

## Modified classification of spinal cord vascular lesions

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The literature on spinal vascular malformations contains a great deal of confusing terminology. Some of the nomenclature is inconsistent with the lesions described. Based on the experience of the senior author (R.F.S.) in the treatment of more than 130 spinal cord vascular lesions and based on a thorough review of the relevant literature, the authors propose a modified classification system for spinal cord vascular lesions.

Lesions are divided into three primary or broad categories: neoplasms, aneurysms, and arteriovenous lesions. Neoplastic vascular lesions include hemangioblastomas and cavernous malformations, both of which occur sporadically and familiarly. The second category consists of spinal aneurysms, which are rare. The third category, spinal cord arteriovenous lesions, is divided into arteriovenous fistulas and arteriovenous malformations (AVMs). Arteriovenous fistulas are subdivided into those that are extradural and those that are intradural, with intradural lesions categorized as either dorsal or ventral. Arteriovenous malformations are subdivided into extradural–intradural and intradural malformations. Intradural lesions are further divided into intramedullary, intramedullary–extramedullary, and conus medullaris, a new category of AVM.

This modified classification system for vascular lesions of the spinal cord, based on pathophysiology, neuroimaging features, intraoperative observations, and neuroanatomy, offers several advantages. First, it includes all surgical vascular lesions that affect the spinal cord. Second, it guides treatment by classifying lesions based on location and pathophysiology. Finally, it eliminates the confusion produced by the multitude of unrelated nomenclatural terms found in the literature.

**KEY WORDS** • spine • arteriovenous malformation • arteriovenous fistula • aneurysm • cavernous malformation • hemangioblastoma • classification

**D**ESPITE the large number of contributions to our present understanding of spinal cord vascular lesions, the literature reflects considerable confusion (Table 1). The existing nomenclature is not always consistent with the lesions described.<sup>9,20,27,45,49–51,75,82</sup> We propose a modified classification system for spinal cord vascular lesions based on pathophysiology, neuroimaging features, intraoperative observations, and neuroanatomy. In the proposed classification system, spinal cord vascular lesions are divided into three primary or broad categories (Table 2): neoplasms, aneurysms, and arteriovenous lesions. Arteriovenous lesions are further subdivided based on their neuroanatomy. This classification system eliminates the confusion inherent in previous classification systems and can be used to determine the appropriate surgical treatment.

*Abbreviations used in this paper:* ASA = anterior spinal artery; AVF = arteriovenous fistula; AVFM = arteriovenous fistulous malformation; AVM = arteriovenous malformation; MR = magnetic resonance.

### Neoplastic Vascular Lesions

Neoplastic vascular lesions include hemangioblastomas<sup>49,52,61,81,85</sup> and cavernous malformations (Table 3),<sup>1,5,31,33,38,47,52,78,84</sup> both of which occur sporadically and familiarly.<sup>5,23,49,52,70</sup> Hemangioblastomas are composed of the following three cell types: endothelial cells, endothelial cell-associated pericytes, and lipid-laden stromal (interstitial) cells.<sup>35,43</sup> Most hemangioblastomas are intramedullary and abut the pial surface, although some occur along nerve roots.<sup>19,43,56</sup> Histopathologically, cerebellar and spinal lesions are similar. The arteries and veins associated with a hemangioblastoma can enlarge and simulate an AVM.<sup>46,52,56</sup> The syrinx associated with some intramedullary hemangioblastomas can simulate an astrocytoma.<sup>29,30,71</sup>

Endothelial cells form the vascular channels in cavernous malformations.<sup>53</sup> Cavernous malformations are always associated with a venous anomaly, which facilitates drainage of normal neurological tissue.<sup>79</sup> Delayed venous infarction is a well-known sequela associated with the resection of these venous anomalies. The method for re-

TABLE 1  
*Old nomenclature for spinal cord vascular lesion*

angioma arteriovenosum
angioma cavernosum
angioma racemosum
angioma racemosum arteriovenosum
angioma racemosum venosum
angioreticuloma
classic AVM
dorsal extramedullary
epidural
extradural
Foix-Alajouanine
glomus
intradural extramedullary
intramedullary
intraspinal extramedullary
intraspinal extramedullary radiculomeningeal
juvenile
metameric
others
perimedullary
radiculomeningeal fistula
retromedullary
Types I, II, III, & IV
varicosis spinalis
ventral

TABLE 2  
*Proposed classification of spinal cord vascular malformations*

neoplastic vascular lesions
hemangioblastoma
cavernous malformation
spinal aneurysms
arteriovenous fistulas
extradural
intradural
ventral*
dorsal†
AVMs
extradural–intradural
intradural
intramedullary
compact
diffuse
conus medullaris

\* Includes the following subtypes: A, small shunt; B, medium shunt; and C, large shunt.

† Includes the following subtypes: A, single feeder; and B, multiple feeders.

TABLE 3  
*Summary of clinical characteristics in neoplastic vascular lesions and spinal cord aneurysms\**

Characteristic	Hemangioblastoma	Cavernous Malformation	Spinal Cord Aneurysm
pathophysiology	tumor, cyst	diapedesis, hemorrhage	blood flow, dissection
presentation	mass effect	mass effect	SAH, stroke
diagnostic modality	MR imaging, angiography	MR imaging, angiography	MR imaging
previous nomenclature	same	same	same

\* SAH = subarachnoid hemorrhage.

section of both cavernous malformations and hemangioblastomas is similar to that used in cases of intradural intramedullary AVMs—that is, sharp dissection of the discrete lesion to separate it from surrounding neurological tissue. Violation of the surface of either lesion can lead to significant blood loss.

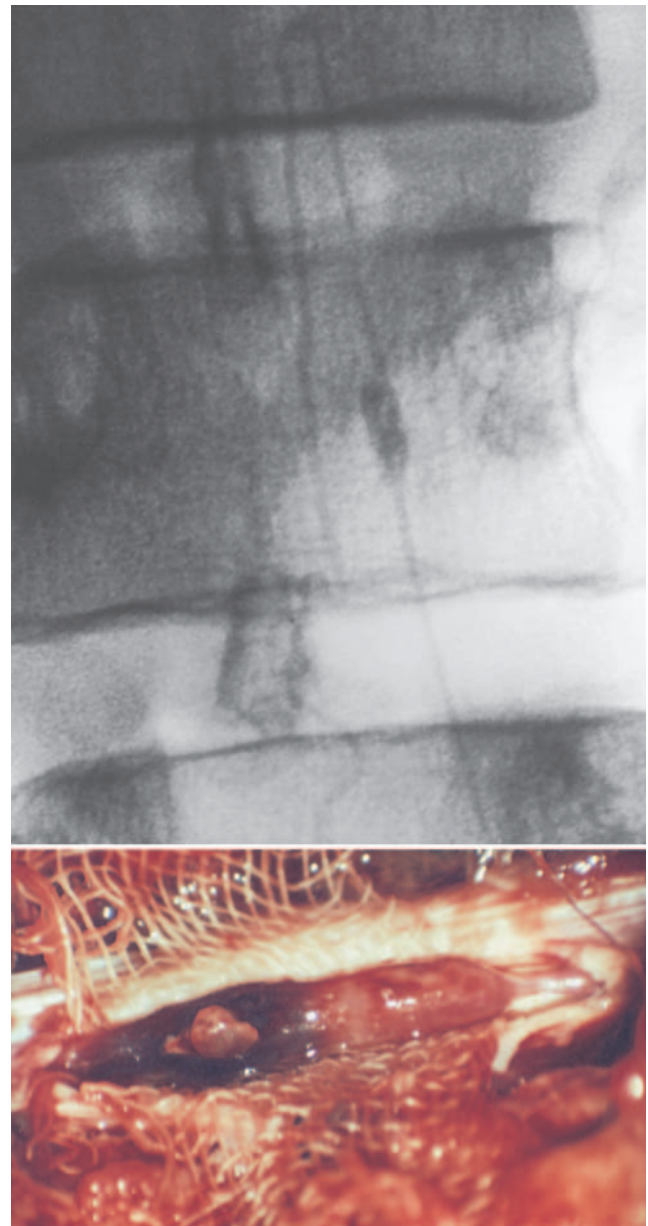


FIG. 1. *Upper:* Anteroposterior angiogram demonstrating subarachnoid hemorrhage associated with a dissecting aneurysm on the artery of Adamkiewicz. *Lower:* Intraoperative photograph. The lesion was wrapped with muslin gauze and has been asymptomatic more than 5 years. From Vishteh AG, Brown AP, Spetzler RF: Aneurysm of the intradural artery Adamkiewicz treated with muslin wrapping: technical case report. *Neurosurgery* 40:207–209, 1997. With permission from Lippincott-Williams and Wilkins.

TABLE 4  
Summary of clinical characteristics in AVFs

Characteristic	Extradural	Dorsal Intradural	Ventral Intradural
pathophysiology	spinal cord compression, venous congestion, vascular steal	venous congestion, rare hemorrhage	compression (venous aneurysm), hemorrhage, vascular steal
presentation	progressive myelopathy	progressive myelopathy	progressive myelopathy
diagnostic modality	MR imaging, angiography	MR imaging, angiography	MR imaging, angiography
previous nomenclature	epidural	dural AVF, long dorsal, Type IA, others	Types IVA (small), B (medium), & C (large), perimedullary

### Spinal Cord Aneurysms

Spinal aneurysms unrelated to AVMs are rare. Their pathophysiology is related to blood flow and dissection (Table 3).<sup>28,74,79</sup> We have treated two cases of spinal cord aneurysms, one located in the thoracic region and the other in the cauda equina. Both patients presented with subarachnoid hemorrhage and sudden-onset low-back pain. The thoracic lesion involved the artery of Adamkiewicz, which precluded resection because primary reanastomosis or bypass of the ASA would create an unacceptable risk to the blood flow to the distal ASA (Fig. 1). Consequently, the thoracic lesion was dissected free from the spinal cord and wrapped with muslin gauze.<sup>79</sup> Five years postoperatively, the patient is functioning normally and has no symptoms. In the other patient, MR imaging and angiography of the spine demonstrated an isolated aneurysm located on a radicular artery coursing along the cauda equina. This lesion was primarily resected, and there were no neurological complications. The results obtained in the latter case demonstrate that an aneurysm of a radicular artery coursing along the cauda equina can be resected safely by sacrificing the involved vessel.

### Arteriovenous Fistulas and Malformations

Spinal cord arteriovenous lesions represent the most confusing category. We have divided these lesions into AVFs and AVMs (Tables 4 and 5). Arteriovenous fistulas are subdivided into extradural and intradural lesions, with the latter being either dorsal or ventral. Arteriovenous malformations are divided into extradural–intradural and

intradural; the latter are further subdivided into intramedullary, intramedullary–extramedullary, and conus medullaris AVMs. Conus lesions are a newly proposed category (Detwiler, et al., unpublished data).

#### Extradural Arteriovenous Fistulas

Extradural (epidural) AVFs (Fig. 2) rarely require open surgery because they can be treated very effectively by endovascular procedures. Their pathophysiology and clinical presentation have been well described (Table 4).<sup>6,33,40,57,72</sup> A direct connection between an extradural artery and vein leads to the development of a high-flow fistula, engorgement of the epidural venous system, compression of the spinal cord, and resultant progressive myelopathy. The high venous pressure in the epidural venous system can lead to intradural venous hypertension by increasing the resistance to outflow. The shunting of large quantities of arterial blood into the venous system can also steal blood flow from the spinal cord.

#### Intradural Dorsal Arteriovenous Fistulas

Intradural AVFs are the most controversial lesions in terms of origin, pathophysiology, and treatment (Table 4). Intradural dorsal (Fig. 3) and intradural ventral lesions (Fig. 4) are distinct entities. Terms for intradural dorsal AVFs have included long dorsal,<sup>49,50</sup> angioma racemosum,<sup>45</sup> dorsal extramedullary, angioma racemosum venosum, and Type I.<sup>45,49,50,59,82</sup> They are the most common type of spinal AVF and usually occur in the thoracic region.

The primary pathophysiology of intradural dorsal AVFs is venous hypertension. In very rare cases do patients

TABLE 5  
Summary of clinical characteristics in AVMs

Characteristic	Extradural–Intradural	Intramedullary	Conus Medullaris
pathophysiology	compression, vascular steal, hemorrhage	hemorrhage, compression, vascular steal	venous hypertension, compression, hemorrhage
presentation	pain, progressive myelopathy	acute myelopathy, pain, progressive myelopathy	progressive myelopathy, radiculopathy
diagnostic modality	MR imaging, angiography, high-flow, multiple feeders	MR imaging, angiography	MR imaging, angiography
previous nomenclature	juvenile AVM, metameric AVM	classic AVM, glomus type	none



FIG. 2. *Left:* Artist's rendering of an extradural AVF. *Right:* Right anteroposterior vertebral artery angiogram revealing a large serpiginous fistula in the epidural space compressing the spinal cord in a 47-year-old woman who developed progressive quadriplegia.

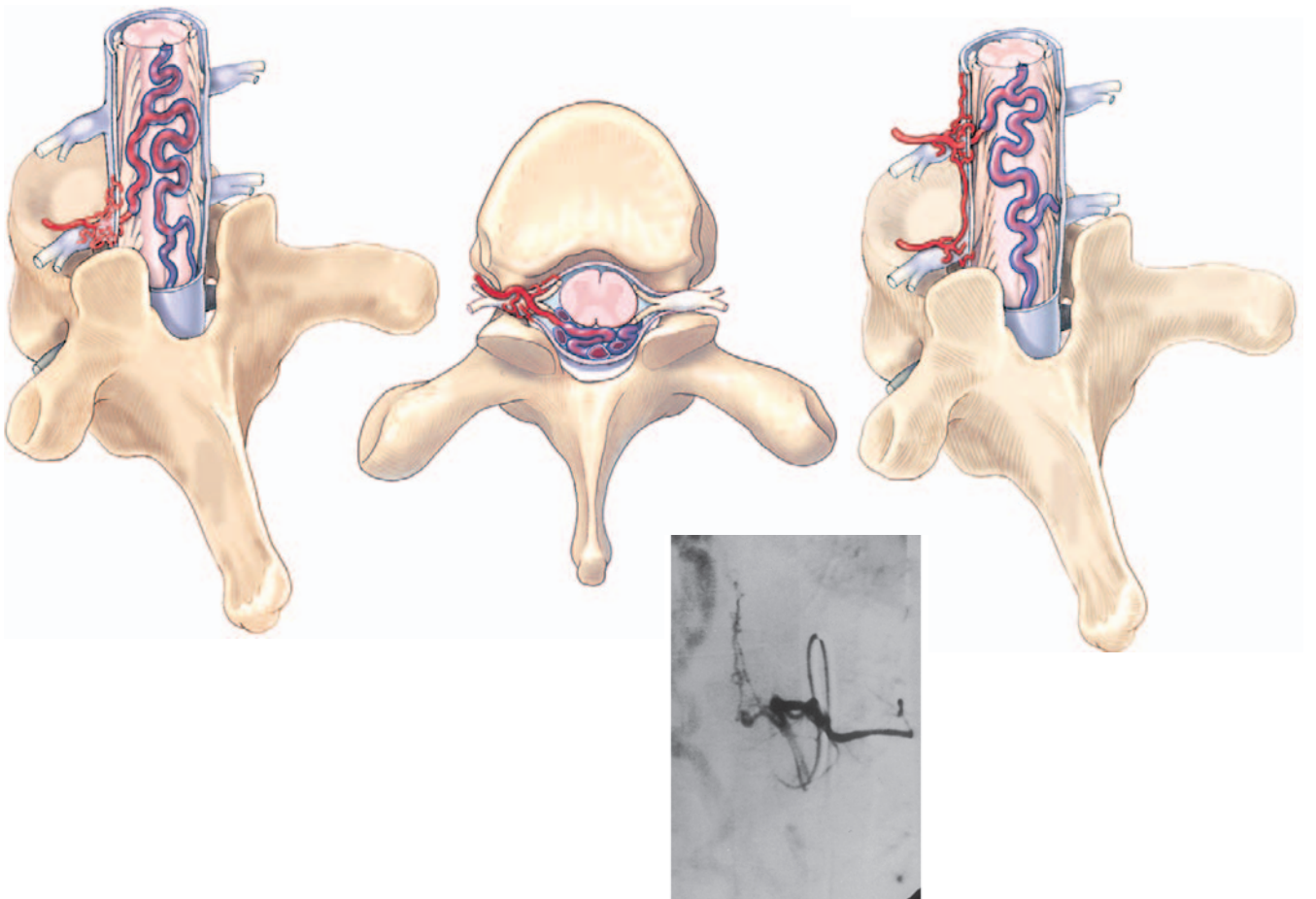


FIG. 3. *Upper Left and Center:* Artist's rendering of an intradural dorsal AVF. *Upper Right:* Artist's rendering of an intradural dorsal fistula that has its recruited blood supply from several levels. *Lower Inset:* Selective angiogram obtained in a 41-year-old woman who developed progressive paraparesis, demonstrating a fistula between the radiculomedullary artery and the coronal venous plexus. The fistula was coagulated and transected.

## Classification of spinal vascular lesions

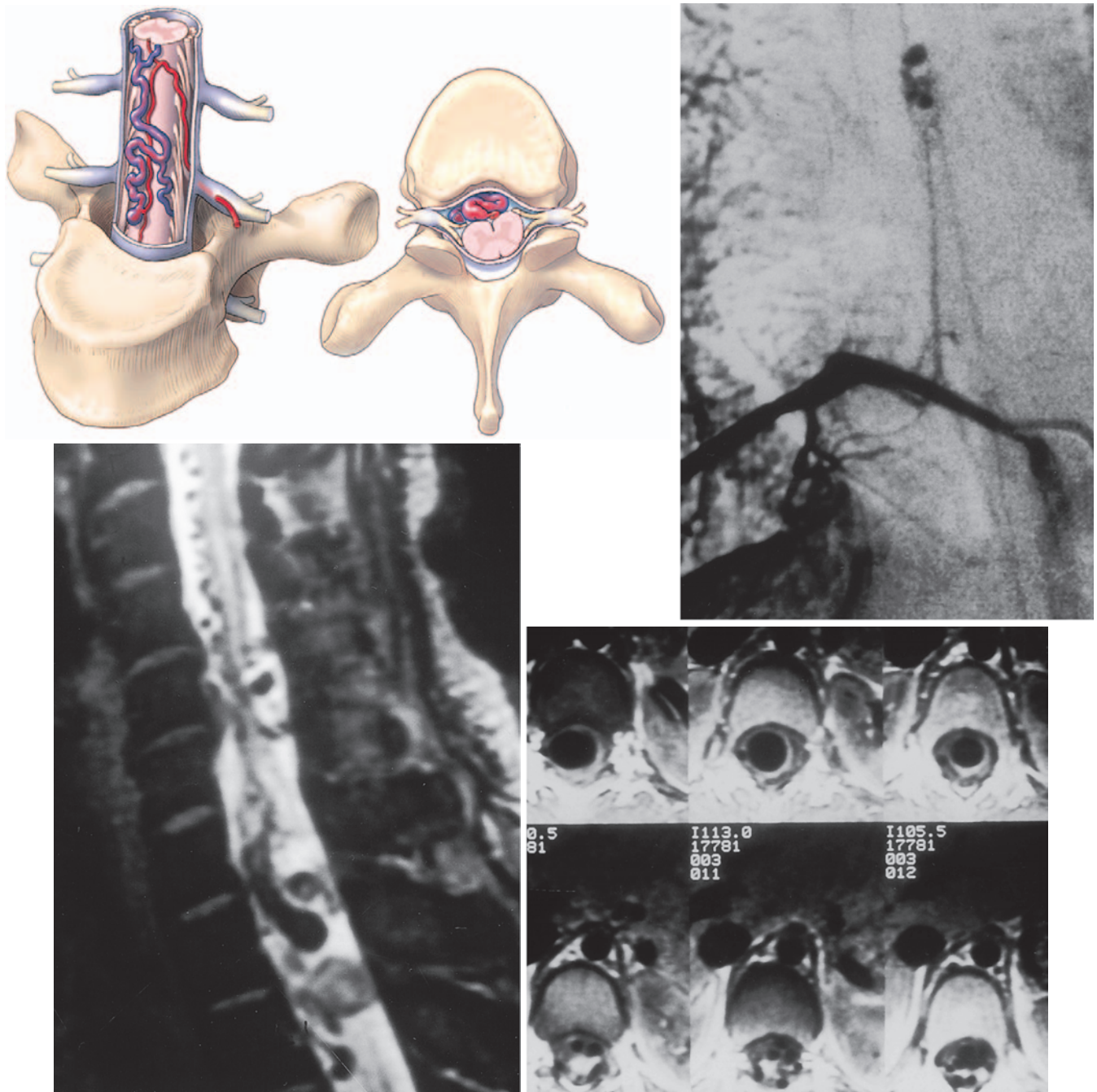


FIG. 4. *Upper Left:* Artist's rendering of an intradural ventral AVF. *Upper Right:* Anteroposterior spinal angiogram obtained in a 16-year-old woman with progressive headache and back pain, revealing a small AVF (Type A) located on the anterior surface of the spinal cord. From Carter LP, Spetzler RF: Spinal arteriovenous malformations. Surgical treatment, in Carter LP, Spetzler RF, Hamilton MG (eds): **Neurovascular Surgery**. New York: McGraw-Hill, 1995, pp 1197–1212. With permission from McGraw-Hill. *Lower Left:* Sagittal T<sub>2</sub>-weighted MR image of the cervical spine obtained in a 34-year-old woman with progressive left upper-extremity pain, revealing serpiginous flow voids ventral and lateral to the spinal cord. The lesion was exposed by a cervical corpectomy and durotomy. The AVF was coagulated and sharply transected, completely relieving the patient's preoperative pain. From Spetzler RF, Koos WT (eds): **Color Atlas of Microneurosurgery, ed 2. Volume III: Cerebral Revascularization, Extracranial Vascular Disease, and Intraspinial Pathology**. Stuttgart: Georg Thieme Verlag, 1999, p 413. With permission from Georg Thieme Verlag Medical, Stuttgart. *Lower Right:* Axial T<sub>1</sub>-weighted MR images obtained in a 10-year-old boy with severe progressive thoracolumbar pain, demonstrating a large, circular flow void in the anterior spinal canal that is indenting the cord. Selective angiography demonstrated a large ventral AVF (Type C). The lesion was occluded using an endovascular approach. With permission from Barrow Neurological Institute.

experience clinical deterioration secondary to hemorrhage.<sup>2,12</sup> Selective angiography demonstrates a characteristic slow-flow pattern produced by the feeding dorsal radiculomedullary artery. The artery enters the dura mater at the dural root sleeve and forms a fistula, arterializing the coronal venous plexus.<sup>39,54,55</sup> These lesions have been divided into two subtypes: Type A, with a single feeding artery, and Type B, with multiple feeding arteries (Fig. 3 right).<sup>4</sup>

Clinically, patients usually present with venous hypertension-induced progressive myelopathy, which can be misdiagnosed as lumbar stenosis. Aminoff and Logue<sup>3</sup> have detailed the clinical prognosis of these lesions in 60 cases. In seven patients who presented with acute-onset symptoms, there was no further progression. In the remaining 53 patients, symptoms progressed gradually, and five of these patients suffered acute neurological events. Within 3 years of the onset of functional lower-extremity impairment, half of the patients had become severely disabled. These outcomes led to the present dogma that progressive neurological deterioration can only be halted by eliminating the fistula.

Intraoperative inspection of the dorsal nerve roots reveals a vascularized pedicle or pedicles, which feed the AVF in the subarachnoid space (Fig. 5). The presence of an AVF in the dural root sleeve has been repeatedly verified histologically, but no extradural arteriovenous abnormality exists; otherwise, engorgement of the epidural venous plexus would be expected. Although there are numerous extradural and dural vessels, they compromise the dural recruitment process to feed the intradural fistula. This same vascularity can be observed when a meningeal feeding vessel supplies a cortical AVM. If the fistula were extradural or within the dura, the drainage would empty into the rich vascular epidural or dural venous system instead of into the high-resistance coronal venous plexus. When more than one feeding vessel is present (Type B), they form a single intradural fistula, providing further support for the intradural location of the AVF.

Repeated measurements of epidural venous pressure, which is the same as central venous pressure, support this contention. The mean invasive pressure recorded in 12 patients with intradural dorsal fistulas was 40 mm Hg within the coronal venous plexus.<sup>12</sup> When the fistula is closed, distal pressure drops to a mean of 23 mm Hg, twice the level of the average epidural venous pressure (same as central venous pressure) into which it drains. Furthermore, angiography routinely demonstrates slow flow;<sup>13</sup> this finding supports the contention that resistance to venous outflow must be high where the coronal venous plexus exits the dura.<sup>39</sup>

#### *Intradural Ventral Arteriovenous Fistulas*

In 1977, Djindjian, et al.,<sup>27</sup> found six intradural ventral AVFs in a series in which there were 150 lesions. Heros, et al.,<sup>41</sup> introduced the term Type IV lesion in 1986, and Gueguen, et al.,<sup>34</sup> divided ventral fistulas into three subtypes: 1, 2, and 3. To avoid confusion, Anson and Spetzler<sup>4</sup> reclassified the subtypes as IV-A, IV-B, and IV-C. The reclassified subtypes have been maintained in the present modification.

These lesions are located ventrally and in the midline

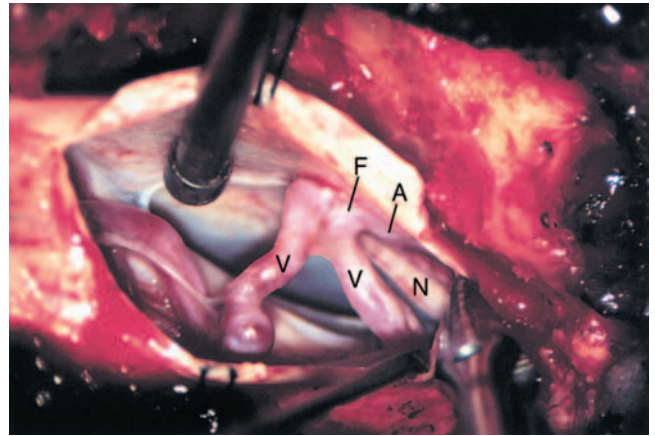


FIG. 5. Intraoperative photograph demonstrating the intradural location of the fistula (F), feeding artery (A), and associated arterialized veins (V). The nerve root (N) is seen coursing below the fistula.

(Fig. 4). They are clearly located in the subarachnoid space. They originate directly from the ASA and have a direct fistula to an enlarged venous network.<sup>10,14</sup> There is no intervening capillary system. Blood flow through the lesions is rapid and can produce flow-related aneurysms and venous hypertension.

Intradural ventral Type A fistulas are small shunts in which blood flow is slow and venous hypertension is moderate. Types B and C have progressively larger shunts; the latter are characterized by a giant fistula and a markedly distended venous network. As the size and flow of the fistula increases, the signs and symptoms attributable to progressive vascular steal and spinal cord compression become more pronounced.

#### *Extradural–Intradural Arteriovenous Malformations*

Fortunately, extradural–intradural AVMs (Fig. 6) are uncommon. In the past they have been referred to as juvenile, metameric, or Type III AVMs (Table 5).<sup>44,69,73,75</sup> They respect no tissue boundary. These lesions require a multidisciplinary approach in which the team emphasizes embolization of the multiple feeding arteries, sometimes followed by staged resection. Complete surgical resection is often difficult without significant risk of neurologic morbidity.

#### *Intramedullary Arteriovenous Malformations*

Intramedullary AVMs have a nidus similar to intracranial AVMs. In the past these lesions have been referred to as classic AVMs, glomus-type lesions, Type II AVMs, angioma arteriovenosum, and angioma racemosum arteriovenous lesions.<sup>5,21,22,63–65,82,83</sup> They can be supplied by multiple branches of the anterior and posterior spinal arteries and are characterized by high pressure, relatively low resistance, and high blood flow (Table 5). Associated aneurysms are common.<sup>17</sup> The nidus can be compact or diffuse. We have therefore subdivided intramedullary AVMs into compact (Fig. 7) and diffuse (Fig. 8).

## Classification of spinal vascular lesions

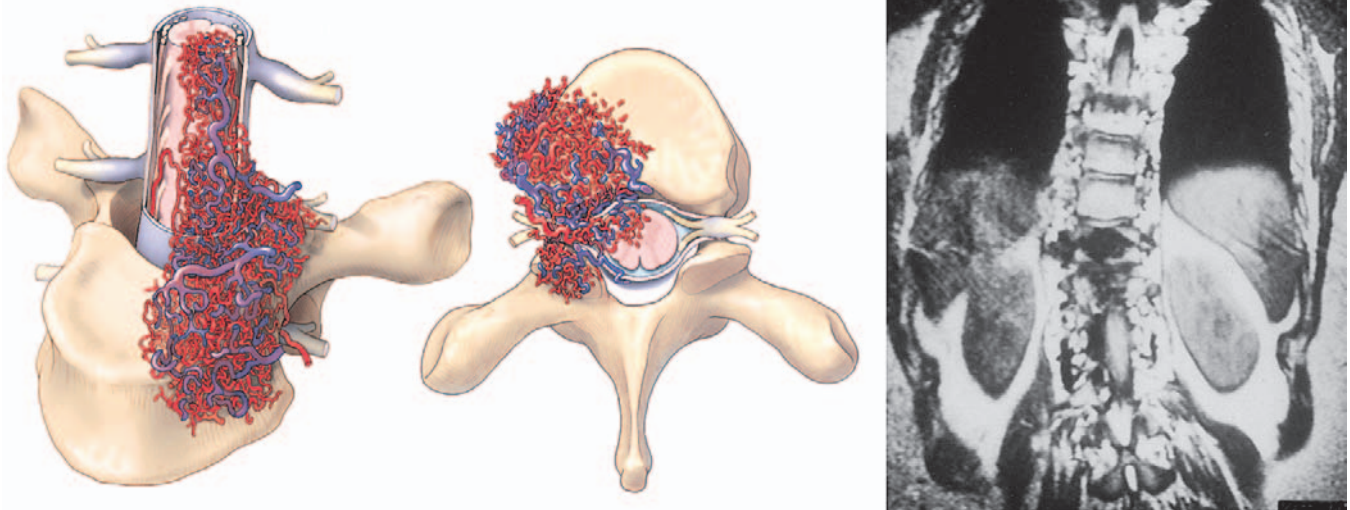


FIG. 6. *Left:* Artist's rendering of an extradural-intradural AVM. *Right:* Coronal T<sub>1</sub>-weighted MR image obtained in an 8-year-old girl who developed severe paraparesis, revealing involvement of the spinal cord, vertebral column, and extraspinal soft tissue.

Based on their experience in the treatment of 26 cases, Djindjian, et al.,<sup>27</sup> recommended subclassifying these lesions into three types based on the volume of the spinal cord affected: normal volume (Type 1), enlarged volume (Type 2), and extramedullary and intramedullary AVM (Type 3) with further subclassifications of each. This system was not adopted clinically because of its complexity. It also provides no useful prognostic information and can be confused with other grading systems.<sup>4</sup>

The most frequent clinical presentation of intradural intramedullary AVMs is hemorrhage- or compression-induced acute myelopathy. Progressive myelopathy can occur secondary to vascular steal.<sup>26</sup> The diagnosis can be made using MR imaging, but angiography is necessary to define the exact angioarchitecture. Obtaining the correct exposure is the cornerstone of treating these lesions successfully.

### *Conus Medullaris Arteriovenous Malformations*

The conus malformation (Fig. 9, Table 5) is a newly proposed category of lesions characterized by multiple feeding arteries, multiple niduses, and complex venous drainage (Detwiler, et al., unpublished data). These lesions fit none of the aforementioned categories. They have multiple direct arteriovenous shunts that derive from the anterior and posterior spinal arteries and have glomus-type niduses that are usually extramedullary and pial based, but they may also have an intramedullary component. They are location specific (that is, they are always located in the conus medullaris and cauda equina) and can extend along the entire terminal filum. Symptomatically, they can manifest with venous hypertension, compression, or hemorrhage. Unlike other spinal arteriovenous lesions, they frequently produce radiculopathy and myelopathy at the same time. The radicular deficits are often prominent but may

improve dramatically over time when these lesions are successfully treated. Their numerous feeding arteries and multiple niduses create a potential for recurrence.

### **Discussion**

In early classification schemes the authors stratified lesions based on pathological type—not location or pathophysiology.<sup>51</sup> In 1969 Yaşargil used the histology-based classification proposed by Bergstrand, et al.,<sup>16</sup> which divided spinal vascular malformations into angioma cavernosum, angioma racemosum, and angioreticuloma. The 17 lesions that he reported were all categorized as angioma racemosum.

Recent classification systems have stressed the concept of the nidus and accounted for the lesion's location. Bao and Ling<sup>8</sup> have reported a series of spinal vascular malformations in 80 patients. Similar to Rosenblum, et al.,<sup>67</sup> they classified lesions as intramedullary AVMs, intradural AVFs, dural AVFs, paravertebral AVMs, and Cobbs syndrome. Intramedullary lesions were subclassified into glomus AVMs and juvenile AVMs. Intradural AVFs were subclassified into Type I, which were small low-flow single-hole fistulas; Type II, which were high-flow single-hole fistulas; and Type III, which were giant high-flow fistulas with multiple feeding arteries. Between Type I (no obvious dilation) and Type III lesions (markedly dilated vein), the intradural feeding artery and draining vein dilate progressively. Ultimately, Bao and Ling concluded that their classification system offered limited guidance for the treatment of spinal vascular malformations. Furthermore, they added that successful treatment requires a precise understanding of the lesion's anatomical location and angioarchitecture.

Rosenblum, et al.,<sup>67</sup> distinguished AVFs from AVMs

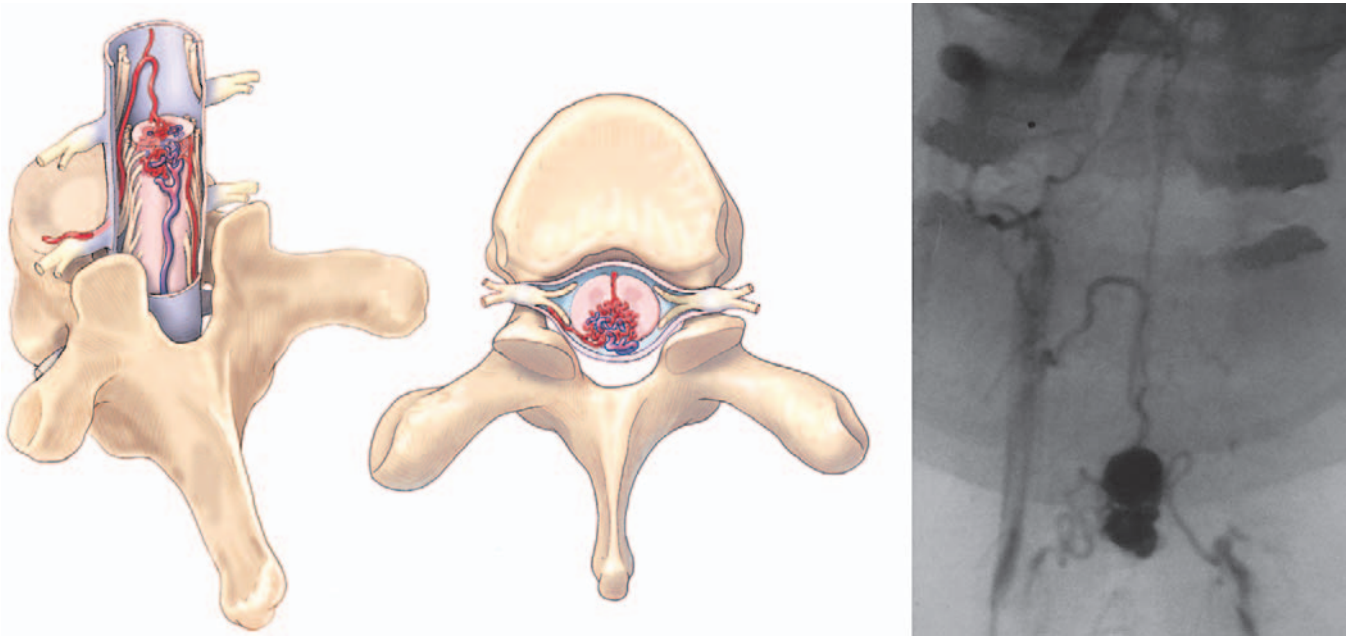


FIG. 7. *Left:* Artist's rendering of a compact intramedullary AVM. *Right:* Anteroposterior right vertebral artery angiogram obtained in a 32-year-old woman who developed acute onset of upper- and lower-extremity weakness, demonstrating multiple fistulas feeding an intramedullary malformation and associated aneurysm. The malformation and associated aneurysm were exposed and resected by a laminectomy and dorsal myelotomy. Postoperative right lateral vertebral angiography (not shown) demonstrated the patency of the ASA and no residual malformation.

based on pathogenesis, pathophysiology, radiographic findings, and response to treatment in 81 patients. Intradural AVMs were subclassified as intramedullary (juve-

nile and glomus) and direct AVFs, which were extramedullary or intramedullary in location. Dural AVFs were supplied by an artery originating from the dura and drain-

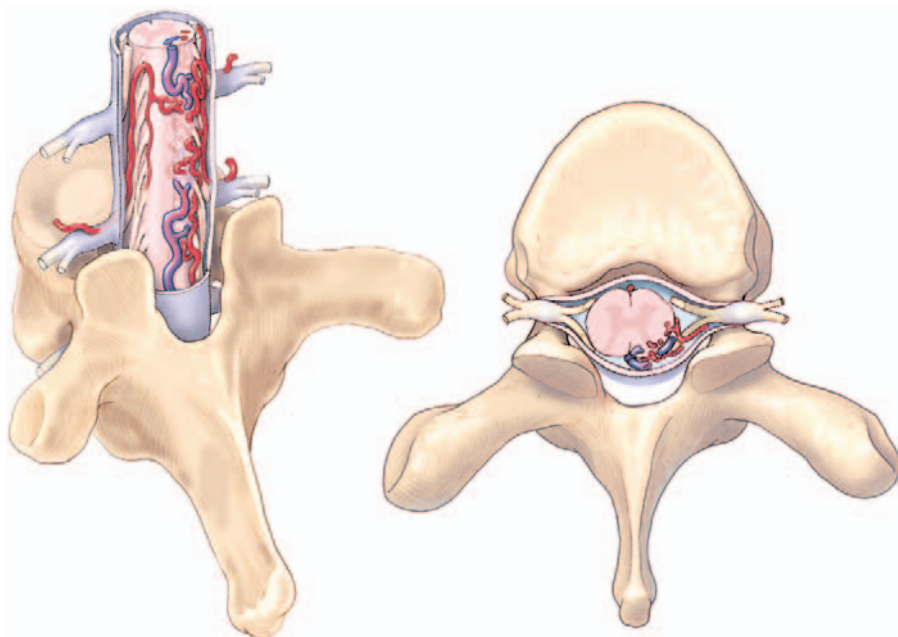


FIG. 8. Artist's rendering of a diffuse intramedullary AVM.



## Classification of spinal vascular lesions

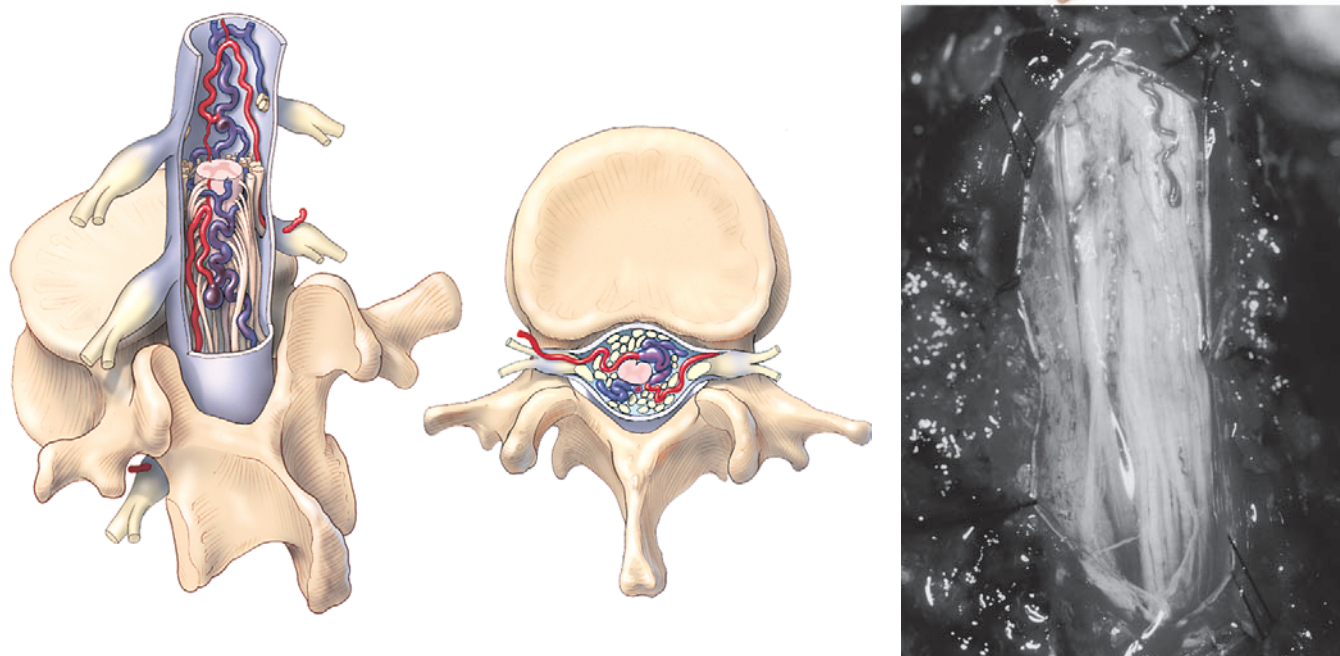


FIG. 9. *Upper:* Artist's rendering of a conus AVM. Selective angiography (not shown) demonstrated a T-10 fistula in a 36-year-old Hispanic man with progressive paraparesis and lower-extremity pain. The fistula fed into the coronal venous system. *Lower:* Intraoperative photograph showing exposure of the conus and cauda equina achieved through a multilevel lumbar laminoplasty. The lesion consisted of multiple fistulas feeding the malformation located on the posterior surface of the conus. Preoperative spinal angiography (not shown) showed only one fistula fed from above. Intraoperatively, three additional fistulas, originating from vessels coursing along the cauda equina nerve roots and being fed from below, were identified. The lesion was resected. The patient's neurological condition remained stable, and her lower-extremity pain resolved.

ing into the coronal venous plexus via an arteriovenous shunt located in the intervertebral foramen.<sup>68</sup> The arteriovenous shunt in the intramedullary lesion was located partially within the spinal cord or pia mater and supplied by the medullary arteries.

Borden and colleagues<sup>18</sup> have proposed a classification for both cranial and spinal dural AVFMs. They introduced the term dural AVFM to account for single dural AVFMs with multiple fistulas. Type I lesions drain directly into the dural venous sinus or meningeal vein intracranially or into the Batson epidural venous plexus in the spine. Type II lesions drain into the venous sinus and have retrograde flow into subarachnoid veins or into both the epidural venous plexus and perimedullary veins. Type III AVFMs drain into subarachnoid veins. They are supplied by a meningeal branch of a radicular artery and drain into the coronal venous plexus. This is the same lesion termed "angioma racemosum venosum" by Wyburn-Mason,<sup>82</sup> Type I by Di Chiro with colleagues,<sup>24</sup> and Wener<sup>25</sup> and intradural dorsal in our modified classification.

Our proposed classification represents an evolution from the earlier, most commonly used classification system (Types I–IV).<sup>4,8,16,18,24,25,67,82</sup> The new classification includes categories for the previously undescribed conus medullaris AVM as well as aneurysms and vascular neoplasms, all of which are being diagnosed with increasing frequency.<sup>79,80</sup> We have adopted this modified classification for several reasons. First, it includes all vascular

lesions that affect the spinal cord. Second, it guides treatment by classifying arteriovenous lesions based on their location and pathophysiology. Finally, it eliminates the confusion produced by the multitude of unrelated nomenclature used in the literature.

### Treatment of Spinal Vascular Lesions

Hemangioblastomas and cavernous malformations should be treated with resection. Embolization plays a

TABLE 6  
Spinal cord arteriovenous lesions treated surgically  
by the authors

	No. of Lesions	Postoperative Status (%)		
		Improved	Same	Worse
extradural AVF	2	100	0	0
dorsal intradural AVF	32	80	10	10*
ventral intradural AVF	7	75	25	0
extradural–intradural AVM	5	100	0	0
intramedullary AVM†	27	68	29	8
conus AVM	16	35	65	0

\* Vein stripping occurred.

† Characterized by 8% residual tumor.

small role in the treatment of hemangioblastomas but not in the treatment of cavernous malformations. In both of our patients with spinal cord aneurysms cure was effected by surgical means.<sup>77,79</sup>

Extradural AVFs are primarily treated endovascularly, as are extradural–intradural AVMs. In the latter case, surgery is intended to relieve compression of the dura and spinal cord and, occasionally, to provide a cure.

Intradural AVFs can be treated with surgery, embolization,<sup>11,15</sup> or both. In some institutions embolization is used to treat these AVFs, but we maintain that surgery is optimum for two reasons. First, the rates of recurrence and progressive myelopathy associated with embolization are high.<sup>37,58,60</sup> Second, in surgical series, the reported morbidity rate is extremely low and the success rate high.<sup>42,45,48,62,76</sup>

Small- and medium-sized intradural ventral fistulas can be treated surgically whereas large-sized fistulas are best suited to occlusion by embolization.<sup>32,36,66</sup> During surgery, the patency of the ASA must be maintained. After resection of a large ventral fistula (Type C), Heros, et al.,<sup>41</sup> found that the ascending and descending ASAs could not be demonstrated on postoperative angiography and the patient's neurological status worsened. Although intradural intramedullary AVMs can be treated with preoperative embolization,<sup>7</sup> resection remains the mainstay of their treatment.<sup>22</sup> In 8% of our cases, complete resection of the entire lesion was not possible. Finally, conus AVMs can be treated with both surgery and embolization. Our continuing experience with this type of lesion indicates that definitive treatment is best achieved using aggressive embolization and subsequent resection.

Based on the experience of the senior author (R.F.S.) in the treatment of more than 130 spinal cord vascular lesions, we have developed the following microsurgery-related tenets. Appropriate preoperative embolization should be preferred. Somatosensory and selected motor evoked potentials should be monitored intraoperatively. Selective intraoperative angiography is needed to identify residual lesion. The ASA must be preserved. The surgical plane should be dissected sharply; too little rather than too much resection is preferable. Surgery-related outcomes achieved using the aforementioned algorithms are reported in Table 6.

## Conclusions

Previous classification systems for spinal cord vascular lesions provide confusing, conflicting, and incomplete descriptions. The classification system proposed here is based on our knowledge of pathophysiology, modern neuroimaging features, intraoperative observations, and anatomy. It accounts for all surgically treatable vascular lesions that affect the spinal cord. The choice of treatment reflects the location and pathophysiology of a lesion, and the confusion produced by the numerous unrelated nomenclature is eliminated.

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