

the neuroradiologist felt free to inject additional O₂. During general anesthesia, this subjective indicator of potentially dangerous elevations in subarachnoid pressure does not exist. One spike of pressure during general anesthesia exceeded 130 cm H₂O. Therefore, it would seem advisable to monitor subarachnoid pressures continuously during gas myelography with general anesthesia, even though, according to our findings, the major source of potentially dangerous high pressure is not inhaled N₂O. Subarachnoid pressures should probably also be measured continuously during gas myelography with local anesthesia, because this would make possible, through controlled injection of O₂, the avoidance of subarachnoid pressures that produce pain.

Not only N₂O but also N₂ and CO₂ diffuse into the subarachnoid bubble during gas myelography. The CO₂ concentration of 7.0 per cent (P_{CO₂} 53 torr) in the subarachnoid gas of Patient 7, who was spontaneously breathing air during local anesthesia, was within the range of normal P_{CO₂} in the CSF of normocapnic man.⁵ Concentrations of CO₂ in gas removed from the subarachnoid space of five of the six patients anesthetized with N₂O, ranging from 3.8 to 5.8 per cent (P_{CO₂} 29 to 44 torr), would be expected during pulmonary hyperventilation. The CO₂ concentration of 9.8 per cent (P_{CO₂} 74 torr) in Patient 4, however, suggested respiratory acidosis. That patient, in fact, did have compensated congestive heart failure and chronic restrictive pulmonary disease with pleural effusion, and showed evidence of respiratory distress

when permitted to breathe spontaneously at the conclusion of myelography. At that time, Pa_{CO₂} was 49 torr, p_{H_a} 7.33.

Although we have shown substantial transfer of gas from CSF or blood into the subarachnoid bubble during gas myelography with N₂O anesthesia, major changes in subarachnoid pressure appear to be related to the pattern of injection and volume of O₂ introduced into the subarachnoid space by the neuroradiologist.

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This study was approved by the Human Subjects Committee of Peter Bent Brigham Hospital.

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Supraclavicular Subcutaneous Emphysema Following Lumbar Epidural Anesthesia

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Epidural anesthesia via the lumbar or sacral approach is widely used for surgical and obstetrical procedures. Although the technique of lumbar epidural anesthesia is relatively simple, problems of epidural space identification¹ and catheter placement² arise. This paper reports a case in which subcutaneous emphysema was present in the cervical and supraclavicular regions of a parturient in labor, six hours after lumbar epidural catheter insertion.

REPORT OF A CASE

A healthy 25-year-old, 70-kg, 165-cm woman, gravida 3, para 2, was admitted to the obstetrical ward in active labor after an uncomplicated 40-week pregnancy. Previous deliveries had been accomplished with local anesthesia. The cardiovascular, pulmonary, musculoskeletal, and neurologic systems review disclosed no abnormality. Results of cardiac and pulmonary examinations were normal, with a supine blood pressure of 120/60 torr and a regular pulse of 72/min. Vertebral landmarks were adequate. The cervix was dilated 4.5 cm and completely effaced. Hematocrit was 32 per cent; urine and chest x-ray were normal.

Lumbar epidural anesthesia was elected and the patient was placed in the left lateral decubitus position and prepped with tincture of benzalkonium chloride (Zephiran). After infiltration of the skin with 1 per cent lidocaine, many attempts at identification of the epidural space were made midline at the L3-4 and L4-5 interspaces with an 18-gauge Tuohy needle, using the technique of loss of resistance to injected air. An estimated total of 30-40 ml of air was injected prior to satisfactorily locating the epidural space. No blood, cerebrospinal fluid, or air was obtained on aspiration.

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FIG. 1. PA radiograph of upper chest and neck 8 hours following epidural catheter placement with supraclavicular and cervical subcutaneous air present.

No side effects followed injection of a 2-ml test dose of 3 per cent chloroprocaine; therefore, an additional 6 ml was injected. A styletted 19-gauge radiopaque Deseret[‡] Teflon[§] epidural catheter was easily inserted cephalad, 5 cm beyond the tip of the needle, which was then removed. Ten minutes later, analgesia from T8 to S1 was present bilaterally. Following an uneventful 1.5-hour labor, a 2,724-g healthy, term infant was delivered vaginally over a midline episiotomy which required supplemental lidocaine infiltration. The epidural catheter was subsequently removed intact.

After returning to the postpartum ward, the patient remained primarily in sitting and semirecumbent positions. Six hours later she complained, "It feels like water is in my neck," although no dyspnea, pain, or meningismus was noticed. Physical examination revealed obvious cervical and supraclavicular crepitus bilaterally and moderate crepitus posteriorly over the upper thoracic vertebrae; minimal crepitus was present over the lower thoracic and lumbar vertebrae.

Cervical, chest, and lumbar x-rays confirmed subcutaneous air present in the neck, especially in the supraclavicular spaces, extending into the upper chest posteriorly (fig. 1). No pneumothorax, mediastinal emphysema, or air in the lumbar region was observed on x-ray. Resorption of the subcutaneous air occurred uneventfully over the next 48 hours and the patient was discharged without sequelae.

DISCUSSION

Occasional complications of lumbar epidural anesthesia, including hypotension,³ inadvertent dural puncture,⁴ total subarachnoid or epidural block,^{3,5} and epidural hematoma,^{6,7} have been well described. Subcutaneous emphysema of the chest and neck, however, has not been associated with epidural anesthesia.

It is likely that the complication described in this paper occurred as a result of the technique chosen for identification of the epidural space, *i.e.*, the loss of resistance technique. Several modifications of this technique, as described by Bonica⁸ and others,² such

as using saline solution,⁸ local anesthetic solution,⁵ or air^{1,9} as the injected medium, may be employed.

In this patient, air was selected as the injected medium. Identification of the lumbar epidural space proved difficult, and as a result, an inordinately large volume of air was injected into the epidural and/or subcutaneous spaces. It is probable that this culminated six hours later in significant subcutaneous emphysema in the cervical and supraclavicular regions, with minimal air remaining in the lumbar region. We theorize that the injected air migrated preferentially cephalad, since the patient remained primarily in an upright position after delivery.

Several anatomic pathways may explain the eventual location of subcutaneous emphysema in the neck. First, air injected into the epidural space may have exited through the lumbar intervertebral foramina and moved cephalad along the deep fascial planes of the back, which are continuous with those of the neck (fig. 2). Subsequently, air in this compartment may escape into the subcutaneous tissue and be felt as crepitus.¹⁰ Second, the epidural air may have migrated cephalad while remaining in the epidural compartment (fig. 2). Since the epidural space terminates cranially at the foramen magnum,¹¹ cephalad movement of injected air would be limited to this level, where it may then diffuse through the cervical intervertebral foramina into the deep fascial planes and the subcutaneous tissues of the neck. Third, any

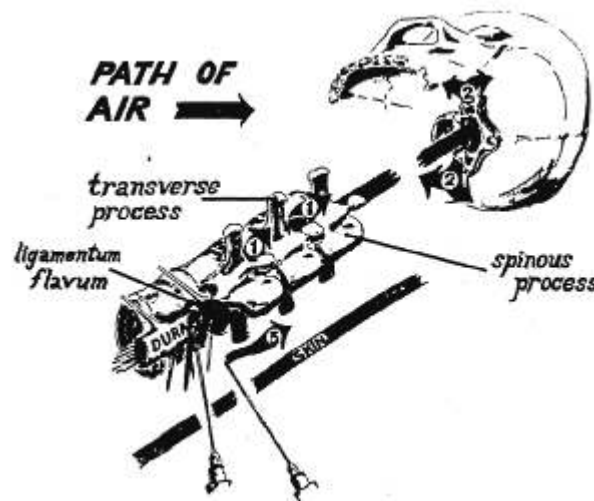


FIG. 2. Artist's illustration of three possible anatomic pathways to explain cervical and supraclavicular subcutaneous emphysema following lumbar epidural catheter insertion. 1, escape of injected air from the epidural space through the lumbar intervertebral foramina with cephalad migration along deep fascial planes of the back continuous with those of the neck. 2, cephalad movement of air within the epidural space with subsequent escape through cervical and thoracic intervertebral foramina into deep fascial planes and eventual migration to the subcutaneous compartment. 3, subcutaneous cephalad progression of air injected in the lumbar subcutaneous region.

[‡] Deseret Pharmaceutical Company, Sandy, Utah.

[§] Teflon is a DuPont registered trademark, Wilmington, Delaware.

air injected subcutaneously in the lumbar area may have migrated cephalad in this compartment, eventually to localize in the neck (fig. 2).

Subcutaneous emphysema may be alarming to the patient, and in large quantities has embolization potential. It may also produce extrinsic compression of vital neck structures, resulting in hoarseness or airway obstruction.¹³ Additionally, air injected into the epidural space may prevent uniform spread of local anesthetic solutions in this compartment and result in an uneven epidural block.¶ Finally, air injected into the body requires a very long period for resorption. Since as long as 16 hours is necessary for absorption of air from a nonventilated but well-perfused alveolus,¹⁴ an even longer period might be expected for resorption of air from poorly perfused subcutaneous tissues.

In contrast, selection of a liquid for the loss of resistance technique will avoid the problems associated with the use of air and may also allow for more sensitive transmission of pressure changes.¹⁵ Finally, the sudden forceful ejection of a liquid from the needle once the epidural space is entered may push the dura away from the needle and lessen the likelihood of inadvertent dural puncture.^{6,7} Sterile physiologic saline solution is preferred to local anesthetic solutions as a liquid injectate for loss of resistance, since its use eliminates the possibility of local anesthetic toxicity from inadvertent intravascular injection.

The case of a patient who had cervical and supraclavicular subcutaneous emphysema after lumbar epidural anesthesia is reported. Theories explaining

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Comparison of Epidural Saline Placement and Epidural Blood Placement in the Treatment of Post-lumbar-puncture Headache

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The occurrence of headache following lumbar puncture is rarely a major complication, but it is

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this event and modifications of technique to prevent its future occurrence are suggested.

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frequently distressing. In addition to the conservative measures of bedrest, hydration and analgesics, more direct methods have been developed to counteract cerebrospinal fluid leakage through the dural puncture site. Epidural or caudal injection of saline solution has been shown to be an effective countermeasure,¹⁻³ with success rates of 71 to 84 per cent

The opinions or assertions in this paper are those of the authors and are not to be construed as official or reflecting the views of the Navy Medical Department or of the Naval Service at large.

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