Review Article

Procedural Mechanics and Pitfalls of Neuraxial Anesthesia in Obstetrics: A Physics-based Approach

Abstract

Neuraxial anesthesia is an effective method of pain control for various procedures in obstetrics and gynecology, pain medicine, and orthopedic surgery. Spinals and epidurals can greatly enhance a patient's quality of life but there often exists a limited understanding of the mechanics of performing such procedures, thus hindering the troubleshooting of non-functional or partially functional epidurals. Understanding how mechanical factors like patient anatomy, clinical history, anesthetic injection rate, pressure gradients between the epidural and intrathecal spaces, and medication infusion are crucial for preventing complications like false analgesic levels, failed epidurals, or total/high spinal. In addition to reviewing knowledge available in anesthesia textbooks, the present article introduces a simplified model for understanding neuraxial anesthesia and postulates a mechanism of action for epidurals. Additionally, this article aims to identify high-risk situations that could lead to a total spinal, while considering the unique anatomical changes experienced during pregnancy.

Keywords: *Anesthesiology, epidural, gynecology, mechanism of action, neuraxial anesthesia, obstetrics, physics, spinal, total spinal*

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Introduction

Neuraxial anesthesia, including spinals and epidurals, is routinely used to provide pain control and anesthetic coverage for various surgical procedures and labor delivery.[1] In 2018, it was reported that approximately 71% of women across the United States received an epidural or spinal anesthesia during childbirth, a 10% increase from 2008.[2] Understanding the nuances of neuraxial anesthesia is of great importance when considering rare yet critical complications such as total spinal anesthesia (TSA) which has a reported incidence in the obstetric population of 1 in 4336 neuraxial blocks.[3]

A total spinal occurs when the administered local anesthetic extends more superiorly than anticipated, affecting a more expansive section of the spinal cord, resulting in reduced sensation and muscle control of both the lower and upper body, reducing the motor function of chest and respiratory muscles, and in severe cases, causing respiratory failure necessitating intubation. A total spinal can stem from various scenarios such as inadvertent medication injection into the subdural or subarachnoid space, migration of an epidural catheter into the intrathecal region, or excessive medication dosage within the epidural space.

There are many nuances involved in successfully tailoring neuraxial anesthetics to specific patient populations and clinical scenarios illustrating the critical impact such subtleties have on anesthetic effectiveness and patient well-being. A better understanding of the mechanics involved in neuraxial anesthesia injections can offer clinicians additional insight on how to minimize risks involved in these procedures, troubleshoot suboptimal epidurals/spinal, and minimize the risk of total spinal.

Though there is a scarcity of literature, this manuscript will provide a discussion of how the mechanics of epidural and spinal injections can lead to failure for either procedure and/or life-threatening complications such as a total spinal. In this paper, a systematic analysis of the theoretical causes for these failures, examine pregnancy-related anatomical/physiological changes affecting outcomes, and assess previous surgical factors

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contributing to failure of neuraxial anesthetics. Additionally, recommendations for managing patients with unsuccessful spinal or epidural injections will be discussed.

Anatomy of the Neuraxial Space(s)

The proper application of neuraxial techniques such as epidural, spinal, and combined spinal epidurals (CSE) hinges on understanding the relationship between the thecal sac and epidural space. The spinal canal houses two distinct anatomical compartments: the intrathecal space and the epidural space [Figure 1].

The epidural space is defined as the area separating the dura mater of the spinal cord and the spinous ligaments (including the ligamentum flavum, interspinous ligament, and supraspinous ligament) and extends from the foramen magnum to the sacral hiatus [Figure 1]. Anatomically, the epidural space is located just outside the dura mater. The epidural space contains connective tissue, blood vessels, and fat. The epidural space is typically used for administering epidural anesthesia, or when performing an epidural blood patch procedure.

The intrathecal space is found directly adjacent to the epidural space, is enclosed by the dura mater, and requires penetration through the dura for access. This space envelops the spinal cord and cauda equina within the spinal canal. It is filled with CSF and functions to provide essential buoyancy and protection for the spinal cord. The intrathecal space is commonly used for spinal injections or lumbar puncture procedures.

The distinction between these spaces lies in their relation to the dura mater: the epidural space is positioned externally, while the intrathecal space is internally encased by it. It is imperative to understand the anatomical relationship of these two spaces to prevent unintended complications, such as total spinal anesthesia, which may arise if the medication spreads too extensively within the spinal canal. This becomes especially relevant when performing a CSE or troubleshooting a failed spinal or epidural, as the volume of anesthetic in each of those spaces directly augments the spread of any administered medication.

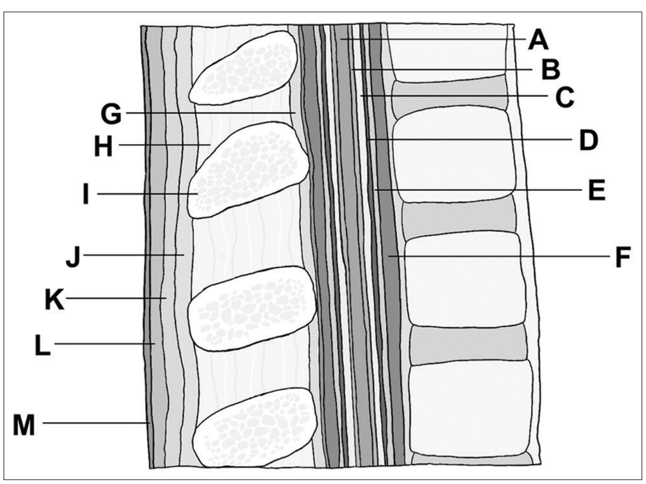

Figure 1: Drawing of Epidural Space and Surrounding Ligaments and Structures. A. ***Spinal cord*****, B.** ***Pia mater*****, C.** ***Subarachnoid space, cerebrospinal fluid*****, D.** ***Arachnoid mater*****, E.** ***Dura mater*****, F.** ***Epidural space*****, G.** ***Ligamentum flavum*****, H.** ***Interspinous ligament*****, I.** ***Spinous process*****, J.** ***Supraspinous ligament*****, K.** ***Muscle layer*****, L.** ***Fat layer*****, M.** ***Skin***

Anatomical studies have shown that the epidural volume varies throughout the cervical, thoracic, lumbar, and sacral regions of the spine. Several factors influence the spread of the epidural block volume of the epidural space, such as patient age, height, positioning, and pregnancy, as outlined below.

While the compartments of the spinal cord (intrathecal space) and the epidural space are well-defined in healthy patients, there are several patient factors that can further alter this region. For example, patients who present with excessive fat tissue either due to pregnancy or a disease state, fibrous membranes due to scar tissue from prior surgeries, neuraxial anesthesia or epidural blood patches, or bony indentations for spine pathologies like spurs or scoliosis may have segmented regions in the epidural space; with each segment having different pressures.[5] [Table 1].

These scar tissues create barriers in the epidural space which may be seen as difficulty threading an epidural catheter or failure of an appropriate anesthetic level to develop. While the CSF will still be continuous through these regions by diffusion, the injection of local anesthetic may create micro pockets bounded by the microsegments that can alter intrathecal compartment compliance in these regions.

Table 1: Factors affecting epidural block level/height[4]

Factor	Effect
Patient age	Decreased epidural space−Decreasing dose requirement with age to achieve adequate block. Increased dura permeability
Patient height	Height determines cephalad spread; shorter patients require less volume per level to achieve block (e.g. 1 mL) vs. taller patients require more (e.g. 2 mL)
Gravity	Patient positioning after an epidural injection; has less effect compared to a spinal. An epidural will settle to patient dependent side i.e., patient positioned in the left lateral decubitus position will have a denser block on the left side.
Volume	The volume of an epidural injection will determine block height. e.g., to achieve a height of T4 block from an L4/L5 injection will require 12 mL (1 mL/segment) to 24 mL (2 mL/segment).
Epidural venous plexus (Batson's plexus)	The epidural venous plexus encircles the epidural space from the base of the skull to the sacrum. Blood volume (hypovolemia, hypervolemia), fluid resuscitation status, and patient position are some of the factors that can cause plexus engorgement leading to a reduction or enlargement in the epidural space volume.

Physics of the Neuraxial Space(s)

When performing spinal anesthesia, local anesthetic, which may be combined with adjuvant medications, is injected into the intrathecal space. In contrast, during epidural anesthesia, an epidural catheter is inserted into the epidural space through which medication is bolused. Typically, when an epidural block is performed, the outflow of the anesthetic drug from the epidural space to the surrounding compartments occurs through diffusion anteriorly through the dura or surrounding connective tissues and egress via the neuroforamina; the destination of outflowing injectate has four pathways[3]: (1) diffusion into surrounding ligaments, (2) distribution into epidural fat, (3) exit into the paraspinal muscle space, or (4) diffusion through the dura mater into the intrathecal space.

To illustrate the concept of an epidural/spinal and how to troubleshoot potential complications, the conceptualization of a simple physical model of the spine consisting of two compartments: the epidural space (compartment 1) and the intrathecal space (compartment 2) is needed [Figure 2].

Generally, the epidural potential space volume is greater than the potential space of the subarachnoid volume at the corresponding level. It is estimated that the epidural space at a specific spinal segment requires about 1.0-2.0 mL of local anesthetic per corresponding vertebral level to achieve an anesthetic block, while the corresponding volume needed to achieve such an anesthetic block is only 0.3 mL in the subarachnoid space.[6] In this two-compartment model, it is important to consider these variables that can change the volume and understand that each compartment is not a fixed volume, but rather there is a diffusion of solution exiting the epidural space through the neuroforamina to surrounding compartments as previously mentioned. However, the rate of diffusion away is relatively slow as compared to the duration of action of the drug. Thus, once injected, the epidural space stays expanded with the injectate.

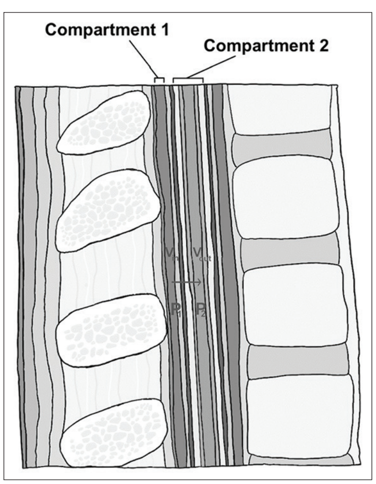

Figure 2: Two Compartment Model for Epidural and Intrathecal Spaces. *P_2 (intrathecal pressure) > P_1 (epidural pressure)*, *Under normal circumstances*

Under normal circumstances, a concentration gradient of the local anesthetic is the primary driver of diffusion into the intrathecal space/surrounding tissues. In a compartmentalized space, a pressure gradient is created either by segmented epidural regions or a large volume of anesthetic drugs in the epidural space. In this two-compartment model, the outflow of drug volume (ΔV_{epi}) in the epidural space shown in equation 1.

Equation 1: $\Delta V_{epi} = V_{in} - V_{out}$.

(ΔV_{epi}) = Change in volume in epidural space; (V_{in}) = Volume into the epidural space;

(V_{out}) = Volume leaving the epidural space

The total change in drug volume (ΔV_{epi}) is the difference between the volume that enters the epidural space (V_{in}) and the volume that leaves the epidural space into the surrounding compartments (V_{out}) [Figure 2].

These values are related to the compliance of the epidural space (C_{epi}) which is being inversely related to pressure changes in the epidural space (ΔP_{epi}), causing leakage of fluid from a compartment of higher pressure to a compartment of lower pressure. The compliance of the spinal canal is defined as the ability of this compartment to hold larger amounts of volume for given changes in pressure.[7] Compliance of the spinal column is thought to provide a protective buffer to patients undergoing neuraxial anesthesia.

As the pressure in the epidural space increases with large volumes of solution or fast rates of injectate injection, there may be an unintended outflow of drugs from the epidural space into the intrathecal space, increasing the anesthetic level in the intrathecal space which may result in a total spinal/high spinal. Depending on the concentration of particles that diffuse across the dura, a total spinal may develop as the particles ascend to the subdural, subarachnoid spaces, and eventually the medulla, thus leading to symptoms like apnea.[8] A notable concern in this scenario is the alteration of CSF distribution in the intrathecal space, attributed to anatomical changes resulting from body habitus. For instance, in a pregnant woman with a substantial central mass and hyperangulated lumbar lordosis, the geometry of the epidural, intrathecal, and spinal compartments can be significantly modified.

Equation 2: $C_{epi} = \dfrac{\Delta V_{epi}}{\Delta P_{epi}}$

C_{epi} = Compliance epidural space; (ΔV_{epi}) = Volume epidural space; (ΔP_{epi})= Pressure epidural space.

The relevance of Equation 2 is with the understanding that the compliance of the epidural space is a constant value dependent on patient presentation,[7] and that changes in pressure in the epidural space (ΔP_{epi}) result in an inversely proportional change in the effective volume of the injected epidural drug (all else being equal), where volume of the epidural space is represented as (ΔV_{epi}). Patients who present with favorable spinal conditions have higher compliance of the spinal column and can accommodate large increases in pressure and volume from neuraxial anesthesia, further extending the protective nature of spinal cord compliance. However, in patients with unfavorable anatomy (such as segmentation of this space), the benefit of this protective feature is exhausted quickly, and additional injection of medication leads to large increases in pressure. As previously mentioned, a direct impact of this is observed in pregnant women. Notably, pregnant women, especially in their third trimester, have a changing center of gravity, which is compensated for with an amplified lumbar lordosis.[9] This change decreases the space between spinous processes, thereby making neuraxial anesthesia technique more difficult.

Scar tissue from trauma or prior procedures may also modify the typical compliance of the neuraxial space. Segmentation of the epidural region due to scar tissue may result in higher pressures than those found throughout the entire epidural space. Previous studies have shown that patients who have a history of prior lumbar spine surgery, such as scoliosis surgery,[10] or previous lumbar epidural anesthesia, have been shown to have reduced analgesic spread due to the accumulation of fibrotic connective tissue.[11] This is the reason that commonly employed troubleshooting methods for issues stemming from the epidural space variations, like catheter retraction or replacement above/below the insertion site, or additional medication boluses are unlikely to yield significant improvements. Administering a bolus in the presence of these clinical scenarios is potentially dangerous, as it could inadvertently lead to a total spinal condition due to increased pressure in these micro-segments as limited movement of fluid in a confined space may further exacerbate epidural pressures [Figure 3].

Through mathematical modeling, it can be concluded that there is a critical value at which any additional doses of medication result in significant increases in epidural pressures.[6] It is estimated that in some instances even a 10 mL bolus of local anesthetic agent injected may result in 100 mmHg or greater pressure increases in the epidural space.[6]

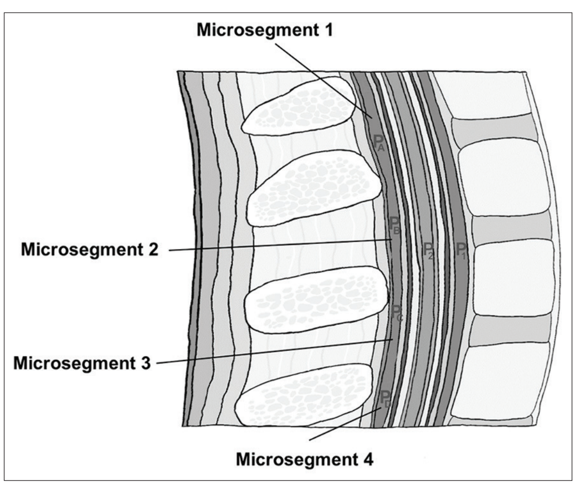

Figure 3: Microsegmented Model as a Result of Fibrous Membranes or Lumbar Lordosis. ***$P_{A, B, C, D}$ (microsegments) > P_1 (general epidural space)***

In a study conducted by Avellanal *et al*.,[12] the authors concluded that intermittent boluses of fluid over a 60-minute period with a maximum volume of 60 mL in the epidural space are insufficient to keep epidural pressures below 60 mmHg – a critical epidural pressure; thus, causing concerns for a high spinal if the dura and arachnoid layers are breached. As previously mentioned, should an epidural fail to develop an adequate level despite appropriate catheter depth, local anesthetic may be trapped in a micro pocket due to adhesions. Perforation of this high-pressurized micro-segment could be catastrophic by channeling the pressure differential into the lower-pressure intrathecal space. This may have potentially devastating effects on the central nervous system. Further dissemination of the fluid from higher pressure compartments to lower pressure compartments translate to other less pressure-tolerant organs resulting in tinnitus or loss of hearing, cranial nerve palsies, blindness, or increased ICP.

Mechanics in the Neuraxial Space(s)

While the unique anatomical and physiological conditions of a patient's spinal cord play a role in the successful injection of neuraxial anesthesia, it is also important to consider the role of injection mechanics and its possible deleterious effects. When a clinician injects neuraxial anesthesia through a needle into the intrathecal space during a spinal, the velocity at which the medication is injected may play a significant role in the pressure generated at the tip of the needle. In a simplified relationship of Bernoulli's equation, the Velocity (V) is directly related to the pressure generation in equation 3

(*Equation 3:* $\Delta P = \frac{1}{2}\rho V^2$).

ΔP = *Pressure;* ρ = *density of fluid;* V = *Velocity*

This is derived from Bernoulli's principle that the total energy of the system is conserved, where the fluid dynamic system in this case is a balance between kinetic energy (fluid movement/injection speed) and potential energy (pressure). Thus, as there is an increase in the velocity, the pressure must decrease to maintain the same energy within the system.

Depending on the volume of medication and the patient's anatomy, this pressure build-up may become larger than the pressure in the intrathecal space. Therefore, it is recommended that the rate of injection through the spinal needle be deliberately slow to avoid massive pressure changes which could disrupt more delicate components of the central nervous system. Often, it would be considered safe for clinicians to remove an epidural that has not been bolused with more than 10 mL of local anesthetic fluid and perform a spinal without complication[4]; however, once an epidural has been bolused with ≥10 mL and an analgesic level has not been obtained that is symmetrical and continuous, the choice to remove the epidural and perform a spinal significantly increases the likelihood of a total spinal and once the integrity of the dura membrane has been compromised with the spinal needle or a dura tear [Figure 4].[5]

The arachnoid layer is the final barrier between the epidural space and intrathecal space. Clinically, the two are rarely violated independently and more often breach of the dura results in the violation of the arachnoid layer. Since the dura and/or the arachnoid is primarily acting as a protective filtration membrane, once a spinal needle is introduced through the dura and/or arachnoid, a direct conduit is created allowing the over-pressurized epidural volume to channel into the now lower-pressure intrathecal space. This will cause fluid from the high-pressure epidural space to flood the low-pressure intrathecal space rapidly, thus increasing the height of the spinal block to cervical segments or beyond [Figure 4].

Even in cases where the dura is not intentionally compromised and remains intact, increased epidural pressures due to over-bolusing local anesthetic has been shown to cause total spinals by mere volume and concentration gradients alone as demonstrated in a case-study.[8] Park described the development of a TSA due to a post-procedure epidural saline injection to hasten the recovery of motor function which inadvertently increased diffusion of long-acting local anesthetics in the epidural space across the dura into the subarachnoid CSF. This demonstrates that even without a compromised barrier to the intrathecal space, a pressure gradient remains a major factor in causing a high spinal after a failed epidural. An explanation of this phenomenon is not reliably discussed in literature. Perhaps, the most understood model of this problem is due to elevated epidural pressures resulting from repetitive epidural local anesthetic injections and the subsequent injection of saline into the epidural space after the procedure can lead to compression of the dural sac. This compression may force cerebrospinal fluid-containing local anesthetic into the intracranial area, potentially triggering a transient spinal anesthesia (TSA). Therefore, healthcare professionals performing epidural spinal blocks should remain vigilant about this secondary cause of TSA. This becomes particularly relevant in situations where clinicians are deciding on the appropriate course of action when an epidural or spinal anesthesia fails to provide adequate sensory coverage for a C-section. Should a failed epidural occur, deciding on whether to perform a spinal after a failed epidural remains a clinical challenge. Another challenge to be considered is deciding the appropriate dosing of the spinal after a failed epidural. This debate has been long-standing and most comprehensively discussed in literature by Stocks and Wilson.[13,14] In 2005, Wilson advocated that performing a spinal after a failed epidural was safe using normal spinal anesthetic doses. However, throughout literature, many studies suggest reducing such spinal doses by 20-30% to avoid the risk of a high spinal; however, the 20-30% reduction is an arbitrary conclusion and fails to prevent the development of a high spinal after a failed epidural.[15,16] Unfortunately, this strategy is not preventative in developing a high spinal after a failed epidural either. In agreement with Stocks, combining a reduced spinal epidural dose using a combined spinal epidural (CSE) with an expansion epidural catheter is the best approach at minimizing the risks of a high spinal. The reduced dose can be approximated by a formula developed by Vadhera *et al*.,[17] as seen in equation 4. Evidence of this dose reduction was observed through formulaic application on 23 parturient with failed epidurals and none had a high spinal.

Equation 4: Reduced Dose of Spinal Anesthetic Dose =

[# segments with no block + (# segments with some block × 0.5) x (standard dose*)/18]

* Standard dose was based on (bupivacaine 11.25 mg with fentanyl 20 mcg)

An added benefit of using an expansion epidural catheter is that it can be utilized to administer additional medication should there be an under-dosing of local anesthetic while using a single-shot spinal with a reduced volume.

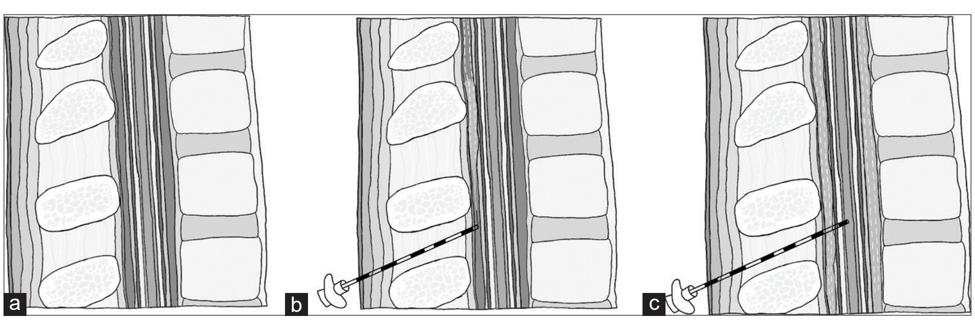

Figure 4: (a) Normal anatomical structure of the vertebral column prior to injection of anesthetic. (b) Needle placement for epidural block. Epidural space begins to expand as a medication is bolused. The dashed line represents the original epidural space. (c) Needle placement for spinal block requires puncturing the dura layer and may pose a risk for total spinal if the epidural layer is expanded from previous medication injection

Mechanistically, epidural, and spinal anesthesia, as employed in a CSE, exhibit a cumulative effect that cannot be fully explained by the quantity of medication administered via the epidural route.[18] Several mechanisms have been proposed to elucidate the upward spread of neuraxial medication, including the passage of epidural anesthetic through the dural puncture needle and the presence of subclinical analgesia at a higher spinal level.[17] However, a more compelling explanation for this augmented cephalad spread of spinal medication is the mechanical compression of the thecal sac due to the additional fluid introduced into the epidural space.[19]

Existing literature demonstrates that an epidural fluid bolus elevates pressure within the epidural space, potentially resulting in mechanical compression of the thecal sac, a phenomenon referred to as epidural volume extension (EVE).[19] In combined spinal-epidural procedures, this thecal sac compression has been observed to promote a greater cephalad spread of any spinal anesthetic introduced into the intrathecal space.[19] An MRI study investigating the impact of epidural saline injection on CSF revealed that the maximum compression of the thecal sac occurs approximately 5 minutes after an epidural fluid bolus and can persist for up to 30 minutes.[19] Research has indicated that a 10 mL epidural bolus of normal saline, administered 5 minutes after spinal anesthesia, increases analgesic levels when compared to spinal anesthesia alone. A similar effect was observed with a 10 mL epidural bolus of local anesthetic administered 5 minutes after spinal anesthesia.[18] Moreover, it has been demonstrated that a 10-mL epidural injection of normal saline raises the upper level of intrathecal contrast from L3 to L1 and from L2 to T12, suggesting that this phenomenon is primarily attributable to the volume administered, rather than solely a pharmacological effect.[18] Consequently, the timing of an epidural bolus in relation to the administration of spinal medication requires careful consideration when troubleshooting incomplete or failed neuraxial anesthesia.

Recommendations for Troubleshooting Epidurals

Understanding the mechanics of the intrathecal space and its surrounding anatomy is important prior to performing neuraxial procedures. It will not only allow for better troubleshooting when complications arise, but also ensure the clinical acumen and safe clinical practice delivered to the patient. Table 2 discusses some general guidelines regarding troubleshooting of a non-functioning or partially functioning epidural and a spinal block.

If an epidural fails to achieve a level with approximately 10 mL of local anesthetic bolus, it is advisable not to perform a spinal block within 30 minutes of the last epidural bolus as the expanded epidural space from administering boluses will have a higher pressure than the intrathecal space.[5] Piercing the dura when performing a spinal after epidural boluses have been administered will cause the local anesthetic to push the intrathecal volume higher in addition to the mechanical effect of an expanded epidural space compressing the dural sac, thus increasing the risk for a total spinal. Simply reducing the volume of the spinal local anesthetic will not effectively decrease the likelihood of this occurrence, as the volume in the epidural space would still flood the intrathecal space. The suggested 30-minute timeframe is not based on empirical data, but rather on a cautious estimate, given the unknown rate of diffusion through the neuroforamina. Thus, a residual risk always remains.[5]

Rapid injection may lead to elevated levels of spinal anesthesia. Mathematical modeling suggests an injection rate of less than 3-4 mL/sec, regardless of syringe size.[5] Therefore, slow injection rate should be employed, less than 3-4 mL/sec when administering medication in the epidural space while being vigilant for signs of resistance during injection, pain, and the swift onset of lower extremity paralysis as these are indicators of intrathecal injections.

Repeated boluses in the epidural space lead to a summative epidural volume. A 10 mL of local anesthetic should be enough to see evidence of an analgesic effect of a correctly placed epidural. At approximately 1.5-2 mL of local anesthetic per epidural space level, a 10 mL local anesthetic bolus should offer coverage for a minimum of five vertebral levels. When factoring in elements that may diminish the epidural space volume, this 10 mL local anesthetic bolus represents a substantial coverage area and quantity of local anesthetic. Furthermore, it is recommended to avoid bolusing more than 60 mL of local anesthetic into the epidural space within a duration of 60 minutes based on mathematical modeling.[11]

Understanding the potential reasons for an epidural or spinal not reaching the desired analgesic level is also critical. Some likely reasons could be – previous back surgeries, prior blood patches, or spinal stenosis. While not commonly described in the literature, it is believed that blood patches will decrease the compliance of the epidural/intrathecal space until the reabsorption of the hematoma. Understanding these pathologies impacts the management of the procedure and influences risks and outcomes. If the patient develops symptoms of headaches or neck pain during a spinal, it is recommended to abort the procedure altogether, as the cause of these symptoms is the patient's underlying spinal pathology (micro adhesions, etc.), and it is unlikely that attempting the procedure again will lead to a favorable outcome. Finally, in the event that a total spinal does occur, it is imperative to have a clear understanding of the crucial medical management that must follow: Intubation, mechanical ventilation, ICU, neuromonitoring, and neuroprotection strategies.

Conclusion

Although neuraxial anesthesia is widely used in obstetrics and gynecology, pain medicine, and orthopedic surgery, a limited understanding of procedural mechanics often

Table 2: Recommendations for troubleshooting epidurals and spinals[4]

Issue	Recommendation
Epidural fails to achieve the desired level with approximately 10 mL of local anesthetic bolus	Avoid performing a spinal within 30 minutes of the last epidural bolus to minimize the risk of a total spinal[5].
Resistance on injection, pain, and/or rapid onset of lower extremity paralysis following epidural anesthetic administration	Inject slowly, less than 3−4 mL/sec, or stop and assess the anesthetic level.
Need for multiple successive boluses	Evaluate the current state of the epidural. Do not over-bolus an epidural; 10 mL should be sufficient to observe an analgesic effect. Based on mathematical modeling, the maximum volume of local anesthetic is 60 mL. Be cognizant that repeated boluses in the epidural space have a summative effect on the epidural volume and concentration gradient.
Epidural or spinal not reaching the desired analgesic level	Consider prior blood patches, spinal procedures, back surgery, spinal stenosis, microadhesions, and their impact on the procedure, risks, and outcomes. Evaluate if alternatives to epidurals/spinal are safer.
Incidence of a high spinal	Understand the medical management, including intubation, mechanical ventilation, ICU, neuromonitoring, neuroprotection strategies, and the option of CSF lavage if deemed safe with central venous pressure monitoring.
Symptoms during spinal procedure	If the patient develops headaches or neck pain during a spinal, consider aborting the procedure as anatomical changes are unlikely to make outcomes more favorable.

hinders troubleshooting for non-functional or partially functional epidurals, emphasizing the importance of understanding factors like patient anatomy, spinal pathology, anesthetic injection rate, compartment compliance, and pressure gradients to prevent complications. Based on a comprehensive literature review, this manuscript proposes a simplified 2-compartment model to help explain the mechanisms of how high or total spinal anesthesia occurs, particularly considering anatomical changes seen in pregnancy or prior surgeries/procedures that alter the dynamics of the neuraxial space. It is recommended that careful consideration of patient history, pathology, and timing of prior neuraxial medications when deciding to troubleshoot suboptimal analgesia while having a clear algorithm for managing complications like a total spinal.

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Conflicts of interest

There are no conflicts of interest.

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