Angiographic study of the effect of laminectomy in the presence of acute anterior epidural masses

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Laminectomies were performed in 16 monkeys to decompress simulated acute epidural masses in front of the spinal cord. When decompression restored normal arterial and venous hemodynamics, the monkeys were neurologically intact in spite of considerable mechanical distortion of the cord. When either the anterior spinal artery or the posterior spinal vein remained obstructed following laminectomy, the monkeys were paraplegic. Acute anterior epidural masses larger than 4 mm in diameter could not be adequately decompressed via the posterior approach. Only minor posterior displacement of the cord is observed following laminectomy in the presence of large anterior masses.

KEY WORDS • acute anterior epidural masses • laminectomy • spinal cord angiography • spinal cord decompression

Acute anterior epidural masses such as massive central disc protrusion and bursting vertebral fractures may be decompressed by laminectomy or via an anterior approach. Proponents of anterior decompression suggest that laminectomy may not adequately decompress the cord in the presence of large anterior epidural masses. The following study was undertaken in monkeys with simulated acute anterior epidural masses to evaluate the effect of laminectomy on spinal cord displacement and blood flow as visualized angiographically.

Sixteen rhesus monkeys weighing 4 to 7 kg were placed supine, sedated with 1 mg/kg of phencyclidine hydrochloride (Sernylan), and anesthetized with frequently repeated intravenous doses of a short-acting barbiturate, sodium thiamylal (Surital). Through a femoral artery cutdown, a catheter was selectively positioned, usually at the level of L-2, in the lumbar artery giving origin to the artery of Adamkiewicz. A control lateral spinal cord arteriogram was performed by injecting 2 ml of methylglucamine iothalamate (Conray). All arteriograms were magnified threefold with a 0.1 mm grid-biased focal spot tube. Filming was continued for 12 seconds

*Sernylan manufactured by Bio-Ceutic Laboratory, Belt Boulevard, St. Joseph, Missouri 64502.

‡Surital manufactured by Parke-Davis, 2211 East Jefferson Street, Detroit, Michigan 48207.

§Conray 60 manufactured by Mallinckrodt, Incorporated, Pharmaceutical Division, Diagnostic Products Division, P.O. Box 5439, St. Louis, Missouri 63147.

§Dynamax 66-A grid-biased focal spot tube manufactured by Machlett, 1063 Hope Street, Stanford, Connecticut 06907.
FIG. 1. Drawing showing measurements used for evaluation of postlaminectomy posterior spinal cord displacement. A = Distance from anterior surface of balloon to posterior spinal vein. B = Distance from posterior vertebral body to posterior spinal vein.

FIG. 2. Graph comparing the neurological status of the 11 monkeys with the diameter of the anterior mass.

to record the venous phase adequately. Occasionally an anteroposterior arteriogram was also obtained. The catheter was always removed from the lumbar artery between injections.

Following the control study, a No. 4 French Fogarty balloon catheter** was percutaneously introduced into the spinal canal and positioned in front of the cord. Opaque silicone rubber was introduced into the balloons over a 1- to 2-minute period, inflating them to diameters ranging from 3.4 to 6.2 mm (unmagnified direct measurement from specimen), and thereby providing a spectrum of epidural masses from moderately indenting to grossly compressing masses. On the basis of our past experience with this model, all masses were of sufficient size to cause paraplegia in the absence of operative intervention. When the silicone rubber had polymerized for 5 to 10 minutes, the catheter was transected and allowed to retract below the skin to prevent displacement of the balloon during subsequent laminectomy or by the awakened animal. Spinal cord arteriography in the lateral projection was repeated for comparison with the control study.

The animal was then turned prone and a three-level laminectomy centered over the balloon was performed to decompress the cord. The interval between inflation of the balloon and completion of the laminectomy was usually 1 hour but never longer than 2 hours. Spinal cord arteriography was repeated following laminectomy with the monkeys supine to compare displacement of the cord with the control and the prelaminectomy studies. The measurements illustrated in Fig. 1 were made on normal and paretic monkeys to evaluate posterior displacement of the cord following laminectomy.

Monkeys were observed for 24 hours and their neurological deficit graded according to the following system: Grade 0 = normal; Grade 1 = able to move legs but unable to bear weight; and Grade 2 = paraplegia, spastic or flaccid. At 24 hours (at 48 hours on two occasions), the animals were reanesthetized, selective spinal cord arteriography was repeated, and the spinal vessels were perfused through the selective catheter with opaque silicone rubber for microangiographic studies. The animals were sacrificed, the thoracolumbar vertebral column was removed and fixed in formalin for 6 to 10 days, and the spinal cord was then removed for microangiographic studies.

Results

In a single animal early in our series, laminectomy was performed at the wrong level and that animal was excluded from sub-

**Balloon catheter manufactured by Edwards Laboratory, Division of American Hospital Supply Corporation, P.O. Box 11150, 17221 Red Hill Avenue, Santa Ana, California 92705.
sequent analysis. Fluoroscopic monitoring of the level of laminectomy during the remainder of the studies eliminated repetition of this error. In one monkey, the balloon was accidentally placed within the cord. This animal was paraplegic at 24 hours and was also excluded from the series. In three monkeys, large anterior balloons (4.5 mm, 4.9 mm, and 5.5 mm) slipped into the lateral gutter at the time of laminectomy. The configuration of the lumbar canal and the shape of the cord tend to favor lateral displacement of large anterior balloons. At 24 hours two of these animals were monoplegic on the side of the balloon and the third showed bilateral weakness (Grade 1). All three were excluded from the study.

There remained for analysis 11 monkeys with acute anterior epidural masses, adequate laminectomies, clinical and angiographic evaluations at 24 hours, and perfusion studies. The diameter of the balloons is compared with the neurological status at 24 hours in Fig. 2. Previous experience with this model has established that without decompression all these anterior epidural masses would result in flaccid (Grade 2) paraplegia. Laminectomy appeared to favorably influence recovery of cord function (Grades 0 and 1) when the anterior epidural mass was less than 4.5 mm in diameter. Larger acute anterior masses, on the other hand, could not be adequately decompressed by a posterior approach since these animals remained paraplegic (Grade 2) in spite of extensive laminectomies.

In the two monkeys with no neurological deficit (Grade 0), the anterior spinal artery was displaced posteriorly but was never obstructed by the balloon. Before laminec-
Fig. 5. Left: Control spinal arteriogram, venous phase. Note the multiple medullary veins emptying into the posterior spinal vein. Center: Prelaminectomy arteriogram, venous phase, shows thinning of posterior spinal vein (arrowheads) behind balloon and absent medullary veins. Right: Postlaminectomy, posterior spinal vein (arrowheads) is better filled but not normal (compare with control). Although cord appears displaced posteriorly, this is largely due to increased flexion of the vertebral column.

tomy, there was always marked thinning or non-filling of the posterior spinal vein behind the balloon (Fig. 3 left). Immediately following decompression, posterior migration of the cord, as outlined during the venous phase, was minimal, but filling of the posterior veins returned to normal (Fig. 3 right). At 24 hours, opacification of the posterior spinal vein was normal in these neurologically intact monkeys. Microangiographic studies showed normal perfusion of the intrinsic cord vasculature at the level of the balloon in spite of mechanical distortion of the cord.

In the four monkeys with postlaminectomy paraparesis (Grade 1), the anterior spinal artery showed more marked posterior displacement and was in one instance narrowed but was never obstructed (Fig. 4). Posterior veins behind the balloon failed to fill or were markedly attenuated before laminectomy. After surgical decompression, posterior venous filling improved but never returned to normal even on the 24-hour studies (Figs. 5-7). Microangiographic studies were normal in three monkeys and showed decreased perfusion of the sulcocommissural arteries beneath the balloon in one.

In the five monkeys with persistent paraplegia following laminectomy, two showed obstruction of the anterior spinal

Fig. 6. Control (left), prelaminectomy (center), and postlaminectomy (right) studies demonstrate absent filling of the posterior spinal vein before decompression (arrows, center). Normal filling (right arrow) is seen following laminectomy (right). Distance between opposing arrows (left and right) remains unchanged indicating no posterior displacement of posterior spinal vein by balloon.
Laminectomy for anterior cord masses

artery by a large anterior balloon both before and after decompression (Fig. 8). In two, complete obstruction of the anterior spinal artery was relieved following laminectomy but prolonged (> 12 seconds) anterior spinal artery opacification and total absence of venous filling persisted. One monkey demonstrated a patent anterior spinal artery (Fig. 9), but prolonged arterial and absent venous filling behind the balloon both before and after laminectomy. Microangiograms in these five monkeys were abnormal with poor sulcocommissural artery perfusion at and below the level of the balloon.

In order to evaluate the magnitude of posterior cord migration following laminectomy, the distances illustrated in Fig. 1 were measured and corrected for magnification on the control, prelaminectomy, and postlaminectomy studies of the normal (Grade 0) and paretic (Grade 1) animals. When the posterior spinal vein directly behind the balloon was not opacified on prelaminectomy studies, its position was estimated by connecting cranial and caudal segments of the opacified vein. The results are presented in Fig. 10. Since the distance from the balloon to the posterior spinal vein (A in Fig. 1) added no further information, only the distances from the vertebral body to the posterior spinal vein (B in Fig. 1) are charted. The average posterior displacement is 0.7 mm and, with a single exception, all posterior migration occurred at the time of balloon inflation, not at the time of laminectomy. Removal of the posterior vertebral arch resulted in improved filling of the posterior spinal vein but the decompression was not generally associated with further retrodisplacement of the cord.

Discussion

This experimental study simulates an uncommon but serious neurosurgical problem, the acute anterior epidural mass. Chronic, slowly enlarging anterior masses, such as hypertrophic spurs, are a more common clinical problem with a totally different pathophysiological evolution: our conclusions apply only to acute anterior cord compression.

We were surprised to observe so little posterior migration of the cord following decompression. We had anticipated that an obvious retrodisplacement of the cord would be apparent on immediate or 24-hour postlaminectomy films. Distances from the posterior vertebral body to the back of the cord behind the balloon were measured before and after laminectomy, and subtracted radiographs of pre- and postlaminectomy angiograms were superimposed; the position of the posterior spinal vein within the spinal canal following laminectomy was shown to have remained unchanged in five of the six monkeys that appeared to benefit clinically from the decompression. The salutary effects of unroofing the spinal canal were clinically apparent in this study but could not be related to a simple posterior displacement of the cord.

When the cord is acutely compressed from the front, the earliest angiographic finding is a thinning or non-filling of the posterior spinal vein behind the anterior epidural mass. The anterior spinal artery, although displaced posteriorly, is more resistant to acute compression and is invariably patent at this early stage. Localized non-filling of the posterior

Fig. 7. Left: Specimen radiograph shows extent of laminectomy and position of balloon in monkey shown in Fig. 6. Right: Microradiography shows deep anterior indentation of the cord but normal filling of the anterior spinal and sulcocommissural arteries.
FIG. 8. Anteroposterior (upper left) and lateral (upper center) selective spinal arteriograms following laminectomy show complete obstruction of anterior spinal artery (large arrowheads) at level of balloon. Small arrowheads indicate artery of Adamkiewicz which in lateral view could be confused with anterior spinal artery below balloon. Animal was flaccidly paraplegic. Anteroposterior (upper right) and lateral (lower left) radiographs and anteroposterior (lower center) and lateral (lower right) microradiographs document the complete obstruction.
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Fig. 9. Arterial (left) and venous (center) phases of selective spinal arteriogram after laminectomy demonstrate patent anterior spinal artery (arrowheads, left) but absent filling of posterior vein behind balloon (arrows, center). Perfusion study (right) reveals non-filling of sulcal arteries beneath balloon. Monkey remained paraplegic.

vein could either be due to compression of the vein against the dura and lamina of the spinal canal or arrest of the microcirculation through the compressed cord segment with secondary reduced venous filling. In spinal arteriograms of normal monkeys, one can see multiple medullary veins draining into the large posterior spinal vein. (Fig. 5 left) Opacification of this latter vessel depends upon circulation through the cord as well as upon ascending flow from below, the normal direction of spinal venous drainage. In normal studies, the posterior vein always opacifies earliest and most densely behind the richly arterialized lumbar enlargement. If a segment of the cord is acutely compressed to the point of compromising transmedullary flow, non-opacification of the corresponding posterior spinal vein might be the earliest angiographic finding. The persistence of poor venous filling after laminectomy in paretic and paraplegic monkeys suggests that reduced circulation through the compressed segment of cord, that is, microcirculatory failure, is the pathophysiological mechanism. However, from these studies, it is impossible to exclude posterior venous compression with secondary intramedullary congestion as the primary pathophysiological mechanism. Historically, it is of interest that Brain, in one of the earliest discussions on anteroposterior compression of the cord, suggested that venous congestion might be more important than arterial insufficiency.
This study also demonstrates that acute anterior epidural masses can be too large for adequate decompression by laminectomy. When the anterior epidural mass obstructed the anterior spinal artery, laminectomy never restored normal spinal cord hemodynamics: these animals remained paraplegic in spite of adequate posterior decompression. Whether deflating the balloon after 1 hour, equivalent to an anterior decompression, would result in better functional recoveries than laminectomy in this group of animals is currently under investigation.

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References


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