

# UNINTENDED SUBDURAL INJECTION: A COMPLICATION OF EPIDURAL ANESTHESIA — A CASE REPORT

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*Epidural anesthesia is practiced in virtually every clinical setting. Its safety and versatility have supported increasing use for more and varied therapies. In a healthy patient in whom near-complete left hemiparesis developed following a routine continuous epidural anesthetic for labor, subdural deposit of the local anesthetic was suspected. The follow-*

*ing case and discussion may help illustrate the mechanism behind this complication and how it can be detected, treated, and, possibly, avoided.*

**Key words:** Complications, dura-arachnoid interface, lumbar epidural, unintended subdural injection.

**T**housands of epidural anesthetics are administered around the world on a daily basis in academic centers, community hospitals, pain clinics, and other care-based facilities. The safety and benefits of regional anesthesia and analgesia, especially continuous lumbar epidural anesthesia, have improved dramatically during the past 3 decades. When successful, the procedure and outcome are usually not given a second thought.

A common occurrence of epidural placement is an asymmetrical or “patchy” block. Often this is attributed to the nonuniform expansion of the potential epidural space, the anatomic obstacles therein, the potential dilution of the familiar medications at our disposal, or the catheter migration to one side.<sup>1</sup> A fifth and less appreciated explanation for a patchy result is a partial subdural block with neuraxial conduction loss that does not fit the pattern of a predictable or intended subarachnoid or epidural anesthetic.

The actual incidence of an accidental subdural deposit of local anesthetic intended for epidural blockade is unknown. However, a recent literature search suggests that it may range anywhere between 0.006%<sup>2</sup> and 17%,<sup>3</sup> and, as demonstrated by subsequent radiographic analysis, this phenomenon of subdural local anesthetic placement may, in fact, occur more often than previously recognized.

An unintended subdural injection may be recognized as an extensive neural blockade that is out of proportion to the amount of local anesthetic injected and may be suspected in the absence of an identified subarachnoid puncture.<sup>4</sup> Subdural injections have been implicated in cases in which the density of block and extensive spread of an intended epidural are disproportionate to and inconsistent with the volume of local anesthetic injected.

Moreover, it has been reported that unintended subdural injection can be of delayed onset as well as

of uneven result. The tendency, therefore, is to subsequently introduce more local anesthetic to effect a solid block. If the catheter or any portion of it remains subdural, this may result in a higher block than intended and may lead to severe hypotension, bradycardia, and prolonged sympatholysis. Furthermore, because the subdural space has a poor blood supply and loss of effect is dependent on diffusion away from the site, the injected medium may stay in that space for a considerable time.<sup>5</sup>

The following case illustrates a situation in which a routine lumbar epidural placement was complicated by a presumed subdural or multicompartmental block.

## Case summary

The patient was a 29-year-old woman, 5 ft, 7 in, 216 lb, ASA physical status III, term parturient who was admitted in active labor with a history of chronic hypertension and new onset superimposed preeclampsia. She had allergies to sulfa, tetracycline, and eggs. Further examination indicated a history of mild asthma, infrequent migraine headaches, and first trimester bleeding. She had no history of neurologic disease.

Her blood pressure on admission was 144/92 mm Hg and, in labor, 170/100 mm Hg, with a pulse consistently around 80 beats per minute. Laboratory results from 5:00 AM that day were as follows: hemoglobin level, 9.1; hematocrit value, 27.1; and platelet count, 253,000. In the sitting position, a continuous lumbar epidural was easily and successfully placed at L3-4 by an experienced and skilled physician who identified the epidural space by loss-of-resistance with an 18-gauge Tuohy needle and the air-saline-air technique.<sup>1</sup> No problems were encountered during placement, and no rotation of the needle was used. A 20-gauge catheter (Periflex, closed-tip, multiport, B. Braun Medical Inc, Bethlehem, Pa) was inserted to a

depth of 3.5 cm and, following negative aspiration, 5 mL of 1% lidocaine was injected, followed by a 5-minute pause for observation. When no signs of subarachnoid or intravascular injection were apparent, fentanyl, 100 µg, 5 mL of 0.25% bupivacaine, and 5 mL of 0.9% sodium chloride was administered at 5:50 PM, for a total of 17 mL. A continuous epidural infusion of 0.1% bupivacaine with fentanyl, 2 µg/mL was begun at 5:55 PM at a rate of 14 mL/h. Although the sensory level was not recorded initially, anesthesia proved adequate throughout labor and delivery.

I assumed management of the anesthetic at 7:00 pm and change of shift. After delivery of a viable infant at 7:53 pm, the patient was taken to the operating room for manual extraction of the placenta. Nursing flow sheets indicated the continuous epidural pump was discontinued at the time of delivery. Blood pressures in the immediate postpartum phase were in the range of 143/88 mm Hg, and the other vital signs were stable.

Exhibiting no motor block from the labor epidural, the patient easily moved herself onto the operating table and was positioned appropriately with both arms on padded arm boards at slightly less than 90°, monitors were applied, and intravenous (IV) fluid (500 mL of multiple electrolytes injection, type 1, US Pharmacopeia) preload was provided. In anticipation of surgical discomfort, 10 mL of 2% lidocaine with epinephrine 1:200,000 and 1 mL of 8.4% sodium bicarbonate were administered slowly via the epidural catheter at 10:12 PM.

Aspiration was negative before and after injection. After her legs were placed in stirrups, the patient was tilted into a modified semi-Fowler position. Straight catheterization of the bladder yielded 200 mL of clear urine. Midazolam, 1 mg, was administered IV along with oxygen at 6 L/min delivered via nonrebreathing mask, and verbal contact was maintained throughout the procedure. An additional 5 mL of 2% lidocaine was provided at 10:22 PM, and onset of surgical analgesia was satisfactory. The patient reported neither discomfort nor any unusual symptoms throughout the procedure. By light scratch, the midline dermatome level was judged to be T8.

Before placental extraction, the patient received 2 metered spray doses of oral nitroglycerine (800 µg total) to provide uterine relaxation, and onset was evident within 30 seconds. The systolic blood pressure during the procedure ranged from 110 to 138 and the diastolic from 56 to 70 mm Hg, and the pulse ranged from 80 to 90 beats per minute. Placental extraction was swift and uneventful, followed immediately by 5 units of oxytocin, IV push, with 35 additional units

placed in the second full liter of multiple electrolytes injection at 10:30 PM. The patient's legs were released from stirrups, and she was prepared to be taken to the postanesthesia care unit (PACU). The total blood loss, including delivery and as reported by the obstetrician, was 900 mL. The total amount of fluid administered in the operating room was 1,000 mL of crystalloid.

As the patient was being moved to the gurney, it was noted that she was unable to move her left arm. She was able to hold her left arm across her chest with the aid of her right arm but had profound motor blockade in the left and reported tingling in both arms.

On arrival to the PACU, the patient's initial blood pressure was 66/32 with a pulse of 70 beats per minute, respirations of 18 breaths per minute and without distress, an oxyhemoglobin saturation of between 96% and 100%, and an axillary temperature of 37.1°C. She was immediately placed in steep Trendelenburg position, IV fluids were opened wide via an 18-gauge catheter, and oxygen mask at 8 L/min was begun. Because of her history of hypertension and recent nitroglycerine administration, it was decided to not use vasopressors at the time. The patient's mentation was appropriate, and she was fully cooperative during this time and during the subsequent neurologic assessment.

Physical examination revealed a good grip in the right hand with slight sensory loss and no grip in the left hand with marked sensory loss. An alcohol swipe was used to determine cold sensation on her chest and revealed T4 sensation level midline and left and slightly lower on the right.

She was able to shrug both shoulders, vigorously on the right and slightly less on the left. Facial grimace revealed equal and symmetrical activity, as did requests to open her mouth, protrude the tongue, blink the eyes, smile, and lift her head. Phonation was intact. Pupils were equal and reactive, and no signs of Horner syndrome were present. The patient reported no sensory loss in the face and neck and did not express any distress with the aforementioned findings or her stay in the PACU.

Blood pressure during the first 30 minutes in PACU ranged from 76/34 to 122/56 mm Hg, with pulse climbing slowly from 70 to 92 beats per minute during the same period. With fluid resuscitation and presumed return of sympathetic tone, her vital signs returned to an acceptable range after 30 minutes, with a blood pressure stable at 110/42 mm Hg, pulse of 88 beats per minute, and an oxyhemoglobin saturation of 100%. She was gradually repositioned to a modified semi-Fowler position, and her condition remained sta-

ble. She was transferred out of the PACU 30 minutes later, after reporting near full sensory and motor return to her affected limbs. Crystalloid administered in the PACU totaled 1,300 mL.

That evening, she was able to ambulate without assistance, hold her baby, and demonstrate a full return to normal activity. A postpartum visit the next day revealed no signs of deficit, complications, or neurologic sequelae. She was discharged 36 hours after delivery.

## Discussion

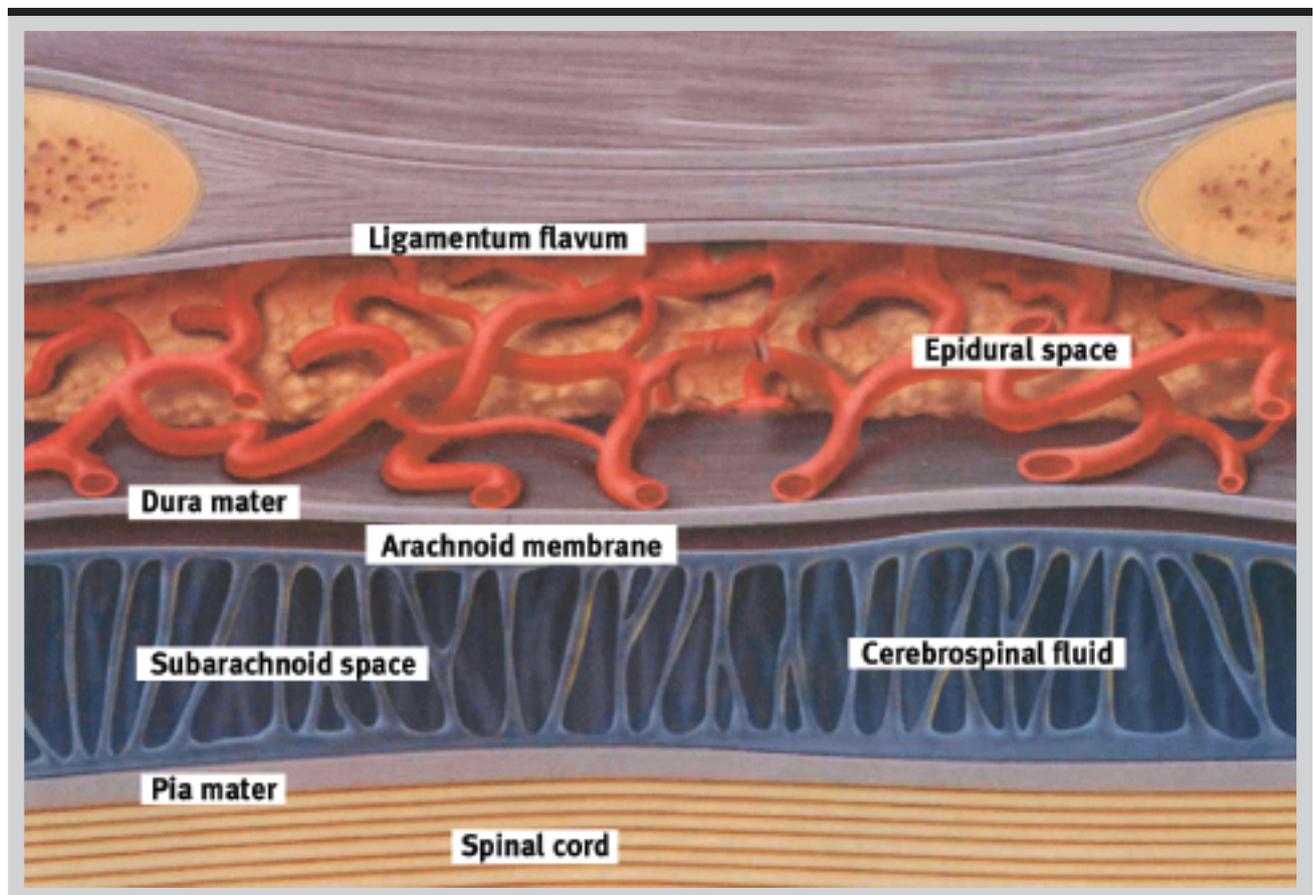
Obstetric anesthesia practice that includes the use of spinal and epidural anesthesia or the combination of both involves the routine application of sufficient local anesthesia and often narcotic in proximity to appropriate neural structures for the purpose of obtaining a degree of conduction block. The blocks are, by conventional execution, designed to be sub-arachnoid or epidural.

The structure of the meninges, particularly the dura-arachnoid interface, is of increasing interest to the anesthesia community. Anatomically, the dura is composed of elongated, flattened fibroblasts and copious

amounts of extracellular collagen. The arachnoid layer is composed of large cells with numerous cellular junctions, no extracellular space, and no extracellular collagen. The dural border cell layer is found at the dura-arachnoid junction and is characterized by flattened fibroblasts, extracellular spaces, but no extracellular collagen and few cell junctions. These features combine to create a layer of inner dura that is, albeit adherent, structurally weak compared with external portions of the dura and the internally located arachnoid. However, under normal conditions, there is no evidence of a naturally occurring space existing at the dura-arachnoid junction.<sup>6</sup>

By using electron microscopy, Reina et al<sup>7</sup> recently described a complex ultrastructure of what they refer to as the *dura-arachnoid interface* (Figure 1) in which “space can appear as neurothelial cells break up because of pressure exerted by mechanical forces creating fissures within the amorphous substance of the interface.” These fissures could grow larger and toward weaker areas, creating a dura-arachnoid space in proportion to the pressure exerted.<sup>7</sup> In cadaver models, it was observed how easily the arachnoid layer could be separated mechanically from the dura

**Figure 1. Schematic view of vertebral arch structures illustrating the dura-arachnoid interface**



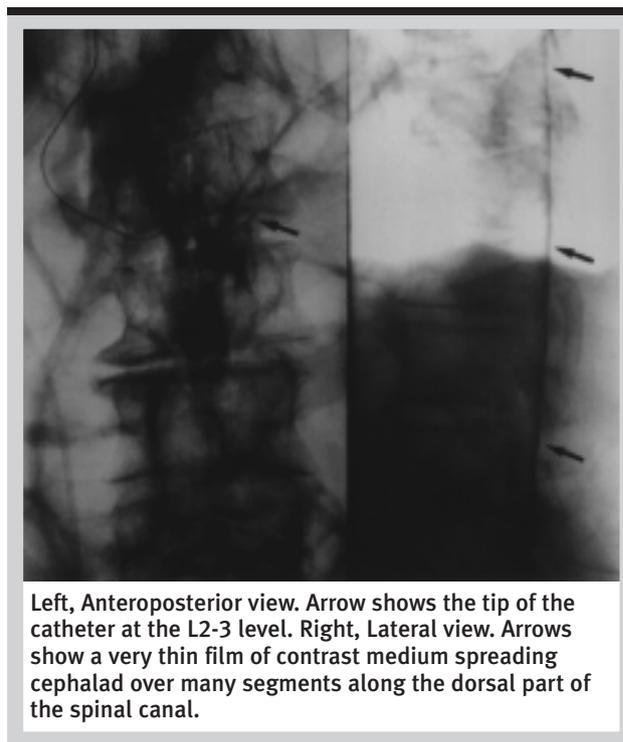
mater, thereby creating a potentially extensive subdural but extrathecal space.<sup>3</sup> Both teams' work also suggest that, in the nontraumatized dura, this cleft within the meninges probably does not exist.<sup>3,7</sup>

An unintended subarachnoid injection following attempted epidural placement usually manifests with a block that is bilateral with profound onset and, even with lower doses of isobaric or hypobaric local anesthetic solution, more intense than usually is seen following administration of drug into the epidural space. However, a subdural, nonintrathecal injection may be suspected if the clinical picture is one of a unilateral, uneven, or inadequate block with delayed onset and a disproportionate sympathectomy secondary to a higher than expected level. Negative catheter aspiration may help rule out subarachnoid or intravascular placement or subsequent migration but is of little value if the catheter rests, in whole or in part, within the dura-arachnoid interface.

If local anesthetic is accidentally injected into the subdural space, an average lumbar epidural dose for abdominal or lower limb anesthesia can easily result in anesthesia in the thoracic and cervical regions (Figure 2). Because the subdural space extends from the lower border of the second sacral vertebrae into the cranial cavity and as high as the floor of the third ventricle,<sup>3</sup> brainstem involvement is possible, with potential apnea and profound bradycardia following. Involvement of the shoulder girdle and neck muscles would indicate a level obtained between the C5 and T1 roots. The rhomboidus major and rhomboidus minor are responsible for scapular rotation and elevation (eg, shoulder shrug), and their innervation is shared between C4 and C5. Because the phrenic nerve originates from C3 to C5, side effects such as hypotension and acute ventilatory failure secondary to respiratory muscle paralysis have been observed.<sup>9</sup> In the aforementioned case, the patient was still able to fully shrug her right shoulder and to partially shrug her left. This suggests that left-sided C3 to C5 involvement was incomplete, possibly sparing or minimally involving the phrenic nerve and diaphragm function.

Several mechanisms seem to be evident and may explain our patient's dramatic change in vital signs in the presence of an unexpectedly high block. The disproportionately higher block, believed to be subdural and predominantly left-sided, led to an extended sympathectomy and further impaired the patient's ability to compensate via the normal mechanisms of vasoconstriction and cardiac output. It is generally accepted that extension of a segmental block that involves cardioaccelerator fibers (higher than T4) often can explain the bradycardia that may accom-

**Figure 2. Radiographs after the injection of 3 mL of contrast medium via the epidural catheter**



(Image courtesy of Ban Tsui, MD. Reprinted with permission from the *Canadian Journal of Anesthesia* and the *Canadian Anesthesiologists' Society*.<sup>8</sup>)

pany epidural anesthesia. However, central volume depletion may have a greater cardioinhibitory vasodepressor effect.<sup>10</sup>

The splanchnic blood reservoir contains between 20% and 40% of the total blood volume. Normal sympathetic activity increases resistance, decreases splanchnic blood volume, and increases systemic blood volume, thereby elevating systemic arterial pressure. If the splanchnic fibers (T6 to L1) are blocked, significant pooling of the blood in the gut and abdominal viscera also may occur.<sup>11</sup>

The venous capacitance vessels contain roughly 80% of circulating blood volume. Venodilatation in the lower extremities has potential for dramatic changes in venous return, reduced right atrial filling pressure, and reduced cardiac output. This decrease in venous return has been shown to result in increased vagal tone.<sup>12</sup> In placing the patient's legs flat after 30 minutes of stirrup suspension, autotransfusion of the core is reduced significantly, effectively preventing adequate venous return and, thereby, reducing cardiac output. In addition, a varying degree of thoracolumbar blockade (T10 and higher) would lead to a reduction in adrenal activity and secretion of available catecholamines.

Vagal predominance is known to be an important factor in the development of bradycardia during regional anesthesia. Reduced venous return in the face of sympathetic inhibition and parasympathetic activation can further lead to vasodilatation, hypotension, and bradycardia. This is termed *vasovagal syncope*, or the *Bezold-Jarisch reflex*, and is likely mediated in part via the afferent nerves from the heart, but also by various noncardiac baroreceptors that may become paradoxically active. The response can occur during regional anesthesia and can become more profound in the face of hypovolemia.<sup>13</sup>

In addition, vasodilatation from oral nitroglycerin can last up to 60 minutes, further compounding the patient's ability to compensate. In retrospect, low-dose pressor therapy with ephedrine or dopamine may have offered more rapid fluid and vascular restabilization. Furthermore, this scenario likely was compounded by relative hypovolemia secondary to increased blood loss between delivery and placental extraction and by conservative IV fluid administration throughout the labor and operative phases because of preeclampsia.

## Conclusion

This article describes an epidural anesthetic complicated by slow onset of left arm paralysis and hypotension despite T8 sensory level. Migration of the anesthetic into the subdural, extrathecal space, or the dura-arachnoid interface, allowing movement of the anesthetic cephalad and into the thoracocervical segments is a possible cause. Even within a solid and intact vertebral arch subsystem, mechanical manipulation of the soft tissue structures can result in microscopic trauma to and dissection of the complex dural layers, allowing fluid (or air) under pressure generated by the force of injection to follow a path of least resistance. The result then would be an absorption of the local anesthetic across the epithelium, resulting in an uneven block with an unpredictable spread, with a result of sensory and motor blockade in disproportion to the level planned.

Many researchers and clinicians now believe this phenomenon of unintended subdural deposit of local anesthetic also may explain the variable onset of an

epidural and extension of neural blockade as an unobserved and occult creation of a subdural space under conditions of microscopic trauma during placement of a routine epidural anesthetic. The present case also represents how multiple physiologic factors can influence outcome and therapy.

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## SUGGESTED READING

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