Hemodynamics of spinal dural arteriovenous fistulas

An intraoperative study

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Local hemodynamics were investigated during nine operations for spinal dural arteriovenous (AV) fistulas. In eight cases, microvascular Doppler sonography was used to measure flow velocities and vasomotor reactivity to CO2 changes. Intravascular pressure recordings of the draining veins on the medullary surface were performed in nine cases. The flow velocities in dural AV fistula feeding vessels were not as high as has been shown in cerebral angioma feeders. The AV fistula feeders often showed low end-diastolic flow velocities as a sign of increased vascular resistance, even in the presence of a downstream AV fistula, thus proving disturbance of venous outflow from the spinal canal. After excision of the fistula, the circulation of the spinal cord vessels improved, with higher inflow and outflow velocities. In the veins formerly draining the fistula, no further flow could be recorded; however, they did not collapse, indicating that some pressure remained. The mean venous pressure in the dural AV fistulas was about 74% of the systemic arterial pressure. It increased concomitantly with the arterial pressure, which may explain the clinical deterioration that occurs during physical activity. Fistulas with a high shunt volume on angiography showed only moderately increased venous pressures and a more pronounced pressure drop compared to low-volume fistulas. The CO2 reactivity of vessels supplying the spinal cord could be demonstrated in all cases, and was normal before and after removal of the fistula.

Key Words • spinal cord arteriovenous malformation • CO2 reactivity • spinal dural arteriovenous fistula • autoregulation • hemodynamics

A spinal arteriovenous malformation (AVM) was reported 100 years ago by Gaupp13 from Tübingen. The lesion, which he described as "hemorrhoids of the pia mater," was probably a spinal dural arteriovenous (AV) fistula, based on the location and clinical signs. The first exact description of what we now call "dural fistula" was given by Brasch4 in 1900. In 1910, Krause11 performed the first surgical exposure of a spinal AVM, but the outcome was poor; the first successful removal with clinical improvement was achieved by Elsberg12 in 1916. Later, in 1926, the first preoperative myelographic diagnosis of spinal AVM was made by Perthes30 (also from Tübingen), and thereafter the malformation was successfully treated by surgery.

Recent studies of spinal AVM's have furnished evidence that one particular type of spinal vascular malformation or anomaly should be reevaluated.10,20,26,33 These malformations, previously classified as dorsal retromedullary angiomas, have been shown to be draining veins from pathological AV shunts with a nidus lying within the dural layer. In contrast to previous classifications of spinal AVM's, it has been suggested that these lesions be categorized into three types.15,33,37 Type I includes intradural AVM's supplied by medullary feeding arteries, with an intra- or extramedullary nidus (fistula), and drained by perimedullary veins (high-flow lesions). Depending on their morphological and angiographic characteristics, lesions in this category may be further divided into various subtypes such as glomerular, juvenile, or fistula types. Type II comprises dural AV fistulas supplied by radiculomeningeal arteries, with the nidus in the dural layer, and drained by perimedullary veins (low- to high-flow lesions). Type III includes extradural AVM's with possible intraspinal extension but which are supplied and drained exclusively by extradural vessels (low- and high-flow lesions).

The various vascular malformations of the spinal cord and spinal cavity differ in etiology, in the time of clinical presentation, in pathological, radiographic, and clinical features, and in location. They also respond differently to treatment.33,39

The clinical findings in cases of dural AV fistula are those of a slowly progressing myelopathy and/or radic-
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ulopathy (that is, spastic or flaccid paraparesis, muscle atrophy, sphincter disturbances, and dorsal-column sensory lesions). These signs result from the presence of often combined lesions of the first and second motor neurons plus transverse lesions at different levels. The symptoms are thought to result from high venous pressure with a reduced AV pressure gradient and, consequently, reduced intramedullary blood flow leading to hypoxic damage. Other possible mechanisms as a cause of clinical deterioration have been discussed elsewhere. These include compression of the cord (mass effect) from dilated vessels, a pulsatile compression mechanism, ischemia due to "parasitic circulation" or a steal phenomenon, subarachnoid or intramedullary bleeding, arachnoiditis, and thrombosis. Compression effects, steal phenomena, and hemorrhage may be factors in the pathogenesis of true intradural AVM's, but they certainly are not the cause of clinical deterioration in cases of dural AV fistula. Rather, in dural AV fistulas, which are probably acquired anomalies, the high venous pressure is presumed to be the cause of clinical symptoms.

Until now, pressure and flow velocity in spinal dural AV fistulas have not been measured. The present study was undertaken to elucidate the hemodynamic conditions of these malformations. Doppler sonography was used to measure blood flow velocities in feeding and draining vessels before and after surgery.

Clinical Material and Methods

Intraoperative studies were performed in nine of 25 patients operated on in the last 5 years for spinal dural AV fistulas. Doppler and pressure measurements were carried out immediately after exposure of the angiographically localized fistula and during clip occlusion of the proximal part of the draining vein. In eight of the nine cases, Doppler sonographic examinations of blood flow velocity and CO2 reactivity were performed in vessels supplying the spinal cord and dural AV fistula. In all nine cases, additional recordings of intravascular pressure of the draining vein were made. After dissection and excision of the dural fistula, the measurements were repeated.

Intravascular Pressure Measurement

Simultaneous recordings of intravascular pressure were performed in the radiculomeningeal artery and in the proximal portion of the draining vein of the AV fistula. The vessels were punctured using a 0.45 x 13-mm needle, a diameter much less than half that of the vessel. Systolic, diastolic, and mean pressure values were recorded and displayed on a Hewlett-Packard monitor.* During and after clip occlusion of the proximal vein near the fistula, the stump pressure of the distal vein was measured.

Measurement of Blood Flow Velocity

The application of intraoperative Doppler ultrasonography for measurement of blood flow velocity in single intracranial vessels was first reported by Nornes, et al., who used 6- and 10-MHz pulsed systems. The present study employed a device originally developed by Cathignol, et al. The high-pulsed Doppler frequency (20 MHz) of our system provides good resolution. Pulse durations were 250, 450, and 850 nsec, axial resolution was 0.4, 0.7, and 1.3 mm, lateral resolution was 0.5 and 1.1 mm, and pulse repetition frequency was 25, 50, and 100 kHz. Depth of measurement could be adjusted in 0.1-mm steps to a maximum of 15 mm. Due to the filter arrangements, the upper limit of recording was at 12.5 kHz of Doppler shift. Doppler signals were evaluated by a real-time frequency analyzer.†

Intraoperative application of the miniaturized autoclavable probes, measuring 5 mm in length and 1 mm in diameter, was simple. The angle of insonation between the instrument-held probes and the vessel was set between 40° and 60°. This results in the recording of comparable relative flow velocities among patients. The probe position and gate depth were adjusted visually and under acoustic control of the signals until the highest frequencies could be obtained. The smallest vessels evaluated measured 0.5 mm in diameter. Use of this Doppler technique in neurosurgery has been described previously by Gilsbach and Hassler for operations on aneurysms and angiomia, and for extracranial-intracranial bypass procedures.

According to Pourcelot, the relationship between the systolic peak flow velocity and the end-diastolic velocity of an artery is indicative of the vascular resistance in the vessel's peripheral distribution. The ratio between end-diastolic (D) and systolic (S) velocity amplitude is normally 0.43:1 to 0.47:1 in intracranial arteries. According to Pourcelot's formula, in which R = (S - D)/S, the resistance index (R) depends mainly on the relative magnitude of the end-diastolic velocity. A low R value corresponds to a low resistance, and relatively elevated end-diastolic flow velocities indicate low peripheral vascular resistance.

Measurements of CO2 Reactivity of Blood Flow Velocity

Cerebral blood flow (CBF) is greatly influenced by the arterial CO2 acting upon the resistance vessels. Hypercapnia results in arteriolar dilatation with increasing CBF, whereas in hypocapnia the arterioles constrict and CBF decreases. Because the diameter of larger arteries does not react to CO2, the flow in these vessels accelerates during arteriolar dilatation (hypercapnia) and decelerates during arteriolar constriction (hypocap-
Changes in flow velocity in these vessels are therefore considered to correlate strongly with CBF changes at varying arterial CO₂ levels. Doppler studies of CO₂ reactivity represent a valuable method of assessing hemodynamic situations because they determine the vasomotor capacity of arterial systems.¹⁶,¹⁷ Until now, spinal blood flow regulation has not been investigated in humans but a mechanism similar to that in brain arteries can be presumed.

For the intraoperative studies of CO₂ reactivity, the end-tidal partial volume of CO₂ in the expired air was documented using infrared analysis.§ This parameter is widely accepted as an indirect measure of the PaCO₂. Doppler curves were documented each time the desired steady state of PaCO₂ had been reached. Flow-velocity measurements during hypercapnia and hypocapnia were performed at a PaCO₂ of 54 mm Hg and 23 to 24 mm Hg, respectively.

§ Normocap infrared analyzer manufactured by Datex Industries, Helsinki, Finland.

Results

Intravascular Pressure

Intravascular pressure of the intrathecal perimedullary draining veins was measured in nine spinal dural AV fistulas. Mean pressures ranged between 54 and 78 mm Hg, which is approximately 60% to 87.5% of the mean systemic arterial blood pressure (SAP) as measured in the radial artery. Pressure pulsations were much lower than in the systemic circulation (Figs. 1 and 2 and Tables 1 and 2). The venous pressure changed with the SAP. The pressure of the draining vein increased with increasing SAP and decreased with decreasing SAP (Fig. 1). After occlusion of the dural fistula the pressure in the veins that formerly drained it dropped markedly to levels between 9 and 45 mm Hg, which corresponds to between 16.4% and 64.3% of the former venous pressure (Figs. 2 to 4 and Table 1). The intravascular pressure and its relative changes did not depend on the segmental level of the dural fistula. Fistulas exhibiting a high shunt volume on the arteriograms showed only moderately increased venous pressures and a more
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FIG. 2. Pressure (p) measurements in a draining vein of a spinal dural arteriovenous fistula. Before occlusion of the fistula, the pressure in the draining vein (VP) was 81 mm Hg (1). After clip occlusion of the vein distal to the pressure-measuring point (2), the VP increased to 107 mm Hg. After removal of the clip, the VP dropped to the former values (1). Clipping of the vein proximal to the measuring point (3) caused the VP to drop to 23 mm Hg (resting pressure of VP after fistula occlusion). The systemic arterial pressure (SAP) measured in the radial artery did not change during these maneuvers. ABP = arterial blood pressure; CVP and ZVD = central venous pressure.

pronounced drop in pressure compared to low-volume fistulas. The initial clinical symptoms and the outcome after operation did not correlate with the height of the intraoperatively measured pressure. After fistula occlusion, SAP changes did not influence the “remaining pressure” in the former draining vein (Fig. 1).

Flow Velocities of AV Fistula Vessel System

Flow velocities in the feeding artery, fistula, and draining veins (proximal and distal to the fistula) are summarized in Table 3. Velocities of the arteries feeding the fistula were much higher than those of arteries supplying the spinal cord (Figs. 3 and 5). The typical valve-closure dicrotic incisura found in normal arteries was lacking in the feeding arteries, and the end-diastolic flow velocity was much higher. The end-diastolic flow velocity in some dural fistulas was lower than in cerebral angioma feeders. This indicates higher outflow resistance (low-flow fistula). Other fistula feeding arteries showed a high end-diastolic flow velocity as a sign of a lower resistance to venous outflow (high-flow fistula).
TABLE 1

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Location of AV Fistula</th>
<th>Estimated Shunt Volume†</th>
<th>Mean SAP (mm Hg)</th>
<th>VP (mm Hg) in Proximal Portion of Draining Vein</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Mean VP Before Occlusion</td>
<td>% of Former VP</td>
</tr>
<tr>
<td>1</td>
<td>62, M</td>
<td>T6-7, rt moderate</td>
<td>92</td>
<td>78</td>
<td>84.8</td>
</tr>
<tr>
<td>2</td>
<td>63, F</td>
<td>T7-8, lt low</td>
<td>67</td>
<td>54</td>
<td>80.6</td>
</tr>
<tr>
<td>3</td>
<td>52, M</td>
<td>T8-9, rt moderate</td>
<td>80</td>
<td>70</td>
<td>87.5</td>
</tr>
<tr>
<td>4</td>
<td>67, F</td>
<td>T10-11, lt moderate</td>
<td>91</td>
<td>56</td>
<td>61.5</td>
</tr>
<tr>
<td>5</td>
<td>62, F</td>
<td>T12-L1, rt high</td>
<td>90</td>
<td>54</td>
<td>60.0</td>
</tr>
<tr>
<td>6</td>
<td>72, M</td>
<td>T12-L1, rt low</td>
<td>90</td>
<td>70</td>
<td>77.8</td>
</tr>
<tr>
<td>7</td>
<td>76, M</td>
<td>L1-2, rt high</td>
<td>105</td>
<td>72</td>
<td>68.6</td>
</tr>
<tr>
<td>8</td>
<td>53, M</td>
<td>L2-3, lt high</td>
<td>90</td>
<td>55</td>
<td>61.1</td>
</tr>
<tr>
<td>9</td>
<td>60, M</td>
<td>L2-3, rt high</td>
<td>96</td>
<td>81</td>
<td>82.0</td>
</tr>
<tr>
<td>mean values</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>73.8</td>
</tr>
</tbody>
</table>

* AV = arteriovenous; SAP = systemic arterial blood pressure.
† Shunt volume was estimated by angiography.

TABLE 2

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Location of AV Fistula</th>
<th>Draining Direction</th>
<th>Draining Veins</th>
<th>Symptoms at Diagnosis</th>
<th>Duration of Symptoms (yrs)</th>
<th>Postop Outcome</th>
</tr>
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<tbody>
<tr>
<td>1</td>
<td>T6-7, rt cranial &amp; caudal &amp; posterior</td>
<td>anterior &amp; posterior</td>
<td>SP rt &gt; lt, STL, SD, walked with support</td>
<td>5</td>
<td>unchanged</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>T7-8, lt cranial &amp; caudal &amp; posterior</td>
<td>anterior &amp; posterior</td>
<td>SP rt &gt; lt, STL, SD, walked with support</td>
<td>2.5</td>
<td>unchanged</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>T8-9, rt cranial &amp; caudal &amp; posterior</td>
<td>posterior</td>
<td>slight SP lt, MAP, walked independently</td>
<td>1.5</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>T10-11, lt cranial &amp; caudal &amp; posterior</td>
<td>posterior</td>
<td>SP lt &gt; rt, STL, SD, unable to walk</td>
<td>6</td>
<td>unchanged</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>T12-L1, rt cranial &amp; caudal &amp; posterior</td>
<td>anterior &amp; posterior</td>
<td>SP rt &gt; lt, STL, SD, walked with minimal support</td>
<td>5</td>
<td>unchanged</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>T12-L1, rt cranial &amp; caudal &amp; posterior</td>
<td>anterior &amp; posterior</td>
<td>SP rt, STL, SD, unable to walk</td>
<td>2</td>
<td>unchanged</td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>L1-2, rt cranial &amp; caudal &amp; posterior</td>
<td>anterior &amp; posterior</td>
<td>SP rt &gt; lt, STL, SD, walked with support</td>
<td>1, acute onset</td>
<td>motor unchanged, sensory improved</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>L2-3, lt cranial &amp; caudal &amp; posterior</td>
<td>anterior &amp; posterior</td>
<td>SP lt &gt; rt, STL, SD, unable to walk</td>
<td>4</td>
<td>improved</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>L2-3, rt cranial &amp; caudal &amp; posterior</td>
<td>anterior &amp; posterior</td>
<td>SP rt &gt; lt, STL rt &gt; lt, SD, unable to walk</td>
<td>2</td>
<td>unchanged</td>
<td></td>
</tr>
</tbody>
</table>

* SP = spastic paraparesis; STL = sensory transverse lesion; SD = sphincter disturbances; MAP = muscle atrophy paresthesia.

TABLE 3

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Age (yrs), Sex</th>
<th>Location of AV Fistula</th>
<th>Feeding Artery</th>
<th>AV Fistula</th>
<th>Proximal Vein</th>
<th>Distal Vein</th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>FV of Sys-tole</td>
<td>FV of Dias-tole</td>
<td>Resistance</td>
<td>FV of Sys-tole</td>
</tr>
<tr>
<td>1</td>
<td>62, M</td>
<td>T6-7, rt</td>
<td>4.22</td>
<td>1.33</td>
<td>0.68</td>
<td>9.77</td>
</tr>
<tr>
<td>2</td>
<td>63, F</td>
<td>T7-8, lt</td>
<td>5.44</td>
<td>2.11</td>
<td>0.61</td>
<td>7.77</td>
</tr>
<tr>
<td>3</td>
<td>52, M</td>
<td>T8-9, rt</td>
<td>5.3</td>
<td>1.76</td>
<td>0.67</td>
<td>7.5</td>
</tr>
<tr>
<td>4</td>
<td>67, F</td>
<td>T10-11, lt</td>
<td>6.4</td>
<td>1.1</td>
<td>0.83</td>
<td>8.8</td>
</tr>
<tr>
<td>5</td>
<td>62, F</td>
<td>T12-L1, rt</td>
<td>6.0</td>
<td>1.66</td>
<td>0.72</td>
<td>7.77</td>
</tr>
<tr>
<td>6</td>
<td>72, M</td>
<td>T12-L1, rt</td>
<td>5.2</td>
<td>1.61</td>
<td>0.69</td>
<td>6.9</td>
</tr>
<tr>
<td>7</td>
<td>76, M</td>
<td>L1-2, lt</td>
<td>4.88</td>
<td>2.55</td>
<td>0.48</td>
<td>7.66</td>
</tr>
<tr>
<td>8</td>
<td>53, M</td>
<td>L2-3, lt</td>
<td>5.62</td>
<td>1.8</td>
<td>0.68</td>
<td>7.6</td>
</tr>
<tr>
<td>mean values</td>
<td></td>
<td></td>
<td>± 0.6± 0.4</td>
<td>± 0.1± 0.1</td>
<td>± 0.8± 1.1</td>
<td>± 0.1± 1.7</td>
</tr>
</tbody>
</table>

* Data are given for eight patients undergoing Doppler sonography. Flow velocity (FV) is measured in kHz. AV = arteriovenous; SD = standard deviation. Data for Cases 1, 4, and 7 correspond to Figs. 3, 4, and 5, respectively.
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The relationship between systolic peak and end-diastolic flow velocities was a sign of distal stream resistance in territories supplied by these arteries. A relatively high end-diastolic flow velocity indicates low stream resistance. According to this, one can differentiate between high-flow fistulas (Fig. 5), moderate-flow fistulas (Fig. 3), and low-flow fistulas (Fig. 4; see also Table 3). The stream resistance was low in high-flow fistulas (up to 0.48, Fig. 5), medium in moderate-flow fistulas (0.68, Fig. 3), and elevated in low-flow fistulas (up to 0.83, Fig. 4).

In the nidus (the shunt) itself the pressure drop was highest due to the abrupt change in the diameter of the whole vessel. The small-caliber meningoradicular feeder emptied into the larger draining vein. Therefore, the flow velocity was increased in the fistula region due to a reduction of stream resistance (Fig. 3). With increasing distance from the fistula, the flow velocity decreased due to the larger diameter of the draining vein. Further from the fistula, the flow velocity in the draining veins was lower, as were the systolic peaks (Figs. 3 and 4).

In most of the nine cases, no significant flow could...
be measured in the large draining veins after occlusion of the fistula (Fig. 4). After several minutes, the red color changed to blue. The diameter of the draining veins decreased slightly, but they did not collapse because of the remaining "resting pressure" in the venous system. This agrees with the assumption that blood from the shunt enters the normal pathways of spinal cord drainage.

After occlusion of the fistula, no further flow was measurable in the arteries feeding it. An exception was a rare dural fistula in which there was a common feeder to the fistula and spinal cord (Fig. 4). In such cases, a reduced flow velocity could be recorded which was directed only to the spinal cord supply.

**Flow Velocity in Vessels Supplying the Spinal Cord**

The arteries supplying the spinal cord showed normal arterial flow patterns with a valve-closure incisura after systolic peak and a low end-diastolic flow velocity (Figs. 3 to 5). The flow velocity was higher or lower depending on the diameter of the artery supplying the spinal cord (Figs. 3 and 5). In one case, we had the opportunity to
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FIG. 5. Flow velocities in a high-flow fistula and in the main spinal cord feeder (artery of Adamkiewicz). In contrast to medullary feeders, the diastolic flow velocity of vessels to and from the fistula was high. In this venous aneurysm, the flow was slow due to the enlarged diameter.

record one of the main feeders of the spinal cord: the artery of Adamkiewicz entering at the T-12 level, one segment above the fistula level. In this case, we could compare the different flow characteristics of a spinal cord feeder and a feeder to a high-flow fistula (Fig. 5).

After occlusion of a fistula, the flow velocity of the spinal cord arteries increased without changes in the shape of the flow profile (Figs. 4 and 6). The increase in flow velocity was seen in large as well as in small arteries supplying the spinal cord.

The veins draining the spinal cord showed a characteristic flow profile, with flow being measurable only in systole (Fig. 2). After occlusion of the fistula, the flow out of the spinal cord also increased (Figs. 4 and 6).

CO₂ Reactivity of Arteries Supplying the Spinal Cord

Flow velocities in the arteries supplying the spinal cord increased with increasing PaCO₂ (Figs. 6 and 7). Systolic flow velocities as well as the diastolic flow velocities were elevated as a sign of lowered stream resistance due to the widening of arterioles of the spinal cord tissue. With decreasing PaCO₂, forced by hyperventilation, the flow velocity decreased markedly as well. This is a sign of raised resistance in the distal supply area of these vessels due to constriction of arterioles. The present investigation showed that CO₂ reactivity of the arteries supplying the spinal cord was normal before fistula occlusion, indicating that the CO₂ vasomotor response seemed to be undisturbed (Figs. 6 and 7).

After occlusion and excision of the fistula, the flow velocity increased in the arteries supplying the spinal cord and the CO₂ response to flow velocity was unchanged (Fig. 6). From these facts one can assume that autoregulation of the spinal cord is not disturbed before or after fistula excision. Therefore, the fistula vessel system does not significantly influence the spinal cord supply, since obviously there are no intimate connections between them.

Discussion

Much confusion exists in the literature concerning the histological and angiographic nomenclature used to describe the varieties of spinal vascular malformations. Without knowledge of the site of the malformation nidus, dilated and elongated vessels on the cord surface were previously considered to represent a spinal angioma. This explains the misinterpretation and inappropriate treatment in those cases which are now classified as dural AV fistula. This most common type of AVM, with an incidence of 80%, is usually encountered in adults and is a fistula of either low or high flow, with the nidus located in the dura near the entry zone of the dorsal nerve root.
FIG. 6. The CO₂ reactivity of the main artery supplying the spinal cord (artery of Adamkiewicz) before and after arteriovenous fistula (AVF) excision. The CO₂ reactivity was undisturbed pre- and postoperatively, demonstrating intact vasomotor response.

It is the dural branch of the spinal ramus of the intercostal or lumbar artery that supplies the AV fistula. Blood flow through the fistula runs through a radicular vein in a retrograde manner to the coronal venous plexus, which becomes dilated and tortuous. Drainage in these veins can be followed up to the cerebral veins or, less frequently, caudally. During angiography there is a noteworthy stagnation of contrast medium in the varicose perimedullary veins (in most cases dorsally), with an absence of any normal epidural venous drainage. This venous dilatation and stagnation is explained by an outflow impairment to the epidural drainage system or blockage of venous return in the caval system. Steal effects have no impact because the arteries that supply the spinal cord are not involved. The basic pathogenic reason for clinical deterioration associated with spinal dural AV fistula is thought to be the elevated pressure in the draining vein caused by arterialization of the spinal cord veins and/or by outflow obstruction. Our measurements of intravascular pressure in the draining veins confirmed this assumption. The high pressure in the vein (70% of SAP) is sometimes the cause of venous aneurysms.

Although impairment of venous drainage from the spinal cord parenchyma is greatest at the level of the shunt, the spread of venous hypertension in the cranial and caudal directions causes damage to the cord over a long distance. Therefore, the location of the shunt (fistula) does not correlate very well with the level and site of clinical symptoms.

Measurements performed under intraoperatively induced systemic hypertension showed that pressure in the drainage vein increased also, resulting in reduced spinal cord circulation. This correlates well with the clinical finding of exacerbation of symptoms by physical activity. After excision of the fistula nidus, pressure in the dilated venous system decreased markedly to between 16.4% and 44.9% (mean 38.3%) of the former pressure. Arteriovenous fistulas with low shunt volume on angiography demonstrated the highest venous pressure before the lowest drop in pressure after shunt occlusion (Table 1). This indicates severe venous outflow obstruction in these cases, whereas drainage of blood from high-volume AV fistulas seems to be less impaired. Due to a remaining pressure that was relatively high, collapse of the former draining veins was not observed as in angioma surgery. In most cases, the Doppler sonographic examinations of veins after fistula occlusion showed no detectable flow signal, indicating “stagnating veins.” This may facilitate thrombosis and thus cause further damage to the spinal cord, which may explain the transient postoperative neurological deterioration in some cases. Treatment with anticoagulant agents after surgery for spinal AV fistulas must therefore be discussed.

Blood flow velocity in AV fistulas is characteristic of
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FIG. 7. The CO₂ reactivity of flow velocity of a small radicular artery supplying the spinal cord. With hypercapnia (up to 54 mm Hg), there was a large increase of systolic and diastolic flow velocity. With hypocapnia, the flow velocity decreased extraordinarily and no further flow was detectable during diastole.

the pathological structure of these lesions. Due to the dural nature of the arterial supply and to disturbed venous outflow, most fistula feeders show relatively high-resistance flow patterns with low diastolic flow, suggesting a low-flow lesion. This is in contrast to cerebral angioma feeders and true spinal AVM's in which high systolic and diastolic flow velocities can be recorded due to reduced vascular resistance.16,17,30

The angiographic and Doppler findings in our study revealed different flow velocities in the dural feeders, suggesting a variable obstruction of venous outflow. However, the varying flow velocities did not correlate with the number of dilated veins and the intensity of neurological disturbances, as suggested previously.23,26

Intraoperative Doppler sonography can easily demonstrate that excision of the fistula nidus or the draining vein has eliminated the pathological AV communications. Therefore, focusing the operative approach on the nidus is an adequate and simple method of surgical treatment.3,19,29,35,39 Stripping of veins by multilevel laminectomy is not only unnecessary and harmful21,22,24 but also removes the physiological draining vessels of the spinal cord.37 We prefer an operative excision rather than embolization in order to exclude the collateral supply and to preserve the small radicular spinal cord feeders, which may originate only a few millimeters outside the dura from a common meningo-radicular artery (Fig. 4).

Investigation of normal arteries supplying the spinal cord showed that their flow velocities were diminished preoperatively, since flow velocities increased after fistula occlusion. Thus, the cause of myelopathy in these patients cannot be the steal phenomenon, as reported previously. This concept is also confirmed by angiography. In most cases, there is no common feeder to the fistula and the spinal cord. In spite of the increased flow velocity in arteries supplying the spinal cord and draining veins after excision of the shunt, there was limited improvement in the hemodynamics. This finding could be expected and is a consequence of insufficient venous drainage.

The CO₂ reactivity test we performed on arteries that supply the spinal cord demonstrated that constricting and dilating mechanisms of the vessel lumen exist in the distal arterial distribution. This is because hypercapnia causes flow acceleration in large and small spinal cord-supplying arteries, whereas hypocapnia causes flow deceleration (Figs. 6 and 7). As compared to CO₂ reactivity studies in brain vessels supplying an angioma,16,17 the CO₂ reactivity of spinal cord arteries seems to be undisturbed. Therefore, the prognosis of these patients after treatment of their AV fistula seems to be related to the extent of problems in spinal venous circulation and to the degree of preexisting irreversible spinal cord damage.

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References