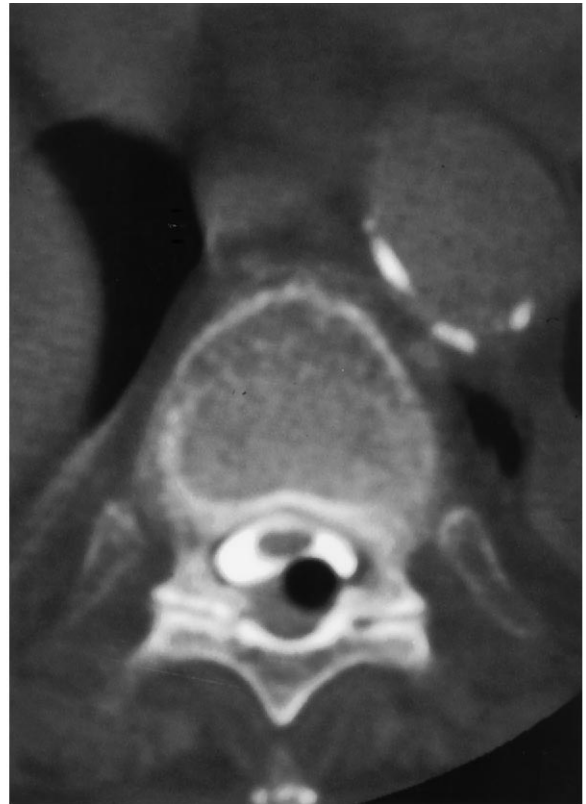


FIG. 3. Computerized tomography (CT) scan in a 75-year-old woman who presented with back pain and paraplegia after an epidural steroid injection for pain control. The CT scan demonstrates epidural soft-tissue attenuation and air dorsal to the thoracic thecal sac. Mass effect on the thecal sac is well visualized.

Radiological Features

Although occasionally an acute SEH may be detected on routine CT of the spine,³⁴ CT myelography is the classic diagnostic study (Fig. 3).¹⁸ The imaging appearance of an extradural lesion is typically nonspecific, and thus hemorrhage usually cannot be distinguished from neoplasm or infection. However, the degree of canal compromise and spinal cord compression is easily visible on CT myelography images, making this study adequate for surgical decision making. More recently, MR imaging has replaced CT myelography as the screening examination of choice for the diagnosis of SEH (Fig. 4).^{1,4,24,26} The multiplanar capability of MR imaging allows parasagittal imaging of the region of interest, which elegantly depicts the extent of the SEH and the degree of cord compression and is sufficiently sensitive for the detection of associated spinal cord edema. Except in cases in which infection must be excluded, gadolinium-enhanced imaging is usually not required.²⁷ In patients being evaluated for spinal cord trauma, MR imaging also allows detection of related injuries.¹⁷

Unlike CT, the MR signal characteristics of various ages of hemorrhage are relatively unique, which can lead to a more specific diagnosis than that obtained by CT myelography. Acute hemorrhage is characterized by a

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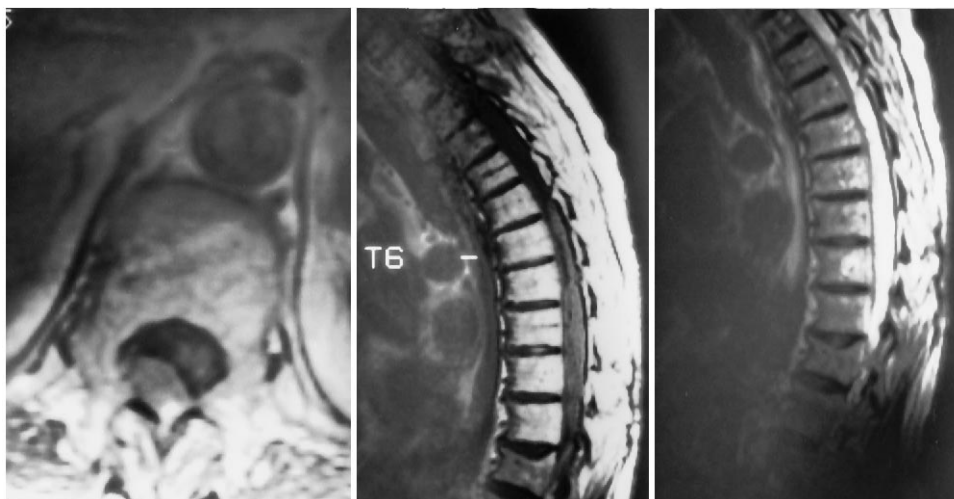


FIG. 4. Magnetic resonance images in an 81-year-old woman who underwent thromboendarterectomy of the lower-extremity vessel with epidural anesthesia and heparinization and presented with back pain and paraplegia. *Left:* Axial T_1 -weighted image demonstrating a lentiform thoracic epidural collection dorsal to the thecal sac, with associated cord displacement. *Center:* Parasagittal T_1 -weighted image to the right of midline demonstrating part of the craniocaudal extent of the hemorrhage. *Right:* The hyperintense hemorrhage is difficult to distinguish from cerebrospinal fluid on the T_2 -weighted image.

marked decrease in signal intensity on T_2 -weighted images. The subacute hematoma demonstrates increased signal intensity on both T_1 - and T_2 -weighted images. Specialized sequences such as gradient-echo methods can alter the contrast.

Angiography is not used routinely in the diagnostic evaluation of SEH unless the appearance of tortuous vessels on MR imaging or myelogram raises the suspicion of a vascular malformation. If an abnormal vascular lesion is found at surgery, an angiogram should be obtained post-operatively to demonstrate complete resection because residual lesions tend to rehemorrhage, as seen in three patients in this series.

Factors Affecting Neurological Outcome

Spinal cord compression has been studied in laboratory animals in terms of: 1) force of compression; 2) duration of compression; and 3) rate of compression.^{8,29-32} Tarlov and Klinger³¹ demonstrated with epidural balloons that recovery from spinal cord compression in dogs depends on the magnitude of the compressive force and the duration of compression. Large forces were tolerated up to 1 minute, whereas small forces were tolerated up to 2 hours. When these time limits were exceeded full recovery did not occur, and when the spinal cord was compressed gradually rather than acutely these time limits for recovery increased.²⁹ Dolan, *et al.*,⁸ used a clip compression model in rats to show that as the force and duration of spinal cord compression were independently increased, functional recovery was correspondingly reduced.

Spinal epidural hematoma represents a clinical situation that resembles these experimental models, because the hematoma compresses the spinal cord in a similar manner. However, in the clinical setting it is difficult to accurately measure force, duration, and rate of compression. The time interval from symptom onset to maximum neurological deficit approximates the rate of cord compression.

^{29,31,32} The time interval from symptom onset to surgery, or from maximum deficit to surgery, approximates the duration of compression.²¹ This study examined these clinical time intervals to determine if the correlation between the timing of decompression and outcome is as valid clinically as it is experimentally.

Rapidity of surgical decompression of the spinal cord correlated with neurological outcome in this patient series. Our findings are consistent with other clinical reports describing this relationship.^{12,21} However, Foo and Rossier⁹ reviewed 158 cases of SEH in the literature and found no such relationship. In their review, they compared outcomes in patients whose time to surgery was either less than or greater than 36 hours, so that patients who underwent "early" surgery had significant treatment delay, which may have diminished their outcome. Their meta-analysis was reexamined with patients treated in less than 36 hours, further subdivided into groups treated in 0 to 12 hours (23 patients), 12 to 24 hours (24 patients), and 24 to 36 hours (13 patients); complete motor recovery was seen in 52%, 25%, and 23%, respectively. Therefore, the meta-analysis of Foo and Rossier demonstrates that patients treated in less than 12 hours did indeed have better outcomes and those treated in 12 to 36 hours had outcomes consistent with what was observed in the late treatment (> 36 hours) groups. It is difficult to establish absolute time limits to recovery in patients with SEH. We have observed neurological recovery even after long delays, and as a result, cannot establish an absolute time limit. Neurological recovery is multifactorial, and surgical timing appears to be a critical factor. The importance of decompressing the spinal cord rapidly is evident from our data. It must be noted, however, that although this patient series is the largest reported, the sample size is small and the trends seen in Fig. 2 are based on smaller subgroups.

The rate of spinal cord compression did not correlate with neurological outcome in our experience. Outcome

was not improved in patients whose symptoms progressed gradually (over hours or days) compared with those whose symptoms progressed acutely (over minutes). When the rate and duration of compression were analyzed together,²⁹ there was nothing to suggest that the rate of symptom progression affected outcome.

The clinical effects of compressive force on neurological outcome cannot be determined in the manner used in experimental models because force cannot be measured directly in the clinical arena. Indirect measures of force, such as the size of the hematoma and the degree of cord displacement have been analyzed, and patients with smaller lesions that minimally displace the cord tend to recover greater function.⁹ The hematoma that arises from venous⁵ or from small arterial bleeding³ may be better tolerated than other lesions whose compressive force arises from trauma.

Factors other than surgical timing affect outcome, most notably a patient's preoperative neurological status.^{9,28,30} In this study, patients with fewer neurological deficits preoperatively had improved recovery of neurological function postoperatively. It is important to note that of the eight patients with complete loss of neurological function (Frankel Grade A), six improved with decompression and two of these regained normal function, thus illustrating that although preoperative neurological status generally predicts outcome, surgery is not contraindicated in SEH patients with poor preoperative Frankel grades. Although many patients have maximum deficits when they present to the neurosurgeon (and rarely does a patient improve spontaneously), most patients present with deteriorating neurological examinations. In these cases rapid intervention is important because loss of function is minimized preoperatively and, in turn, outcome is improved.

Recovery of neurological function can be understood from the microscopic anatomy of spinal cord injury. Harrison and McDonald¹⁴ showed that increasing spinal cord compression in cats resulted in loss of function, as determined by somatosensory evoked potentials. Functional recovery was associated with demyelination in spinal cord white matter. In contrast, failure to recover was associated with axonal disruption and degeneration. Therefore, the patient with an SEH who recovers after surgical decompression probably had a demyelinating lesion, whereas the patient who does not recover after surgery had an axotomizing lesion.

Conclusions

Spinal epidural hematomas tend to develop after therapeutic anticoagulation and surgical procedures involving the epidural space. Patients with suspected SEH should be rapidly evaluated using MR imaging or CT myelography. This large series of patients with SEH demonstrates a relationship between surgical timing and neurological outcome. Outcome correlates inversely with the time interval from symptom onset to surgery and the duration of maximum deficit, which both reflect the duration of spinal cord compression. Outcome also correlates with the severity of preoperative neurological deficits. Immediate surgical evacuation of the hematoma is recommended. Surgery can be performed safely (mortality 3%, neurological morbidity

0%), with neurological improvement expected in 87% of patients. Complete recovery (Frankel Grade E) was observed in 43% of patients and functional recovery (Frankel Grade D or E) was observed in 87%. Even patients with complete motor and sensory deficits or long-standing compression can improve with surgery.

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Manuscript received January 28, 1994.

Accepted in final form August 8, 1994.

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