

3. Greenblatt GM, Denson JS: Needle nerve stimulator-locator. *Anesth Analg* 41:599-602, 1967
4. Wright BD: A new use for the Block-Aid Monitor®. *ANESTHESIOLOGY* 30:236-237, 1969
5. Montgomery SJ, Raj P, Nettles D, Jenkins MT: The use of the nerve stimulator with standard unsheathed needles in nerve blockade. *Anesth Analg* 52:827-831, 1973
6. Chapman GM: Regional nerve block with the aid of a nerve stimulator. *Anaesthesia* 27:185-193, 1972
7. Magora F, Rozin R, Ben-Menachem Y, Magora A: Obturator nerve block: An evaluation of technique. *Br J Anaesth* 41:695-698, 1969
8. Smith BL: *Forum. Anaesthesia* 31:185-193, 1976
9. Koons RA: The use of the Block-Aid Monitor® and plastic intravenous cannulas for nerve blocks. *ANESTHESIOLOGY* 31:290-291, 1969
10. Winnie AP: An "immobile needle" for nerve blocks. *ANESTHESIOLOGY* 31:577-578, 1969
11. Bosomworth PP, Egbert L, Hamelberg W: Block of the brachial plexus in the axilla: Its value and complications. *Ann Surg* 154: 911-914, 1961
12. Lofstrom B, Wennberg A, Widen L: Late disturbances in nerve function after block with local anesthetic agents. An electro-neurographic study. *Acta Anaesthesiol Scand* 10:111-115, 1966
13. McClain DA, Finucane BT: Interscalene approach to the brachial plexus. Paresthesia versus nerve stimulator (Abstract). *Reg Anesth* 8:39, 1983
14. Galindo A: Electrical localization of peripheral nerves: Instrumentation and clinical experience (Abstract). *Reg Anesth* 8: 49-50, 1983
15. Ford DJ, Pither CE, Raj PP: Electrical characteristics of peripheral nerve stimulators: Implications for nerve localization. *Reg Anesth* 9:73-77, 1984
16. Pither CE, Raj PP, Ford DJ: The use of peripheral nerve stimulators for regional anesthesia. *Reg Anesth* 10:49-58, 1985
17. Thompson GE, Rorie DL: Functional anatomy of the brachial plexus sheaths. *ANESTHESIOLOGY* 59:117-122, 1983

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Precordial Ultrasonic Monitoring during Cesarean Delivery

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Venous air embolism occurs during a wide variety of surgical procedures. Recently, a massive air embolism necessitating cardiopulmonary resuscitation was reported during cesarean delivery.¹ The purpose of this study was to investigate the complaints of chest pain and dyspnea frequently reported by parturients during cesarean delivery and to find a possible cause. Such chest pain is ascribed to many etiologies. In our experience, it is dismissed as secondary to surgical traction on the peritoneum or intraperitoneal structures (*e.g.*, the uterus). We believe that this chest pain could be secondary to venous air embolism;

specifically, air entrained in venous sinuses opened during hysterotomy and subsequent delivery of the fetus and placenta. In this study, we used a precordial ultrasonic Doppler monitor to detect intracardiac air.²

METHODS AND MATERIALS

Eighty-nine ASA Class I patients who sequentially presented for elective cesarean delivery and had indicated a preference for either epidural or spinal anesthesia were selected for study. Informed consent was not obtained, nor judged to be necessary, since the ultrasonic Doppler is a common intraoperative monitor.

All parturients were hydrated according to a protocol of administering 1½ to 2 l of lactated Ringer's solution during the 15-30 min immediately before the induction of regional anesthesia.³ A Travenol solution administration set (2 C0649) with a 50 ml in-line hand pressure pump (air trap) was used to ensure that no air was accidentally infused intravascularly. Spinal anesthesia was induced in the right lateral decubitus position using a 26-gauge needle and hyperbaric 5% lidocaine or a 1% tetracaine/10% procaine mixture of local anesthetic.⁴ Alternatively, continuous lumbar epidural anesthesia was induced in the following manner: after identifying the epidural space at L₂/L₃ or L₃/L₄ with a 17-gauge Weiss needle, a test dose of 3 ml of 2% lidocaine with 1:200,000 epinephrine was administered, and a 23-gauge catheter

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was inserted. Incremental 3 ml doses of 2% lidocaine with 1:200,000 epinephrine were given *via* the catheter to produce surgical anesthesia. Left uterine displacement was instituted once the anesthesia was initiated. Maternal arterial blood pressure was monitored by cuff or Dinamap®. Maternal systolic blood pressure was kept above 100 mmHg with 5–10 mg of ephedrine. There were no episodes of hypotension after the skin incision requiring vasopressor therapy. Routine administration of oxygen (8–10 l/min) by face mask was given until delivery of the infant. All patients had a T₂/T₄ dermatome level of sensory anesthesia to pinprick before the start of the surgical procedure. All patients asserted to the anesthesiologist that they were “comfortable” upon direct questioning. No further questions regarding patient comfort were directed by the anesthesiologist after the skin incision was made.

A Parks Medical Electronics Model 915C Dual Frequency Ultrasonic Doppler was used to generate an audible Doppler signal. A 2 MHz transducer was placed parasternally over the fourth right intercostal space. Position of the transducer was confirmed after a bolus infusion of crystalloid through the peripheral intravenous infusion⁵ created a change in the audible signal. The patients were informed of the use of this “electronic stethoscope.” The audible Doppler was directed away from the patient and the operating team and was monitored by an observer (one of the authors). The Doppler signal volume was adjusted so that it was easily audible only to the monitoring observer. Patients’ unsolicited complaints of chest pain and/or dyspnea were recorded, as were the occurrence and timing of Doppler changes. A positive Doppler change was defined as the occurrence of a “broken-up, roaring” change in the rhythmic “swishing” sound of the normal precordial Doppler signal, which lasted for at least 15 s. Also noted were whether the uterus was exteriorized for hysterotomy repair and the occurrence of sustained maternal systemic arterial hypotension (30 or more mmHg lower than preoperative values) or dysrhythmias.

An iv oxytocin infusion (20 units in 1 liter of lactated Ringer’s solution) was begun immediately after the delivery of the neonate. Any iv injection of medication (*e.g.*, antibiotics or oxytocin) was made proximal to the air trap in the iv infusion to avoid the possibility of intravascular turbulence. Epidural fentanyl (50 µg in 5–10 ml of normal saline) was given to all patients with an epidural catheter sometime after closure of the hysterotomy.

Results were analyzed with a Chi-square test with a Yates correction when applicable. A *P* value of less than 0.05 was considered statistically significant.

RESULTS

A positive Doppler change occurred in 46 of 89 women (52%) during the procedure: 74% at the time of hyster-

TABLE 1. A Statistical Association Exists Between Doppler Change and Chest Pain

	Chest Pain	
	Yes	No
Doppler Change		
Yes	24	22
No	2	41
	<i>P</i> < 0.01	

otomy; 2% with the delivery of the baby; 13% with the delivery of the placenta; and 11% during the hysterotomy repair. The positive Doppler changes in the audible signal were mostly brief in duration; some were recurrent. All Doppler signals had reverted to normal by the end of the surgical procedure.

Chest pain was spontaneously reported in 24 of the 46 women with a previous Doppler change, but only two of the 43 women without a Doppler change spontaneously reported chest pain. The relationship between unsolicited complaints of chest pain and a positive Doppler change was statistically significant (table 1). In the 24 women with positive Doppler changes and chest pain, the discomfort occurred within 1–10 min after the initial Doppler change. The pain was described as heavy in quality, retrosternal, and non-radiating. All episodes of chest pain were transient (less than 5–10 min in duration), and none lasted into the postoperative period.

When the results were evaluated according to the type of anesthesia given (spinal *versus* continuous lumbar epidural), there was still a significant association between the occurrence of chest pain and an occurrence of Doppler change.

Nine of the 46 women with a Doppler change (20%) complained of dyspnea. Only one of the 43 women without Doppler change complained of dyspnea. The relationship between the occurrence of dyspnea and a previous Doppler change was statistically significant (table 2). The complaints of dyspnea were transient and, in all instances, disappeared before the surgery was completed.

Eight of the 26 women with chest pain complained of dyspnea. The onset of dyspnea was coincidental with the onset of chest pain. Only two of the 63 women without

TABLE 2. A Statistical Association Exists Between Dyspnea and Doppler Change

	Dyspnea	
	Yes	No
Doppler Change		
Yes	9	37
No	1	42
	<i>P</i> < 0.02	

TABLE 3. A Statistical Association Exists Between Dyspnea and Chest Pain

	Dyspnea	
	Yes	No
Chest Pain		
Yes	8	18
No	2	61
	$P < 0.01$	

chest pain complained of dyspnea. This association was statistically significant (table 3).

There were no associated changes in vital signs during these episodes of chest pain and/or dyspnea. There was no statistical association between the occurrence of chest pain or dyspnea and the type of anesthesia. There was no statistical association between the occurrence of chest pain, dyspnea, and whether or not the uterus was exteriorized for hysterotomy repair. Finally, there was no statistical association between the choice of observer (AMM as compared to the other authors) and the occurrence of Doppler changes.

DISCUSSION

We found that Doppler changes occur in about 50% of all patients undergoing elective cesarean delivery under regional anesthesia. A Doppler change usually first occurs at the time of hysterotomy, during incision through the lower cervical segment, possibly opening venous sinuses. The occurrence of chest pain is associated with Doppler changes. Chest pain is not associated with the type of anesthesia given, nor with surgical exteriorization of the uterus for hysterotomy repair.

Precordial Doppler signal change is evidence of an embolic event. The most likely emboli to occur during cesarean delivery are from amniotic debris or air. There are both similarities and differences between air and amniotic emboli in both clinical presentations and ultimate pathophysiologic courses.

Amniotic fluid embolus classically presents with dyspnea and cyanosis, hypotension, and coma.⁶ Afflicted parturients do not often initially complain of chest pain.^{6,7} Diagnosis of amniotic fluid embolism is usually one of exclusion, and is often made post-mortem. Perhaps small, subcritical quantities of amniotic debris are being liberated into the maternal circulation during cesarean delivery. Aspiration of amniotic debris from the maternal pulmonary circulation in completely asymptomatic parturients was recently reported.⁷

A precordial ultrasonic Doppler monitor is capable of detecting as little as 0.1 ml of intracardiac air.² Air emboli, especially small ones, cause complaints of dyspnea and

chest pain. Cardiac dysrhythmias, cyanosis, and hypotension are sometimes seen. Most of the experience with venous air embolism has occurred during general anesthesia for neurosurgery. Complaints of chest pain and dyspnea associated with small volumes of intracardiac air present in the parturient. These unsolicited complaints correlate with previous Doppler changes. These episodes are of brief duration, and are not associated, in this study, with significant cardiorespiratory changes. Yet, massive venous air embolism in obstetrics has occurred in both cesarean and vaginal deliveries.¹ A precordial Doppler was not used on parturients having vaginal deliveries, as we thought it more difficult to control the state of hydration and the parturients' positioning on the delivery table. More evidence of venous air embolism, including more detailed and quantitative ultrasonic or echocardiographic measurements of air volume, invasive and non-invasive monitoring of cardiac output during cesarean delivery, and measurement of end-tidal gases is needed to further investigate our hypothesis. Direct confirmation of intracardiac air with a central venous catheter should also be attempted.

Further studies will need to include better attempts to quantify the severity and timing of the chest pain, dyspnea, Doppler signal, and hemodynamic data. Hard-copy data can then be collected, recording the electrocardiograph, systemic arterial, central venous and pulmonary artery pressures, and end-tidal gases. Only the occurrence of the already defined change in arterial blood pressure or heart rate was noted in our study. Smaller changes, perhaps consistent with smaller volumes of intracardiac air, might have been missed. The hemodynamic data in our study were not recorded any more frequently than on the anesthesia records. Similarly, only the occurrence of a Doppler change, chest pain, or dyspnea was recorded. The duration of and the intervals between such events were not precisely noted.

We conclude that venous air embolism may commonly occur during cesarean delivery. Venous air embolism may or may not present with complaints of chest pain and/or dyspnea, perhaps depending on the venous air volume. In this study, complaints of chest pain and/or dyspnea by parturients are highly associated with ultrasonic Doppler evidence of venous air embolism.

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REFERENCES

1. Younker D, Rodriguez V, Kavanaugh J: Massive air embolism during cesarean section. *ANESTHESIOLOGY* 65:77-79, 1986
2. Michenfelder JD, Riller RH, Gronert GA: Evaluation of an ultrasonic device (Doppler) for diagnosis of air embolism. *ANESTHESIOLOGY* 36:164-197, 1972

3. Datta S: Cesarean delivery, Manual of Obstetric Anesthesia. Edited by Ostheimer GW. New York, Churchill Livingstone, 1984, pp 221-238
4. Chantigian RC, Datta S, Burger GA, Naulty JS, Lambert D, Ostheimer GW: Anesthesia for cesarean delivery utilizing spinal anesthesia: Tetracaine versus tetracaine and procaine. Reg Anesth 9:195-199, 1984
5. Naulty JS, Ostheimer GW, Datta S, Knapp R, Weiss JB: Incidence

of venous air embolism during epidural catheter insertion. ANESTHESIOLOGY 57:410-412, 1982

6. Inglis MS, Morgan M: Management of the patient with amniotic fluid embolism, Obstetric Analgesia and Anaesthesia II, Clinics in Anaesthesiology. Edited by Ostheimer GW. London, WB Saunders, 1986, pp 359-371
7. Mulder JI: Amniotic fluid embolism: an overview and case report (discussion). Am J Obstet Gynecol 152:430-435, 1985

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Epidural Ketamine or Morphine for Postoperative Analgesia

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Intrathecal and epidural narcotics have been widely used since 1979 to relieve pain and provide postoperative analgesia.^{1,2} However, their use has been tempered by the potential of respiratory depression.^{1,2} Thus, other substances have been tried that have the advantages of opioids but not their drawbacks. Ketamine has analgesic properties which apparently are mediated by various pathways and, especially, opiate receptors.³⁻⁷ Yet, Fratta *et al.*⁸ have disproven fixation to opiate receptors. Still, Ketamine analgesia is antagonised by naloxone, an opioid antagonist.⁹ Ahuja¹⁰ showed that intrathecally administered ketamine could produce analgesia, but its effect is of short duration and inconsistent. As iv ketamine administered at the usual doses does not produce respiratory depression, Ahuja¹⁰ thought it would be the same by intrathecal route. Animal experiments¹¹ showed that ketamine with preservative had probably no neurotoxic effect, despite what had been presented in two studies.^{10,12} However, the neurotoxic effect observed are questionable

(*i.e.*, very high doses in one case¹⁰ and difficulties for lumbar puncture in the other case¹²). Moreover, according to five clinical observations,¹³⁻¹⁷ ketamine should, therefore, be a good choice for epidural analgesia. In the above mentioned studies, epidural ketamine seemed to be a potent and safe method for postoperative analgesia. However, this method had never been compared to a technique using epidural opioids. This is, therefore, the purpose of this work where morphine was chosen as the reference opiate.

PATIENTS AND METHODS

This study was conducted with the informed consent of 20 patients and with the approval of the Hospital Ethic Committee. Patients had undergone orthopedic surgery (table 1). The operative anesthesia was provided by a lumbar epidural anesthesia (catheter inserted at the L₃₋₄ interspace) with or without supplementation by inhalation of enflurane. The patients were allocated by computerized randomisation into two groups of ten: one "ketamine" group, and one "morphine" group. This study was a single blind study.

Pain was assessed according to a five-step scale (equivalent to a visual analog scale¹⁸), as follows: 0 = no pain; 1 = very slight pain, simple bother; 2 = slight pain that can be temporarily forgotten; 3 = permanent and tolerable pain that cannot be forgotten; 4 = marked pain that leads the patient to request an analgesia; and 5 = intolerable pain with screams and restlessness.

This five-step scale was explained preoperatively to the patient, and pain was measured by asking the patient (when the patient was asleep, pain was estimated at the "0" level).

Monitoring was under the direct responsibility of an anesthesiologist in an intensive care unit. Analgesia was

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