Neuraxial Anesthesia in Parturients with Intracranial Pathology

A Comprehensive Review and Reassessment of Risk

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ABSTRACT

Parturients with intracranial lesions are often assumed to have increased intracranial pressure, even in the absence of clinical and radiographic signs. The risk of herniation after an inadvertent dural puncture is frequently cited as a contraindication to neuraxial anesthesia. This article reviews the relevant literature on the use of neuraxial anesthesia in parturients with known intracranial pathology, and proposes a framework and recommendations for assessing risk of neurologic deterioration, with epidural analgesia or anesthesia, or planned or inadvertent dural puncture. The authors illustrate these concepts with numerous case examples and provide guidance for the practicing anesthesiologist in determining the safety of neuraxial anesthesia.

Despite the decrease in case-fatality rates for general anesthesia for cesarean delivery from 16.8 per million (1991–1996) to 6.5 per million (1997–2002),1 neuraxial anesthesia remains the technique of choice for the majority of these procedures.2,3 A timely and safely administered neuraxial technique can potentially avoid (1) instrumentation of the parturient’s airway, (2) fetal exposure to the potentially toxic effects of general anesthetics on the developing brain,4–7 (3) a high-risk setting for increased risk of awareness under general anesthesia,8 and (4) the uterine relaxant effects of volatile anesthetics. In addition, neuraxial anesthesia for cesarean delivery allows the parturient and partner to experience the birth. For postoperative analgesia, the literature supports the use of epidural opioids for improved efficacy in comparison with intermittent injections of IV or intramuscular opioids.2 For labor, neuraxial (epidural) analgesia has been shown to significantly decrease parturients’ pain scores when compared with IV meperidine9 or continuous midwifery support with supplemental intramuscular meperidine, nitrous oxide inhalation, or other nonpharmacological adjuncts.10,11

Parturients with intracranial lesions are often assumed to have increased intracranial pressure (ICP), and the risk of herniation is frequently cited as a contraindication to neuraxial anesthesia.11–18 To care for these parturients and ascertain which of them can safely undergo spinal or epidural analgesia or anesthesia,19–21 we must understand the factors that contribute to clinically significant brain tissue shifts (i.e., brain herniation), such as increased ICP, brain edema, or hydrocephalus. Anesthesiologists can then most effectively weigh the relative risks and benefits of neuraxial anesthesia for these patients and engage in productive multidisciplinary peripartum planning.

We review the relevant intracranial anatomy and physiology, and integrate cases from the obstetric anesthesia and neurological literature, to clarify the risks of neuraxial...
Neuraxial Anesthesia and Intracranial Lesions in Pregnancy

Intracranial Anatomy and Physiology

**The Three Primary Intracranial Elements**

In order to estimate the association between ICP and brain herniation, it is vital to understand intracranial compliance. Compliance is the relationship between the ICP and the volume of the primary intracranial elements—the brain, cerebrospinal fluid (CSF), and blood—within the rigid structure of the cranium. Figure 1 illustrates the relative volumetric contributions of these three components under normal circumstances. Disorders of each of these intracranial contents and the physical laws that govern compliance are presented here.

**Brain Tissue.** The brain represents approximately 1,400 cc of volume and comprises mostly intra- and extracellular water (fig. 1), which is relatively noncompressible. Brain tissue volume can increase through abnormal growth in benign or malignant tumor cells or via an increase in intra- or extracellular water. Malignant brain tumors, one common form of space-occupying lesion, are often associated with localized edema, necrotic tissue, and hemorrhage, which further increase brain volume. Anesthesiologists are often reluctant to proceed with neuraxial anesthesia in patients with these pathologies for fear of precipitating herniation or neurologic deterioration.12–18

**CSF.** The intracranial volume of CSF is approximately 150 cc (fig. 1) and is contained in the cerebral ventricles, a system of communicating chambers that connect the intracranial cavity with the spinal subarachnoid space and lumbar cistern.22–24 CSF circulates between the two lateral ventricles, the third ventricle, the aqueduct, and the fourth ventricle. It then flows through the foramen magnum at the base of the skull into the spinal subarachnoid space, which terminates in the lumbar cistern. CSF is produced in the choroid plexus within the intracranial portion of the system at a rate of approximately 20 cc/h. Under normal circumstances, this intracranial CSF freely communicates back and forth with the extracranial subarachnoid space. CSF is ultimately reabsorbed in the arachnoid granulations, which then empty into the intracranial venous sinuses, which are noncompressible channels cut into the bone of the skull (fig. 2). The CSF system plays an important role in the exchange of nutrients in and waste removal from the central nervous system and also acts like a compressible bladder and cushion within and around the brain. Ordinarily, approximately 400–500 cc of new CSF is produced daily. Each of the intracranial and spinal compartments of the CSF system typically contains 150cc of CSF, and the remainder is continuously reabsorbed. Normal CSF pressure ranges from 13 to 20 cm H₂O when measured by manometer, or less than 10–15 mmHg by strain gauge or fiberoptic device.

If the flow of CSF is impeded within or between the ventricles, “noncommunicating” or “obstructive” hydrocephalus develops (fig. 3).24 Hydrocephalus (Greek for
“water on the brain”) is a radiological finding of increased ventricular volume. An increased ventricular volume is not always associated with increased pressure. For example, when cerebral atrophy causes dilation of the ventricles ("ventriculomegaly") and "hydrocephalus ex-vacuo", the ICP is usually normal. Hydrocephalus, which is associated with

Fig. 2. Schematic illustration of the cerebrospinal fluid (CSF) circulation in the sagittal plane (A) and coronal plane (B). The direction of CSF flow is indicated by black arrows, and the choroid plexus where CSF is produced is indicated by red.

Fig. 3. Schematic illustration in the coronal plane of a normal brain without hydrocephalus or mass effect (A), and hydrocephalus due to obstruction at the level of the cerebral aqueduct (B). Magnetic resonance imaging shows obstruction at the level of the 3rd ventricle, as seen in the coronal T1-weighted, spoiled gradient echo (C) and axial fluid attenuated inversion recovery images (D) of the same lesion.
increased ICP, may be due to impaired absorption of CSF (communicating hydrocephalus) or obstructed flow of CSF (obstructive hydrocephalus). In general, whereas sudden or major obstruction in intracranial CSF flow will produce symptoms within minutes to hours, the onset of neurological deterioration due to partial obstruction can be significantly delayed by days or weeks. As described below, parturients with noncommunicating hydrocephalus are typically at greater relative risk of acute neurological deterioration than those with communicating hydrocephalus in the setting of an intentional or inadvertent dural puncture.  

**Cerebral Blood Volume.** The intracranial vessels and the blood within them serve several vital functions including facilitating the exchange of oxygen, carbon dioxide, glucose, and amino acids with the brain tissue.\(^{22,25}\) The normal cerebral blood volume, approximately 150 cc (fig. 1), is influenced by a variety of factors, including the degree of vasoconstriction or vasodilation in response to local tissue environments, or the partial pressure of arterial carbon dioxide. In addition to providing a physical barrier to impede the entry of many large molecules or harmful substances, the blood–brain barrier regulates the flow of nutrients, ions, and fluids to the brain. Although it is a complex, multilayered membrane that is designed to support optimal neuronal electrical activity and metabolism, it is not as impermeable as many believe. Even under normal circumstances, the blood–brain barrier permits the flux not only of desirable but also undesirable molecules, such as alcohol and other illicit substances. In the presence of malignant tumors or conditions such as eclampsia, disruption of the blood–brain barrier frequently leads to vasogenic edema, which increases the extracellular water content, and subsequently the tissue volume, within the skull.\(^ {26–29}\) Abnormalities in intracranial blood vessels can also impact intracranial volume in a variety of ways. Defects in the arterial or venous walls can produce dysplastic segments or aneurysms, arteriovenous malformations, or fistulas which can bleed, dissect, or occlude. Small bridging dural veins, and tiny collateral arteries in obstructive vasculopathies, such as Moyamoya, are frail and at high risk of rupture. When vessel rupture occurs, it produces intracranial hemorrhage (e.g., subdural, intracerebral, or subarachnoid hemorrhage). Venous occlusion can also lead to secondary intracerebral hemorrhage. Any intracranial hemorrhage or other sudden increase in intracerebral blood volume has the potential to increase ICP by impeding the free flow of CSF either by exerting mass effect on the ventricular system, or by causing thrombosis within the ventricles. Alternatively, arterial or venous thrombosis can produce cytotoxic or vasogenic edema, which can increase ICP by increasing tissue volume. Any of these circumstances can increase the risk of neurological deterioration related to neuraxial anesthesia.  

**Intracranial Compliance**  
Within the noncompliant bony skull, the sum total of the intracranial volume remains a constant, such that an increase in the volume of any one component causes a compensatory decrease in the volume of another. This “zero-sum game” of the volumes of the brain, CSF, and blood is known as the Mono–Kellie doctrine.\(^ {23,30–32}\) Intracranial compliance (C) is defined as the change in volume (ΔV) for any given change in pressure (ΔP), or \( C = \Delta V/\Delta P \). The intracranial compliance curve is derived from the summation of the compliance of each of the three intracranial elements. The brain volume contributes to the steep section of the curve because it is composed mostly of intracellular and extracellular water, which is inherently noncompressible. The initial slope of the intracranial compliance curve is flattened because, under normal circumstances, an equivalent volume of blood and CSF can be displaced out of the cranium when intracranial tissue volume increases. Once blood and CSF are unable to further translocate out of the skull (either because of obstruction, or depletion), they then contribute to increased pressure, just like any other noncompressible substance within the rigid skull. Because small, slow increases in brain volume often successfully lead to displacement of CSF or blood into their respective extracranial reservoirs, the intracranial compliance curve is initially flat (fig. 4). However, once this adaptive element of compliance in the intracranial system is exhausted, further small increases in intracranial volume will cause a substantial and steep increase in ICP (fig. 4).  

**The Impact of Baseline Physiologic Perturbations and the Changes during Pregnancy and Labor.** In normal subjects, magnetic resonance imaging of the brain shows that CSF shifts from the intracranial to the spinal subarachnoid space during cardiac diastole. This phenomenon is thought to be a physiologic response to the corresponding increased brain-blood volume that occurs with each systolic contraction (an example of homeostasis based on the Mono–Kellie doctrine). During diastole, CSF returns to the intracranial compartment. Hence, physiologic alterations in cerebral blood volume results in a transient, well-tolerated increase in ICP followed by rapid reequilibration, which does not compromise neurologic function.\(^ {31,33–35}\) The respiratory cycle has also been found to produce smaller, well-tolerated oscillations of CSF pressure.\(^ {36,37}\)  

There have been several investigations of the effect of pregnancy, labor, or prolonged valsalva on CSF volume and ICP. Hogan et al.\(^ {38}\) demonstrated that obesity and external abdominal compression, simulating the impact of the gravid uterus, result in decreased lumbar CSF volume. They postulated that the mechanism of lumbar CSF displacement was compression of the spinal subarachnoid space via the surrounding soft tissue at the sites of intervertebral foramens in the abdomen and thorax. In the 1960s, Marx et al.\(^ {39}\) measured lumbar CSF pressure in 20 normal pregnant women, at rest and during labor, and found increased CSF pressure with contractions only when the parturient was panting, bearing down, or otherwise changing her respiratory muscle function. Subsequently, using more precise information
from continuous intrauterine pressure catheters, Hopkins et al.\textsuperscript{37} were able to show that CSF pressure did increase with contractions in a predictable fashion, with a mean rise of 2.5 mmHg. This rise corresponded to an increase in central venous pressure, which persisted even during sleep or total sensory blockade. These findings illustrate that ICP does not exist in a vacuum, but is influenced by the pressures of the adjacent compartments of the thorax and abdomen and the fluids within them. The pressures in these other compartments are most influential when subjects are on the steep portion of the intracranial compliance curve.

**The Impact of Lumbar Epidural Injection.** Injection of medication into the lumbar epidural space compresses the dural sac, alters the compliance of the spinal subarachnoid space, and displaces CSF upward toward the cranium.\textsuperscript{40,41} Pregnancy and labor have been found to increase baseline lumbar epidural pressure, an effect that is more pronounced than the effect of pregnancy on lumbar CSF pressure.\textsuperscript{42,43} This increase in epidural pressure occurs gradually during pregnancy and is thought to be due to the space-occupying effect of the increased blood volume in distended epidural veins. In the nonpregnant population, studies in animals\textsuperscript{40} and humans\textsuperscript{41} have confirmed that subjects with baseline-elevated ICP have more pronounced transient increases in ICP after epidural injection, than subjects with normal preinjection ICP. Specifically, in a patient with preinjection-elevated ICP, a 10-cc bolus of local anesthetic given over 20–30 s caused an average increase of 21 mmHg in ICP (from 18.8 to 39.5 mmHg) lasting for 4.5 min compared with an average increase of 6 mmHg (from 9.3 to 15.6 mmHg) for 2.3 min in a patient with preinjection-normal ICP. Decreasing the volume of injection to 5 cc in the patient with preinjection-elevated ICP dramatically reduced the change in ICP to 5 mmHg for a duration of 2.8 min. In a porcine model with elevated ICP, epidural injection was shown to correlate with more than 90% transient reduction in cerebral blood flow.

**Intracranial Pathology and the Risks of Neuraxial Anesthesia and Analgesia**

Under normal conditions, the total intracranial volume (\textit{i.e.}, brain, CSF, and cerebral blood) is low enough so that, despite the routine physiologic perturbations associated with the cardiac and respiratory cycles, as well as those in pregnancy and vaginal delivery, the ICP fluctuates within the normal range and no neurologic consequences occur. However, pathologic changes in brain tissue, CSF, or cerebral blood volume can disrupt this balance and potentially result in substantially increased ICP, brain tissue shifts, or rupture of intracranial vascular lesions. In this section, we will apply the principles of intracranial compliance to case examples to help determine when harm could result from the reduction in lumbar CSF volume, which can occur with an intentional spinal anesthetic, accidental dural puncture during epidural catheter placement, or epidural catheter dosing. We will
specifically address several generalizations frequently cited in the literature and in clinical practice and show under what circumstances they do and do not apply.

**The Impact of a Space-occupying Lesion**

The diagnosis of a brain mass during pregnancy can be devastating. Fortunately, the incidence of malignant brain tumors during pregnancy is very low, although not well quantified. Pregnancy is not thought to change the basic rate of developing a brain tumor. Of the intracranial tumors discovered during pregnancy due to detection of neurologic symptoms, gliomas represent the majority, followed by meningioma, and acoustic neuroma. Tumors such as meningiomas and pituitary adenomas can be hormone responsive and therefore, may enlarge during pregnancy.

Critical to assessing the potential for neurologic deterioration from neuraxial anesthesia in the setting of a brain tumor or other space-occupying lesion is an analysis of its effect on intracranial compliance. Although many factors can be considered, the most relevant lesion characteristics are its location and size, the rapidity with which the overall brain tissue and associated volume has increased, and the presence of imaging evidence of preexisting fluid or tissue shifts or obstruction to CSF pathways.

To safely perform any dural puncture, there should be preservation of continuous flow of CSF and the absence of a substantial pressure differential between the intracranial and intraspinal compartments. If a pressure differential exists, then loss of sufficient CSF volume via the dural puncture could force brain tissue to shift from one compartment to another. In addition, because there will undoubtedly be some loss of lumbar CSF after a (lumbar) dural puncture, there should be ample remaining intracranial CSF so that CSF will shift to equalize the pressure rather than brain tissue. The ultimate decision will be the result of balancing many factors simultaneously, as there are often competing risks.

The following generalizations about parturients with space-occupying intracranial lesions are not supported by evidence:

- A space-occupying lesion is always associated with increased ICP
- Normal ICP necessarily implies low risk of herniation after dural puncture
- Increased ICP always implies high herniation risk after dural puncture
- An epidural anesthetic is a safe alternative when a dural puncture, even with a small-gauge spinal needle, is considered to be too dangerous
- A spinal anesthetic with a small-gauge needle is a safe alternative when an inadvertent dural puncture during epidural placement is considered to be too dangerous
- When all forms of neuraxial anesthesia pose some risk of herniation, general anesthesia is always preferable
- A parturient with an intracranial arterial or venous pathology should not undergo neuraxial anesthesia due to increased risk of vessel rupture

1. Is a space-occupying lesion always associated with an increased ICP? If a primary brain tumor or brain metastasis is located in a region which is remote from CSF pathways, and is of small-to-moderate size or grows slowly over time, it may cause little-to-no ventricular compression, and have no impact on CSF flow. A common example would be a small, slow-growing, and low-grade glioma in the anterior frontal lobe. This tumor may cause minimal mechanical distortion of brain tissue or mass effect. As described above, there may then be little-to-no increase in ICP because the increase in brain volume is offset by caudal displacement of CSF or cerebral blood volume (fig. 5). A sudden loss of lumbar CSF volume at the time of dural puncture will cause a transient pressure gradient across the foramen magnum. In this scenario, CSF rather than brain tissue will be displaced from the intracranial to the lumbar CSF compartment. In other words, there should be no herniation of brain tissue. Similarly, one would expect that in this patient with normal preprocedure ICP, the transient increase in ICP associated with epidural injection would also be well tolerated.

There are several illustrative case examples reported in the literature in which parturients with benign or malignant brain tumors have had successful epidural anesthetics (or unspecified “neuraxial anesthetics”) for cesarean or low vasa la va vaginal deliveries. Typically, these patients lacked clinical symptoms or signs of increased ICP (e.g., headache, nausea, vomiting, decreased alertness, recent seizure, hemiparesis, or pupillary abnormalities) or imaging evidence suggestive of increased ICP (table 1).

In contrast, if the intracranial lesion partially or completely obstructs the free flow of CSF, then the risk of brain herniation after either intentional, or unintentional dural puncture will be increased. This is particularly likely to happen if the lesion is located at an anatomically narrowed segment of the ventricular system (e.g., near the third ventricle or cerebral aqueduct) or at the foramen magnum. As the lesion grows, it displaces intracranial CSF caudally, or in the case of an obstruction to CSF outflow, it causes increased ventricular volume and hydrocephalus. Space-occupying lesions that narrow the ordinarily large opening of the foramen magnum, or are situated in the posterior fossa and cause a bottleneck to CSF flow, can place the parturient at significant risk of herniation. This can occur from benign or malignant tumors anywhere in the posterior fossa or at the opening to the foramen magnum. It can also occur from low-lying cerebellar tonsils, either due to a preexisting Arnold–Chiari malformation, or due to intracranial hypotension from a persistent CSF leak. If the intracranial CSF has been exhausted and the lumbar CSF pressure suddenly drops from a dural puncture, then brain tissue itself will be displaced into the neighboring intracranial
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compartment (i.e., transtentorial or uncal herniation) or into the spinal canal (tonsillar herniation). In their review on brain herniation from lumbar puncture, the neurologist van Crevel et al. described “brain shift” imaging findings “anatomically (by) tense dura, flattened gyri, narrowed sulci, effaced cisterns, compressed (or, in obstruction, dilated) ventricles, and in advanced stages, herniation, i.e., displacement of brain from one intracranial compartment into another.” Evidence regarding the safety of diagnostic lumbar puncture in patients suspected of having acute bacterial meningitis also provides important lessons for the management of parturients with intracranial pathology and increased ICP. Lumbar puncture in this setting is usually performed with a 20-gauge Quinke spinal needle because smaller gauge needles do not allow for adequate drainage of viscous, infected spinal fluid. The abrupt decrease in lumbar CSF pressure associated with a lumbar puncture is often implicated as contributing to the neurological deterioration of some patients who herniate immediately after the procedure, as well as others who manifest symptoms 12–24 h later. Preexisting clinical signs of impending or preexisting fatal herniation, specifically deteriorating level of consciousness, brainstem signs (e.g., pupillary changes or asymmetry, eye movement abnormalities, dysconjugate gaze, facial weakness, swallowing dysfunction, irregular respirations, or limb posturing), recent new onset seizure, or the presence of papilledema and/or hemiparesis are thought to be the best indicators of individuals at high risk. If signs of herniation develop, then prompt attention to airway, breathing, cerebral, and systemic circulation are of paramount importance. The recommended maneuvers to rapidly lower ICP include hyperosmolar therapy (e.g., rapid IV infusion of mannitol 100 g or 23.4% saline 15–30 cc), intubation, and hyperventilation (with a goal of PaCO₂ of 25–30 mmHg) while emergency neurosurgical consultation is obtained. If a spinal needle is in place, the stylet should be reinserted and kept in situ until the ICP normalizes to avoid ongoing large volume CSF leak.

Fig. 5. Schematic illustration in the coronal plane of a normal brain with normal ventricular volume (A), and in the axial plane of a brain with a tumor surrounded by edema in the left occipital pole that exerts no visualized mass effect on the ventricles and no midline shift (B). The lesion is demonstrated on fluid attenuated inversion recovery coronal (C) and axial (D) magnetic resonance images.
Table 1. Features Associated with Increased ICP

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<th>Clinical features</th>
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<td>Pupillary changes or asymmetry</td>
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<td>Papilledema</td>
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<td>Hemiparesis</td>
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<td>Facial weakness</td>
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<td>New onset seizure</td>
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<td>Decreased level of consciousness</td>
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<td>Radiologic features on CT or MRI</td>
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<td>Tense dura</td>
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<td>Flattened gyri</td>
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<td>Narrowed sulci</td>
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<tr>
<td>Effaced cisterns</td>
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<tr>
<td>Compressed (or in obstruction, dilated) ventricles</td>
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<tr>
<td>Lateral shift of midline structures</td>
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<td>If advanced, displacement of brain tissue from one compartment to another</td>
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CT = computed tomography; ICP = intracranial pressure; MRI = magnetic resonance imaging.

2. Does normal ICP always imply low risk of herniation after dural puncture? If expansion of an intracranial mass happens very slowly, over weeks or months, it may cause little-to-no symptoms associated with elevated ICP and these pressures might be at the upper end of normal. What is critical is that these patients have exhausted all reserves for tolerating any pressure differential across the foramen magnum so that any small change in intracranial volume will cause a shift of brain tissue rather than of blood volume or CSF. A large right hemisphere tumor with significant surrounding edema causing major mass effect and shift of brain tissue (subfalcian herniation) is shown in figure 6. If a dural puncture were attempted, this patient would surely be at increased risk for herniation.

Parturients with Arnold–Chiari malformations pose an interesting challenge for assessment of the safety of neuraxial anesthesia. Because, by definition, the cerebellar tonsils are descended at least 5 mm through the foramen magnum, there can be partial or intermittent obstruction of CSF and dynamic or static herniation of brain tissue.33,35 Parturients with the most common form, type I, may be asymptomatic or may manifest symptoms, including headache, ataxia and/or sensorimotor impairments of the extremities.33,54 Patients with type II Chiari malformations usually present in infancy, with associated myelomeningoceles. In type II or “classic” Chiari malformation, both cerebellar and brain stem tissue extend into the foramen magnum, resulting in episodic apnea, cranial nerve dysfunction, and upper extremity weakness. In type III Chiari malformations, there is further protrusion of the cerebellum and the brain stem into the foramen magnum and the spinal cord, resulting in severe neurologic dysfunction. Type IV disease involves cerebellar hypoplasia. Successful surgical correction of the Arnold–Chiari malformation with extrarachnoidal craniocervical decompression and duraplasty removes the obstruction and ameliorates these abnormal phenomena. However, some of the associated anomalies, such as a tethered spinal cord, may persist and may represent important contraindications to neuraxial anesthesia.

Multiple individual reports and a case series of 30 delivers document successful spinal and epidural analgesics and anesthetics in a heterogeneous group of patients with Chiari malformations. Among them are patients in whom the condition was or was not previously diagnosed, and among those previously diagnosed, there were some who had and others who had not gone in for surgical correction.53,55 One of the parturients with an uncorrected Arnold–Chiari malformation in the published case series underwent a combined spinal–epidural and developed a spinal headache. Fortunately, she was successfully treated with an epidural blood patch. She was the only parturient to have an anesthetic-related complication in that series. In a separate report, a new diagnosis of Arnold–Chiari malformation was made in a parturient who developed a persistent headache after a failed epidural anesthetic, followed by a spinal anesthetic that required multiple attempts.56 When the headache was not responsive to an epidural blood patch, a subsequent brain magnetic resonance imaging revealed the Arnold–Chiari malformation. There are an increasing number of published reports of patients with persistent headache after dural puncture, wherein subsequent brain magnetic resonance imaging shows cerebellar descent consistent with an acquired Arnold–Chiari malformation.57 These cases confirm that large-gauge dural punctures or persistent CSF leaks can lead to cerebellar descent, even in patients without preexisting Chiari malformations. As such, it may be reasonable to proceed with a spinal or epidural analgesic or anesthetic in a parturient with an asymptomatic, type I Chiari malformation with minimal initial tonsillar descent. However, if she has an inadvertent large-gauge needle dural puncture and/or develops a persistent headache or other neurologic symptoms, a neurologic consultation and consideration of early epidural blood patch is recommended.

3. Does increased ICP always mean a high rate of herniation after dural puncture? Idiopathic or benign intracranial hypertension (also known as pseudotumor cerebri) is a common condition, in which increased ICP does not imply herniation risk after dural puncture. This disorder, usually occurring in obese women of childbearing age, is defined by increased ICP (>20 cm H2O) with normal CSF composition and the absence of a known underlying cause (e.g., a space-occupying or vascular lesion, mass effect, or hydrocephalus).26,58–61 Extraventricular CSF volume and ICP are thought to be elevated in these women, who often experience symptoms of headache, neck stiffness, papilledema, and visual loss. However, there is no obstruction to CSF flow and no baseline pressure differential between the intracranial and spinal CSF compartments. Therefore, a sudden drop in CSF volume during lumbar dural puncture will be rapidly accommodated by caudal flow of CSF and should not result in brain shift or herniation. In fact, serial
lumbar punctures for deliberate removal of large volumes of CSF, coupled with weight control, diuretics, and steroids are mainstays of therapy for the disease. Although there are individual case reports of both increased and decreased spinal anesthetic effects in these patients after deliberate spinal fluid removal, neuraxial anesthesia has been used effectively for parturients with benign intracranial hypertension with or without shunts. Slow, incremental dosing of epidural medication may be better tolerated in symptomatic patients who might otherwise experience exacerbation of their symptoms due to predelivery increase in ICP. Dosages in the range of 5 cc every 5–7 min have been recommended.  

4. Is an epidural anesthetic a safe alternative when a dural puncture, even with a small-gauge spinal needle, is considered to be too dangerous? Alternatively, is a spinal anesthetic with a small-gauge needle a safe alternative when an inadvertent dural puncture during epidural placement is considered to be too dangerous? Because epidural placement always entails the risk of a dural puncture even in the experienced hands, it is never a completely safe alternative if a spinal anesthetic is contraindicated. This is especially true because when inadvertent dural puncture does occur during epidural placement, it is with a much larger gauge needle. Conversely, even a small-gauge spinal needle causes a dural tear and potential CSF leak, so spinal anesthesia is not without risk when seeking to avoid significant loss of CSF.

In vitro experimental models have indicated that dural punctures with smaller gauge needles are correlated with a shorter duration of CSF leak: fluid loss ceased within 5 min in 10% of dural punctures made with a 22-gauge needle and in 65% of those made with a 29-gauge needle. Pencil point versus Quincke needle design also minimized fluid loss. Results of studies to determine the impact of the angle of needle alignment relative to the dural fibers were inconsistent. When the dural leaks from six 17- to 20-gauge epidural needles were compared in cadaveric tissue, the 20-gauge Tuohy needle produced the slowest rate of fluid loss. Noninvasive, in vivo measurements of CSF leak after lumbar puncture have shown considerable variation in the amount of CSF leak, even when the same needle is used, and that predominantly...
cortical, sulcal CSF is depleted.\textsuperscript{75} In summary, there is ample evidence that the leakage of CSF can persist after dural punctures with both spinal and epidural needles and that to date, nothing including postprocedure patient positioning, prophylactic blood patch, or IV medications can reliably prevent the intracranial hypotension headache syndrome.\textsuperscript{76–78} However, there are two salient cases that illustrate the perils of neuraxial anesthesia when dural puncture is considered risky. Su \textit{et al}.\textsuperscript{81} described a parturient with fatal brain herniation after accidental dural puncture associated with epidural analgesia for labor and delivery. Over the course of her labor, she developed a severe headache and systemic hypertension and ultimately, postdelivery, deep coma. Urgent brain computed tomography revealed a slightly hyperdense tumor in the left hemisphere, midline shift to the right and transtentorial herniation. The most likely explanation for this patient’s neurologic deterioration is that the increased intracranial volume, associated with her unrecognized tumor, had exhausted her compensatory mechanisms, and put her on the steep portion of the intracranial compliance curve before her epidural procedure. The increased intracranial volume during labor contractions and valsalva during delivery likely pushed her ICP to dangerous levels. This increased ICP likely resulted in the translocation of most of the intracranial CSF into the lumbar compartment with associated collapse of the ventricles. The inadvertent dural puncture added ongoing loss of lumbar CSF, which created a further pressure gradient across the tentorium. When there was no remaining intracranial CSF to shift caudally, brain tissue then translocated, leading to transtentorial herniation.

Another parturient with a known, extensive, frontoparietal tumor was induced for vaginal delivery under epidural analgesia because of signs of increased ICP.\textsuperscript{92} Despite her expeditious, nonvalsalva delivery and lack of known dural puncture, she suffered increasingly intense intrapartum headaches. Urgent postpartum computed tomography revealed substantially increased tumor volume and contralateral obstructive hydrocephalus. This was due to a trapped lateral ventricle that could not empty because its drainage, into the rest of the ventricular system via the foramen of Monro, was blocked. Although her tumor was urgently decompressed, she developed progressive mass effect from edema, and ultimately fatal brain herniation. These cases highlight that epidural placement can be just as risky as a spinal anesthetic in patients with poor intracranial compliance. In some cases, the use of a small-gauge spinal needle for deliberate dural puncture may be the lesser of two evils, but all options should be explored to assess that this is the technique with the most favorable risk/benefit ratio.

5. \textit{When neuraxial analgesia or anesthetics poses a risk of herniation, is general anesthesia always preferable?}\n
General anesthesia can pose significant risks for neurological deterioration in parturients with compromised intracranial compliance.\textsuperscript{12–14,18} Care should be taken to minimize valsalva during the rapid sequence induction and emergence of general anesthesia, although blunting the airway reflexes in these patients may further increase their pregnancy-related risk of aspiration. Strategies to blunt the sympathetic response to intubation include combining induction agents with opioids (\textit{e.g.}, fentanyl 2–5 \mu g/kg or remifentanil 1 \mu g/kg over 1 min) or labetolol in 5-mg increments.\textsuperscript{83–85} Some experts advocate replacing succinylcholine, which can theoretically cause a transient increase in ICP due to contraction of the muscles of the abdomen and thorax, with a nondepolarizing muscle relaxant (\textit{e.g.}, rocuronium 1.2 mg/kg) for a rapid sequence induction.\textsuperscript{13} Paradoxically, while hyperventilation is beneficial to the mother by decreasing ICP and maximizing cerebral blood flow in patients with increased ICP, it can be detrimental to the fetus by decreasing placental blood flow. To balance fetal and maternal cerebral perfusion, the recommendation is to keep maternal \textit{PaCO}_2 at approximately 25–30 mmHg and maternal blood pressure close to baseline values.

Whereas the risk of herniation in a parturient may limit the desirability of a neuraxial approach, Semple \textit{et al}.\textsuperscript{86} makes a compelling case for doing an epidural anesthetic for cesarean delivery, if there are equally or more compelling risks associated with a general anesthetic. They discuss the management of one such parturient with a Chiari malformation who had no papilledema or other symptoms associated with raised ICP, but whose airway was characterized as a Mallampati class III, with a high arched palate, receding chin, and protruding incisors. The authors initially considered a general anesthetic (awake fiberoptic or elective tracheostomy) but in collaboration with the patient, decided to proceed with an epidural anesthetic instead because of her unfavorable airway. They slowly titrated 23 cc of a combination of 2\% lidocaine and 0.5\% bupivacaine with 50 mics of fentanyl over 30 min, and she delivered without acute or subacute neurologic complication. Another case report describes a parturient with a glioblastoma with mass effect and a “fourth ventricle narrowed but not obstructed” who was successfully managed for cesarean delivery, with a spinal anesthetic using a 24-gauge pencil-point needle.\textsuperscript{79} The authors noted that this parturient was at particularly high risk for aspiration due to cranial nerve dysfunction, and at low risk for herniation because “anatraumatic small-bore, pencil-point needle, even under slightly raised ICP, presumably causes only minimal CSF leakage, if at all”. On the basis of the evidence and rationale discussed above, the authors may have been overly optimistic in their confidence of the safety of a dural puncture in this patient. An alternative, and perhaps, more prudent approach for cesarean delivery could have been a controlled general anesthetic, thus securing her tenuous airway, and avoiding the small but very real risk of a persistent CSF leak from the spinal anesthetic.

In summary, there are no published randomized controlled trials comparing the safety of neuraxial versus general anesthesia.
anesthesia in patients with intracranial lesions, nor are there likely to be any. As with all published case reports, there is an inherent bias in the cases that are chosen for reporting. Therefore, for each parturient with an intracranial lesion, there needs to be a collaborative team discussion, which includes anesthesia, obstetric, neurologic, and neonatology experts, and a rational exploration of the likelihood of increased ICP and the potential for related negative effects. To make recommendations on the proper anesthetic choice for any individual case, it is necessary to evaluate the relative contribution of each of the identified risks, in both severity and likelihood, and weigh them against the potential benefits.

6. Is it unsafe for a parturient with intracranial arterial or venous pathology to undergo neuraxial analgesia or anesthesia due to increased risk of vessel rupture or mass effect? Some authors advocate avoiding neuraxial anesthesia in parturients with intracranial hemorrhage or derangements of the intracranial vasculature out of concern for precipitating neurological complications.87 Because there are many attendant risks to general anesthesia in this population and wide variability in blood vessel fragility across patients, knowledge of the precise details of the vascular abnormality and its risk of rupture is key to making informed decisions about anesthetic management. Neurological or neurosurgical or endovascular consultation is generally required to undertake appropriate risk/benefit analysis. Although urgent or elective cesarean delivery has been the predominant mode of delivery recommended for those with serious vascular lesions prone to hemorrhage, the degree to which pregnancy and the mode of delivery are actually risk factors for hemorrhage in these cases is controversial.83,84,87-91

Historically, the reason that pregnant patients with asymptomatic unruptured brain aneurysms have been counseled to avoid expulsive efforts is the concern for the following chain of events: the associated increase in cerebral blood and CSF volume during prolonged valsalva can elevate ICP; this increase in ICP can cause a compensatory increase in arterial blood pressure to maintain constant cerebral perfusion, which may in turn increase the transmural pressure across the already weakened aneurysmal vessel wall and precipitate aneurysmal rupture. In addition, intracranial hypotension due to a persistent CSF leak, after a spinal anesthetic or inadvertent dural puncture during epidural placement, could theoretically cause a compensatory increase in cerebral blood volume. This could trigger similar increases in transmural pressure across the vulnerable aneurysmal wall. To our knowledge this complication of neuraxial anesthesia has not been reported.

Of note, in a recent retrospective review of women with aneurysmal subarachnoid hemorrhage, Tiel Groenestege et al.90 did find that pregnancy, labor, or the puerperium did not appear to increase the risk of aneurysmal subarachnoid hemorrhage. The authors concluded that there was no need to advise women, with a previous history or family history of aneurysmal subarachnoid hemorrhage, against pregnancy or vaginal delivery. We recently reported that in the National Inpatient Sample, a large U.S. administrative dataset, a significant proportion of subarachnoid hemorrhage in the puerperium is likely nonaneurysmal in etiology.92 Whereas the precise etiologies require further investigation, advancing age, African American race, Hispanic ethnicity, hypertensive disorders, coagulopathy, tobacco, drug or alcohol abuse, intracranial venous thrombosis, sickle cell disease, and hypercoagulability were identified as key risk factors for pregnancy-related subarachnoid hemorrhage. The risks associated with neuraxial anesthesia in the setting of these nonaneurysmal etiologies are not known.

If a pregnant patient develops subarachnoid hemorrhage due to aneurysmal rupture, then there is consensus that the neurosurgical management should be the same as if she were not pregnant.44,87,88 There are multiple reports of craniotomy and embolization occurring simultaneously with or in advance of cesarean delivery.84,85 For these combined procedures, general anesthesia is typically used with volatile agents plus IV anesthetics or a total IV anesthetic technique.83 For cesarean delivery that does not require aneurysm repair, a neuraxial anesthetic (spinal, epidural, or combined spinal–epidural anesthesia) can be used in a stable patient and has the advantage of (1) avoiding the valsalva potentially associated with intubation, (2) minimizing fetal exposure to general anesthetics, and (3) allowing the mother to be awake and participate fully in the birth experience. Hypotension is typically well-controlled with a prophylactic phenylephrine infusion 25-120 µg/min or intermittent 120-µg boluses.93,94

The natural history of arteriovenous malformations during pregnancy is also a matter of some debate, although when arteriovenous malformations do bleed during pregnancy, the results can be devastating.89,95,96 In 1990, Horton et al.90 did a retrospective review of pregnant women with untreated, symptomatic intracerebral arteriovenous malformations and found these patients to be at the same risk of nonfatal hemorrhage as their nonpregnant counterparts. However, because these women were retrospectively identified by referral for proton beam therapy, the ascertainment strategy did not include those parturients with fatal bleeds or those with previous arteriovenous malformation-related surgery. In contrast, in a recently published retrospective review of women with angiographic diagnosis of arteriovenous malformation, Gross and Du97 found an increased risk of hemorrhage from arteriovenous malformations during pregnancy. Acknowledging their small sample size, retrospective design, and the assumption that arteriovenous malformations are congenital, the authors concluded that therapeutic intervention is indicated ideally before pregnancy, particularly if the arteriovenous malformation has previously bled.

Of particular anesthetic concern in parturients with intracranial hemorrhage from ruptured arteriovenous malformations is the possibility of CSF flow obstruction or raised ICP.96 As in patients with other space-occupying lesions, the key to deciding whether it is safe to proceed with a neuraxial anesthetic is to determine the likelihood of creating a
pressure differential between intracranial versus intraspinal compartments, causing brain tissue to translocate downward. An additional priority of anesthetic management of a parturient with recent intracranial hemorrhage is to provide strict hemodynamic stability. Hypertension can occur after aggressive treatment of neuraxial-induced hypotension or during induction or emergence from general anesthesia, and increase the risk of fatal hemorrhage in lesions predisposed to bleeding. Hypotension, however, can reduce maternal brain and fetal-placental perfusion.

In the case of an unruptured arteriovenous malformation found during pregnancy, experts recommend case-specific multidisciplinary discussions about the relative risks of neurosurgical intervention versus cesarean delivery before neurosurgical intervention.53 Spinal and epidural analgesics and anesthetics have also been successfully used for cesarean and vaginal deliveries in parturients with intact, partially or fully resected arteriovenous malformations.97–99 In these cases, the anesthetic options have been guided by the usual principles.

Moyamoya syndrome, an oblitative vasculopathy affecting the distal internal carotid and proximal middle and anterior cerebral arteries, invokes the same principles of anesthetic management as those of the other cerebrovascular pathologies discussed.100–107 In Moyamoya, multiple tiny collateral arterioles develop at sites of proximal arterial occlusion. These vessels are prone to rupture, but lowering blood pressure can increase the risk of ischemic brain infarction by decreasing collateral flow. The anesthetic goals for parturients with known Moyamoya syndrome are to avoid hypertension, which can precipitate the hemorrhage and avoid hypotension or hypocapnea that can reduce placental perfusion and the already compromised cerebral blood flow. Most affected parturients undergo successful cesarean deliveries under neuraxial anesthesia (spinal, epidural, or combined spinal–epidural anesthesia), often with invasive arterial hemodynamic monitoring.

Peripartum cerebral venous thrombosis is a rare but likely underdiagnosed intracranial pathology that occurs most often in the postpartum period but can present before delivery.108,109 Cerebral venous sinus thrombosis is typically associated with severe headache, sometimes worse when lying down, and can be accompanied by visual changes, nausea, vomiting, seizures, or lateralizing neurologic signs. Diagnosis of cerebral venous sinus thrombosis is usually confirmed by computed tomography or magnetic resonance venography.110 Anticoagulation therapy is strongly recommended by clinical experts, even in many patients with existing hemorrhagic infarction.111,112 Case reports describe the use of both catheter-based and systemic thrombolysis in selected patients with severe neurologic symptoms due to cerebral venous sinus thrombosis, but there are no relevant published randomized controlled studies. Although some brain swelling has been reported to occur in up to 50% of nonpregnant patients with this entity, osmotic therapy, with or without decompressive surgery, is thought to be necessary in only 20% of patients. Large volume lumbar puncture has been recommended in selected patients to reduce the raised ICP, which is caused by the increased blood volume associated from impaired venous drainage. Balancing the timing of lumbar puncture with the need for continuous anticoagulation therapy remains challenging.

Successful spinal anesthesia for cesarean delivery has been reported in at least one parturient with extensive cerebral venous sinus thrombosis that was being treated with IV unfractionated heparin therapy.113 Despite her mild hypertension and intermittent bradycardia suggesting some degree of increased ICP, the authors opted to perform a spinal anesthetic with a small-gauge, pencil-point needle. These practitioners were depending on the presence of an ample volume of unobstructed CSF that could flow caudally so that downward herniation of the brain would not occur. In their estimate, this was less risky than trying to blunt the increased ICP related to induction and emergence of general anesthesia. In other published reports, the symptoms of cerebral venous sinus thrombosis have been mistaken for postdural puncture headache or have occurred concomitantly with postdural puncture headache.114,115 Interestingly, dural puncture, itself, has been proposed as a risk factor for subsequent cerebral venous sinus thrombosis.

When the integrity of the blood–brain barrier is disrupted, vasogenic edema may occur. This increased intracranial tissue water content can cause increased ICP and has been documented in some parturients with eclampsia.28,29,116,117 Because pregnant patients are not routinely monitored for ICP, the overall incidence of asymptomatic or mildly symptomatic increased ICP in patients with eclampsia is not known. Using optic nerve sheath diameter as a noninvasive proxy for ICP, a recent pilot study confirmed higher ICP in preeclamptic patients compared with healthy pregnant women. The authors were not, however, able to demonstrate a relationship between the magnitude of optic nerve sheath diameter enlargement and the severity of preeclampsia.116 As discussed by Rollins and Flood in their accompanying editorial, it is important to consider whether the increased ICP identified in this study is of any clinical significance. Most preeclamptic patients undergo spinal or epidural analgesics and anesthetics without neurologic symptoms or other untoward events. The current American Society of Anesthesiologists guideline encourages the “early insertion of a spinal or epidural catheter for high-risk parturients such as those with preeclampsia.”2 The perceived advantage of doing so includes decreasing circulating catecholamines,118 and providing an in situ catheter that can be dosed to provide surgical anesthesia as an alternative to general anesthesia in these parturients who are at increased risk for cesarean delivery.

**Individual Patient Assessment**

As with all high-risk parturients, ensuring antepartum anesthesia consultation and multidisciplinary planning is
of paramount importance for pregnant patients with intracranial pathology. Decisions regarding the mode of delivery are, by their nature, linked to the anesthetic plan. As such, timely assessment of whether the parturient is a candidate for a neuraxial anesthetic is necessary. Using the basic anatomic, physiologic, and radiologic principles described, anesthesiologists can work collaboratively with their neurologic colleagues to evaluate a parturient’s appropriateness for neuraxial anesthesia. The decision tree (fig. 7) that accompanies this chapter synthesizes the critical elements to be considered, when assessing the risks of neuraxial anesthesia in parturients with space-occupying lesions, and helps to provide the framework for informed consultative questions. This requires the availability of a brain scan (usually magnetic resonance imaging) that accurately reflects the current pathology and an individual with neurologic or neuroradiologic expertise to interpret it. Unless the parturient has neurologic symptoms, or a tumor that is likely to be hormone responsive as described above, then repeat imaging is often not necessary. Ultimately, decisions about which kind of imaging is most appropriate and whether or not repeat imaging is necessary for optimal predelivery planning is best made by a provider with neurologic expertise. In light of the lack of evidence of fetal harm due to performance of maternal brain magnetic resonance imaging, this diagnostic test should not be withheld during pregnancy if needed.119

To summarize, in the absence of unrelated contraindications to neuraxial anesthesia, parturients with space-occupying lesions that have no mass effect, hydrocephalus, or clinical or imaging findings suggestive of increased ICP are likely to have minimal to no increased risk of herniation from a dural puncture. Parturients at high risk of herniation from a dural puncture are those with lesions that compress normal brain tissue and cause it to shift across the midline (i.e., “midline shift”) or downward, with or without obstruction to the flow of CSF. Understanding a parturient’s risk of neurological deterioration not only allows appropriate low-risk candidates to reap the potential benefits of neuraxial anesthesia, but also allows proper preparation for a ICP-controlled general anesthetic for high-risk parturients when needed.

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Fig. 7. Decision tree summarizing the critical elements for assessing the risks of neurological deterioration from neuraxial anesthesia in patients with intracranial space-occupying lesions. CSF = cerebrospinal fluid.
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