muscular origin and should be considered early in the course of GBS when other modalities are not effective.

References

1. Oxman T, Denson DD: Antidepressants and adjunctive psychotropic drugs, Practical Management of Pain. Edited by Prithvl RP. Chicago, Year Book Medical Publishers, Inc., 1986, pp 528–538
2. Rosenfield B, Burel C, Hanley D: Epidural morphine treatment of pain in Guillain-Barré syndrome. Arch Neurol 43:1194–1196, 1986
3. Ropper A, Shahani B: Pain in the Guillain-Barré syndrome. Arch Neurol 41:511–514, 1984
4. Haymaker W, Kernohan J: Landry-Guillain-Barré syndrome: Fifty fatal cases and a critique of the literature. Medicine 28:59–141, 1949
5. Henschel E: The Guillain-Barré syndrome. A personal experience. ANESTHESIOLOGY 47:228–231, 1977
6. Melzac R, Wall PD: Pain mechanisms: A new theory. Science 150: 971–979, 1965

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Beneficial Effect of Delivery in a Patient with Adult Respiratory Distress Syndrome

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The management of adult respiratory distress syndrome (ARDS) often requires support of respiratory and cardiovascular function and is particularly difficult in pregnant patients. ARDS has been reported to occur in pregnancy secondary to many different etiologies.[1–15] However, there is limited information in these studies concerning the effect of delivery on gas exchange in ARDS. We report a patient in her third trimester who developed ARDS requiring mechanical ventilation and PEEP. Following delivery her pulmonary status improved markedly.

Case Report

A 49-kg, 160-cm, 19-yr-old woman G1PO was hospitalized with a complaint of a 2-day history of abdominal pain. Her 31-week pregnancy had been uneventful except for hyperemesis in the first trimester. She denied any previous medical problems. Her vital signs were temperature 38.5° C, blood pressure 93/41 mmHg, pulse 102 beats/min, and respiratory rate 16 breaths/min. Physical examination revealed an enlarged uterus consistent with 31-week gestation and right lower quadrant tenderness. Uterine contractions were occurring every 2–5 min. Laboratory studies included 19,900 leukocyte/mm^3 with 64 polymorphonuclear cells, 32 bands, and 4 lymphocytes. Urinalysis was normal. A magnesium sulfate infusion was begun to treat preterm labor at 3:40 A.M. of her first hospital day. No sympathomimetic drugs were administered.

At 5 A.M. on the first hospital day, the patient was taken to the operating room with a diagnosis of acute appendicitis. A spinal anesthetic was performed at the L3–4 interspace with the patient in the left lateral decubitus position; 1.2 ml of bupivacaine 0.75% in 8.5% dextrose with 0.2 ml of epinephrine 1:1,000 was administered. Exploratory laparotomy revealed an erythematous appendix and no other abnormalities. Intraoperatively, the only additional drug the patient received was 1.25 mg droperidol for nausea. She vomited during the laparotomy but did not develop clinical signs of aspiration. She was discharged from the peri-anesthetic care unit (PACU) to her room at 8:30 A.M. with a respiratory rate of 26 breaths/min. At 9:40 P.M. on the second hospital day, 39 h after operation, she developed tachypnea of 40–50 breaths/min. Arterial blood gases (ABG) while breathing room air were *p*H 7.47, Pa_{CO_2} 33 mmHg, and Pa_{O_2} 42 mmHg (table 1). Magnesium sulfate was discontinued and she was transferred to the surgical intensive care unit (SICU). Upon admission to the SICU, ABG while breathing 100% O_2 *via* a nonrebreathing mask were *p*H 7.44, Pa_{CO_2} 34 mmHg, and Pa_{O_2} 83 mmHg. Continuous positive airway pressure (CPAP) therapy *via* a mask failed to improve her ventilatory status sufficiently and her trachea was intubated at 4 A.M. on the third hospital day, 46 h after operation. Total fluid intake from hospital admission to intubation (50 h) was 6,010 ml and total output was 4,835 ml. Ventilator settings included tidal volume of 600 ml with an intermittent mandatory ventilation (IMV) rate of 10. Radial and pulmonary artery catheters were inserted. Initial hemodynamic measurement included pulmonary capillary wedge pressure (PCWP) of 15 mmHg and cardiac index of 4.59 $l \cdot min^{-1} \cdot m^{-2}$ (table 2). A chest x-ray revealed extensive bilateral pulmonary edema. Fetal monitoring demonstrated a heart rate of 140 beats/min, good variability, and occasional contractions. PEEP was increased to 10 cmH_2O to decrease FI_{O_2} to 0.4. These ventilator settings resulted in Pa_{O_2} 60 mmHg, Sa_{O_2} 92.4%, cardiac index 4.63 $l \cdot min^{-1} \cdot m^{-2}$, and Q_s/Q_t 28%. Five hours prior to delivery, she received 50 mg of meperidine iv for analgesia. Spontaneous labor progressed to unassisted vaginal delivery at 8:36 P.M. on the third hospital day, 62 h after operation. Peripartum arterial blood gas (table 1) and hemodynamic data (table 2) demonstrated the impact of delivery on the patient's ARDS. The patient's gas exchange was stable for several hours prior to delivery. At the time of delivery arterial desaturation was noted by pulse oximetry and FI_{O_2} was increased to 1.0. Blood gases and hemodynamic data collected during delivery revealed a 47% increase in oxygen consumption ($\dot{V}_{O_2}I$) but only a 17% increase in cardiac index. Consequently, $S\bar{v}_{O_2}$ decreased to 54.1% and oxygen extraction ($Sa_{O_2} - S\bar{v}_{O_2}/Sa_{O_2}$) increased to 0.37. Following delivery gas exchange returned to predelivery levels within 2 h. The patient's ventilatory status improved sufficiently to allow tracheal extubation 18 h postpartum. Maternal fluid intake from delivery to extubation was

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TABLE 1. Selected Arterial Blood Gases during the Peripartum Period

Hospital Day	Time	FIO_2	PEEP (cmH_2O)	pH	$PaCO_2$ (mmHg)	PaO_2 (mmHg)	SaO_2 (%)
2	2212	0.21	—	7.47	33	42	—
3	0035	1.0	—	7.44	34	83	—
	0620	0.55	5	7.43	38	67	91.6
	0900	0.55	7.5	7.50	32	56	91.6
	1017	0.65	10	7.39	42	139	97.5
	1400	0.40	10	7.47	34	60	92.4
	2023	0.40	10	7.41	39	71	93.7
Delivery	2036	1.0	10	7.45	34	51	86.1
	2047	1.0	10	7.40	36	62	91.4
	2150	0.40	10	7.40	38	66	—
	2220	0.40	10	7.39	39	62	90.9
	2330	0.40	10	7.40	39	80	97.0
4	0300	0.40	10	7.38	37	107	97.4
	0615	0.40	5	7.59	22	135	97.4

2,596 ml and measured output was 1,046 ml. She was discharged from the hospital 4 days later. Figure 1 shows intrapulmonary shunt and oxygen consumption index during the peripartum period. The infant weighed 1,500 g and at 1, 5, and 10 min had Apgar scores of 2, 7, and 8, respectively. Umbilical cord gases were as follows: arterial *p*H 7.24, Pa_{CO_2} 59 mmHg, Pa_{O_2} 15 mmHg and venous *p*H 7.25, $P\bar{v}_{CO_2}$ 55 mmHg, and $P\bar{v}_{O_2}$ 16 mmHg. The baby initially required tracheal intubation for poor inspiratory effort, which improved sufficiently to permit extubation of the trachea on the same day.

DISCUSSION

ARDS is defined as a group of findings that include refractory hypoxemia, diffuse infiltrates on chest x-ray, PCWP < 20 mmHg, and no other obvious cause for these changes. Our patient met these criteria upon admission to the SICU. However, the etiology of ARDS in our patient was not clear. Because the patient vomited during the operation, aspiration is a possibility but not likely. Our patient was alert when vomiting occurred and there was no coughing. Pulse oximetry in the operating room and the PACU did not reveal hypoxemia. Furthermore, her respiratory distress was not noted until a day and a half later. Infection[12,13] and the administration of magnesium sulfate[9] for tocolysis have both been associated with the development of pulmonary edema and ARDS in pregnant patients. Hatjis and Swain reviewed the charts of 527 patients receiving tocolysis (ritodrine or magnesium sulfate) and found that 52 had evidence of infection.[16] The incidence of pulmonary edema was 21% in the group with infection compared with only 1% in the rest of the patients.[16] Thus, there is an association between the development of pulmonary edema and maternal infection in patients receiving treatment for preterm labor. Both of these factors, infection and administration of magnesium sulfate, were present in our patient, placing her at high risk for the development of pulmonary edema. Our patient had evidence of systemic infection (fever and leukocytosis with a left shift). However, neither laparotomy nor cultures of blood, sputum, or peritoneal fluid revealed a source of infection.

Although rarely addressed in the literature, a key question in the care of the critically ill pregnant patient

TABLE 2. Hemodynamics during Peripartum Period

Hospital Day	Time	MAP (mmHg)	PA-M (mmHg)	PCWP (mmHg)	CVP (mmHg)	CI ($l \cdot min^{-1} \cdot m^{-2}$)	SvO_2 (%)	Q_s/Q_t (%)	$\dot{V}O_2I$ ($ml\ O_2 \cdot min^{-1} \cdot m^{-2}$)	O_2ER
3	0900	88	22	15	12	4.59	72.0	28	142	0.25
	1400	73	23	15	16	4.63	65.9	28	155	0.29
	2023	88	26	20	18	4.86	67.3	26	156	0.29
Delivery	2036	107	22	30	15	5.39	54.1	47	209	0.37
	2047	86	19	15	12	4.51	65.6	46	147	0.28
	2220	86	24	13	8	3.68	60.3	26	146	0.34
	2330	88	28	25	9	4.09	70.2	21	127	0.28
4	0300	90	33	23	12	7.83	83	8	175	0.16
	0615	95	33	25	10	4.99	82.2	13	125	0.17

MAP = mean arterial pressure; PA-M = mean pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; CVP = central venous pressure; CI = cardiac index; $S\bar{v}_{O_2}$ = mixed venous oxygen saturation; Q_s/Q_t = intrapulmonary shunt; $\bar{V}_{O_2}I$ = oxygen consumption index; O_2ER = oxygen extraction ratio $\frac{Sa_{O_2} - S\bar{v}_{O_2}}{Sa_{O_2}}$.

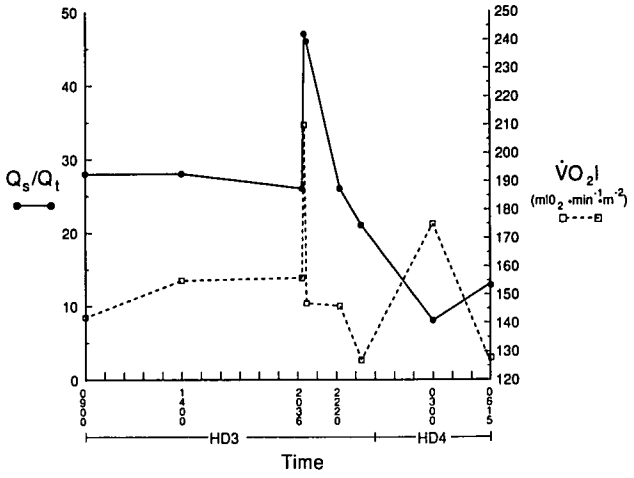

FIG. 1. Intrapulmonary shunt (Q_s/Q_t) and oxygen consumption index ($\dot{V}_{O_2}I$) during peripartum period. $\dot{V}_{O_2}I = CI \times [(SaO_2 - SvO_2) \times Hgb \times 1.39]$.[23] Maximal shunt and oxygen consumption occurred at partuition, which occurred at 2036 h on hospital day (HD) 3.

is whether termination of pregnancy might substantially improve the mother's condition. The hypoxemia in ARDS is at least partly due to a decrease in functional residual capacity (FRC) and an increase in the number of lung units that are perfused but not ventilated.[17] During the third trimester of pregnancy, FRC is reduced an average of 18%[18] due to an equal and proportional decrease in its two components; the expiratory reserve volume (ERV) and the residual volume (RV).[19] Thus, hypoxemia in our patient may have been due to the combined decrease in FRC due to both ARDS and pregnancy. Because FRC increases postpartum,[20] we believed that termination of pregnancy would result in improvement of gas exchange in our patient by reversing this component of her hypoxemia.

One of our concerns with withdrawal of tocolytic therapy was whether labor and delivery would increase oxygen demand above our patient's ability to compensate. Increases in oxygen needs should be met by increases in cardiac output, a determinant of oxygen delivery. Failure of oxygen delivery to increase sufficiently to meet oxygen needs may result in decreased mixed venous oxygen saturation ($S\bar{v}_{O_2}$), increased oxygen extraction ratio (O_2ER), and tissue hypoxemia. Pregnancy[18] and ARDS are associated with increased oxygen consumption. Labor and delivery result in additional increases in oxygen consumption; uterine contractions are associated with a 60% increase.[21] These increases in oxygen demand induced by labor and delivery are normally met, at least partially, by increases in cardiac output.[22] Our patient increased her oxygen consumption by 47% at delivery while her cardiac output increased by only 17%. This relatively greater increase in oxygen demand compared with oxygen delivery resulted in a dramatic increase in the O_2ER of 48% at delivery and, along with her increased Q_s/Q_t, contributed to her decreased Pa_{O_2}.

The direction and magnitude of the hemodynamic changes we observed during labor and delivery in our patient were consistent with those reported in partuition not complicated by ARDS.[18,21,22] However, in the presence of pulmonary disease, these changes produced considerable effects on Pa_{O_2}.

Our data document the hemodynamic changes and the increased oxygen demand of delivery as well as the beneficial effect of emptying the uterus on pulmonary function in a patient with ARDS.

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REFERENCES

1. Nimrod CA, Beresford P, Frais M, Belenkie I, Tyberg J, Fremit A, Smith E: Hemodynamic observations on pulmonary edema associated with a β-mimetic agent. J Reprod Med 29:341–344, 1984
2. Jacobs MM, Knight AB, Arias F: Maternal pulmonary edema resulting from betamimetic and glucocorticoid therapy. Obstet Gynecol 56:56–59, 1980
3. Russi EW, Spaetling L, Gmur J, Schneider H: High permeability pulmonary edema (ARDS) during tocolytic therapy: A case report. J Perinat Med 16:45–49, 1988
4. Stubblefield PG: Pulmonary edema occurring after therapy with dexamethasone and terbutaline for premature labor: A case report. Am J Obstet Gynecol 132:341–342, 1978
5. Benedetti TJ, Hargrove JC, Rosene KA: Maternal pulmonary edema during premature labor inhibition. Obstet Gynecol 59(suppl):33–37, 1982
6. Bowen RE, Dedhia HV, Beatty J, Schiebel F, Koss W, Granados J: ARDS associated with the use of sympathomimetics and glucocorticoids for the treatment of premature labor. Crit Care Med 11:671–673, 1983
7. MacLennan FM, Thomson MAR, Rankin R, Terry PB, Adey GD: Fatal pulmonary oedema associated with the use of ritodrine in pregnancy: Case report. Br J Obstet Gynaecol 92:703–705, 1985
8. Davies AE, Robertson MJS: Pulmonary oedema after the administration of intravenous salbutamol and ergometrine: Case report. Br J Obstet Gynaecol 87:539–541, 1980
9. Elliot JP: Magnesium sulfate as a tocolytic agent. Am J Obstet Gynecol 147:277–283, 1983
10. Andersen HF, Lynch JP, Johnson TRB: Adult respiratory distress syndrome in obstetrics and gynecology. Obstet Gynecol 55:291–295, 1980
11. Burton WN, Vender J, Shapiro BA: Adult respiratory distress syndrome after placidyl abuse. Crit Care Med 8:48–49, 1980
12. Boucher M, Yonekura ML, Wallace RJ, Phelan JP: Adult respiratory distress syndrome: A rare manifestation of listeria monocytogenes infection in pregnancy. Am J Obstet Gynecol 149: 686–688, 1984
13. Cunningham FG, Leveno KJ, Hankins GDV, Whalley PJ: Respiratory insufficiency associated with pyelonephritis during pregnancy. Obstet Gynecol 63:121–125, 1984
14. Keefer JR, Strauss RG, Civetta JM, Burke T: Noncardiogenic pulmonary edema and invasive cardiovascular monitoring. Obstet Gynecol 58:46–51, 1981

15. Benedetti TJ, Kates R, Williams V: Hemodynamic observations in severe preeclampsia complicated by pulmonary edema. Am J Obstet Gynecol 152:330–334, 1985

16. Hatjis CG, Swain M: Systemic tocolysis for premature labor is associated with an increased incidence of pulmonary edema in the presence of maternal infection. Am J Obstet Gynecol 159: 723–728, 1988

17. Dantzker DR, Brook CH, DeHart P, Lynch JP, Weg JG: Gas exchange in adult respiratory distress syndrome and the effect of positive end-expiratory pressure. Am Rev Respir Dis 120:1039–1052, 1979

18. Cugell DW, Frank NR, Gaensler EA, Badger TL: Pulmonary function in pregnancy. I. Serial observations in normal women. Am Rev Tuberc 67:568–597, 1953

19. Cheek TG, Gutsche BB: Maternal physiologic alterations during pregnancy, Anesthesia for Obstetrics. Edited by Shnider SM, Levinson C. Baltimore, Williams & Wilkins, 1987, pp 3–13

20. Knuttgen HG, Emerson K: Physiological response to pregnancy at rest and during exercise. J Appl Physiol 36:549–553, 1974

21. Hagerdal M, Morgan CW, Sumner AE, Gutsche BB: Minute ventilation and oxygen consumption during labor with epidural analgesia. ANESTHESIOLOGY 59:425–427, 1983

22. Ueland K, Hansen JM: Maternal cardiovascular dynamics. III. Labor and delivery under local and caudal analgesia. Am J Obstet Gynecol 103:8–18, 1969

23. Shoemaker WC: Physiologic monitoring of the critically ill patient, Textbook of Critical Care Medicine. Edited by Shoemaker WC, Ayres S, Grenvik A, Holbrook PR, Thompson WL. Philadelphia, Saunders, 1989, pp 145–159

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Intraoperative Diagnosis of Acute Subarachnoid Hemorrhage Using Continuous Pressure Monitoring *via* a Lumbar Subarachnoid Catheter

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Lumbar cerebrospinal fluid (CSF) drainage is often helpful in reducing intracranial volume during the surgical management of intracranial aneurysms, thereby decreasing the need for brain retraction and improving surgical exposure.[1,2] At our institution the surgeon often requests that a catheter be inserted in the lumbar subarachnoid space to withdraw CSF intraoperatively. It is our routine practice to continuously monitor the lumbar CSF pressure (LCSFP), usually a reflection of intracranial pressure (ICP).[3]

We report a case of a patient about to undergo clipping of a middle cerebral artery aneurysm, in whom aneurysmal rupture occurred shortly after induction of general anesthesia. The information provided by the lumbar catheter led to a major change in the operative plan and subsequent patient management.

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CASE REPORT

A 38-yr-old white woman was admitted to the hospital following a subarachnoid hemorrhage (SAH). Ten days after admission she was brought to the operating room for clipping of a middle cerebral artery aneurysm, at which time her Hunt clinical grade was III. Arterial and intravenous (iv) catheters were inserted and general anesthesia was induced with sufentanil, thiopental, esmolol, and lidocaine. Blood pressure and heart rate remained essentially unchanged during induction. General anesthesia and paralysis were maintained with sufentanil, oxygen, 60% nitrous oxide, and pancuronium. The patient was placed in the lateral decubitus position and a lumbar subarachnoid catheter was inserted at the L4–5 interspace, using an epidural anesthesia set. The CSF was clear and flowed freely; however, care was taken to avoid loss of more than 1–2 ml of CSF. The catheter was attached to a pressure transducer, and the LCSFP was continuously displayed by analog waveform and digital display. A waveform that fluctuated with respirations was observed, and an initial pressure of 11 mmHg was measured. The patient was positioned supine and placed in a Mayfield fixation apparatus without significant change in monitored parameters. During preparation and prior to skin incision, a sudden increase in the LCSFP from 10 to 150 mmHg was noted (fig. 1). This was accompanied by a sudden increase in the arterial blood pressure from 108/36 to 200/108 mmHg, and by an increase in heart rate from 52 to 130 beats/min. The patient was immediately treated with iv thiopental and esmolol, and 1% inspired isoflurane was added. The LSCFP, blood pressure, and heart rate gradually decreased and returned to baseline values within 30 min, at which time isoflurane was discontinued. Arterial blood gases, serum electrolytes, and complete blood count values were measured as normal following this event. Small aspirates of CSF from the lumbar catheter were initially clear but became blood-tinged within 5 min: cell count, 40,000 red blood cells (RBC) and 7 white blood cells (WBC) per millimeter.[3] Based upon this