Brief Reports

'Postmortem' Cesarean Section With Recovery of Both Mother and Offspring

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● A 27-year-old primigravida of 37 weeks' gestation suffered cardiopulmonary arrest after massive hemoptysis. After extensive advanced cardiopulmonary resuscitation measures, it was thought that the mother could not be resuscitated and a cesarean section was performed. Immediately after delivery of the fetus, the mother's pulse was palpated, and both the mother and infant are alive without neurological sequelae 20 months later. The reversal of the supine hypotensive syndrome, which was precipitated by massive blood loss, may be the mechanism to account for the restoration of the mother's cardiac output after delivery.
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CESAREAN sections were performed in ancient Rome in 715 BC, when Numa Pompilius decreed that if a pregnant woman died, the fetus was to be cut out of her abdomen.[1] The intent was to keep the fetus from being buried with the mother and not to save the infant.[2] In 237 BC, the first reported infant who survived a postmortem cesarean section was Scipio Africanus, who subsequently became the Roman general who defeated Hannibal.[2] It was not until 1500 AD, when Jacob Nufer sectioned his own wife, that the operation was performed on a live woman with survival of both the mother and the infant.[3]*

More than 153 successful postmortem cesarean sections have been reported in the medical literature.[4] Of infants delivered by this method, approximately 15% survive and are discharged from the hospital in good condition.[2,4,5] The survival of these infants depends on how soon they are extracted, their maturity, the duration and nature of the mother's illness, the performance of postmortem cardiopulmonary resuscitation (CPR) measures on the mother, and the availability of neonatal intensive care.[6]

We report a case of a woman in whom cesarean section was performed after she had a cardiopulmonary arrest.

Report of a Case

A 27-year-old primigravida of 36 weeks' gestation was admitted complaining of hemoptysis. She had emigrated from Hong Kong 1½ years before admission. Hemoptysis had occurred twice before, once in Hong Kong and once one year before this admission. A bronchogram report from Hong Kong described bronchiectasis. There was no history of pneumonia, asthma, tuberculosis, bleeding disorder, or cigarette smoking. She had documented thalassemia minor with a hemoglobin level of 10.5 g/dL.

In the 24 hours before admission, the patient sporadically coughed up approximately 500 mL of blood. She had no fever, chills, pleuritic pain, or dyspnea. Her pregnancy had been uncomplicated.

Physical examination revealed a well-nourished, frightened woman with mild respiratory distress, intermittently coughing up blood-tinged sputum. Her BP was 110/70 mm Hg without orthostatic fall, her pulse rate was 80 beats per minute and regular, respirations were 16/min, and temperature was 36.7 °C. Examination of the head and neck demonstrated no abnormalities. The right lung was clear to percussion and auscultation. Wheezes, rhonchi, and rales were heard in the left upper lung field. A grade 1/6 systolic ejection murmur was heard along the left sternal border. The abdomen revealed a 36-week gravid uterus. No contractions were noted, but fetal heart tones were heard at 140 beats per minute. Extremities revealed no clubbing, cyanosis, or edema.

Laboratory data at the time of admission revealed a normal chest x-ray film. The platelet count was 256,000/cu mm. The prothrombin time and the partial thromboplastin time were normal. The hemoglobin level was 10.5 g/dL, with a mean corpuscular volume of 74 cu μm and the mean corpuscular hemoglobin level of 23.8 pg. The WBC count was 9,600/cu mm. The arterial pH was 7.43, Po_2 was 82 mm Hg, Pco_2 was 26 mm Hg, and the serum creatinine level was 0.8 mg/dL.

Pulmonary consultation was requested with a view to performing bronchoscopy. The patient began to have massive hemoptysis (Table). She became cyanotic, apneic, and pulseless. Intubation was attempted and CPR was begun. The ECG showed a junctional rhythm of 40. A catheter was inserted into the jugular vein, and a peripheral line was placed in an antecubital vein. After eight minutes, the patient was successfully intubated. Vigorous suctioning produced copious amounts of blood from the trachea. Because of continued hemoptysis and the need for suctioning as well as external cardiac compression, the patient could not be placed in the left lateral decubitus position. The ECG showed sinus tachycardia at a rate of 140, but no pulse was palpable between compressions. Arterial blood gasses on 100% oxygen revealed a Po_2 of 240 mm Hg, Pco_2 of 33 mm Hg, and pH of 7.17. Two ampules of sodium bicarbonate (44 mEq/dL each ampule) were given.

A cardiothoracic surgeon, pediatrician, and gynecologist were called. After 25 minutes of advance CPR, the patient had cessation of hemoptysis but remained without femoral or carotid pulses despite a sinus tachycardia. No fetal heart tones were audible. The physicians present believed that the mother would not respond. Cardiopulmonary resuscitation was continued, and a bedside cesarean section was performed. A baby girl was retrieved within four minutes. No anesthesia was used, and the procedure was bloodless.

The infant had an initial Apgar score of 1 and a pH of 6.9. Cardiopulmonary resuscitation was initiated on the infant, and after five minutes her Apgar score was 2 (slow heart rate and mild irritability). She was moved to the neonatal intensive care unit (ICU) and received ventilatory assistance.

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*The Mishnah, compiled in the third century AD in Hebrew, mentions that a woman who gives birth vaginally is required to bring a sacrifice seven or 14 days later. A woman who gave birth by cesarian section was not required to bring this sacrifice. This law assumes survival of the mother and may represent documentation of maternal survival after cesarian section as early as the third century AD.

Clinical Event	Massive Hemoptysis	CPR Begun	Intubation					Cesarean Begun	Cesarean Completed	Decerebrate Rigidity
Rhythm	Sinus	Junctional	ST	ST	ST	ST	ST	ST	ST	ST
HR, beats per min	80	40	140	140	140	136	140	140	130	130
BP, mm Hg	110/70	0	0	0	0	0	0	0	80 (palpable)	140/80
PO_2, mm Hg	82	...	...	...	240	...	...	...	...	210
pH	7.43	...	...	...	7.17	...	...	...	...	7.33
Time, min	−4	0	4	8	12	16	20	24	28	32

*CPR indicates cardiopulmonary resuscitation; ST, sinus tachycardia; HR, heart rate.

The amount of uterine bleeding was minimal. Total resuscitation time now was nearly 30 minutes. Immediately after removal of the fetus, the mother regained femoral and carotid pulses, with a systolic BP of 80 mm Hg. An ECG showed a sinus tachycardia of 130 beats per minute. She was given uncrossmatched blood, and bilateral chest tubes were inserted because of subcutaneous chest crepitations. Her BP rose to 140/80 mm Hg. She was transferred to the ICU, and 1,000 mg of phenytoin sodium, oxytocin, 10 mg of dexamethasone acetate, and cimetidine were given intravenously (IV). The patient continued to receive ventilatory assistance. She was noted to have decerebrate rigidity, and generalized seizures were present intermittently for six hours.

The following morning, the patient was alert without focal neurological findings. Hemoptysis had stopped. Bronchoscopy was performed using a flexible bronchoscope, but no bleeding site was observed. Massive hemoptysis recurred the next day, and the patient underwent a rigid bronchoscopy revealing bleeding from the left lung. However, the precise origin could not be discerned because of the massive flow of blood in the left main bronchus. A left pneumonectomy was performed.

Gross examination of the left lung showed organized blood throughout the entire bronchial tree. The blood vessels did not contain any thrombi or thromboemboli. The pleura was intact.

Microscopically, intra-alveolar hemorrhage was prominent. No bronchiectasis, aneurysms, tumor, or other lesions were noted on microscopic examination to explain the bleeding. The mucosal lining was normal. Mild, acute, and chronic inflammation without fibrosis was noted in the submucosal tissue. No arterial or venous malformations were seen. Immunofluorescent staining of the lung did not reveal any pathological condition.

The infant was weaned from the ventilator and at the time of discharge from the hospital had no focal neurological deficits, but did show hypotonia and decreased sucking.

Twenty months later, the mother and child are alive and doing well. The mother has normal cranial nerve, motor, and sensory function and no recurrent hemoptysis or seizures. All medication has been stopped. She rides a bicycle daily. The child is well and is currently receiving no medications. Neurological examination reveals appropriate development for the child.

Comment

In the supine position, in late pregnancy, the vena cava is completely obstructed in 90% of women, and venous return occurs through the azygous lumbar and paraspinal veins.[7,8] Systolic BP is maintained by an increase in peripheral resistance[7]; however, in the case of hemorrhage, this compensation is lost. In this case, the state of severe and prolonged shock seemed to have resulted from a combination of massive hemorrhage from the bronchial tree, supine hypotensive syndrome, and an initial vasovagal reflex with bradycardia.

The stroke volume and cardiac output increase by 30% when a preterm pregnant female changes from the supine position to the lateral decubitus position.[9] However, in our patient, we could not take advantage of this phenomenon because it was impossible to perform CPR in the lateral decubitus position. A wedge pad under the right side of the patient's back with displacement of the uterus to the left (as used by anesthesiologists during cesarean operation) may have improved vena cava return without interfering with CPR procedures.

Immediately after spontaneous delivery, cardiac output rises by 60% to 80% of prelabor values.[10] After cesarean section, the rise in cardiac output is 30% of prelabor values.[11] The smaller increase associated with cesarean delivery is due to anesthetic-induced hypotension and blood loss, which usually averages 1,000 mL.[11] However, in our patient, no anesthetic was used, and the procedure was bloodless because of the absence of BP. There-fore, it is possible that the rise in cardiac output in our patient may have been higher than that observed in the routine cesarean section.

In the supine position, the uterus displaces and partially obstructs the aorta and its branches late in pregnancy, and maternal hypotension markedly enhances this effect.[12] Our patient was in the supine position during the resuscitative procedure. In addition, the pregnant uterus compresses the inferior vena cava and further reduces the venous return from the lower abdomen and extremities. These two factors may have contributed substantially to the shock syndrome. In addition, bradycardia as initially documented in our patient has also been reported in the supine hypotensive syndrome.[7] This phenomenon might contribute to but could not explain entirely the absence of femoral pulses in our patient. It is likely that our patient's recovery was due to an improvement in cardiac output from removal of uterine compression on the vena cava and redirection of cardiac output away from the uterus.

It is also possible, but less likely, that continued CPR and IV fluids would have allowed her to recover. Although sequestration of circulating blood mass caudal to uterine vena cava aggravated the degree of hemorrhagic shock, sufficient replacement volume had been given IV.

There are two important clinical lessons to be learned from this case report: First, if CPR fails and the cardiac rhythm is electromechanical dissociation, then before pronouncement of death, the woman should momentarily be turned to the lateral decubitus position to see if a rise in cardiac output is possible. The bradycardia that can frequently accompany the supine hypotensive syndrome may also disappear when the patient is

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turned into the lateral decubitus position.

Second, if CPR fails to resuscitate the mother, it must be continued while performing a postmortem cesarean section. Although certain authors have stressed the importance of continuing CPR during the postmortem cesarean section, this is often neglected.[4,13] In these cases, CPR should be done to enhance fetal survival. In addition, it would be appropriate to check the mother's pulse after the cesarean section.

It would be erroneous to conclude that the widespread use of cesarean sections in preterm pregnant patients who are moribund or who are undergoing CPR is justified based on our single case report. Nevertheless, such antemortem cesarean sections might improve infant survival but would probably sacrifice the mother's

life, since the adverse consequences of the surgery would likely outweigh the hemodynamic benefits we have described. We recognize, however, that it may be difficult to determine exactly when, during CPR, resuscitation is no longer possible. Physicians should be aware of the hemodynamic consequences we have described and the fact that the longer one waits the more fetal viability diminishes.

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Dangers of Intravenous Ritodrine in Diabetic Patients

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● **Two cases are reported in which administration of ritodrine hydrochloride, a β-2-sympathomimetic agent, was followed by diabetic ketoacidosis when it was used for inhibition of premature labor in insulin-requiring diabetic patients. Ketoacidosis occurred shortly after intravenous ritodrine, despite previous administration of large subcutaneous doses of insulin. Early recognition and treatment of this serious complication are critical for fetal survival.**
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RITODRINE hydrochloride (Yutopar) is a β-2-sympathomimetic agent that has recently been introduced in the United States for inhibition of premature labor. The drug has been shown to decrease the frequency and intensity of uterine contractions. In controlled clinical trials, ritodrine has decreased the incidence of pre-

term birth, perinatal death, and infant respiratory distress syndrome.[1,2]

Adverse reactions have been reported with this and other β-sympathomimetic agents, however. These include maternal pulmonary edema, adult respiratory distress syndrome, hypokalemia, angina, tachycardia, and other cardiovascular complications. It is not generally appreciated that β-agonists may precipitate noticeable decompensation of the diabetic state when used in insulin-requiring diabetic patients. Furthermore, although some concern has been expressed about the release of ritodrine for use in the United States,

exacerbation of diabetes in the mother has not been a matter of great concern.[3] In Europe, where β-agonists have been used since 1972, there are three published reports of the occurrence of this phenomenon when ritodrine was used to prevent premature labor in diabetic patients.[4-6]

It is the purpose of this communication to report two additional cases in which intravenous (IV) ritodrine administration led to noticeable decompensation of diabetes mellitus. Prevention and treatment of this problem will be considered. It is hoped that this will alert the medical community to a potentially serious complication of such therapy in diabetic patients.

Report of Cases

CASE 1.—A 30-year-old diabetic woman, gravida 4, para 0, abortus 3, in her 29th week of pregnancy with twins was admitted to the Medical University of South Carolina, Charleston, in active labor. After her condition had been managed by diet therapy for 15 years, she had