THE VERTEBRAL VENOUS PLEXUS*

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It is a pleasure to be asked to deliver this lecture honouring Dr. Harry Shields, particularly with Harry Shields in the audience. A pioneer in medical education and in anaesthesia practice, Dr. Shields is considered by many of my contemporaries as one of a powerful Canadian triumverate of another generation, the other two, of course, being Harold Griffith and Wesley Bourne. I am delighted to participate in paying homage to Harry Shields.

DURING A SERIES of studies on the effects of deliberate hypotension on the circulation, several interesting observations were made relative to the return of venous blood from the brain to the heart.[1] First, during radical neck dissections with the systolic blood pressure at about 50 mm Hg and with the patient in a head-up tilt of 30°, the jugular veins were often noted to be collapsed. Despite this, the carotid arteries were obviously transporting blood and the lack of postoperative cerebral damage suggested cerebral blood flow to be adequate.[2] Secondly, needles placed in the superior jugular bulb for the purpose of withdrawing cerebral venous blood samples failed to allow free withdrawal of blood in the head-up and hypotensive state, whereas when the patients were supine and normotensive, samples could be withdrawn easily without readjustment of the needle. The volume of blood within the jugular bulb appeared inadequate to permit free sampling in the head-up position. Thirdly, increasing intrathoracic pressure with the patient head up 30° and hypotensive did not increase the bleeding in a neck wound. Intrathoracic pressure is often elevated during this technique to reduce venous return to the heart, pool blood in the dependent portion of the body and decrease cardiac output, thus allowing blood pressure to be controlled more readily. Ordinarily, increasing intrathoracic pressure causes venous engorgement of the face and neck and might be expected to cause more bleeding in a wound. The fact was that, under these conditions, it did not. The only conclusion one could draw from these three observations was that blood was returning to the heart by a route other than the internal jugular veins, probably the vertebral venous plexus.

The vertebral venous plexus is generally described as a thin walled, valveless network of veins within and surrounding the vertebral column and extending from cranium to pelvis. It consists of an internal division which lies within the epidural space with direct connections to the vertebral bodies. The internal plexus anastomoses freely with an external division which courses paravertebrally and empties into the cervical, thoracic and lumbar veins.[3]

Generally, three routes of exit for cerebral venous blood have been described; the internal jugular veins, the emissary veins, and the vertebral venous plexus. Many

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textbooks of anatomy completely ignore the vertebral plexus in this regard and place entire emphasis on the internal jugular veins. No one appears to have explored the vertebral venous plexus as a major route of exit of cerebral venous blood nor does anyone appear to have studied the variability of cerebral outflow in the three venous routes.

Our observations were discussed with the distinguished anatomist, Oscar Batson, who called attention to unpublished work which would lend support to the thesis that the vertebral plexus might, under some circumstances, be a principal outflow tract. Batson had injected a radiopaque substance into the sagittal sinus of a recumbent monkey and by cinefluorographs had observed the material move down the sagittal sinus through the transverse sinus into the sigmoid sinus and then back out again, finally descending by way of the vertebral plexus. He opined that his data and ours were confirmatory and that additional studies should be made on this phenomenon.

Radiological Studies

Accordingly, a series of roentenological observations were made, primarily the work of my associates, Herbert Epstein and Harry Linde, with able radiological and neurosurgical assistance, the results of which were recently published in *Anesthesiology*.[4] The technique consisted of insertion of a plastic catheter via a burr hole into the sagittal sinus of anaesthetized rhesus monkeys and injecting a contrast medium through the catheter into the sinus, with the animals in either the supine or vertical position and with or without increased airway pressure. In brief, the results of these experiments indicate that an appreciable quantity of blood escapes from the brain by way of both the internal jugular veins and the vertebral venous plexus when the animals are supine and breathing quietly at ambient pressure. If airway pressure is increased, a larger quantity of blood goes through the vertebral plexus and blood from the vertebral plexus outflow appears to reflux into the inferior portions of the jugular veins. In upright animals essentially all blood leaves the brain by way of the vertebral plexus and little, if any, contrast medium is observed in the jugular veins. When airway pressure is increased and maintained the vertebral veins are visible down to at least the seventh thoracic vertebra, there is considerable reflux into the subclavian and axillary veins and there is increased density of the vertebral bodies suggesting that the contrast medium is taken up by bony matter. In none of these circumstances do the emissary veins appear to carry blood from the brain. Under the conditions of these experiments, therefore, one must conclude that the vertebral plexus always plays a part in cerebral venous drainage and in the upright position is apparently the principal route of drainage.

Anatomical Studies

The anatomy of the vertebral venous plexus was first described in the mid 1820s by Breschet[5] in France and Bock[6] in Germany. Not much was added for 100 years until the studies of Batson[7] who investigated the plexus as a pathway by which tumor or infection could be carried from pelvis to brain. Most of the subsequent

studies were concerned with the same subject, or with the role of the vertebral system in bypassing obstruction of the vena cava. An exception was Herlihy,[8] who envisioned the vertebral plexus as a separate circulatory bed, with multiple connections to other vascular beds of the body. No one appears to have delved seriously into the physiological significance of the plexus other than as an avenue for the transmission of disease.

In order to further examine the vertebral venous plexus as a cerebral outflow tract and to compare our roentgenograph studies in monkeys, we have studied the system minutely in two human cadavers.[9] One was a 50-year-old embalmed male, weighing 185 pounds, 68 inches tall and the second was a fresh specimen of a 21-year-old male, 170 pounds, and 73 inches in height. The vertebral plexus and anastomotic channels of the preserved cadaver were filled with 400 ml of a rubber latex solution injected into the right jugular vein after the system was cleaned with hydrogen peroxide. The vertebral plexus of the second specimen was filled by injecting 1000 ml of the same solution into the superior vena cava in such manner that all latex appearing in the systemic veins had to cross through the cerebral venous system. The inferior and superior vena cavae were ligated near the heart.

The vertebral plexus was found to extend within the spinal canal from the cranial dural sinuses to the pelvis. The veins of this internal plexus were most prominent laterally and were several millimeters in diameter. There were anastomoses between the two sides at each vertebral level; posteriorly, these were only visible in the thoracic area (Figure 1) and were missing in the cervical and lumbar regions; anteriorly (Figure 2) there was a filiform plexus of anastomotic veins overlying the bodies of the first several cervical vertebrae, and from this point downward, a vein or veins lying directly against the body of each vertebra joined the two sides of the internal plexus. It was apparent during dissection that the distended plexus exerted pressure upon the dura and hence upon the subarachnoid space.

At each vertebral level the internal plexus joined with the external vertebral plexus which coursed freely up and down the length of the spinal column. The anastomoses with external veins seemed most abundant in the cervical region with multiple connections to the deep cervical and vertebral veins. In the thoracic area, the external plexus not only connected with the intercostal, azygos and hemiazygos system deep in the thorax, but also joined extensively with superficial venous plexi (Figure 3). The injected veins were found on the lateral chest wall and in some instances, these veins were 5 mm in diameter. All of the thoracic drainage system as well as superficial anastomoses were well filled with the latex solution.

In the lumbar region, internal anastomoses with the external vertebral plexus were rich, but there appeared few connections with superficial venous plexi. The external plexus joined with lumbar veins and interestingly, a sizable branch joined the renal vein (Figure 4). Again, the veins of the abdominal wall, the inferior caval system and the renal venous system were filled with latex solution.

A comparison of the vertebral plexus venograms obtained in the monkey with the anatomical dissections from man show a striking similarity. Pending confirmatory roentgenographic studies in man, there seems little reason to question that what was seen in the monkey is directly applicable to man.

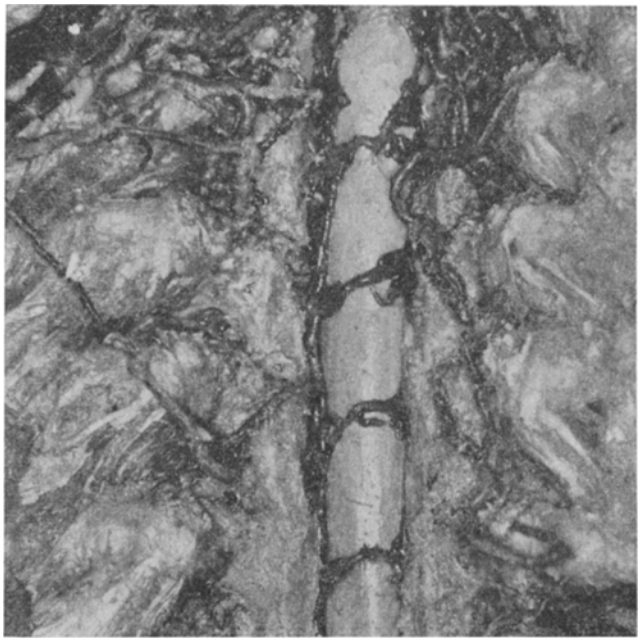

FIGURE 1. Posterior view of vertebral venous plexus at the upper thoracic level.

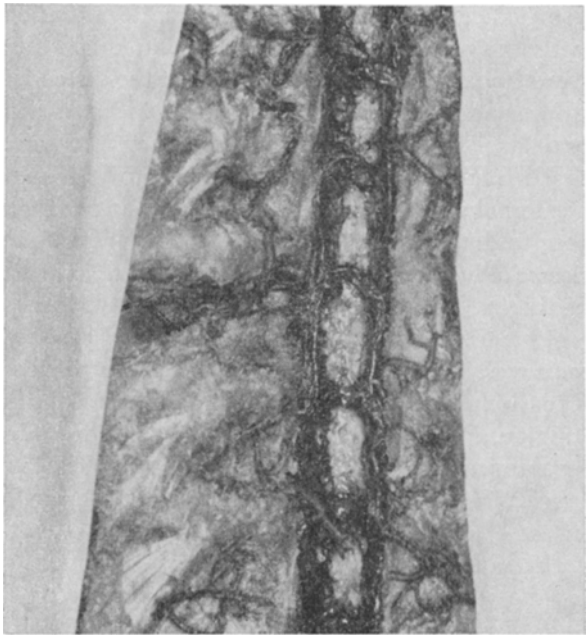

FIGURE 2. The spinal cord has been removed showing the anterior anastomoses of the lateral components of the vertebral venous plexus, and anastomoses with intercostal veins. Upper thoracic level.

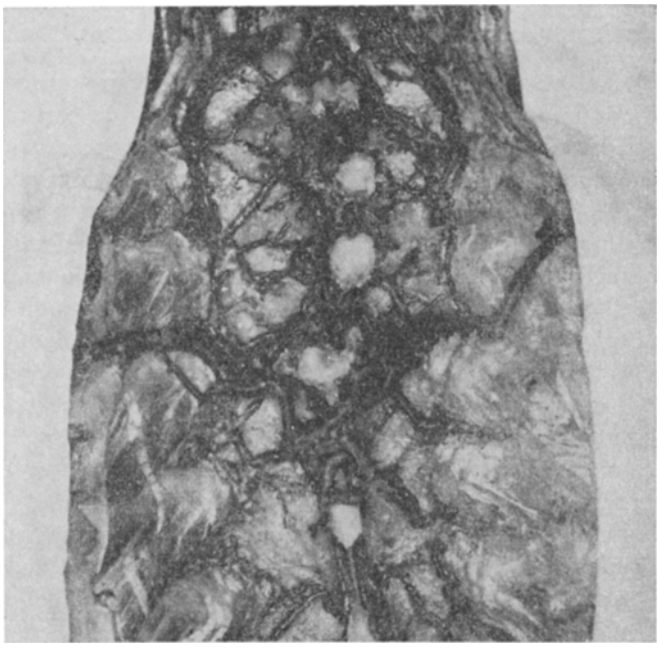

FIGURE 3. View of upper thoracic and lower cervical area. Vertebral column intact. External venous plexus visible as is anastomoses with transverse cervical plexus.

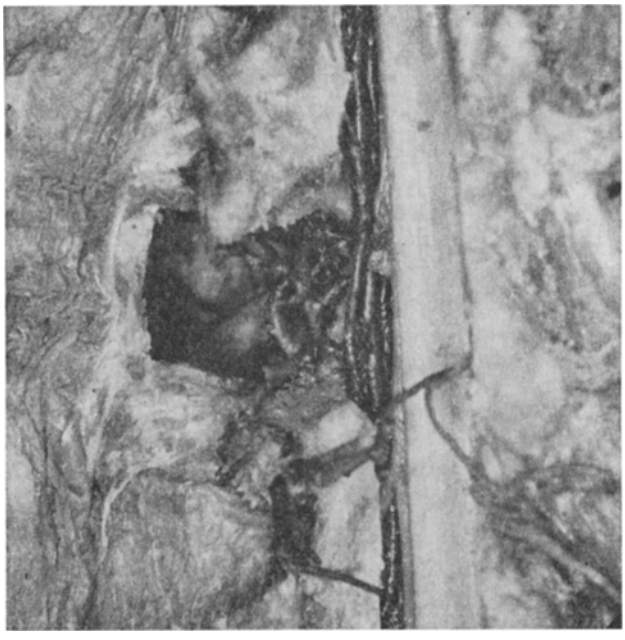

FIGURE 4. View of lumbar area, with spinal cord retracted laterally. Anastomosis of longitudinal veins of vertebral venous plexus with extra vertebral plexus and third lumbar vein. All join the renal vein.

The Physiological Significance of the Vertebral Plexus

On the basis of the observations so far summarized, one can draw certain reasonable conclusions and perhaps arrive at a hypothesis. The data clearly show that the vertebral venous plexus is a major cerebral venous outflow tract and that it is a dynamic system, not a static one. In the supine position during quiet breathing, blood leaves the brain by way of the internal jugular veins, but the vertebral plexus functions as an alternate route. In the upright position, the vertebral plexus is the major outflow tract and relatively little blood leaves via the jugular system. An increase in intrathoracic pressure causes more blood to go through the vertebral system. In the upright position, a marked elevation of intrathoracic pressure results in cerebral blood being carried well down into the thoracic vertebral plexus, into the bodies of the cervical spine, and out into the peripheral subcutaneous venous beds. The vertebral plexus, therefore, appears to be a protective device to the brain, assuring a path of exit for cerebral blood, even though blood flow back into the thorax is momentarily impeded.[10]

There are many measurements on both the blood and cerebrospinal fluid (CSF) pressure that would support this thesis. The volume of the skull and spinal canal are reasonably fixed, so much so that the translocation of 1 or 2 ml of CSF from skull to spinal canal upon standing is enough to produce significant pressure changes within the skull. CSF pressure in the supine position is essentially the same throughout the cerebrospinal axis, e.g. 100 mm H_2O. Upon standing, CSF pressures in the lumbar area rise markedly (400 mm H_2O) while those beneath the vertex fall to a subatmospheric level (Verjaal has measured −50 mm H_2O at the cisternal level, 15 cm above the heart level).[11] Blood flowing across the brain is subjected to a below atmospheric pressure in the venous circulation and further, the thin walled vessels do not collapse, since they are protected from ambient atmospheric pressure by the skull. In addition, blood flows in the direction of pressure gradients, since there are no valves in the vertebral venous plexus. We can thus envision blood being siphoned across the brain by the negative venous pressure, and then falling by gravity and pressure gradients down within the spinal canal, draining off into the cervical or thoracic veins as the pressure gradients between the subarachnoid space and the vertebral plexus equalize. The rhythmic intrathoracic pressure changes likewise facilitate drainage from the vertebral system. One cannot help but speculate that the connection between the plexus and the renal vein has a Venturi effect. This might be especially important during prolonged periods of increased intrathoracic pressure, such as during weight lifting. While the vertebral bodies and anastomotic channels could take up appreciable quantities of cerebral blood, a mechanism in the upper lumbar area that withdraws blood from the plexus and returns it to the systemic venous system would be quite a protective device. The monkey roentgenograms clearly indicate that cerebral venous blood moves well down into the lower thoracic spine.

The implication is apparent that the supine position is not necessarily the best one to facilitate cerebral blood flow. It would seem wise to slightly elevate the head to improve pressure gradients and thereby flow of blood through the vertebral plexus. There is little to support the notion that the head-down position is optimal

for cerebral blood flow. Some have thought that the deliberate hypotensive technique was safer for the brain in this position, but the data do not substantiate this in the presence of a normal blood volume.

The thought of a bellows to help pull blood across the brain is an intriguing one. Cerebral blood flow is markedly increased with elevation in P_{CO_2}. It is impossible, under these circumstances, for blood to be stored in the brain during each exhalation when intrathoracic pressure rises; it must go into the vertebral plexus and then be withdrawn during an ensuing inspiration. One cannot help but wonder what hypercarbia would do if the vertebral plexus were blocked. Is there a relationship between the sighing respiration of fainting, of deep shock, or of agonal premortal gasping, and the activation of a protective mechanism to improve cerebral blood flow?

The Anaesthetist and the Vertebral Plexus

The interest of the anaesthetist in the vertebral plexus is not confined to the part it may play in cerebral blood flow. There are other implications having to do with epidural and spinal anaesthesia and with positioning patients on the operating table. The spinal canal is a rigid bony cage, containing neural elements suspended in CSF reasonably constant in volume, and surrounded by the semipermeable dura. Between dura and bone there is the epidural space containing the vertebral plexus. Only one of the aforementioned elements is subject to rapid change and that is the volume within the vertebral plexus. There is evidence that the pressure within the plexus determines that in the CSF.

If there is obstruction of the vena cava, blood will be shunted into the vertebral plexus, increasing the volume of blood there. In order to maintain homeostasis, if the increased volume persists for long, fluid will leak out of the subarachnoid space. In pregnancy near term, there is often pressure on the vena cava, elevation of venous pressure below the obstruction and blood is shunted into the vertebral plexus to ascend and leave above the site of obstruction. This leads to a decreased volume of CSF without appreciable alteration from normal in the CSF pressure. In all likelihood, this explains the need for smaller doses of intrathecal local anaesthetics for spinal anaesthesia for Caesarean section.

Engorgement of the vertebral venous plexus under these same circumstances may account for some of the toxic responses to epidural anaesthesia in the parturient. If the patient is properly positioned in the lateral recumbent position, with the uterus falling forward relieving pressure on the vena cava, the epidural space may be relatively empty. If there is improper positioning, or if the patient has a contraction during or immediately following the injection, there may be a rapid absorption of the local anaesthetic via the engorged venous plexus.

It is also possible that the high incidence of headache following spinal anaesthesia for vaginal delivery may relate to the vertebral venous plexus. If the plexus in the lumbar space has increased in volume and, as a consequence, the CSF volume has diminished, then the stage is set for headache after spinal anaesthesia. A hole is made in the dura and in the pia arachnoid. The infant is delivered, the obstruction to vena caval flow relieved, the engorgement of the vertebral plexus subsides and

now suddenly there is a disproportion between the volume of CSF versus that in the epidural space. Fluid continues to leak from the hole in the slack membranes. With assumption of the upright position, there is greater displacement of fluid from cranium to spinal canal and more tension on pain sensitive meningeal membranes and nerve roots, with resulting headache. Anything that will increase the volume of the spinal canal will relieve the headache. An inflated basketball bladder contained in a tight abdominal binder (presumably again producing vertebral vein engorgement), saline injected into the epidural space, or fluid injected into the CSF will relieve headache. While all are successful, the latter technique is certainly no longer recommended. Why does the incidence of spinal headache appear to be less today than 15 years ago? Perhaps this relates to better hydration of the patients, resulting in a larger blood volume or the routine use of smaller needles for spinal puncture.

Concerning positioning patients on the operating table, earlier mention was made that the vertebral venous plexus was a passive system of veins extending from pelvis to cranium, without valves and entirely responsive to the pressure gradients acting upon it. Considering these facts along with the rich anastomosis of the plexus with other venous systems, it should be apparent that bleeding, during any operation which encroaches upon the confines of the vertebral system, may be strikingly influenced by physical factors affecting the system. Any body position that will increase vertebral plexus pressure will increase blood loss at operation. Any position or condition that will retard flow of blood out of the vertebral system will increase bleeding.

The causes for concern among anaesthetists and surgeons would be operations upon the spine and cranium. It is common practice to do suboccipital craniotomies or cervical laminectomies with patients in the prone position with the head lower than the thoracic spine. The physical principles already outlined indicate that blood loss will be increased under these circumstances. Either the prone body should be tilted to keep the operative site parallel to the floor or the sitting position should be considered. The latter requires vigilance to predict, control, and treat hypotension should it occur.

Excluding cervical and cranial operations, careful positioning for other operative procedures in the prone position requires considerable forethought and planning. The anaesthetist must position patients so that blood drains away from the spine – not toward it. There must be no impediment to flow in the inferior cava to cause blood to be diverted into the vertebral plexus. There must be no restriction to respiratory function so that blood is dammed within the vertebral plexus. It follows that the caval vessels and those that feed into it must be unobstructed by pressure exerted externally or indirectly as, for example, by increased muscle tension. An improperly positioned patient for a herniated disc may have excessive bleeding because of illiac vein or inferior vena caval compression, or from increase in intra-abdominal pressure from failure of freedom of abdominal action, or from increased muscular tension from light anaesthesia. Similarly, respiratory activity must be free. If the body position interferes with free exchange of air, increases intrathoracic pressure, or leads to obstruction of flow from the vessels feeding into the superior cava, then bleeding will be increased. Failure of appreciation of the importance of proper positioning may influence the outcome of an operation.

There are further ramifications of the importance of the vertebral venous plexus. The ones so far quoted require scientific proof, but are reasonable. Why does the patient with congestive failure have dyspnea? Is it related in part to his inability to pull blood through his brain by his respiratory efforts? What is the relationship of Cheyne Stokes respiration to the activity of the vertebral plexus? Is this a respiratory effort pulling blood through faster, or is it a cerebral effort dilating cerebral vessels and improving respiratory center blood supply? Why does a patient with congestive failure need to sit upright or in the semi-Fowler's position? Is it a pulmonary response (lowered diaphragm and increased vital capacity) or is it a circulatory response (better blood flow through vertebral plexus and better cerebral blood flow)?

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