were located in the right lower quadrant on the anteroposterior roentgenogram, which confirms the observations of Benumof *et al.*[8]

The pulmonary vasculature has primarily been described as being analogous to the Starling resistor.[10–12] A measurement of PAOP that accurately and reliably reflects LAP depends on a patent vascular system between the left atrium and the catheter tip. Early reports of experiences with flotation pulmonary-artery catheters did not mention experience with coincident mechanical ventilation with PEEP. When alveolar pressure increases with mechanical ventilation and high PEEP to cause collapse of the pulmonary vasculature, PAOP will reflect airway pressure. However, PAOP accurately reflects LAP in animals with high PEEP when the catheter tip is vertically below the left atrium.[4–6] Combining these findings with those of the present study, PAOP should accurately reflect LAP in supine patients when the transducer is referenced to the left atrium.

The authors gratefully acknowledge the editorial assistance of Ms. Mary Wondra and Ms. Virginia Swim.

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51:469–471, 1979

Massive Trophoblastic Embolization and PEEP Therapy

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Embolization of trophoblastic tissue to lung is a potentially fatal complication of uterine evacuation of a benign hydatidiform mole. It appears that tissue emboli may result in a progressive hypoxemia and cardiopulmonary instability. To our knowledge all previously reported cases of massive trophoblastic embolization in which ventilatory support has been necessary have been fatal. We present a case in which a patient with this diagnosis was successfully supported with combined ventilatory support and positive end-expiratory pressure (PEEP) therapy.

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Report of a Case

A 19-year-old previously healthy white girl complained of orthopnea, dyspnea on exertion, and a nonproductive cough for six days prior to a scheduled uterine evacuation of a hydatidiform mole. Copious vaginal bleeding necessitated admission to the hospital on an emergency basis, at which time the patient was alert and oriented,

TABLE 1. Clinical Data in the Six Hours Immediately after Operation

Time	$F_{I_{O_2}}$	pH_a	$P_{a_{CO_2}}$ (torr)	$P_{a_{O_2}}$ (torr)	Blood Pressure (torr)	Pulse Rate (/Min)	Respiratory Rate (/Min)	V_T
0200	1.0	7.30	39	99	160/100	120	48	240
0730 (before tracheal reintubation)	1.0	7.24	48	33	170/120	130	52	200
0800 (after tracheal intubation, CPAP 5 cm H_2O)	1.0	7.13	56	32	170/10	130	55	200

TABLE 2. Clinical Data after the First Six Hours

Time	$F_{I_{O_2}}$	pH_a	$P_{a_{CO_2}}$ (torr)	$P_{a_{O_2}}$ (torr)	$P\bar{v}_{O_2}$ (torr)	$Ca\text{-}vD_{O_2}$ (torr)	$\dot{Q}_S/\dot{Q}_T$* (Per Cent)	PWP (torr)	PAP (torr)
0900 IMV 800 × 8 Spontaneous ventilation 100 × 40 PEEP 5	1.0	7.11	60	33	25	2.7	75	18	60/30
1000 IMV 1000 × 8 Spontaneous ventilation 75 × 30 PEEP 20	1.0	7.38	40	83	35	4.5	30	20	35/20
1900 IMV 1000 × 4 Spontaneous ventilation 200 × 25 PEEP 20	0.5	7.39	36	83	40	3.1	26		

* $\frac{\dot{Q}_S}{\dot{Q}_T} = \frac{Cc_{O_2} - Ca_{O_2}}{Cc_{O_2} - C\bar{v}_{O_2}}$.

with a blood pressure of 180/100 torr, a pulse rate of 90/min (regular), and non-labored respiratory rate of 30/min, and a rectal temperature of 99.2 F. Auscultation of the chest revealed coarse basilar rales bilaterally. Examination of the extremities demonstrated 2+ pitting ankle edema but no evidence of clubbing or cyanosis. Tendon reflexes were hyperreflexic bilaterally. Laboratory tests revealed hemoglobin 10.3 g/dl, leukocyte count 13,200, with a normal differential, and a normal platelet count. Urine was unremarkable except for 3+ protein. Chest x-ray on admission revealed diffuse patchy infiltrates bilaterally. Arterial blood-gas values with the patient breathing room air were *p*H 7.46, P_{CO_2} 25 torr, and P_{O_2} 67 torr.

At 0030 hours on the day of operation, a rapid-sequence induction was performed and anesthesia maintained with N_2O, 4l, O_2, 2l, and 0.05 mg fentanyl, iv. Uterine evacuation confirmed the diagnosis of a hydatidiform mole. At the end of the procedure the patient was awake, taking deep breaths on command. The trachea was extubated. Vital signs were stable: blood pressure was 160/100 torr and pulse rate, 120/min. The patient was transferred to the intensive care unit for careful postoperative monitoring. Vital signs, general clinical status, and arterial blood gases progressively deteriorated over the following six hours (table 1). Initially the patient received oxygen therapy through a properly fitting high-humidity face mask with gas flows exceeding 50 l/min. At 0600 hours a portable roentgenogram of the chest was interpreted as "progressive infiltrates suggestive of pulmonary edema." Despite aggressive diuretic therapy and careful fluid balance, her condition continued to deteriorate, and by 0730 progressive refractory hypoxemia (table 1) and respiratory distress necessitated reintubation of the trachea. In spite of the addition of 5 cm H_2O PEEP and bronchial hygiene, the work of breathing necessitated mechanical ventilation.

At 0830 hours a #7-Fr Swan-Ganz catheter was introduced percutaneously via the right internal jugular vein. Table 2 lists the hemodynamic and blood gas findings at 0900. Over the following hour, PEEP was increased incrementally to 20 cm H_2O, at which point the patient's condition was dramatically improved (1000 hours, table 2). Inspired oxygen concentration ($F_{I_{O_2}}$) and rates of intermittent mandatory ventilation (IMV) were then gradually decreased over the next seven hours, maintaining PEEP at 20 cm H_2O. The patient remained alert, without signs of cardiovascular instability.

Inspired oxygen concentration was reduced to 0.3 and the rate of IMV to 2/min by 0700 hours the following day. The patient's condition continued to imrpove, and she maintained adequate oxygenation and alveolar ventilation breathing spontaneously with $F_{I_{O_2}}$ 0.3 for the next six days. However, PEEP therapy could not be completely withdrawn without dramatic increases in intrapulmonary shunting until six days after the surgical procedure. The day after withdrawal of PEEP, the patient's trachea was extubated, and thereafter arterial blood gases were normal during breathing of room air.

DISCUSSION

Pulmonary embolization of trophoblastic tissue secondary to uterine evacuation of a hydatidiform mole may represent an exaggeration of a process that commonly occurs during normal pregnancy. Trophoblastic cells have been found in the veins of the broad ligament in normal pregnancies[1] and have been identified in the lungs of women with normal pregnancies dying of other causes.[2] Since trophoblastic cells characteristically invade blood vessels, it is not surprising that pulmonary embolization may result and cause severe respiratory malfunction.[3]

Following uterine curettage, maternal death has

resulted secondary to massive embolization of trophoblastic cells.[4,5] Autopsy findings in these patients consistently show patchy areas of pulmonary infarction and the presence of hyaline membrane formation. The larger pulmonary arteries remain patent, but numerous smaller arteries and arterioles are completely occluded by trophoblastic cells.[4,5]

Unlike most pulmonary embolization secondary to venous blood clots, this case of trophoblastic embolization resulted in marked intrapulmonary shunting. In addition, the severe hypoxemia was refractory to 100 per cent inspired oxygen. The initial measured shunt in our patient was 75 per cent. Another possible explanation for this large degree of shunt is opening of an arteriovenous pulmonary anastomosis, which became patent when many of the small arterioles were occluded by trophoblasts. This type of intrapulmonary shunting secondary to an acute increase in pulmonary vascular pressure has been demonstrated experimentally in animals and angiographically demonstrated in patients with metastatic choriocarcinoma.[6]

Our patient's response to appropriate PEEP therapy, carefully monitored, is well documented, and we believe it was a primary factor in her survival. Therefore, we believe this therapeutic regimen must be kept in mind whenever a patient has significant respiratory distress, with associated refractory hypoxemia, following uterine curettage of a hydatidiform mole.

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Anesthesiology
51:471–473, 1979

Plasma Catecholamine Levels during Local Anesthesia for Cataract Operations

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Cataract operations may be performed either with general anesthesia[1] or after retrobulbar injection of local anesthetic[2] (usually with added epinephrine) into the eyelid and retrobulbar area. Many patients who have cataracts are elderly and have significant cardiovascular disease[1] such as hypertension, angina, and previous myocardial infarctions.[3] The goal of safe anesthetic management of these patients is to reduce the stress response. Significant increases in the release of catecholamines, as reflected by plasma levels, may cause hemodynamic changes resulting in tachycardia, hypertension, ischemia, angina, or dysrhythmias. With retrobulbar block there are two possible sources of catecholamines: those endogenously released in response to stress, and exogenous epinephrine added to the local anesthetic solutions.

The purposes of this study were to determine whether: 1) there is hemodynamically significant systemic uptake of exogenous epinephrine following eyelid infiltration and retrobulbar block; and 2) patients undergoing this procedure manifest elevated endogenous plasma norepinephrine levels.

Methods and Materials

Seventeen patients, ASA class II, scheduled for elective cataract operations with local anesthesia, were selected. The age range was 43 to 84 years (mean, 64.5 years) and weight range was 60 to 94 kg (mean, 71.8 kg). These patients were premedicated with meperidine, 0.7 mg/kg, im, and promethazine, 0.4 mg/kg, im, an hour before operation.

An 18-gauge heparin lock intravenous catheter was placed in a forearm vein 20 min before the patient was

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Received from the Anesthesia Services of the Massachusetts Eye and Ear Infirmary and Massachusetts General Hospital and the Department of Anesthesia, Harvard Medical School, Boston, Massachusetts 02114. Accepted for publication April 21, 1979. Supported in part by Harvard Anesthesia Research Center Grant No. 705-9718-2 and the Charles A. King Trust.

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