Dura Mater: Anatomy

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Cranial Dura Mater

The dura mater (*Greek: tough mother*) or pachymeninx (*Greek: thick + membrane*), commonly called simply "the dura," is a membranous sac of mesenchymal origin whose outer surface lies in firm contact with the inner surface of the skull and whose inner surface surrounds the arachnoid mater (*Latin: spider + in the image of + mother)* of the cranial vault and spine. (See excellent review by Lopes [1])

Embryology [2, 3]

In early embryogenesis, a layer of mesenchyme, the meninx primitiva *(Greek: membrane + Latin: original)*, composed of neural crest cells and mesenchyme, covers the neural tube. The outermost layer becomes the dura mater, also called the pachymeninges and is entirely of mesenchymal origin. The inner layer of the meninx primitiva, which contains neural crest cells, becomes the leptomeninges (*Greek: thin + membrane*), and this layer develops into two distinct thin layers, the arachnoid mater *(Greek: spider-like + Latin: mother)* and pia mater (*Latin: pious mother*). The latter two will not be addressed. Calvarial bone develops from ossification centers within the meninx primitive.

The outer layer of the cranial dura mater, the *endosteal* layer, is continuous, through foramina with cranial periosteum, and can be thought of as the pericranium of the inner surface of the cranial bones. This layer does not extend beyond the foramen magnum, and therefore, there is no endosteal layer in the spinal dura mater. The endosteal layer of the dura also terminates near the external end of each cranial foramen and fuses with the epineurium of nerves.

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The inner layer of the dura mater, the *meningeal* layer, is a tough fibrous membrane, which is tightly adherent to the endosteal layer of the dura and less tightly adherent to the arachnoid mater. The meningeal layer of the dura mater continues through the foramen magnum to become the spinal dura mater and through the cranial foramina as sheaths to become continuous with the epineurium surrounding the nerves.

Structure [4]

In the cranium, the *dura mater* consists of two layers, the *endosteal layer* and the *meningeal layer*. (See excellent reviews by Yamashima [5] and Seker et al. [6]). The outermost or endosteal layer is the periosteum of the inner surface of the cranial vault and is composed of fibroblasts and osteoblasts with a large amount of collagen [7]. Along the cranial sutures, it is continuous with the periosteum covering the outer surface of the skull and is most densely adherent to the bone near the cranial sutures, at the foramen magnum, and over the base of the skull. It forms sheaths around nerves, including the optic nerve, as they exit the cranial vault. In children, particularly newborns and infants, this endosteal layer has relatively loose attachment to the frontal and parietal bones and the squamous portions of the occipital and temporal bones. The endosteal layer of the dura mater becomes progressively more adherent to the bone with advancing age and can be very adherent over the entire surface in the elderly.

The meningeal layer consists of fibrous and elastic tissue bands having little or no pattern of arrangement and is adherent to the endosteal layer. A layer of flat fibroblasts, the dural border cell layer, exists at the junction of the dura with the arachnoid and has prominent extracellular spaces, no extracellular collagen, and few cell junctions, thereby forming a relatively weak inner structural layer in contact with the arachnoid [8]. The meningeal layer of the dura mater has several double-layered infoldings or processes extending into the cranial vault, and several of these restrict rotary displacement of the brain. These include the falx cerebri, tentorium cerebelli, falx cerebelli, the diaphragm sella, and the roof of Meckel's cave (named for Friedrich Meckel, the Elder). The periosteal layer of the dura mater does not extend into these structures. The meningeal layer also surrounds the venous sinuses of the dura mater. Melanin-containing cells are often present in the dura, particularly around the base of the cranial vault.

The *tentorium cerebelli* (*Latin: tent of the cerebellum*) is a double-layered fold of the dura and separates the supratentorial and infratentorial spaces. The tentorium is fixed laterally to the superior edges of the petrous part of the temporal bones, and anteriorly the tentorium is attached to the anterior clinoid process. The tent shape transfers the weight, and hence downward force, of the cerebral hemispheres laterally toward the walls of the cranium and away from the foramen magnum [9].

The *falx cerebri* (*Greek: scythe of the brain*) is a sickle-shaped extension of the meningeal dura mater continuous with the dura over the midline cranial convexity

and is positioned between the two cerebral hemispheres. The narrow anterior end of the sickle is attached to the crista galli and to the dura mater of the floor of the anterior fossa. The much wider posterior portion of the falx is attached along the upper surface of the tentorium cerebelli, straddling the straight sinus. The two layers of the falx cerebri separate to enclose the *superior sagittal sinus* along the inner convexity of the skull. The smaller *inferior sagittal sinus* lies within the lower free edge of the falx cerebri.

The roof of each *Meckel's cave* or trigeminal cave, is a small, arachnoid lined pouch in which lies the trigeminal ganglion surrounded by CSF. It is formed by a double-layered fold of meningeal dura, which is located at the posterior edge of the middle cranial fossa and at the posterolateral extent of the cavernous sinus immediately lateral to the internal carotid artery with its intracranial opening facing the posterior fossa [10].

The *diaphragma sellae (Latin: partition saddle)* is a circular double-layered fold of the meningeal layer of the dura mater, which covers the sella turcica *(Latin: Turkish saddle)* and forms the hypophyseal (pituitary) fossa. The infundibulum of the pituitary gland passes through a round opening in the diaphragma [11, 12].

The *falx cerebelli (Latin: scythe of the small brain)* is a small, sickle-shaped, double-layered sagittal infolding of the meningeal dura. Superiorly it is attached to the tentorium cerebelli, and inferiorly it extends downward in the vallecula cerebelli and posterior cerebellar notch toward the foramen magnum. Its margin contains the *occipital sinus*. The falx cerebelli in most patients is small and may not be identifiable in its lower half. In small children, it occasionally divides inferiorly with an extension for several millimeters to each side of the foramen magnum [7].

The *dura mater of cranial base* has the same structure as that of the convexity but is densely adherent to the bone, except across the floor of the frontal fossa.

The *arachnoid villi* are one-way valves in the paths of egress of CSF from the subarachnoid space into the venous blood, but they do not appear until 7–8 months of age [13, 14]. An arachnoid villus consists of subarachnoid tissue, surrounded by arachnoid membrane projecting into a parasagittal venous lake or into the venous sinus, and is covered by a thin layer of endothelium. The arachnoid villi are soft small protrusions of thin spots of mater, usually near the sagittal sinus but occasionally near a transverse sinus, which act as one-way valves [13]. They are not present in infants and children but are usually present by 7 years of age. Overlying bone is absorbed, probably caused by pressure from the expanding arachnoid against the overlying thin dura mater. This accounts for the extensions of the dura into depressions on the inner surface of the calvarium. As arachnoid mater in these regions expands and penetrates the dura, it comes into direct contact with the vascular endothelium of the dura along the large venous sinuses.

A *subdural space* does not exist in normal meninges, except as being a *potential space*. However, in response to trauma or other pathologic process, a cleavage plane develops near the dura–arachnoid continuum, in the *border cell layer* of the dura and therefore not, in a literal sense, beneath the dura [8].

Arteries of Cranial Dura Mater [3, 15]

The arterial supply of the meninges is almost exclusively concerned with the endosteal layer of the dura mater. Although the middle meningeal artery is the dominant supplier for the supratentorial dura mater, dura requires little supply, and the major end organ supplied by this vessel is the calvarial bone. Arteries in the dura mater have extensive anastomotic connections with one another, particularly near the dura of the falx, tentorium, and parasellar region.

The arterial supply to *dura of the anterior cranial fossa* is from the meningeal branches of the anterior ethmoidal and posterior ethmoidal arteries, the ophthalmic artery, and from a frontal branch of the middle meningeal artery. The arterial supply to *dura mater of the middle cranial fossa* is primarily from the middle meningeal artery, but there is also contribution from the ascending pharyngeal artery and the internal carotid artery.

The dura mater of the posterior cranial fossa receives supply from the meningeal branches of the ascending pharyngeal artery (via hypoglossal canal and jugular foramen) and meningeal arteries from the occipital artery (via jugular or mastoid foramen) and from the vertebral artery (via foramen magnum) [16].

In summary, small branches of the internal carotid artery supply the medial-most dura mater of the anterior and middle cranial fossae and a small amount of the posterior fossa. Branches from the external carotid artery supply the much larger lateral dural territories of all three fossae, and the vertebral arterial system supplies the more medial dura mater of the posterior cranial fossa and the dura mater near the foramen magnum.

Anterior Meningeal Artery (Falx Artery or Anterior Falcine Artery)

The anterior meningeal artery is a branch of the anterior ethmoidal artery, which is a branch from the ophthalmic artery as it accompanies the nasociliary nerve in the orbit and then through the anterior ethmoidal foramen. This anterior meningeal artery supplies a variable area of bone and dura mater of the anterior fossa, including the dura over the cribriform plate. One small branch enters the falx and becomes the anterior falx artery.

Confusingly there is another artery with the name “anterior meningeal artery.” It is a branch of the vertebral artery at the C2 or C3 level and supplies various adjacent structures including the bone and an area of the dura mater at or below the foramen magnum.

Middle Meningeal Artery

The middle meningeal artery is a branch from the retromandibular part of the maxillary artery, which is a branch of the external carotid artery. It runs near and often directly through this nerve and then through the foramen spinosum of the sphenoid bone in the floor of the middle fossa. The middle meningeal artery is the largest of the arteries that supply the cranial bone and meninges and, throughout much of its proximal intracranial course, lies within a groove along the greater wing of the sphenoid bone and on the undersurface of the frontal and parietal bones. It is very

often surrounded with bone along the lateral extent of the greater wing of the sphenoid bone and occasionally for several millimeters of the parietal bone. Its branches spread upward to the vertex and backward into the occipital region. The largest branch of the middle meningeal artery is the anterior branch, and it typically lies approximately beneath the pterion. The posterior branch of the middle meningeal artery courses posteriorly along the undersurface of the squamous part of the temporal and parietal bones. There are anastomotic connections through the superior orbital fissure, with the ophthalmic artery and lacrimal artery. Occasionally the ophthalmic artery arises as a branch of the middle meningeal artery or lacrimal artery. Rarely there is an accessory meningeal artery or pterygomeningeal artery, which supplies a small area of the dura mater in the floor of the middle fossa, the trigeminal ganglion, and some muscles before entering the cranium.

The tentorium cerebelli receive arterial supply from the external carotid, internal carotid, posterior cerebral, and vertebral arteries. The meningohypophyseal artery arises from the internal carotid artery and divides into the tentorial artery of Bernasconi and Cassinari and dorsal meningeal arteries. The former courses in the tentorium near its free edge and supplies anterior and lateral portions of the tentorium and often the oculomotor, abducens, and trochlear nerves. The artery of Davidoff and Schechter, a branch of the P2 segment of the posterior cerebral artery, courses beneath the superior cerebellar artery and enters the undersurface of the tentorium cerebelli near the midpoint of the incisura to supply the medial portion of the tentorium cerebelli [17].

Posterior Meningeal Artery

The posterior meningeal artery enters the cranium through the jugular foramen, hypoglossal canal, or foramen magnum. It is usually a branch of the ascending pharyngeal artery but can arise from the occipital artery. It is the largest vessel supplying the bones and dura mater of the posterior fossa.

Veins of Cranial Dura Mater

Meningeal veins course within the endosteal layer of dura mater. The larger of these tend to run near or in contact with the larger arteries in the dura and drain into the pterygoid venous plexus or sphenoparietal sinus. At the site of dural folds, large venous structures, called venous sinuses, develop between the opposing meningeal layers of the dura mater. The venous sinuses drain blood and hence cerebrospinal fluid from the brain into the internal jugular veins. *Bridging veins* connect the veins of the underlying neural tissue with the dural sinuses and therefore lie within the subarachnoid space.

Veins Attached to Arachnoid Mater

Many cerebral veins and bridging veins are attached to the arachnoid mater by fibrous arachnoidal strands. These adhesions must be disrupted while reflecting a

dural flap, without disrupting the vessel. This can be done with a small blunt instrument, and others must be cut with a small, pointed knife or with microscissors.

Venous Sinuses

The *superior sagittal sinus* is a large, midline intracranial venous structure coursing along the upper extent of the falx cerebri, from the foramen cecum anteriorly, to the tentorium posteriorly. Its posterior few centimeters usually tilts toward the right at the confluence of sinuses (torcula Herophili) and therefore drains primarily into the right transverse sinus. Valve-like lamellae, the *chordae willisii*, exist within the lumen of the superior sagittal sinus, chiefly in its parietal and occipital sections, and can partially cover entering cerebral veins [18, 19]. Their precise function is not known, but they are thought to influence laminar flow. The two *transverse sinuses* lie in the posterior edges of the tentorium, receive blood from the superior sagittal sinus, and drain into the sigmoid sinuses. The *occipital sinus*, which is prominent in infants but rudimentary or absent in adults, lies along the edge of the falx cerebelli and also drains into the confluence of sinuses.

Lymphatics of Cranial Dura Mater [20–22]

It was long taught that lymphatic vessels did not exist in the dura mater, but there is now a growing literature on a functioning dural lymphatic network, which drains toward the superior sagittal sinus, along the cranial nerves, and through the cribriform plates. These vessels have a role in drainage of CSF.

The lymphatic drainage of the falx cerebri occurs via the meningeal lymphatic vessels, which run parallel to the dural sinuses. These lymphatic vessels drain primarily along a similar path as the dural sinuses, pass through the jugular foramen, and empty into the deep cervical lymph nodes. Lymphatic channels from the falx cerebri drain anteriorly through the cribriform plate into the lymphatic channels of the nasal mucosa. Lymphatic vessels of the meninges have few if any valves [23].

The glymphatic drainage from the brain probably empties into the lymphatic vessels of the dura and falx and then into the extracranial lymphatic system [24]. Details of this system are not within the scope of this book.

Innervation of Cranial Dura Mater

Dura mater of the anterior cranial fossa receives innervation from the ophthalmic division of the trigeminal nerve, through the anterior ethmoidal, posterior ethmoidal, and nasociliary nerves. Dura mater over the middle fossa is innervated by branches from the ophthalmic nerve and some small contributions from the maxillary and mandibular divisions of the trigeminal nerve. The upper surface of the tentorium receives innervation from both the maxillary and mandibular nerves, whose branches course posteriorly along the dura of the cavernous sinus. The undersurface of the tentorium and the dura mater of most of the posterior cranial fossa are

innervated by branches of the vagus nerve (X) and glossopharyngeal nerve (IX) with contribution, near the foramen magnum, from cervical nerves C2 and C3. Afferent fibers in the dura mater reside in the trigeminal ganglion and sympathetic fibers are from the superior cervical ganglion. The falx cerebri is innervated by all three branches of the trigeminal nerve [25–27].

Abnormal Cranial Dura Mater

Thin Dura

There are no criteria for what constitutes abnormally thin dura, but nonetheless the designation has clinical usefulness. Dura in neonates and infants is noticeably thin. Dura is often thin in patients who have chronically elevated intracranial pressure. Elderly people are often said to have abnormally thin dura, perhaps because of the difficulty surgeons may encounter when separating the dura from the bone. Little if anything has been published on unusually thin dura.

Very thin dura can be opened with little or no difficulty, but it is easily torn by retraction. It becomes desiccated more quickly than does the normal dura and therefore requires more attention to keeping it moist with wet Cottonoids® and frequent irrigation. The act of suturing can easily tear the dura, and needle puncture sites may stretch larger than expected. Thin dura should be gently closed using 4-0 or 5-0 sutures to achieve continuous approximation of edges.

Thick Dura

Abnormally thick dura occurs in several settings and usually comes to attention as it is being incised, for example, in patients who have undergone prior surgical manipulation, in local regions of plexiform neurofibromatosis, and occasionally in elderly patients with Alzheimer disease. Dura can also be abnormally thick in settings of chronic intracranial hypotension.

Opening abnormally thick dura presents little difficulty unless it is hypervascular, infiltrated with tumor, or adherent to the brain, as can occur following prior surgery (irradiation or infection). Retraction and closing of thick dura rarely present a significant difficulty.

Hypervascular Dura

Hypervascular dura may occur in neurofibromatosis, vascular malformation, or neoplastic involvement and can be occasionally encountered unexpectedly in normal patients. It also may occur in infected dura and in the dura that has had undergone surgery. Hypervascular dura may have normal attachment to bone or can be adherent, making surgical separation difficult, often with significant bleeding from both dura and bone. The opening, retraction, and closing of hypervascular dura must be done slowly with constant attention to hemostasis. Tamponade with Cottonoids® and mild manual pressure is effective for temporary control. Persistent bleeding can be managed with liberal use of bipolar electrocautery, Weck® clips, and multiple ligating sutures. In association with neoplasia, much of the hypervascularity is a

component of the lesion and will be excised. Extensive coagulation can cause substantial dural contraction, making closure problematic.

Neoplastic Involvement of Dura

Neoplastic involvement of the dura is commonly associated with meningiomas. Infiltration of the dura also occurs in malignant tumors of the cerebrum and in the cranial base. These may grow through the dura, for example, chordomas of the clivus. Neoplastic infiltration of the dura can result in difficult hemostasis, particularly when the tumor has grown through the dura and into the bone, as occurs with meningiomas, malignant brain tumors, and a few primary tumors of bone such as chordoma, neuroesthesioma, and melanotic progonoma. Melanin-containing cells are often present in the dura, particularly around the base of the cranial vault, and their presence is not always indicative of neoplasia; however, there are several melanin-containing tumors of dura, including melanotic meningioma, melanotic progonoma, and meningeal melanomatosis.

Dura involved with tumor is commonly managed with circumferential resection. Arteries and veins in the surrounding normal appearing dura can be cauterized, but large arteries supplying the lesion will require ligation.

Neurofibromatosis

Hypervascular dura associated with neurofibromatosis type 1 tends to be confined to small regions. Hypervascularity associated with neurofibromatosis type 1 tends to be confined to regions of grossly identifiable abnormality, and hemostasis in the region can be difficult and time-consuming. Dura in patients with plexiform neurofibromatosis, particularly when extending intracranially from the orbit, can be very hypervascular and have a network of vascular channels that respond poorly to electrocoagulation. Compression with multiple hemostats combined with extensive cauterization or placement of sutures around identified bleeding sites may be required. Such measures result in desiccation and contraction of the dura. Hemostasis obtained by packing may achieve temporary success, but bleeding usually starts anew when the packing is removed.

Dense Adherence of Dura to Bone

Dura is densely adherent to the cranial bone along the cranial sutures in normal neonates, infants, and young children and becomes less adherent as the sutures close and generally more adherent over the entire cranial convexity in elderly patients. Dense adherence is also normal at all ages across most of the cranial base, except for the floor of the frontal fossa. Dura along the edges of prior twist drill sites, burr holes, and craniotomy sites is almost always densely adherent. Densely adherent dura can be thinner than normal, in elderly patients, or often appears so to the neurosurgeon when attempt is being made to preserve its integrity. Dura in an irradiated field, as in patients with many types of brain tumors, can be quite adherent to overlying bone or almost unattached. Prolonged intake of phenytoin, as was common in years past in patients with epilepsy, resulted in dense adherence of the dura to the knobby irregular inner surface of the cranial bone, also caused by the drug. These

patients are now in midlife or older. Dura that has been irradiated can be very adherent but is occasionally loosely attached.

Adherence of Galea to Brain Through Defect in Bone

If brain parenchyma comes into firm contact with galea, as can occur following craniectomy for decompression in which no intervening barrier was implanted, or through a dural defect resulting from a dehisced durotomy in a setting of high intracranial pressure, the brain parenchyma very commonly becomes fibrotically attached to the galea. At the time any of future surgery in the area, the two can be difficult or impossible to surgically separate without considerable injury to the brain.

Excess Dura

Bulging Dura in Craniotomy Sites

An intact dural envelope, whether overlying brain or over a CSF apace, can displace a bone flap whenever intracranial pressure exceeds atmospheric pressure and then expand into the cranial defect. Early displacement of a flap is more likely to occur when the flap is poorly secured, but chronically elevated pressure may elevate a flap after weeks or months. Also, intact dura occasionally will bulge through a cranial defect following craniectomy or come to attention during surgery for cranioplasty. If the excessive dura is not addressed at the time of subsequent cranioplasty, there is significant risk of delayed displacement of the flap, regardless of the material used. Free fragments of bone immediately conform to the contour of the underlying dural envelope. If overlying bone fragments, bone flap, or synthetic material is used to eliminate the dural bulge by compression, there is significant risk of delayed displacement of the flap and need for repeat surgery.

Dural Bulge in Infants

Normal intact, non-bulging dura lying beneath a postoperative cranial defect of an infant may begin to bulge within a few days or weeks following surgery and cause progressive resorption of bone, similar to that which occurs in an expanding skull fracture. This is most often seen following surgery for correction of craniosynostosis and is not apparently related to elevated intracranial pressure.

Surgical correction is required and must include reduction of the dural bulge by either imbrication or by desiccation of the dura with electrocautery, followed by secure repair of the cranial defect.

Macrocrania Secondary to Hydrocephalus

Within a vault that has functioning cranial sutures, the envelope can steadily expand over months or years in response to intracranial hypertension. Surgery to reduce cranial volume, reduction cranioplasty, is rarely done except in extreme cases and should be understood as being a long and high-risk surgical undertaking. These patients have severe ventriculomegaly, and a thin cerebral mantel. Reduction cranioplasty results in excess dura.

Abnormally Shaped Dural Envelope

Patients with craniosynostosis have abnormally shaped cranial vaults and, consequentially, abnormal dural envelopes. Most surgeries for correction of craniosynostoses alter the cranial vault toward a more spherical configuration. Therefore, less dura is required for enclosure. Some surgeons ignore the dura in these cases, but others, including the author, often use electrocautery to shrink the bulging regions by desiccating multiple spots or lines across the dura. If a bulging region of dura is ignored, it may slowly displace the overlying bone and return to the abnormal cranial shape.

Dural Defects

Adverse Consequences

Adverse consequences of dural defects are influenced by the size and site of the defect, patient's age, intracranial pressure, operant pathologic processes, the integrity of cranial bone and scalp, and by the neurosurgical interventions, which may have produced the defect. Problems associated with dural defects include leakage of CSF, herniation of brain, pneumocephalus, bacterial contamination of the CSF and brain, displacement of bone flaps, and expanding fracture of overlying bone; however, many small defects in dura have no recognizable adverse consequences.

A dural defect is a window through which the brain can herniate whenever centripetal force of intracranial pressure exceeds the opposing force. An intact dural envelope in neonates, infants, and young children has an especially important role of being a restraining envelope within which intracranial pressure is distributed approximately equally in all directions as the brain grows. Most neurosurgical operations that include durotomy, excluding twist drill holes and operations done for decompression, require some form of closure to reestablish a dural envelope. This is particularly important in pediatric patients because the normal growth of the brain produces a steady centrifugal force against the dural envelope, and a large defect in the dura is a region of lesser resistance to expansion of the brain, which will result in slow cerebral herniation. Adults with normal intracranial pressures may tolerate relatively large persistent dural defects beneath a cranial bone flap; however, a dural defect beneath a craniectomy site results in the brain being in contact with the pericranium, to which it commonly adheres. This can cause neurosurgical difficulty if that region needs to be exposed in the future.

Defects in the cranial dura of any size predisposes to CSF leakage and to the herniation of the brain. A large dural defect through which the brain is expanding, for example, a growing brain of a child, or high intracranial pressure may at any age allow herniation of the brain tissue through the opening.

CSF Leakage

Defects in the cranial dura of any size predispose to CSF leakage into the epidural space and beyond. Leakage can occur through any size defects, even tiny ones, but is much more likely to occur in sites where the dura is not in contact with the brain

tissue, for example, over cisterns, large cerebral sulci, in sites overlying atrophic brain, and sites of resection of tumor or brain tissue. Needle holes and openings of 1 or 2 mm are usually not problematic but can result in leakage of CSF when CSF pressure significantly exceeds the pressure against which it must flow. High intracranial pressure greatly increases the likelihood of CSF leakage. CSF that leaks through an opening in the cranial dura can accumulate in the epidural space, subgaleal space, through a scalp incision, or any combination of these. CSF drainage requires a space into which to flow, for example, epidural space resulting from postoperative dura not being adherent to the bone. Persistent flow of CSF through a tiny dural defect can impair healing and result in the development and persistence of a pocket of CSF in an epidural or subgaleal space, and the tract can become epithelialized. (The clinical details of CSF leakage, their diagnoses, and management will not be addressed.)

Herniation of Brain at Sites of Dural Defects

Brain not uncommonly bulges progressively through a dural defect resulting from its being intentionally unclosed or in which the dural suture line became disrupted following surgery. A dural defect in which brain is expanding, for example, a growing brain or high intracranial pressure at any age, allows the enclosed brain to herniate through the opening. In older children and adults, this bulging may occur only if there is high intracranial pressure, and the bulge will often be flat or concave in the upright position.

Large dural defects are stretched ever larger by steadily growing brain, thereby allowing progressively worsening herniation. When a large defect is present, the expanding brain, whether from normal growth or pathologic cause, herniates the dural opening and steadily enlarges, and the overlying bone is resorbed. Over several months, the mid portion of the brain can shift. After the first year of life, this risk diminishes, except in settings of elevated intracranial pressure.

Risk of herniation diminishes after the first year of life except in settings of elevated intracranial pressure. However, in children with large dural defects beneath a cranial bone slowly progressive outward expansion of the bone flap can occur. In patients who have undergone craniectomies for decompression with the dura left widely open, the brain parenchyma becomes adherent to the galea and can become difficult or impossible to surgically separate without considerable cortical injury.

Patients who have large cranial defects and normal CSF dynamics often experience significant neurological problems when sitting or standing (syndrome of the trephined).

Fungus Cerebri [28, 29]

Fungus cerebri is a herniation of brain through a defect in scalp at the site of a cranial defect and having a mushroom-like gross appearance. The herniation of brain occurs in response to severe cerebral edema, often but not always following surgery, which is followed by dehiscence of durotomy and scalp; however, instances have occurred into the mastoid and middle ear. Historical treatment included frequent change of dressings, often followed by shaving of slices of brain until the scalp could be securely repaired, uncommonly but not universally followed by death. The

entity of fungus cerebri disappeared, with rare exceptions, following the introduction of steroids to reduce cerebral edema, and the evolution of surgical techniques and procedures including decompression craniectomies. The treatment of fungus cerebri requires wide surgical exposure, excision of any herniated necrotic brain tissue, and the secure closure of the dura and scalp. Cranioplasty should probably be delayed because of wound contamination.

Causes of Dural Defects

The most common cause of insufficient dura for primary closure is contraction caused by desiccation, electrocoagulation, or by prolonged intraoperative exposure to air, particularly if the dura has not been tightly retracted during surgery. Some neurosurgical operations require the resection of sections of dura, for example, when there is neoplastic invasion. Iatrogenic injury of dura occurs when a router penetrates the dura and shreds a path along the course of an osteotomy, particularly when the dura is tightly adherent to overlying bone. Traumatic laceration of the dura is common, but avulsion is extremely rare.

Encephalocele

The brain at birth is found to be bulging through an area of missing cranial bone. The dura surrounding an encephalocele is often hypoplastic, and the herniated brain is usually if not always structurally abnormal. These occur in or near the cranial midline, and size can vary from 1 cm to larger than the cranial cavity. Severe dural attenuation and dural defects may exist occur in association with encephaloceles in the floor of the frontal fossa.

Meningocele

This is a CSF-filled outpouching of the dura, often, but not consistently atretic, of the dural envelope. The term is most often used for a congenital abnormality in or near the cranial midline. A meningocele differs from an encephalocele by not containing the brain tissue. Typically, the meningocele has a small communication with the cranial dural envelope. The term is also used for post-surgical outpouching of the dura, regardless of the location.

Surgery for Neoplasia

Most dural defects related to surgery for neoplasia are the result of resection with the tumor. Tumors such as meningioma and neuroesthesioma can grow through the dura of the floor of the frontal fossa.

Aplasia Cutis Congenita

This is a birth defect in which there is one or more regions of missing or severely atretic skin and may occur any part of the body. When involving scalp, there may also be a cranial defect or cranial and dural defect. Defects that include the cranium

typically overlie the superior sagittal sinus and, in the hours and days after birth, and if not managed appropriately, desiccation often leads to cracking across the sinus and exsanguination.

Trauma

Trauma to the low forehead or midface is the most common cause of dural disruption in the anterior cranial base and can occur at any age but much more commonly in older children and adults. This may come to attention with the identification of CSF leakage or when directly visualized during surgery. These CSF leaks are often through dural disruptions in the cribriform plate and can be difficult to identify radiographically and only identified at time of surgical reconstruction.

A raw acute edge of fractured cranial bone, when acutely driven inward, can lacerate dura, with significant concomitant injury to the underlying brain. The dural edges immediately contract from the facture line by a few millimeters and, if not treated, will contract by a centimeter or more over the following days.

When the same injury occurs in neonates or infants (usually under 1 year of age), the normal growth of brain will, over weeks and months, stretch the lacerated dura and allow progressive cerebral herniation with resorption of overlying calvarial bone, hence the name expanding or growing fracture. Expanding fractures can also occur from an unrecognized or unrepaired elective durotomy in an infant.

Iatrogenic (Greek: Healer + Produced) Injury

The most common cause of dural defects coming to neurosurgical attention is the contraction of dura caused by desiccation during long neurosurgical procedures, particularly if the dura was not tightly retracted during surgery. A path of injury to the dura can be the result of accidental penetration and shredding by a router while making an osteotomy.

Dural puncture by a drill bit of 3–4 mm diameter, commonly called a twist drill, only rarely causes a problem, which requires neurosurgical attention. Although an artery of the dura can be disrupted but the relatively dull tip and its rotary action as it advances usually stretches and pushes aside arteries directly in the path. Single puncture of the dura with a sharp needle produces a much smaller hole but does not push the vessels aside and therefore may penetrate any vessel in the path of puncture. Multiple needle punctures made to produce a larger hole in the dura or greatly weaken the dura for more easy passage of a larger instrument greatly increases the risk of penetration of a cerebral artery.

Iatrogenic dural injury may occur when a router penetrates the dura and shreds a path beneath the course of an osteotomy, particularly in areas where the dura is tightly adherent to the overlying bone. The path of shredded dura is always several millimeters wider than the diameter of the bit and the osteotomy.

Surgical Resection of Dura

Portions of dura may be resected during neurosurgical operations, for example, resection of meningiomas and vascular malformations of the dura.

Dural Defects by Location

Region of Sella Turcica

Defects in this region present with CSF rhinorrhea following transsphenoidal surgery for tumors (e.g., pituitary adenomas or craniopharyngiomas). Spontaneous CSF leaks in this region are rare but reported and are often associated with an empty sella. Fluid in the sphenoid sinus that is apparent after cranial trauma can be CSF that has leaked through a dural rent associated with a non-displaced fracture, and the fracture can be quite difficult to radiographically identify.

Anterior Cranial Base

Trauma to the low forehead or midface is a common cause of dural disruption in the anterior cranial base and can occur at any age but more commonly in older children and adults. This may be immediately visibly apparent or on early neuroimaging of a victim of trauma, but not uncommonly this may first come to attention with the identification of CSF rhinorrhea hours or days after trauma. Delayed CSF leaks are often through dural disruptions over the cribriform plate or into the frontal sinus. These sites can be difficult to identify radiographically and even at the time of surgical exploration.

Dural defects or severe attenuation of the dura occurs in association with encephaloceles in the floor of the frontal fossa, almost always in or near the midline, and is commonly associated with significant craniofacial abnormality that includes hypertelorism.

Floor of Middle Fossa

Defects in the dura of the floor of the middle fossa are commonly a result of resection of tumors, for example, meningiomas. Many neurosurgeons place an onlay patch of the pericranium or a synthetic material, for example, DuraGen®, over the defect. Others leave these dural defects unrepaired, and adverse consequences are rare. It is difficult to impossible to suture a graft in this location.

Trauma that fractures the temporal bone can disrupt the attached dura and allow CSF to flow into the middle ear. If the tympanic membrane is disrupted, the result is CSF otorrhea, but if the tympanic membrane remains intact, CSF can flow through the Eustachian tube and present with rhinorrhea or as an intermittent salty taste. If the leakage of CSF ceases spontaneously within a few days, no treatment is required, but persistent leakage has significant risk of meningitis. Details of management of rhinorrhea and otorrhea are not within the scope of this book.

Posterior Fossa

Dural defects in the posterior fossa, which come to neurosurgical attention, are almost exclusively post-surgical and are commonly the result of problematic surgical closure. These defects present as post-surgical CSF leaking or accumulation of CSF in the extradural space or within soft tissues. Elevated intracranial pressure greatly increases this risk. Postoperative CSF leaks following surgeries within or near the cerebellopontine angle are usually avoidable.

Which Dural Defects Require Repair?

Elective durotomies are, with a few exceptions, meticulously closed at the end of all intracranial surgeries. It is particularly important in children to close dura and hence reestablish the dural envelope. Most neurosurgeons ignore needle holes and dural openings beneath burr holes unless there is a persistent postoperative CSF leak. Some neurosurgeons do not repair mild to moderately large dural gaps in the floor of the middle fossa. Very large dural openings, such as the stellate durotomies accompanying decompression craniectomies, are intentionally left unrepaired to allow cerebral herniation; however, the dural opening site is usually covered with a synthetic material, such as DuraGen®, to prevent adherence of the brain to the galea.

A strong attempt should be made to achieve a secure closure of all openings in the spinal dura. There are settings, however, in which this may not be safely accomplished, for example, the ventral defect after resection of diastematomyelia, and when surgery was done to decompress unresectable tumor.

Irradiated Dura

Irradiated dura often has a gross appearance of being thin, dry, inelastic, and grayer in color than normal dura, but if intact, its function as an enveloping membrane is normal. It rarely presents a surgical problem when being incised but can be difficult to close.

Spinal Dura Mater [5, 30, 31]

The spinal dura mater is an extension of the meningeal layer of the cranial dura mater, and it is attached firmly to the surrounding bone only at the foramen magnum. The spinal dura mater is structurally similar to the meningeal layer of cranial dura mater; however, its fibrous and elastic tissues are arranged in bands, which generally course parallel with one another in a longitudinal arrangement. Spinal dura is composed of an fibroelastic outer layer, a middle layer, and, like cranial dura, an inner dural border cell layer.

Spinal dura mater has no endosteal layer; however, the spinal representation of the periosteal layer of cranial dura mater exists in the spine as the periosteum of the vertebral canal and is not a component of the spinal dura. There may be small fibrous connections of the posterior side of the spinal dura mater to the C2 and C3 vertebral arches and a few connections to the posterior longitudinal ligament in the mid sacral region. In the lower sacral region, the dura mater is tightly attached to the filum terminale, and this structure is tightly attached to the periosteum of the coccyx.

The tubular compartment formed by spinal dura is the theca (*Greek: box or place to put*) or thecal sac, which surrounds the spinal cord, cauda equina, and CSF.

Vasculature of Spinal Dura Mater

The arterial supply to the spinal dura mater is primarily arises from the anterior and posterior radicular arteries [15, 32]. There is a network of arterial vessels and veins in the epidural space, which have connections to vessels of the dura mater [33]. Veins tend to follow arteries in the dura and drain into segmental veins. A spinal venous sinus has been described near the midline of the dorsal lumbosacral dura [34].

Innervation of Spinal Dura Mater [35]

The spinal innervation of dura mater has two components. The ventral dura mater contains a dense plexus of longitudinally coursing nerves having contributions from *sinuvertebral nerves*, nerve plexus from the posterior longitudinal ligament, and the radicular branches of segmental arteries. Sinuvertebral nerves, also known as meningeal branches of the spinal nerves, recurrent meningeal nerves (sinuvertebral nerves or *recurrent nerves of Luschka*) are small nerves arising from each spinal nerve somewhere near the anterior and posterior rami. They pass through the intervertebral foramina to supply facet joints, posterior longitudinal ligament, posterior-lateral part of the annulus fibrosis, and the ligaments and periosteum within the spinal canal. These nerves may span as many as eight segments, with considerable overlapping. There are fewer nerves in the dorsal portion of spinal dura mater, and they do not form a plexus and may not reach the most medial dorsal region. The dorsal nerves are derived from the ventral dural plexus at the "intersleeval" parts of the dura mater. The curled bundles of nerve fibers allow displacements of the spinal dura mater during normal flexion and extension of the spine.

Abnormal Spinal Dura

Openings in spinal dura, regardless of size, will result in leakage of CSF into epidural space and often beyond. In settings in which the spinal cord is in continuous contact with a durotomy site, the spinal cord can herniate through the opening if the dura is not securely repaired.

Iatrogenic Defects

Persistent CSF leakage following lumbar puncture causes abnormally low intracranial pressure and can be a cause for positional headache and meningeal enhancement on MRI [36].

Unintended surgical disruption of the spinal dura can occur during laminectomy for spinal decompression, particularly lumbar decompressions and in the course of exposing a spinal nerve.

Chiari malformations, lipomyelomeningocele, spinal meningocele, myelomeningocele, and split cord malformation (diastematomyelia).

The dural abnormalities associated with these disorders are addressed individually in Chap. 18.

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