punctures, use of positive-pressure breathing following an attempted block, and familiarity with the anatomic landmarks (such as the interscalene groove). While Moore[1] stated an incidence of 0.5 to 4 per cent, Brand[2] reported an incidence of 6.1 per cent. It has been claimed that the classic interscalene approach of Winnie[3,4] offers the least chance of producing pneumothorax. The single-injection subclavian perivascular approach can produce pneumothorax, though the incidence is very low.

To our knowledge, hemopneumothorax following brachial block in heparinized patients has not been reported. Spontaneous hemopneumothorax in patients undergoing heparin therapy for thromboembolic disease has been reported.[5–7] In most of these cases the primary disease was pulmonary infarction. The presumed mechanism in these instances was either rupture of the hemorrhagic infarction into the pleural cavity or spontaneous bleeding from the pleural membrane. In these cases the onset was delayed, and all had hemothorax that was not associated with pneumothorax. In the case presented, the onset was relatively acute following brachial block and heparin therapy. The clinical manifestations occurring on the third day indicated either slow leakage of blood into the pleural cavity or dislodgement of clot and sudden hemorrhage subsequent to the puncture of lung tissue and the continued heparin therapy, respectively. Though it could have been a spontaneous hemopneumothorax following anticoagulant therapy, the fact that it occurred on the same side as the brachial block, and the retrospective review of the routine immediate postoperative chest x-ray showing a 10 per cent pneumothorax, substantiate the cause of the hemopneumothorax in this patient as injury to the lung during brachial block. Anatomically, injury to the pleura and lung on the left side is less likely because the left pleural dome is at a lower level in the neck compared with that on the right.

The normal platelet count in the postoperative period rules out the possibility that this bleeding was due to either idiopathic thrombocytopenia or heparin-induced immune thrombocytopenia.[8] The prolonged partial thromboplastin time indicates that the bleeding was due to the anticoagulant therapy. Most patients who have had hemothorax subsequent to pulmonary embolism have had some permanent sequelae. Many of these patients have had to undergo further surgical procedures such as decortication, whereas our patient did not.

The purpose of reporting this case is not to discourage the relatively safe technique of brachial block. It is to alert anesthesiologists to the rare possibility of development of this complication in patients heparinized during or after operation.

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Cerebrospinal Fluid Pressure and Subarachnoid Gas Composition during Nitrous Oxide Anesthesia for Gas Myelography

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During gas myelography, a bubble of O_2 is introduced into the subarachnoid space to outline abnormalities such as atrophy or edema of the spinal cord or obstruction of cerebrospinal fluid pathways. When air is used as a contrast medium during pneumoencephalography in man, the introduction of 70–75 per cent N_2O into the inspired anesthetic mixture can lead to more than a doubling of the subarachnoid pressure.[1] However, data on the behavior of air introduced into the cerebral ventricles cannot be assumed to apply to O_2 confined to the spinal subarachnoid space. Therefore, we have examined some aspects of the transfer of N_2O from blood into

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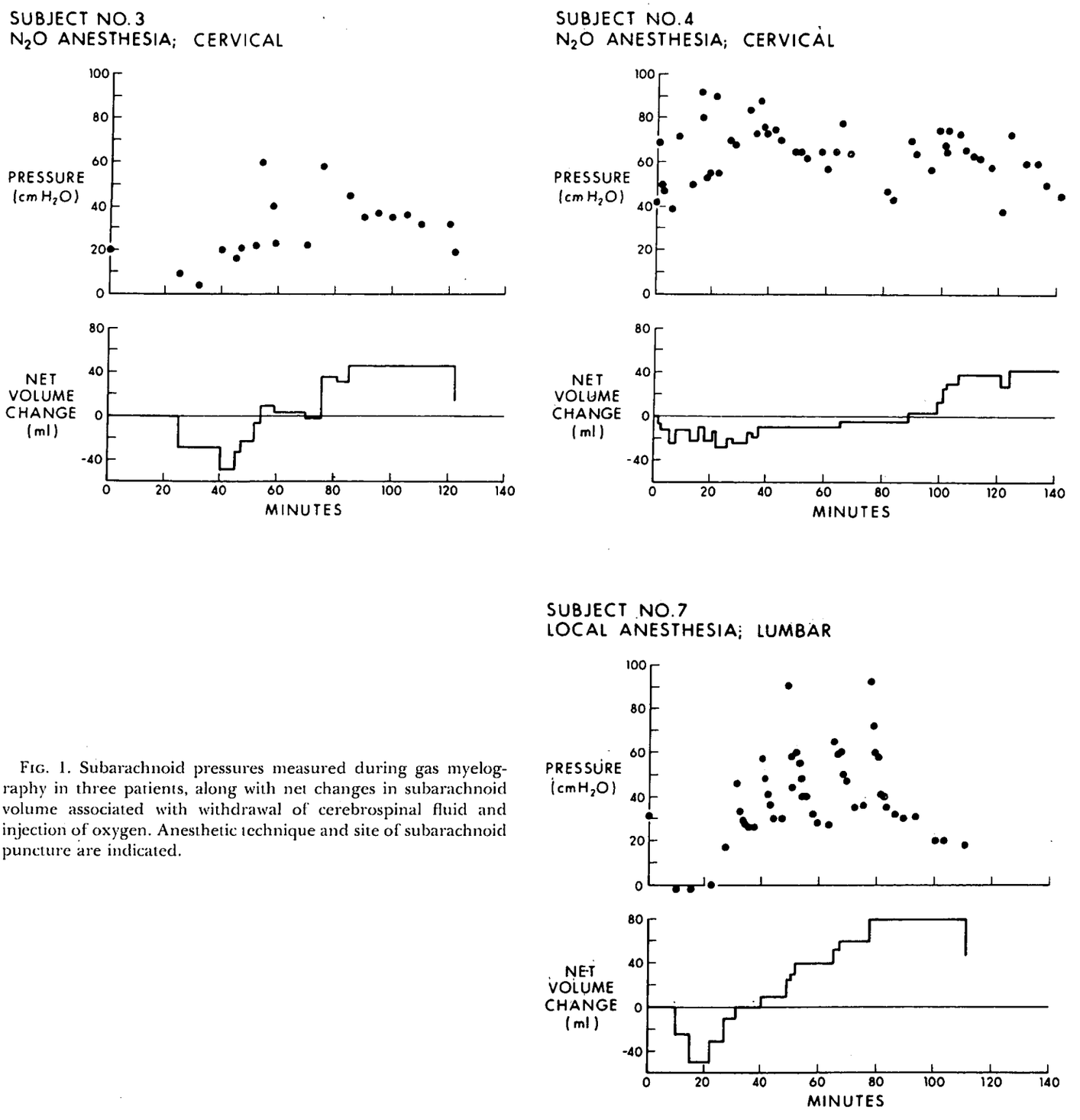

FIG. 1. Subarachnoid pressures measured during gas myelography in three patients, along with net changes in subarachnoid volume associated with withdrawal of cerebrospinal fluid and injection of oxygen. Anesthetic technique and site of subarachnoid puncture are indicated.

the subarachnoid O_2 bubble during gas myelography in man and have found that, despite substantial gas transfer, subarachnoid pressures appear to be determined predominantly by the pattern of injection and volume of O_2 introduced into the subarachnoid space.

METHODS

We studied four men and four women, aged 18–74 years, while they were undergoing gas myelography, six during general and two during local anesthesia. Approximately 60 per cent N_2O formed part of each general anesthetic mixture. O_2 was administered by mask for 5 minutes before induction of anesthesia with thiopental and intubation of the trachea during neuromuscular blockade with succinylcholine. N_2O in O_2 was then administered, 6:3 l/min during the first 15 min, 3:2 l/min for the remainder of the procedure. Two patients were given halothane, while four received morphine and pancuronium to supplement the N_2O. Ventilation was manually controlled in all six patients who received general anesthesia. Those given

TABLE 1. Changes in Subarachnoid Gas Volumes

	Age (Years), Sex	Anesthetic Agents	Site of Puncture	Volume CSF Withdrawn (ml)	Volume O_2 Injected (ml ATPD*)	Time Gas in Subarachnoid Space (Min)
General anesthesia						
Patient 1	57, F	Thiopental N_2O Halothane	Lumbar	30	55	100
Patient 2	58, F	Thiopental N_2O Halothane	Cervical	65	70	115
Patient 3	18, M	Thiopental N_2O–morphine Pancuronium	Cervical	48	94	77
Patient 4	55, F	Thiopental N_2O–morphine Pancuronium	Cervical	85	127	133
Patient 5	42, M	Thiopental N_2O–morphine Pancuronium	Cervical	89	80	50
Patient 6	74, M	Thiopental N_2O–morphine Pancuronium	Cervical	52	54	53
MEAN				62	80	88
SD				23	28	34
Local anesthesia						
Patient 7	56, M	Lidocaine	Lumbar	50	130	89
Patient 8	69, F	Lidocaine	Cervical	108	90	62
MEAN				79	110	76

* Ambient temperature and pressure, dry.
† Body temperature and pressure, saturated.
‡ He concentration measured by gas chromatography.
§ Estimated.

local anesthesia breathed air spontaneously during myelography.

After induction of general anesthesia, the patients were placed in the lateral position for subarachnoid puncture at the cervical (six patients) or lumbar level (two). After opening pressure was measured, 30–108 ml of cerebrospinal fluid (CSF) were withdrawn. Before injection of O_2, the patient was positioned 15–30 degrees head-down to prohibit entry of gas into the cerebral ventricles. The O_2 was introduced in divided portions until the anatomic areas to be visualized radiographically were satisfactorily outlined by the gas.

In three of the patients given N_2O anesthesia, myelography was performed using U.S.P. O_2 as the contrast medium. In the remaining five patients, with each patient's written informed consent, a gas mixture of 98.00 per cent O_2, 1.63 per cent He, and 0.37 per cent N_2 was used. The inertness and relative insolubility of He in CSF made this a safe tracer whose dilution could indicate changes in mass of the subarachnoid gas.

During the myelography, we continuously measured CSF pressure through the indwelling spinal needle used to withdraw CSF and to inject O_2. Zero reference for all CSF pressures was at the hub of the subarachnoid needle. Pressure was measured with a Sanborn strain gauge transducer (Model 268B), a Sanborn carrier preamplifier (Model 350-1100), and a direct-current voltmeter to measure output from the preamplifier. This system was calibrated with a water manometer prior to each study.

Upon completion of myelography, as much of the gas as possible was withdrawn from the subarachnoid space, usually 15–30 ml. Composition of this gas was analyzed in all but one case with a mass spectrometer (Perkin-Elmer MGA 1100A) that provided simultaneous readings of N_2O, O_2, CO_2, N_2, and He concentrations. Water vapor pressure in the bubble was assumed to be 47 torr, which, when divided by barometric pressure, gave an estimate of the fraction of water vapor in the subarachnoid gas. In one patient (Patient 8), only He concentration was measured by gas chromatography. The mass spectrometer was calibrated with gases of known concentration, ranging from zero to 100 per cent; intermediate mixtures were

and Concentrations during Gas Myelography

Final Subarachnoid Gas Composition (Vol Per Cent)							Calculated Final Volume of Gas (ml BTPS†)	Percentage Increase in Volume of Gas	Volume N_2O Added to Bubble (ml BTPS)	Volume O_2 Lost from Bubble (ml BTPS)
N_2O	O_2	N_2	H_2O	CO_2	He	Total				
44.5	42.8	2.5	6.2	3.8	—	99.8	—	—	—	—
54.7	31.6	1.9	6.2	5.8	—	100.2	—	—	—	—
44.5	37.5	5.6	6.2	5.8	—	99.6	—	—	—	—
40.8	40.8	1.2	6.2	9.8	0.82	99.6	252	98	103	27
40.2	37.6	8.9	6.2	5.4	0.70	99.0	187	134	75	11
42.4	39.4	6.5	6.2	4.8	0.75	100.1	116	115	49	9
44.5	38.3	4.4	6.2	5.9	0.76	99.7	185	116	76	16
5.3	3.8	3.0	0.0	2.1	0.06	0.4	68	18	27	10
0.0	80.5	5.0	6.2	7.0	1.39	100.1	155	19	—	8
—	—	—	—	—	1.51‡	—	98	9	—	(6)§
—	—	—	—	—	1.45	—	127	14	—	—

analyzed by the method of Scholander.[2] Maximum expected error in spectrometric measurement of N_2O, O_2, CO_2, and N_2 in our laboratory is 0.05 vol per cent. He concentration should have been accurate to within 0.01 vol per cent; therefore, errors in calculated gas volumes caused by limitations in measuring He concentration should have been less than ±1.4 per cent.

Final volume of gas in the subarachnoid space was calculated from dilution of He. We corrected for He carried away by blood perfusing the spinal cord and for He lost through dissolving in and being carried away by bulk flow of CSF. It was assumed that the mean fraction of He lost into the blood or CSF was midway between the initial and final fractions of He in the gas bubble and that losses of subarachnoid He and O_2 into the blood were proportional to their effective solubilities in blood. The solubility of He in blood at 38 C is 0.0098 ml/ml,[3] which is about one-fifth the effective solubility of O_2 in venous blood already containing 15 vol per cent O_2 and capable of holding an additional 5 vol per cent, or 0.05 ml/ml.

In correcting for loss of He into CSF, we assumed that He is equally soluble in CSF and water and that the CSF volume dissolving He is 150 ml (undoubtedly an overestimation). The rate of production and absorption (bulk flow) of CSF in normal man is 0.40 ml/min.[4] Therefore, the distribution volume for He in CSF would have been as much as 150 ml + 0.40 ml/min × 90 min = 186 ml during a typical myelographic study. Because the solubility of He in water is 0.0097 ml/ml at 38 C,[3] it follows that 186 ml CSF would contain 1.80 ml He when fully saturated at one atmosphere. The volume of dissolved He would increase by a factor of 800/760, according to Henry's law, to 1.90 ml at 800 torr, a realistic value for absolute subarachnoid pressure. To estimate the loss of He through CSF, 1.90 ml was multiplied by the average fraction of He in the gas bubble during myelography.

The following equation describes the dilution of He and loss of He through blood and CSF:

$$(IFHe)(IVG) = (FFHe)(FVG) + (0.5)(IFHe + FFHe)(0.2)(LVO_2) + (0.5)(IFHe + FFHe)(1.90) \quad (1)$$

where IFHe is initial fraction of He, IVG is initial

volume of gas, FFHe is final fraction of He, FVG is final volume of gas, and LVO_2 is lost volume of O_2. The quantity (0.5) (IFHe + FFHe) represents the mean fraction of He in the gas bubble, while the factor (0.2) accounts for the fact that He is about one-fifth as soluble as O_2 in venous blood. Volume of O_2 lost from the subarachnoid bubble was calculated using the equation:

$$LVO_2 = (IFO_2)\,(IVG) - (FFO_2)\,(FVG) \qquad (2)$$

where IFO_2 is initial fraction of O_2 and FFO_2 is final fraction of O_2. Combining equations 1 and 2 and solving for FVG gives the equation used for calculating the final volume of gas in the subarachnoid bubble:

$$FVG = \frac{(IVG)(IFHe - (0.1)(IFHe + FFHe) \times (IFO_2)) - (0.95)(IFHe + FFHe)}{FFHe - (0.1)(IFHe + FFHe)(FFO_2)} \qquad (3)$$

Before carrying out calculations using equations 2 and 3, we multiplied the initial gas volume by the ratio of body to room temperature, each expressed in degrees Kelvin. This resulted in calculated gas volumes expressed under BTPS conditions. The increase in volume owing to saturation with water vapor was accounted for by dilution of He.

Results

We observed wide fluctuation in CSF pressure during gas myelography, whether the procedure was performed under general or local anesthesia. As can be seen in figure 1, pressure fell during withdrawal of CSF, sometimes to values below atmospheric pressure. Pressure usually returned to control levels as the volume of O_2 introduced approximated the volume of CSF removed. Thereafter, additional increments of O_2 frequently produced large elevations in pressure. Peak pressures seemed to be unrelated quantitatively to net changes in volume resulting from withdrawal of CSF or injection of O_2 (fig. 1). The data do not suggest any difference in peak or "average" pressures between patients breathing air and those breathing N_2O:O_2.

During gas myelography performed with general anesthesia using approximately 60 per cent N_2O, volume of the subarachnoid gas bubble increased by 116 ± 18 (SD) per cent. This more than doubling of volume resulted from diffusion of N_2O, CO_2, N_2, and water vapor into the bubble, while a substantially smaller volume of O_2 diffused outward. Increase in temperature also contributed to the increase in volume. The concentration of N_2O in gas withdrawn at the conclusion of myelography (44.5 ± 5.3 per cent) showed that N_2O in the subarachnoid bubble was approximately 80 per cent equilibrated with the inspired N_2O concentration (55.9 ± 2.4 per cent) measured in three of the patients. N_2O in the anesthetic gases contributed substantially (76 ± 27 ml) to the increase in mass of the bubble.

In contrast, when myelography was performed during breathing of air, the mass of gas in the subarachnoid bubble increased by only 14 ± 7 per cent. The volume of CO_2, N_2, and water vapor that diffused into the gas bubble was only slightly greater than the volume of O_2 that diffused outward.

Table 1 gives details of each procedure, including final composition of the subarachnoid gas and calculated changes in volumes of gas in the subarachnoid bubble.

Discussion

The marked increase in subarachnoid pressure during pneumoencephalography with air as contrast medium with N_2O anesthesia has been explained by Saidman and Eger as resulting from the high solubility of N_2O in blood relative to that of N_2.[1] The number of molecules of N_2O transferred to the ventricular gas bubble exceeds the number of N_2 molecules leaving the bubble, and pressure rises. In contrast, we found during myelography with O_2 as the contrast medium that subarachnoid pressures with N_2O anesthesia are not consistently higher than when air is inhaled. The reason for this does not seem to be that loss of O_2 from the subarachnoid bubble compensates for gain of N_2O, because the percentage increase in volume of the bubble was clearly higher when patients breathed N_2O:O_2 than when they breathed air (table 1).

Our finding that there were no consistent differences between subarachnoid pressures in patients breathing N_2O:O_2 and those breathing air may be explained by the fact that the neuroradiologist was primarily concerned with the volume of subarachnoid gas and not with the amount of gas introduced. He injected increments of O_2, monitoring progress with frequent radiographs, until the total volume of gas was just sufficient to outline subarachnoid structures. N_2O entering the subarachnoid gas bubble simply contributed to adequacy of the gas volume for radiologic contrast. Mean volumes of O_2 injected into the subarachnoid space in the two groups of patients, 80 ml during N_2O and 110 ml during local anesthesia, support this interpretation; the difference, however, is not statistically significant (table 1).

During subarachnoid injection of increments of O_2 during local anesthesia, patients often complained of severe headache, which the neuroradiologist used as a signal to stop injection. Complaint of headache was usually accompanied by a transient spike in subarachnoid pressure (fig. 1). When the pressure returned toward baseline, the headache subsided, and

the neuroradiologist felt free to inject additional O_2. 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 H_2O. 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 N_2O. Subarachnoid pressures should probably also be measured continuously during gas myelography with local anesthesia, because this would make possible, through controlled injection of O_2, the avoidance of subarachnoid pressures that produce pain.

Not only N_2O but also N_2 and CO_2 diffuse into the subarachnoid bubble during gas myelography. The CO_2 concentration of 7.0 per cent (P_{CO_2} 53 torr) in the subarachnoid gas of Patient 7, who was spontaneously breathing air during local anesthesia, was within the range of normal P_{CO_2} in the CSF of normocapnic man.[5] Concentrations of CO_2 in gas removed from the subarachnoid space of five of the six patients anesthetized with N_2O, ranging from 3.8 to 5.8 per cent (P_{CO_2} 29 to 44 torr), would be expected during pulmonary hyperventilation. The CO_2 concentration of 9.8 per cent (P_{CO_2} 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_{CO_2} was 49 torr, pH_a 7.33.

Although we have shown substantial transfer of gas from CSF or blood into the subarachnoid bubble during gas myelography with N_2O anesthesia, major changes in subarachnoid pressure appear to be related to the pattern of injection and volume of O_2 introduced into the subarachnoid space by the neuroradiologist.

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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 identification[1] and catheter placement[2] 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.

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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.