Internal vertebral venous plexuses

Related terms:

- Vertebral column
- Epidural
- Spinal canal
- Epidural space
- Venous plexus
- Ascending lumbar vein
- Intervertebral disc
- Spinal nerve
- Dura mater
- Adipose tissue

Learn more about Internal vertebral venous plexuses

General Characteristics of the Spine

Gregory D. Cramer, in Clinical Anatomy of the Spine, Spinal Cord, and Ans (Third Edition), 2014

Internal Vertebral Venous Plexus

The internal vertebral venous plexus is located beneath the bony elements of the vertebral foramina (e.g., laminae, spinous processes, pedicles, and vertebral body). As mentioned, it is embedded in a layer of loose areolar tissue known as the epidural (extradural) adipose tissue, although less adipose tissue surrounds the veins located in the anterior aspect of the epidural space. The internal vertebral venous plexus provides an alternate route of venous return when the jugular veins of the neck are compressed, when the flow through the inferior vena cava is obstructed, and when intrathoracic or intraabdominal pressures are increased. They also provide a protective cushion for the important contents of the vertebral canal (Stringer et al., 2012). The internal vertebral venous plexus is a clinically important plexus, and perhaps for this reason it has been given many names. It is known as the internal vertebral venous plexus, the epidural venous plexus, the extradural venous plexus, and also as Batson's channels.

The internal vertebral venous plexus consists of approximately four interconnected longitudinal channels. Two course along the posterior aspect of the vertebral canal, and two channels of larger diameter course along the anterior aspect of the canal (Stringer et al., 2012). The posterior channels are rudimentary in the cervical region, but well developed in the thoracic and lumbar regions (Chaynes et al., 1998). The posterior channels are located along the posterolateral aspect of the vertebral canal in the thoracic region and are more laterally placed in the cervical (rudimentary channels) and lumbar regions (Stringer et al., 2012).

The anterior channels are located on either side of the posterior longitudinal ligament and drain the vertebral bodies via large basivertebral veins. The basivertebral veins pierce the center of each vertebral body and communicate posteriorly with the internal plexus and anteriorly with the external vertebral venous plexus. Posteriorly the basivertebral veins connect with horizontally oriented transverse veins that course anterior to the posterior longitudinal ligament. These transverse veins connect the left and right longitudinal channels of the anterior internal vertebral venous plexus (Stringer et al, 2012).

The veins of the internal vertebral venous plexus contain no valves; therefore the direction of drainage is posture and respiration dependent. Inferiorly this plexus is continuous with the prostatic venous plexus of the male, and superiorly (in both sexes) it is continuous with many veins and also to dura mater venous sinuses of the posterior cranial fossa. These superior connections include

continuous with many veins and also to dural mater. Venous sinuses of the posterior cranial fossa. These superior connections include the following: vertebral veins, occipital veins, occipital sinus, sigmoid sinus, and inferior petrosal sinus (Stringer et al., 2012). Therefore prostatic carcinoma may metastasize via the intervertebral venous plexus to all regions of the spine and to the meninges and brain. Because of venous communications in the thoracic region, lung and breast cancers can metastasize to these veins as well. However, the veins of the internal vertebral venous plexus eventually (by means of intervertebral and ascending lumbar veins) drain into large veins. These large veins include the vertebral, for the cervical region; and the azygos, hemiazygos, and right highest intercostal veins, for blood draining the thoracic and lumbar regions. These large veins each have one or two valves at their entrance to the brachiocephalic veins (for the vertebral veins) or at their entrance to the azygos vein (for the right highest intercostal and hemiazygos veins). The azygos vein then drains directly into the superior vena cava, and there is also a valve at this entrance. These valves act as a protective mechanism, preventing reflux of blood (and the accompanying increase in pressure) into the internal vertebral venous plexus and the important neural tissues they serve (Scapinelli, 2000). However, diminished right-sided heart function can lead to congestion and engorgement of the intervertebral veins, coursing through the IVFs, and veins of the internal vertebral venous plexus. Such venous congestion, usually when coupled with narrowing (stenosis) of the vertebral canal, can exacerbate inflammation of dorsal roots and/or dorsal root ganglia and lead to pain typically associated with prolonged recumbency (e.g., during sleep) (Parke, 2005).

The walls of the longitudinal veins of the internal vertebral venous plexus are very distinct. These walls have many trabeculae composed of collagen and smooth muscle that create a series of interconnected, parallel channels for blood flow within the individual longitudinal veins. The trabeculae are supplied by small nerve endings and arteries. The trabeculae are thought to prevent overdistension or collapse of the vessels and may help to regulate the direction and velocity of blood flow within the vessels (Stringer et al., 2012). However, segments of the longitudinal veins, and all of the horizontal connecting and intervertebral veins (the latter coursing through the intervertebral foramina), are devoid of these reinforcements. These trabeculae-free regions appear to be more vulnerable to distension and collapse. Isolated regions of the veins have been found to become dilated (varices of epidural veins; that is, epidural venous engorgement) (Parke, 2005). These regions are likely related to the trabeculae-free areas of the veins. The varices that can develop in these regions, and in the intervertebral veins, may compress the exiting spinal nerves and cause radiculopathy (i.e., pain coursing along the distribution of the dorsal root that contributes to the formation of the spinal nerve). Radiculopathy from this cause can mimic that more commonly caused by an IVD protrusion (Wong et al., 2003). In addition, the veins can collapse from the pressure of an IVD protrusion. This fact has been used in a procedure known as epidural venography (Fig. 2-17) to aid in the diagnosis of IVD disease. In epidural venography, radiopaque dye is injected into the epidural veins and x-ray films are taken. This allows the veins filled with dye to be visualized (Jayson, 1980). Pressure from a disc protrusion prevents the veins from filling and is seen as an area devoid of dye on the x-ray film.

Spinal epidural hematoma is a condition in which bleeding occurs into the space surrounding the dura mater. It is usually the result of a ruptured epidural vein and is rather rare, with only 250 cases reported in the literature. Of these cases, approximately 50% are spontaneous and of unknown cause. The causes of the remainder of the cases include trauma, anticoagulant therapy, and arteriovenous malformation. Spinal epidural hematoma may simulate IVD protrusion but can usually be identified through MRI (Mirkovic & Melany, 1992). The treatment protocol usually involves the release of pressure (decompression) by the removal of a lamina (laminectomy), although several cases with spontaneous recovery have been reported (Sei et al., 1991).

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General Anatomy of the Spinal Cord

Susan A. Darby, in Clinical Anatomy of the Spine, Spinal Cord, and Ans (Third Edition), 2014

Venous Drainage of the Spinal Cord

The venous system found within the vertebral canal consists of an epidural, or internal, vertebral venous plexus, located in the epidural space (see Chapter 2), and also an irregular venous plexus lying on the cord. This entire venous system is devoid of valves and anastomoses freely with itself. Please see Chapter 2 for a full description of the internal and external vertebral venous plexuses.

The venous system of the cord is a tortuous plexus that consists of six variable longitudinal veins: three anterior and three posterior (Fig. 3-17, *A*). The anteromedian vein is located deep in the ventral median fissure and has an average diameter ranging between 0.5 and 1.5 mm. It is a single vessel in the lumbar region but is accompanied by two anterolateral veins in the thoracic and cervical segments (Bowen & Pattany, 1999). These anterior venous channels receive blood from the neural tissue via sulcal veins and subsequently drain into anterior radiculomedullary veins. (**Note:** The nomenclature used for these vessels is inconsistent [Brockstein, Johns, & Gewertz, 1994; Bowen & Pattany, 1999; Krauss, 1999]. This text uses radiculomedullary to identify the veins draining the cord [which are variable in number] and roots [which are present at every level].) The anterior radiculomedullary vessels range in number from 10 to 20 (Bowen & Pattany, 1999), and empty into the epidural venous plexus. In the lumbar region, there may be one large anterior radiculomedullary vein, the vena radicularis magna (Carpenter, 1991; Brockstein, Johns, & Gewertz, 1994; Bowen & Pattany, 1999; Krauss, 1999), which usually courses with one of the left nerve roots located between the T11 and L3 spinal cord segments. The posterior aspect of the cord has a similar venous pattern. A midline posteromedian vein is the dominant vessel in the lumbar area with two posterolateral veins accompanying it in other regions. These drain the dorsal funiculus, dorsal gray horns, and their adjacent lateral white matter. These posteromedian and posterolateral veins in turn become tributaries of posterior radiculomedullary veins, which number the same as, or more than, the anterior radiculomedullary veins (Bowen & Pattany, 1999). The posterior radiculomedullary veins also empty into the epidural venous plexus. At the level of the dorsal root ganglion, the posterior radiculomedullary vein receives blood from the parenchyma of the dorsal root ganglion via a periganglionic venous plexus located on the surface of the ganglion. Because this plexus lies immediately underneath the dura, an external force could compress the dura onto the underlying plexus and impede venous flow. This would alter the permeability of the vessel wall and lead to endoneurial edema. The edema would press the venous plexus against its overlying covering of dura, resulting in chronic venostasis. If this condition continued it could lead to increased sensitivity, endoneurial fibrosis, and ectopic firing of the ganglion neurons (Parke & Whalen, 2002). A large posterior radiculomedullary vein also is found in the lumbar region, and usually is seen accompanying a L1 or L2 root. Using MR angiography images, the intersection of either an anterior or a posterior radiculomedullary vein with a spinal vein (e.g.,

Using MR angiography images, the intersection of either an anterior or a posterior radiculomedullary vein with a spinal vein (e.g., anteromedian vein) can be identified by its “coat hook” appearance, which distinguishes it from the “hairpin” look of the artery of Adamkiewicz (Bowen & Pattany, 1999). As also seen in the arterial system, an encompassing venous vasa corona interconnects the six longitudinal veins.

The epidural (internal vertebral) venous plexus, which drains blood not only from the cord but also from bone and red bone marrow, consists of several anterior and posterior longitudinal and interconnecting vessels. At the level of the foramen magnum, it forms a dense network that communicates with vertebral veins. In addition, it is continuous with the dural venous sinuses (occipital and sigmoid) and venous channels (basilar plexus, venous plexus of the hypoglossal canal, and the emissary veins of the condyles [Standring et al., 2008]) within the skull (see Chapter 2). This plexus also drains into intervertebral veins in the IVFs and also into another longitudinally arranged plexus, the external vertebral venous plexus (Fig. 3-17, *B*). From intervertebral veins, venous blood drains into segmental veins such as the vertebral, intercostal, lumbar, and lateral sacral veins (Standring et al., 2008). These segmental veins lie outside the vertebral column. Chapter 2 provides a full description of the external and internal vertebral venous plexuses.

A metastatic tumor in the epidural space may damage the cord by impeding venous return and cause vasogenic edema (Grant et al., 1991). Also, because the internal vertebral venous plexus lacks valves, blood flow through the intervertebral veins (which communicate with segmental veins) is allowed to be reversed. This provides the opportunity for cancer to metastasize from such areas as the prostate, lung, breast, and thyroid gland to the brain and vertebral bodies (FitzGerald & Folan-Curran, 2002; Standring et al., 2008) (see Chapter 2).

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Four Stages of Skeletal Axis Development

Meridel I. Gatterman, in Whiplash, 2012

Vascular Components

External Vertebral Venous Plexus

The external vertebral venous plexus is a network of veins surrounding the external aspect of the vertebral column. It is associated with both posterior and anterior elements of the vertebral column and can be divided into an anterior plexus surrounding the vertebral bodies and a posterior plexus associated with the neural arches. These plexuses communicate with segmental veins throughout the spine, including deep cervical veins, intercostal veins, lumbar veins, and ascending lumbar veins in addition to the internal vertebral venous plexus that lies within the vertebral canal. The external and internal vertebral plexuses communicate through the IVF and also directly though the vertebral bodies. The veins that run through the IVF, connecting the two plexuses, surround the exiting spinal nerve and form a vascular cuff around the nerve.[7]

Internal Vertebral Venous Plexus

The internal vertebral venous plexus is located beneath the bony elements of the vertebral foramina (laminae, spinous processes, pedicles, and vertebral body). It is embedded in a layer of loose areolar tissue know as the epidural (extradural) adipose tissue. The internal vertebral venous plexus is made up of many interconnected longitudinal channels, some that run along the posterior and anterior aspects of the vertebral canal. They have no valves and therefore drainage is dependent on posture and respiration.

Arterial Supply to the Spine

The external aspect of the vertebral column receives its arterial supply from branches of regional deep arteries. The internal aspect of the vertebral canal receives its arterial supply from segmental arteries that send branches into the IVF. The spinal segmental artery divides into three branches on entering the IVF. One branch courses posteriorly, supplying the posterior arches of neighboring vertebrae; an anterior branch supplies the posterior longitudinal ligament, the posterior aspect of the vertebral body, and the surrounding tissues; and a third branch, the neural branch, runs to the mixed spinal nerve.

A close relationship exists between the extensive and abundant blood supply of arterial branches that form the anterior lateral and posterior spinal arteries of the spinal cord.[7]

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The Peripheral and Central Nervous System

S. Franklin, in Conn's Translational Neuroscience, 2017

Meninges of the Spinal Cord

The spinal cord is a relatively soft structure and must be supported within vertebral column by membranous layers called the meninges. The outermost layer is called the dura mater. It is attached to the rim of the foramen magnum and extends down the length of the vertebral canal and ends around vertebral level S2 creating a closed dural sac. The epidural space of the spinal cord is positioned between the dura mater and the vertebra that is typically filled with fat and the internal vertebral venous plexus.

Deep to the dura mater is a very thin layer called the arachnoid membrane. Deep to the arachnoid membrane is the subarachnoid space that is filled with CSF. The subarachnoid space of the neurocranium is continuous with the subarachnoid space of the spinal

space that is filled with CSF. The subarachnoid space of the neurocranium is continuous with the subarachnoid space of the spinal cord. The arachnoid membrane is not fused to the overlying dura mater, but the pressure of the CSF in the subarachnoid space prevents this layer from collapsing on to the surface of the spinal cord.

Arising from the arachnoid layer are thin filaments that traverse the subarachnoid space called arachnoid trabeculae. Arachnoid trabeculae fuse with the 3rd layer of meninges called pia mater that intimately covers the surface of the spinal cord. The pia mater forms two special structures to support the spinal cord within the dural sac. Denticulate ligaments arise from the pia mater on the lateral edge of the spinal cord and fuse to the overlying dura mater and the filum terminale extends from the conus medullaris to the end of the dural sac in order to anchor the inferior tip of the spinal cord.

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Obstetric Analgesia and Anesthesia

Mark D. Rollins, Mark A. Rosen, in Avery's Diseases of the Newborn (Ninth Edition), 2012

Maternal Circulatory System

Hypotension can occur when a pregnant woman is in the supine position because of compression of the vena cava by the gravid uterus. Significant aortoiliac artery compression occurs in 15% to 20% of parturients and vena caval compression is universal, often as early as 13 to 16 weeks' gestation. Vena caval compression contributes to lower extremity venous stasis and can cause ankle edema and varices despite increased collateral circulation. Venous compression by the gravid uterus diverts some blood returning from the lower extremities through the internal vertebral venous plexus, the azygos, and the epidural veins. This increases the likelihood of entering an epidural vein with spinal or epidural anesthetic techniques. Anesthetic interventions that diminish sympathetic tone can further exacerbate the effects of vena caval compression induced by supine positioning, potentially causing profound hypotension. Therefore supine positioning is avoided during anesthetic administration in the second and third trimesters. Significant lateral tilt is used in all operative deliveries and frequently during labor analgesia to help preserve uterine blood flow and fetal circulation.

Cardiac output increases during pregnancy, reaching an output 50% greater than the prepregnant state by the third trimester. During labor, maternal cardiac output increases during the first and second stages, reaching an additional 40% above prelabor values in the second stage (Robson et al, 1987). Each uterine contraction results in the autotransfusion of 300 to 500 mL of blood back into the maternal central circulation. The greatest increase in cardiac output occurs immediately after delivery, when values can increase as much as 75% above predelivery levels. This abrupt increase in cardiac output is secondary to the loss of aortocaval compression, autotransfusion from the contracted uterus, and decreased venous pressure in the lower extremities (Kjeldsen, 1979).

Physiologic (dilutional) anemia of pregnancy occurs as a result of a greater increase in plasma volume (45%) than in red blood cell volume (20%) at term. Average blood loss at delivery—Approximately 500 mL for vaginal delivery and 1000 mL for cesarean section—is well tolerated because of this expanded blood volume and autotransfusion (normally in excess of 500 mL) from the contracted uterus after delivery (Cheek and Gutsche, 2002).

Read full chapter

Small Animal Spinal Cord Disease

Alexander de Lahunta DVM, PhD, DACVIM, DACVP, Eric Glass MS, DVM, DACVIM (Neurology), in Veterinary Neuroanatomy and Clinical Neurology (Third Edition), 2009

FIBROCARTILAGINOUS EMBOLIC MYELOPATHY

FCEM is a spinal cord lesion that is common in dogs but is uncommon in other species of domestic animals.* It is most common in young adult dogs of the larger breeds but it can occur as young as 3 months of age and it is common in the miniature schnauzer, Labrador retriever, and boxer breeds, in our experience. The clinical signs are peracute in onset and usually stabilize within 24 hours. Rarely, clinical signs may progress for 48 hours. Following that, there is no further progression or there is improvement, depending on the degree of ischemia or infarction that has occurred. The source of the fibrocartilage is assumed to be the intervertebral disk that is undergoing degeneration. This embolic fibrocartilage has the same collagen type that is found in the nucleus pulposus. How this degenerate fibrocartilage gains access to the spinal cord vasculature remains speculative. These emboli are more common in small arteries but also can be found in veins. Arteriovenous anastomoses do occur in the blood supply of the spinal cord and have been implicated in the distribution of the emboli. Protrusion of degenerate disk material into the adjacent ventral internal vertebral venous plexus has occasionally been observed at necropsy. It has been suggested that the normally avascular intervertebral disk is invaded by new growth of arteries when degeneration occurs in the annulus fibrosis and this is a route for these emboli to enter the arterial vasculature. We find this mechanism difficult to accept. In humans, degenerate intervertebral disk material can protrude into the adjacent vertebral body where there is ready access to the blood vessels in the marrow of the vertebra. One route of venous drainage from this marrow is into the ventral internal vertebral venous plexus within the vertebral canal. These intramedullary protrusions are referred to as Schmorl nodes. They are rare, or at least rarely identified in dogs, which may be because of dogs' quadruped posture and the thick layer of cortical bone that is adjacent to the intervertebral disk. Reverse venous blood flow may be involved in the distribution of these emboli. Whenever an animal strains by contracting its trunk muscles with the glottis closed, the increased pressure in the thorax and abdomen interferes with the venous return to the heart and forces the venous blood into the vertebral venous plexus. This is the Valsalva maneuver, and it may play a role in the ability of these emboli to gain access to the spinal cord vasculature. The involvement of the intervertebral disk as the source of these emboli is also supported by the observation that these lesions occur primarily in the spinal cord. One report of brainstem lesions with fibrocartilaginous emboli indicated a possible source of emboli from cervical intervertebral disks.[5] Magnetic resonance (MR) imaging often shows intervertebral disk degeneration at the level

of the FCEM lesion in the spinal cord. These FCEM lesions can be unilateral or bilateral at any level of the spinal cord, and they affect various combinations of the gray and white matter. The lesions are usually limited to a few adjacent spinal cord segments. There are many examples in the following case examples that involve the various regions of the spinal cord. Caudal brainstem signs are rare and probably are associated with emboli arising from the cervical intervertebral disks.

Because of the extensive collateral circulation to the spinal cord (Fig. 10-4), multiple blood vessels must be compromised to cause the degree of infarction and severe clinical signs seen in dogs similar to Brittney. This suggests that a sudden shower of emboli must occur at one time. At necropsy, these emboli can be found in many blood vessels in or near the lesions. Usually, this shower affects the blood vessels to a few adjacent spinal cord segments and the associated lesions often are scattered and asymmetric within these segments. Thus, the clinical signs are usually focal and often asymmetric, as seen in Brittney. FCEM may be much more common than we realize and not be extensive enough to cause clinical signs or cause only transient clinical signs. Most veterinarians have had the experience of being called by a distraught owner who has just found their pet dog collapsed and unable to stand, but by the time the dog arrives at the hospital for examination, the dog is walking normally. We believe that some of these transient episodes of collapse may be due to transient spinal cord ischemia caused by FCEM.

Many dogs in which you make this clinical diagnosis will recover spontaneously. This is more common in dogs with paresis and ataxia due to interruption of the UMN and GP pathways. The more severe the involvement of the gray matter in the intumescences, the more guarded the prognosis. As a rule of thumb, if there are no signs of improvement in 10 to 14 days after the onset of clinical signs, it is unlikely that any recovery will occur. In a few patients, after an initial mild improvement, there may be a period of weeks before they rapidly regain the ability to walk. We usually tell owners that it may take up to 10 weeks before final improvement occurs.

An interesting observation is that FCEM is very rare in the chondrodystrophic breeds in which the chondroid metaplastic form of intervertebral disk degeneration is so common. FCEM also is observed in dogs as young as 3 months of age in which you do not expect intervertebral disk degeneration to occur. In these young dogs, the source of cartilage may be the vertebral growth plates. Often, there is an associated history of mild trauma such as a sudden fall or vigorous playing and jumping as with catching a frisbee. This relationship between young age and vigorous handling is associated with FCEM in young feeder pigs; it occurs during their transportation in crowded trucks.

The three primary disorders that can cause an acute onset of relatively nonprogressive spinal cord dysfunction are external injury by objects in the environment, most commonly vehicles; internal injury resulting from intervertebral disk extrusions; and vascular compromise resulting from FCEM. The history usually permits substantiation or exclusion of external injury. Lacking that, vertebral column radiographs should provide that answer. To differentiate between the other two causes of these clinical signs, evidence of discomfort by the patient is more suggestive of an intervertebral disk extrusion than of FCEM, but exceptions are common for both of these disorders. Ultimately, immediate imaging is necessary because a diagnosis of an intervertebral disk extrusion usually requires emergency surgery. Myelograms in dogs with FCEM are helpful only in the small percentage of dogs in which intramedullary swelling is extensive. MR imaging is much more reliable in detecting the spinal cord edema that accompanies the ischemia or infarction caused by the fibrocartilaginous emboli.[25] Be aware that MR imaging that is done in the first 24 to 48 hours after the embolic shower occurs may occasionally be normal.

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The Lumbar Region

Gregory D. Cramer, in Clinical Anatomy of the Spine, Spinal Cord, and Ans (Third Edition), 2014

Intervertebral Foramina Proper

When viewed from the side, the lumbar IVFs face laterally. A typical lumbar IVF is sometimes described as being shaped like an inverted teardrop or an inverted pear (Fig. 7-14). The specific dimensions of the lumbar IVFs are given in Chapter 2 (see Tables 2-6 and 2-7Table 2-6Table 2-7).

The spinal nerve is located in the upper third of each lumbar IVF. As it enters the IVF, the spinal nerve is close to the medial and inferior aspect of the superior pedicle that forms the upper boundary of the IVF (Crock, 1981). Here the nerve is accompanied by a branch (or sometimes branches) of the lumbar segmental artery; by the superior intervertebral (segmental, or pedicle) veins, which connect the external and internal vertebral venous plexuses; and by the sinuvertebral nerve (Rauschning, 1987). The spinal nerve occupies approximately one third of the IVF in the lumbar region. However, the spinal nerve can be as close as 0.4 mm (range, 0.4 to 0.8 mm) to the bony anterior and posterior IVF borders (Giles, 1994). This allows crowding by the articular facets during extension (Bose & Balasubramaniam, 1984; Rauschning, 1987). The inferior aspect of the IVF is usually narrowed to a slit by the anulus fibrosus, which normally bulges slightly posteriorly. The inferior aspect of the IVF is also narrowed by the posteriorly located ligamentum flavum. The inferior intervertebral (segmental, discal) veins usually lie in this narrow space. As with the superior intervertebral (pedicle) veins, the inferior veins also unite the internal (epidural) vertebral venous plexus with the external vertebral venous plexus and the ascending lumbar vein.

The lateral borders of the IVFs are covered with an incomplete layer of transforaminal fascia (Paz-Fumagalli & Haughton, 1993). This fascia condenses in several locations at each IVF to form the accessory ligaments of the IVF (see Chapter 2). An exiting spinal nerve could be affected by these structures as it leaves the IVF (Bachop & Janse, 1983; Bachop & Ro, 1984; Bose & Balasubramaniam, 1984; Rauschning, 1987; Bakkum & Mestan, 1994; Qian, Qin, & Zheng, 2011). In addition to compression by accessory ligaments or transforaminal fascia, Rauschning (1987) states that lateral disc herniations and bony structures such as the TPs (usually of L5, but at higher lumbar levels on rare occasions) “may compress, kink, or constrict the lumbar nerves beyond the foraminal outlet.” The author calls this region the extraforaminal region or the postcanal zone.

Also, the lateral borders of the L1 through L4 IVFs are associated with the origin of the psoas major muscle. In fact, because of the

posterior origin of the psoas major muscle from the front of the TPs and its anterior origin from the lumbar vertebral bodies and IVDs, the psoas major almost completely surrounds the lateral opening of the first four lumbar IVFs. Therefore the anterior primary divisions (ventral rami), by necessity, run through the substance of the psoas major muscle, frequently uniting with neighboring ventral rami within the muscle to form the branches of the lumbar plexus. In addition to the protection given by the dural root sleeve and the meningovertebral (Hoffmann) ligaments (see previous discussion and Fig. 7-9), the psoas major muscle may provide some protection for the dorsal and ventral roots during traction of the peripheral nerves of the lumbar plexus (dePeretti et al., 1989). Such traction may occur as a result of hyperflexion or hyperextension of the lower extremity.

The boundaries of the IVF can be imaged well with both MRI and CT (Cramer et al., 1994) (Fig. 7-15; see also Fig. 7-14). Occasionally ossification of the superior attachment of the ligamentum flavum results in foraminal spurs, which can be seen on CT. These spurs are considered to be normal variants; may project well into the IVF, posterior to the dorsal root ganglion; are frequently bilateral; and are usually asymptomatic (Helms & Sims, 1986).

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Pelvic Limb Paresis, Paralysis, or Ataxia

Michael D. Lorenz BS, DVM, DACVIM, ... Marc Kent DVM, BA, DACVIM, in Handbook of Veterinary Neurology (Fifth Edition), 2011

Fibrocartilaginous Embolic Myelopathy and Spinal Cord Infarction

Although fibrocartilaginous embolism can affect any spinal cord segment, it occurs most frequently in the caudal lumbar area and less frequently in the midcervical to caudal cervical spinal cord. For this reason, we have elected to discuss it with the other L4-S3 disorders. Fibrocartilaginous embolism has a brief progressive course (a few hours) and then becomes nonprogressive. We have therefore classified it as a nonprogressive disease.

Pathogenesis

Although emboli to the CNS can develop from a variety of sources such as endocarditis, sepsis, and fat, the most common form causing spinal cord infarction is a fibrocartilaginous material that histochemically stains in a manner similar to nucleus pulposus. The cause of this disease is unknown. Fibrocartilaginous embolic myelopathy occurs most frequently in dogs but also in horses, cats, pigs, sheep, and humans.[270-276] Fibrocartilaginous material found in arterioles and veins of the meninges and spinal cord results in an ischemic necrotizing myelopathy (Figure 6-28). Exactly how this material is distributed into the circulation of the spinal cord is not known, but several theories have been proposed. Most of these are based on the belief that fibrocartilaginous emboli originate from the intervertebral disks. The most probable mechanism is herniation of the nucleus pulposus into the body of the vertebrae, followed by entrance into an internal vertebral venous plexus and then into an arteriovenous anastomosis. The material could then enter the spinal cord in arteries, veins, or both.[270,273]

Larger-breed dogs have been studied most intensely, but spinal cord infarction occurs in smaller-breed dogs as well. A breed predisposition may exist for miniature schnauzers, GSDs, and Irish wolfhounds.[277-280] Fibrocartilaginous embolism occurs more frequently in nonchondrodystrophic breeds. Interestingly, chondrodystrophic breeds with a predisposition for Hansen type I disk disease are affected infrequently.[281]

Clinical Signs

The key clinical features of fibrocartilaginous embolism are acute onset, nonprogressive course (except for the first few hours), and nonpainful, asymmetric paresis.[272,282] The clinical signs develop acutely and progress rapidly within 1 to 2 hours from initial pain to unilateral or bilateral paralysis. Spinal hyperesthesia may be present at onset of signs but is absent after the clinical signs stabilize. Trauma is not in the history, but dogs are frequently reported to be exercising at the time of onset. Asymmetry is not found in every case but is a valuable sign when present. Asymmetry is explained by the frequency of unilateral branches of the central branch of the ventral spinal artery. Lateralization of signs is very suggestive of fibrocartilaginous embolism because spinal cord compression generally causes bilateral signs. Although asymmetry may be present in disease processes that result in compression (e.g., IVD herniation), the asymmetry rarely is strikingly disparate as is often the case in a fibrocartilaginous embolism. With a fibrocartilaginous embolism, it is not uncommon to have one pelvic limb lack voluntary motor ability while the contralateral limb displays minimal deficits. Despite this presentation, bilateral signs also occur in a fibrocartilaginous embolism. The degree and character of the neurologic deficit correspond to the site and the extent of the spinal cord infarction. Infarction of gray matter of an intumescence may cause LMN signs in affected limbs. Absence of spinal hyperesthesia distinguishes embolic myelopathy diseases in which there is compression, such as Hansen type I IVDD, neoplasia, or vertebral fractures and disease processes in which there is inflammation such as meningomyelitis.

Diagnosis

Diagnosis is based on history, clinical findings, and exclusion of other causes. No definitive antemortem diagnostic procedure exists for fibrocartilaginous embolic myelopathy. The diagnosis is supported by evidence that rules out the presence of spinal cord compression. Survey radiography and myelography findings are usually within normal limits. The myelogram may show a slight swelling of the spinal cord for the first few days. The CSF may contain a slight increase in protein. MRI has become the imaging modality of choice for a presumptive diagnosis while excluding other causes. The MRI findings are characterized by focal, intramedullary, hyperintense lesions on T2W images with varying degrees of contrast enhancement (Figure 6-29).[283,284] Lesions are often present in a segment of the spinal cord overlying an IVD in which the nucleus pulposus has undergone degenerative changes resulting in a loss of signal intensity on T2W images. The severity of clinical signs is associated with the presence and extent of the

resulting in a loss of signal intensity on T2W images. The severity of clinical signs is associated with the presence and extent of the lesion on MRI.[283,284] Animals should be evaluated for conditions such as hypertension and hypothyroidism that might predispose to CNS vascular occlusion and infarction.

Treatment

Corticosteroid therapy for fibrocartilaginous embolism is considered controversial. Corticosteroids such as methylprednisolone sodium succinate are aimed largely at reducing spinal cord edema and inflammation. However, a study revealed lack of significant association with corticosteroid administration and outcome.[284] Neurologic improvement may be noted within a few days, but functional recovery may require several weeks. Although rest is advocated, physical rehabilitation may have a positive role during the recovery phase.[272]

Prognosis

The clinical signs of complete paralysis, loss of pain perception, or LMN involvement have been associated with a poor prognosis.[272,282] If motor neurons are destroyed in an infarcted area of the spinal cord that innervates the limbs or bladder, the deficit is likely to be permanent. Because recovery from white matter damage is more likely to occur, animals with UMN deficits have better prognoses. Animals with functional recovery in 2 weeks seem to have a better prognosis.[282] Severity of neurologic signs and lesion extent on MRI are associated with outcome in dogs with ischemic myelopathy.[284] Many dogs, especially smaller dogs, make satisfactory recoveries, so early euthanasia is discouraged.

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Volume 1

Jose Alberto Landeiro, ... Cassius Vinícius Corrêa Dos Reis, in Schmidek and Sweet Operative Neurosurgical Techniques (Sixth Edition), 2012

Exposure of the Extradural VAs

The VAs is divided into four segments. V1 is the segment that runs from the origin of the artery at the subclavian artery and ends at the vertebral foramen of C6. V2 runs within the vertebral foramina from C6 through C1. V3, which is the horizontal segment of the vessel, begins at the transverse foramen of the atlas, runs through a groove on the upper surface of the posterior arch of the atlas, and ends by piercing the dura of the posterior fossa, medial and to the right of the OC. V4 is the intradural segment of the VAs and joins the opposite side vessel to form the basilar artery[13] (Fig. 43-1).

Exposure and transposition of the VAs is not needed in the basic far lateral approach, in which drilling of the OC is not required.[15,16] To transpose the vessel in the other variations of the far lateral approach, dissection and manipulation of the venous plexus around the VAs, which is sometimes referred to as the suboccipital cavernous sinus, is needed. The suboccipital cavernous plexus is connected to the suboccipital plexus through the suboccipital triangle and via the anterior vertebral vein.[4] It is also connected to the internal vertebral venous plexus, posterior and anterior condylar veins, and occipital marginal sinus. To avoid intense bleeding from the plexuses, subperiosteal detachment of the VAs from its groove in C1 is recommended. To transpose the VAs, unroofing of the C1 transverse process is also mandatory. After detachment of the VAs and plexus, laminectomy of the C1 arch as laterally as possible can be performed to expose the OC for drilling.[1,2,4,5,10]

The V3 segment of the VAs has some branches that need to be coagulated during the approach. The first and largest is the anterior VAs, which passes through the suboccipital triangle to reach the muscles of the posterior neck. The posterior meningeal artery is another branch that can be coagulated. Care should be taken not to coagulate a posteroinferior cerebellar artery (PICA) or a posterior spinal artery that arises extradurally from the V3.

Osseous Stage: Suboccipital Craniectomy and Hemilaminectomy

The target points of the osseous stage of the approach are (1) exposure of the borders of the sigmoid and transverse sinuses, (2) resection of the ipsilateral margin of the FM, (3) resection of the squama of the occipital bone to the midline, and (4) resection of the ipsilateral border of the posterior arch of C1. If additional lateral space is needed, the OC can be removed in a subsequent step.

The landmarks for orientation of the craniotomy are (1) the asterion, (2) the midline, (3) the posterior border of the mastoid, (4) the inion, and (5) the superior nuchal line. The asterion is closely related to the lateral portion of the sulcus of the transverse sinus, especially with its inferior margin. To expose the lateral angle of the junction between the transverse and the sigmoid sinuses, a bur hole is placed immediately posterior and inferior to the asterion. This retrosigmoid point is the keyhole to the lateral suboccipital approach and exposes the posterolateral border of the cerebellar hemisphere.[17] The inferior margin of the transverse sinus is located over a 50-mm line beginning at the inion and running across the superior nuchal line. This is the upper limit of the lateral suboccipital approach.

A high-speed drill is used to thin the squama of the occipital bone, and rongeurs are used to perform an occipital craniectomy. Another option is to perform a craniotomy of the posterior fossa. The mastoid air cells are the lateral limit of the suboccipital approach and are drilled until the borders of the sigmoid sinus and jugular bulb are exposed. The ipsilateral border of the FM is removed, and the occipital bone is removed to the point where it joins the OC.

To improve the inferior dural exposure, a hemilaminectomy of C1 is performed after detachment of the VAs and its plexus from its

groove in C1. If additional exposure is needed, the C2 and C3 laminas can be removed.

Small tumors without rostral extension require small craniectomy; however, craniotomy is preferred for tumors with rostral extension, as replacing the bone protects the dura and limits the postoperative occipital pain.

Condylar Stage

The OC, which is an oval-shaped osseous structure located at the base of the occipital bone, articulates the skull in relation to the cervical spine. The anterior portion of the condyle is directed anteriorly and medially toward the basion. The posterior portion ends at the level of the middle portion of the FM and blocks the angle of view to an anterior portion of the FM and of the craniovertebral junction. The resection of the posterior aspect of the condyle increases the angle of exposure, reduces brain stem retraction, and increases the working area of the posterior fossa.[4-8,10,18,19] The presence of small anterior tumors, an elongated FM, a short distance between the foramen and the brain stem, and relatively large OCs represent the ideal conditions for resection of the condyle.[20]

A high-speed drill is used to remove the posterior portion of the condyle after displacement of the VAs to avoid injury of the vessel. The amount of condyle that can be safely removed is controversial; however, biomechanical studies showed that the removal of more than 50% of the condyle leads to considerable hypermobility of the craniocervical junction, in which case fusion is indicated.[5,21] The removal of the cortical bone (which forms the external capsule of the condyle) exposes cancellous bone (which forms the core of the condyle). Drilling of this bone exposes the lateral aspect of the intracranial portion of the hypoglossal canal; this landmark is approximately at the limit of the posterior third of the condyle. Another maneuver that can be used to achieve a better view of the anterior portion of the clivus is the removal of the JT, a bony prominence situated above the hypoglossal canal, in cranial and medial extensions of the tumor.[8,11]

Many variants of the far lateral approach have been proposed, according to the amount of bony resection at the condylar region.[4,5,10,18] The basic far lateral approach comprises the steps described earlier, without condylar drilling. An occipitoatlantal transarticular transcondilar approach is performed after the condyle and the C1 superior articular facet are removed. The occipital–transcondylar approach exposes the clivus and the lower medulla and is performed after drilling the atlanto-occipital joint, condyle, and lower border of the hypoglossal canal. A supracondylar variant increases the exposure of the lateral aspect of the clivus and is directed above the condyle. During the transtubercular approach, the JT above the hypoglossal canal is removed to expose the area in front of the lower cranial nerves. The paracondylar approach is achieved via drilling of the area lateral to the condyle to resect lesions of the jugular process and of the posterior aspect of the mastoid.[5,11,13]

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