CEREBRAL VASCULAR DISEASES

SEVENTH CONFERENCE

TOOLE
MOOSSY · JANEWAY

Seventh Conference

CEREBRAL VASCULAR DISEASES

JAMES F. TOOLE,
Chairman

JOHN MOOSSY and RICHARD JANEWAY,
Editors

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Preface

THE SEVENTH Princeton Conference on Cerebral Vascular Diseases took place January 7, 8, and 9, 1970, with Dr. James F. Toole serving as chairman for the second time. On the Planning Committee for the conference were Drs. Stuart Bondurant, Albert Heyman, Clark Millikan, John Moossy, James F. Toole, and Jack P. Whisnant. Dr. Irving Wright was an honorary member of the committee. Dr. Richard Janeway served as a contributing member by invitation. The editors extend their thanks to the chairman and other members of the Planning Committee for their counsel and cooperation. It is also appropriate to acknowledge once again the sponsorship of the American Neurological Association and the American Heart Association, Council on Cerebrovascular Disease. We wish to express our gratitude for the support of the National Institute of Neurological Diseases and Stroke and the National Heart and Lung Institute of the National Institutes of Health.

Only a few alterations have been made in the format of the edited transactions. When references to the medical literature were used in Open Discussion, they were listed at the end of each session if the participants furnished the proper citations to the editors. Some of the formal presentations contained a large number of illustrations and references. We have only reordered figure numbers and performed minimal editorial manipulations. We have followed this course because of our conviction that only the authors and the critical reader are entitled to judge the adequacy and accuracy of documentation in a scientific conference of this type.

The editors are pleased to offer special thanks to many co-workers and friends who have assisted them. Mrs. Diane Porter cheerfully and efficiently assumed the heavy burdens of typing and rendered the high caliber secretarial assistance necessary for the job. Miss Suzanne Pickett aided the chairman and the editors in the important phases of planning and execution of the conference. Mrs. Bette Pou also deserves our thanks for attending to numerous details in her quiet and dependable fashion. The recording of the conference was again ably handled by Mr. Cyril Lichtensteiger and his staff.

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Contents

Introductory Remarks

JAMES F. TOOLE

Bowman Gray School of Medicine
Winston-Salem, North Carolina

CHAIRMAN DR. JAMES TOOLE: This is the Seventh Princeton Conference on Cerebral Vascular Diseases. This meeting, which has been held in Princeton at intervals since 1954, is co-sponsored by the American Neurological Association and the American Heart Association. It is supported jointly by the National Institute of Neurological Diseases and Stroke and the National Heart and Lung Institute. We are grateful to these associations and institutes for their sponsorship and support.

I am pleased, at this time to recognize Dr. Clark Millikan.

DR. CLARK MILLIKAN: It's a great pleasure to be here for the Seventh Princeton Conference and to anticipate another meeting marked by the friendliness and the warmth and vigor of the exchange in debate which have characterized these conferences in the past. At this time I want to introduce our guests from outside the United States and ask them to stand so that they can be identified and made welcome throughout the meeting.

From Canada, Drs. H. J. M. Barnett and Charles Drake are here from London, Ontario, and Dr. Ian Turnbull from Vancouver; from the United Kingdom, Mr. Lyndsay Symon, London; from Italy, Dr. Cesare Fieschi; and from Mainz, West Germany, Dr. Mario Brock. We welcome you to full participation in this somewhat unusual conference.

Also with us tonight are representatives from the National Institutes of Health, the Regional Medical Program, and the Lasker Foundation: Dr. Murray Goldstein, Dr. Mathilde Solowey, and Dr. Edward MacNichol from the National Institute of Neurological Diseases and Stroke; Dr. Jerome Green and Dr. Gardner McMillan from the National Heart and Lung Institute; Dr. Philip Klieger and Dr. Margaret Sloan from the Regional Medical Program; and Mrs. Edward McSweeney from the Lasker Foundation. We welcome you all and invite you to participate fully in the activities of this Seventh Princeton Conference.

Questions that recur from time to time are whether there is going to be a continuing need for this particular kind of forum and, if so, what its format should be in the future. Thumbing through the transactions of the first six conferences, you will note that the core of each conference has been human disease and the patient. Although we have on many occasions wandered some distance from the bedside, from office practice, and from the laboratory study of human patients, it has always been with the general motivation of shedding additional light on the subject of human disease. The six volumes of transactions which have been the final products of these conferences not only reveal the developmental aspects of cerebral vascular disease investigation at all levels over this period of 16 years but also contain a ready and ample source of references concerning prevention, pathogenesis, diagnosis, treatment, and prognosis in these diseases.

Returning to the question, "What about the need for this kind of get-together?" one need only consider the increasing complexity of the subject of cerebral vascular disease and the fashion in which this kind of conference can

direct itself to a specific focus or target to be convinced that there is very likely to be a continuing need for Princeton Conferences — meetings of experts who are willing to debate and exchange ideas in order that all those in attendance may be stimulated to reinspect some of the pressing questions that are raised.

Within the past ten days I've been asked five times whether the subject of low-molecular-weight dextran was going to be discussed at this conference. I simply mention this topic (which is not on the program) as an example of the kind of question that might be pursued in considerable depth, not just for the benefit of those here but, through the lucid volume of transactions that will be produced, for all those interested in cerebral vascular disease.

People who have been to these conferences before say that they receive a kind of stimulus from the interchange, whether during the formal sessions, during meals, or after the evening meetings. They leave here not only with new ideas to pursue, but perhaps also with a willingness to ask themselves new questions.

With this motivating philosophy, the Princeton Conferences have continued and now number seven. This year, for the first time, we are devoting a significant portion of the conference to the spinal cord and its vasculature. This has been done in accord with our custom of introducing at each conference an item that has not been adequately covered in the past.

DR. TOOLE: Thank you, Clark. Although the emphasis in this meeting is on flexibility, there are some things about which we are *inflexible*. One is the time allotted to each speaker for his presentation. Because we are most anxious to allow ample time for discussion, I will exercise the chairman's prerogative of "calling time" on those few speakers who seem to speak too long.

Another inflexible rule is that each discussant identify himself. A third is that all manuscripts be given to the editors at this meeting so that the transactions can be published without undue delay.

Without further ado, I call upon Dr. Lois Gillilan, who will speak to us about the arterial and venous anatomy of the spinal cord.

Spinal Cord Vascular Diseases:

Arterial and Venous Anatomy of the Spinal Cord

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ALTHOUGH DESCRIPTIONS of the vascular anatomy of the human spinal cord were published by Adamkiewicz[1-3] and Kadyi[4,5] in the late 1800s, it was not until Tureen's article[6] appeared in 1938 that similar information became available in the English language. A year later, other descriptions were published in English by Suh and Alexander[7] and by Herren and Alexander.[8] Since then, most investigators[9-13] have been essentially in agreement concerning the vascular anatomy of the spinal cord, differing only concerning details and terminology.

ARTERIES

The superficial arterial vascular tree supplying the spinal cord is made up of several sets of branches: spinal arteries, radicular arteries, medullary arteries, an anterior median spinal artery, and a posterior arterial plexus. The *spinal arteries* arise from the posterior segmental branches of the descending aorta and from the common iliac, supreme intercostal, ascending and/or deep cervical, and vertebral arteries. At each intervertebral foramen, an *anterior* and a *posterior radicular ramus* join the anterior and posterior nerve roots. These supply the root fibers and the spinal ganglion but do not contribute significantly to the blood supply of the spinal cord. Occasionally, a posterior radicular artery may join the posterior pial plexus.

Medullary arteries also arise from the segmental arteries, course along the nerve roots without giving off branches, and join either the anterior median spinal artery or the posterior plexus. There are approximately six to nine anterior medullary arteries, which are asymmetrical in their locations (Fig. 1A). The largest of these, the *great anterior medullary artery* (of Adamkiewicz), usually accompanies the left L_2 nerve root, although it may be located anywhere between T_8 and L_4.[4,7,12] When the artery reaches the anterior median fissure, it turns abruptly caudad and tapers downward over the lumbosacral enlargement and the filum terminale. Rostrally it anastomoses with the thoracic portion of the anterior median spinal artery, which has become very much attenuated.

The second largest of the anterior medullary arteries joins the anterior median spinal artery in the region of the cervical enlargement, usually at C_5 or C_6. This vessel divides into an ascending and a descending limb, each of which becomes continuous with the adjacent segment of the artery above and below. There are one or two (rarely three), additional medullary arteries going to the cervical cord, two or three to the thoracic

cord, and occasionally a second small lumbar artery below the great anterior medullary artery.

The *anterior median spinal artery* varies in diameter from level to level, being directly proportional to the diameter of the medullary artery feeding it. It is largest in the lumbosacral region and smallest in the thoracic region. There is a watershed of effective blood flow about halfway between any two of the medullary vessels. The cranial end of the anterior median spinal artery is said to be formed by the union of the *anterior spinal rami* arising from the intracranial portion of the vertebral arteries. In fact, however, the midline vessel formed by these rami, from both or often from only one side, dwindles in its course over the lower end of the medulla. If it does not disappear entirely, it becomes so small that, by the time it anastomoses with the spinal artery, it is at best only a potential source of collateral blood supply. Only in the case of chronic obstruction above or below the point of anastomosis does the connection attain any size.

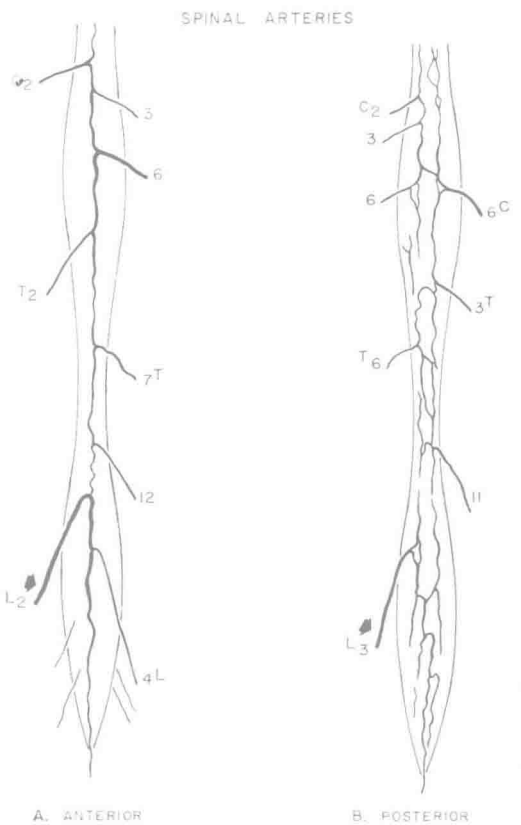

Fig. 1. Composite diagrams made from arterial injections of infant spinal cords. A. Eight anterior medullary arteries form the anterior median spinal artery, whose caliber is greatest over the lumbar and cervical enlargements. The major blood supply to the lumbosacral cord is furnished by the great anterior medullary artery (arrow). B. The superficial arteries on the posterior aspect form bilateral, irregularly anastomosing channels with which the posterior medullary arteries unite. The great posterior medullary artery is indicated by the arrow.

Twelve to 16 smaller *posterior medullary arteries* join the *posterior plexus*, which consists of two irregular and incompletely anastomosing channels running longitudinally along the line of entrance of the posterior nerve roots (Fig. 1B). These posterior plexiform channels are frequently but erroneously referred to as "posterior spinal arteries." The plexuses anastomose superiorly with the *posterior spinal rami* of the vertebral arteries, and inferiorly with the anterior median spinal artery through the fine, wide-meshed coronal (pial) arteriolar plexus that surrounds the cord.[7,12]

Arteries entering the spinal cord are of two types: (1) the central (sulcal) arteries, through which the arterial blood flows centrifugally into the anterior gray and white matter, and (2) the centripetal system of vessels which arises from the coronal arteriolar plexus and penetrates the entire periphery of the cord. The *central arteries* leave the anterior median spinal artery singly, pass dorsally through the anterior median fissure without branching, and turn alternately to the right and to the left[4,6,8,12] with only an occasional bifurcating vessel. Embryologically, these arteries stem from paired anterior spinal channels; although these channels eventually fuse into a single midline artery, the central arteries retain their laterality.[14-17] Levantovskii[10] reported that, in the

cervical and lumbar regions, 30 to 40 per cent of the central arteries bifurcate as they enter the spinal cord. Lazorthes[18] and Soutoul et al.[19-21] have stated recently that almost all central arteries in the human being as well as in experimental animals bifurcate. It should be noted, however, that these French authors based their observations on radiographs of sections from spinal cords injected with radiopaque dye.

Upon reaching the anterior horn, the central artery (Fig. 2) branches immediately into precapillaries and capillaries that form a plexus about the cells within the gray matter of the anterior horn, the gray commissure, the lateral horn, and the base of the posterior horn. This intrinsic capillary plexus is continuous throughout both the gray and the white matter of the entire cord. In addition to the capillary plexus, intersegmental arterioles extend upward and downward on either side of the midline to anastomose with adjacent intersegmental vessels. The density of the plexus in the gray matter is proportional to the number of cellular elements present.

In the white matter, the capillary plexus takes the form of wide rectangular meshes oriented in the direction of the fiber bundles. The transition from the dense meshwork in the gray matter to the loose plexus of the white matter occurs abruptly at the border between them. The white matter of the lateral and anterior funiculi receives its blood supply not only from the central arteries but also from the *penetrating arterioles* entering around the periphery. The anterior two-thirds of the spinal cord receives its major blood supply through the anterior median spinal artery and its 250 to 300 central tributaries.[1,2]

The posterior horns receive blood only through the rami penetrating from the posterior arterial plexus. The vessels enter

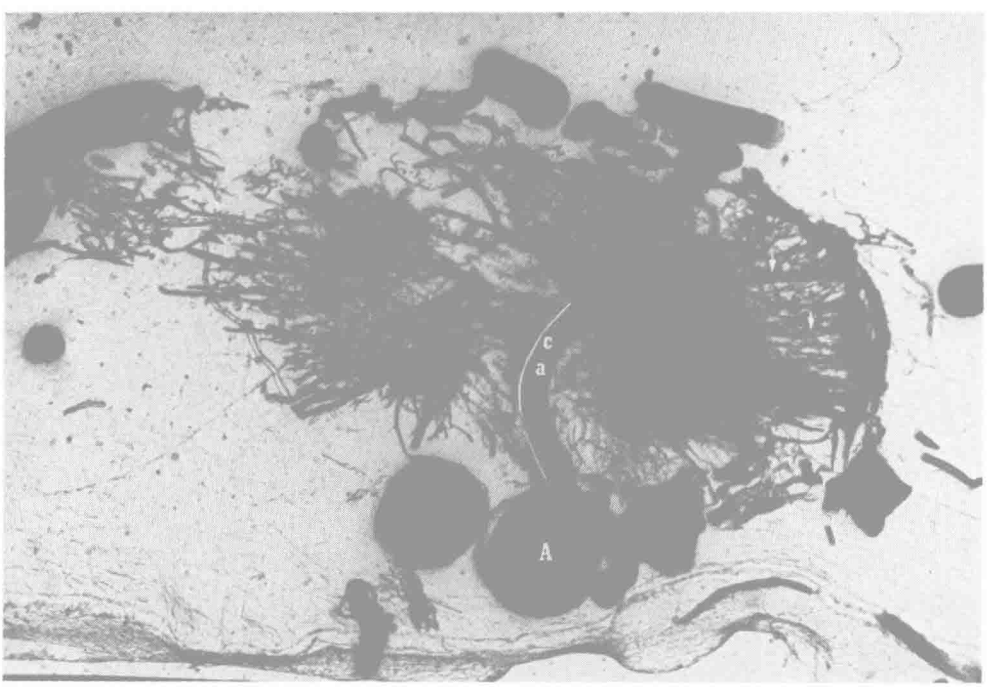

Fig. 2. Arteries in a transverse section of a corrosion preparation of an infant spinal cord injected with methyl methacrylate. Central arteries (ca) are seen turning to the right and to the left. The dense capillary plexus outlining the gray matter is connected with the coronal arterial plexus by penetrating arterioles that traverse and supply the white matter. A. Anterior median spinal artery; *arrows* show radial arteries.

along with the fibers of the medial division of the posterior root. At the border of the gray matter they divide and then subdivide immediately into precapillaries, forming the "fan" described by Kadyi.[4] The posterior funiculus receives other penetrating arterioles, the largest of which traverse the posterior median and intermediate septa. The posterior third of the spinal cord is supplied entirely by penetrating arterioles arising from the superficial posterior plexiform channels. Presumably because of this anatomic arrangement, this part of the cord seems less susceptible to arterial vascular accidents than the anterior two-thirds, which is served mainly by the central artery system.

Veins

The venous drainage of the spinal cord has been, for the most part, neglected. The confusion about this subject is reflected in the varied and inaccurate accounts that exist in current textbooks. The earliest reliable description is that of Kadyi,[4,5] which was followed after half a century by those of Tureen,[6] Suh and Alexander,[7] and Herren and Alexander.[8]

Intrinsic spinal cord veins comprise two morphologic groups or systems: (1) a *central vein* or anterior median *group,* and (2) a *radial group*[22] (Fig. 3).

In the region of the anterior white commissure a number of venules converge in a stellate pattern to form the *central veins* that drain the anterior gray commissure, the medial part of the anterior horn, and the anterior white matter bordering the anterior median fissure. The central (sulcal) veins collect from both halves of the spinal cord, and their intersegmental anastomoses extend upward and downward to adjacent regions. In the anterior median fissure there are additional extensive anastomoses between the central veins themselves.

At the periphery of the gray matter, two

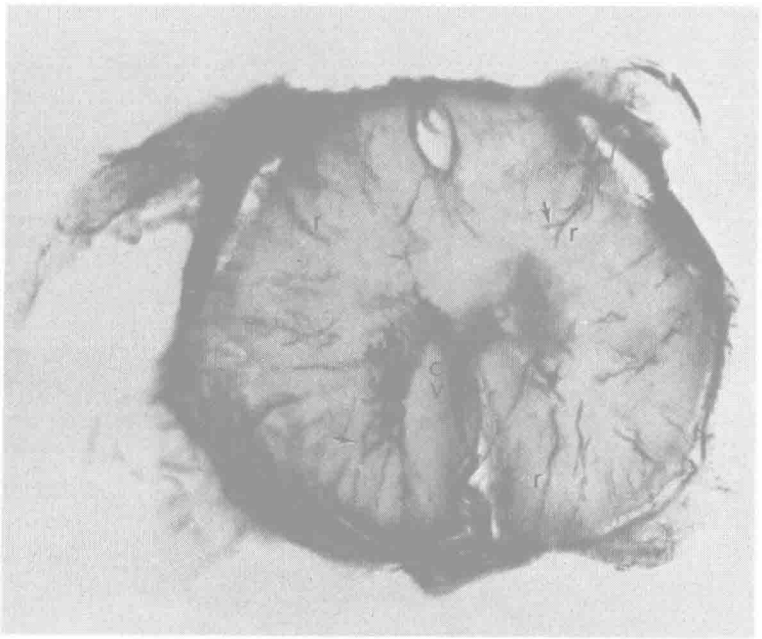

Fig. 3. Transverse section from an adult spinal cord in which the veins were injected with a blue pigment in aqueous gum acacia and cleared by the Spalteholz technique. The central veins (cv) are formed by veins converging in a stellate pattern from both sides. Radial veins are seen around the entire periphery. Arrows show the formation of two radial veins.

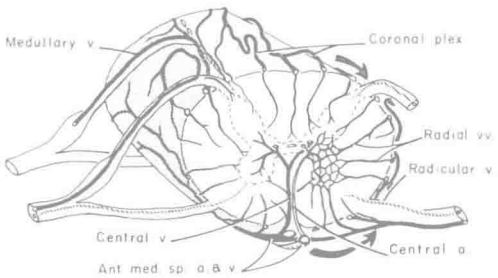

Fig. 4. Diagram of cleared preparation of an adult spinal cord in which the veins had been injected as in Fig. 3 and to which a central artery and the capillary plexus in the anterior horn have been added. A central vein and its tributaries are shown, as are medullary and radicular veins, the formation and disposition of the radial veins, and the coronal venous plexus.

short venules arise from the capillary plexus and join to form a *radial vein* (Figs. 3 and 4). As the radial veins course toward the surface, they may be joined by short venules from the plexus in the white matter. Among the veins of the radial group, three series or groups are noteworthy because of their recurrence in particular locations: (1) In the cervical region a series of venules arises in the reticular formation and in the retrodorsolateral cell column and passes obliquely toward the coronal veins near the posterior nerve roots. This series continues through the thoracic region, where the veins collect from the plexus in the lateral horn.

(2) A second series extends from the level of C_8 to L_3, draining Clarke's nucleus. These veins pass through the posterior intermediate septum into the posterior plexus. (3) Each of the veins of a third series found throughout the cord is formed by the union of two small vessels from either side of the cord just dorsal to the gray commissure; it then traverses the posterior median septum. Herren and Alexander,[8] as well as Kadyi,[5] described an occasional large vein connecting the intersegmental vein with a "posterolateral vein" on the surface. This they called the "centromedian anastomosis."

The superficial veins are essentially plexiform in character. The *anterior median spinal vein* overlies the anterior median fissure (Fig. 5A). It varies in diameter from level to level and may be double for part of its course. Entering this longitudinal channel are the central veins and many small, tortuous tributaries from the anterior pial plexus. A

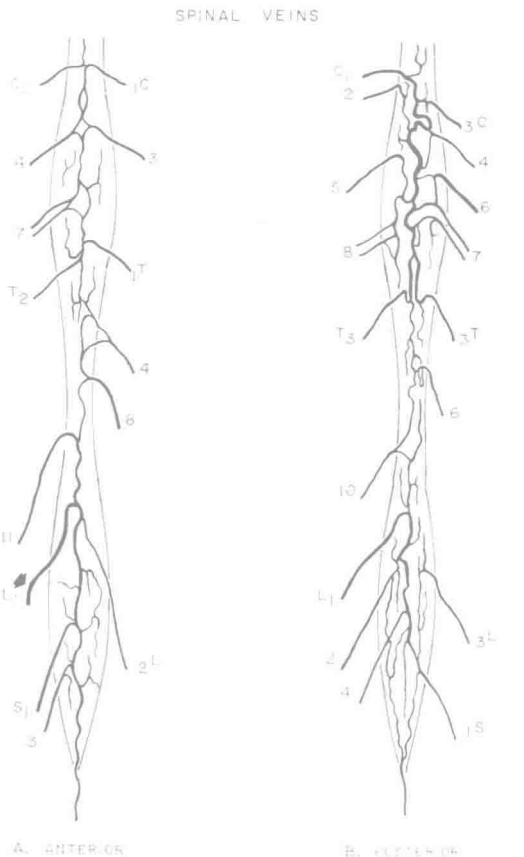

Fig. 5. Diagrams made from adult spinal cords in which veins were injected. A. The anterior median spinal vein extends longitudinally along the anterior median fissure. Anterior medullary veins, arising mainly from this vessel, drain the anterior half of the cord; note their lack of symmetry. A great anterior medullary vein is indicated by the arrow. B. The posterior superficial veins are plexiform and are especially large and convoluted over the lumbar and cervical enlargements. The posterior medullary veins, which drain the posterior half of the cord, are numerous in the cervical region. The radicular veins that accompany each rootlet are not shown.

fairly continuous vessel just posterior to the emerging anterior roots drains the anterior half of the lateral coronal (pial) plexus. Blood from both the anterior median spinal vein (the central vein system) and the coronal veins of the anterior half of the cord leaves the spinal cord through approximately 8 to 14 *anterior medullary veins*.[7,22] Like the medullary arteries, these veins are asymmetrically located. A *great anterior medullary vein* leaves the cord at the upper level of the lumbar enlargement, usually from the left side.

The posterior superficial veins are large and convoluted. They tend to be interrupted plexiform vessels overlying the posterior median and intermediate septa and located along the line of entrance of the posterior nerve roots (Fig. 5B). All intrinsic radial veins from the posterior horns and posterior funiculus empty into the posterior plexus. Radial veins emerging from the posterior half of the lateral funiculus drain into a discontinuous vein lying anterior to the posterior nerve roots. Anastomoses between the anterior and posterior halves of the coronal plexus are infrequent in the upper half of the cord, but they increase in number over the lumbosacral region. Blood from the posterior half of the spinal cord is carried outward by the *posterior medullary veins*, which are especially numerous in the cervical region.

In addition to the medullary veins, which serve only the spinal cord, every nerve rootlet is accompanied by its own *radicular vein*. The radicular veins are tiny, often invisible to the naked eye, and they do not carry blood away from the cord.

In the vicinity of the intervertebral foramen, the medullary veins and the radicular veins pierce the dura mater and join a coarse plexus, which marks the confluence of these intradural vessels with tributaries from the epidural (internal) plexus and from the external vertebral plexus. The *intervertebral veins* are found at every level and constitute the final common pathway for blood leaving the spinal cord and the perivertebral plexus. They anastomose with the venae cavae, and with the azygos and the hemiazygos veins. Through the valveless perivertebral plexus, blood from the spinal cord can be channeled not only into the veins of the thoracico-abdominal cavity but also into the dural sinuses and cerebral veins above and into the pelvic plexus below. Backflow of venous blood into the spinal cord, however, is limited by valves (described by Clemens[23] in 1961 and located at the juncture of the medullary veins with the intervertebral veins) as well as by the small caliber of the medullary veins and the acute angle at which most of them enter the plexus.[24] These factors may account for the rarity of metastatic intramedullary tumors of the spinal cord.

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