Medical Record Analysis
Of
P. C.

Mrs. P.C. was a previously healthy, 31 year old black female who was 37 weeks pregnant when at 11:45pm on March 12, 1994 the protective sack around the baby had prematurely and spontaneously ruptured. She was admitted to Southside Community Hospital in Farmville on March 13 at 1:20am. She had reported to them that the amniotic fluid was clear and that there was no vaginal bleeding. She did not report any complications during her pregnancy except a urinary tract infection at the beginning of her pregnancy. She also reported that she did not use any alcohol, but admitted to smoking 2 cigarettes/day. She had a previous surgery to her right knee in 1985 without any complications. She was taking prenatal vitamins and iron. She was allergic to penicillin and codeine. She had two ultrasounds during her pregnancy. She had a family history of high blood pressure, asthma and diabetes. Her blood work during her pregnancy revealed that she was negative for chlamydia and gonorrhea, but she had a cervical culture that was positive for Group B Streptococcus bacteria.

The physical exam on admission was unremarkable. Her height was 5'3" and her weight was 227 pounds. Her contractions were mild, irregular and lasting about 15 seconds. She was connected to an external monitor that picked up the baby's heart beat. She had a needle inserted into her arm to receive intravenous fluids and antibiotics; her contractions and vital signs also were monitored. An order was given that no one perform a pelvic exam to decrease the chances of bacteria being inserted into the vagina. Dr. O. P., her obstetrician, was in to do a pelvic exam at 10:30am and found that she was only 2-3 cm dilated. He decided to administer Pitocin intravenously by an IV pump to stimulate and improve her contractions. At this point internal monitoring was begun to allow for a more direct and continuous monitoring of the mother's labor and the baby's tolerance of the labor.

Uterine contractions were recorded by placing a catheter into the uterine cavity behind the baby's head. The fetal EKG is obtained by clipping a small electrode to the head.

Mrs. C. had a foley catheter placed into her bladder to continuously drain her urine. The only pain medication she had received was Demerol at 3:15pm. At about 8:30 that night, she had an epidural catheter placed for pain control by G. M., CRNA. [This procedure is done with the patient sitting on the side of the bed with her shoulders bent forward to expose and elongate the spine. A 3 inch needle is then maneuvered (under local anesthesia) between the vertebrae into the lumbar area into the narrow epidural space surrounding the spinal cord. A very thin catheter is threaded through the needle into the epidural space and the needle is removed.] Local anesthetic medication was injected through this catheter by means a computerized precision pump. Mr. M. was called to check this catheter after the nurse reported the pump was reading that there was some type of blockage and the medication wasn't being administered. At 11:32 p.m., the first epidural catheter was removed intact and replaced. Before Mr. M. left Mrs. C.'s bedside, she told him that she was starting to get numb over her abdominal area.

Although she continued to have irregular contractions 1-6 minutes apart throughout the day, Mrs. C.'s cervix was not progressively dilating. Dr. P. decided by that a C-section needed to be performed. The Pitocin infusion was stopped and she was taken to the operating room at 12:20 a.m., more than 24 hours after her membranes had ruptured. The risks and possible complications of a C-section, including bleeding and infection, were explained and the consent obtained.

Once in the operating room, Mrs. C.'s level of epidural anesthesia was tested and found to be inadequate for surgery. Dr. P. decided that general anesthesia was needed. Mr. M.'s assessment of the patient's airway was that she had a short, fat neck, and that her range of motion was adequate.

At 12:40 a.m., Mr. M. administered 100% oxygen by face mask and 2.5 cc of Fentanyl (a narcotic) in preparation for the administration of general anesthesia. (Narcotics are usually not given prior to induction, as they may cross the placenta and cause respiratory depression in the baby). Monitors were applied; baseline vital signs were normal, including an oxygen saturation of 100%.

At approximately 12:45 am, a Rapid-Sequence Induction of general anesthesia was done by Mr. M.; this is the standard technique used for C-section patients. A sleep-dose of Pentothal was given, followed immediately with a paralyzing dose of Anectine, while cricoid pressure (Sellick Maneuver) was held to diminish the risk of aspiration of stomach contents. These drugs exert full effect after about 60 seconds. As the patient lost consciousness and stopped breathing (due to the Anectine), Mr. M. attempted twice to place an breathing tube through her vocal cords into her trachea, using a laryngoscope. In between attempts, she was still able to be ventilated manually with a face mask and breathing bag; the monitor showed adequate oxygen saturation of her blood at 97-98% (normal 95-100%). This showed that she still had an adequate natural airway at this point, although she was not breathing on her own. After these attempts at intubation, Dr. P. instructed Mr. M. to stop trying to intubate her, and just use the mask and breathing bag to provide 100% oxygen through her natural airway to her lungs, even though she was not yet breathing on her own, after the Anectine paralysis. Dr. P. further instructed him to reverse whatever sedation Mrs. C. had received and to no longer proceed with general anesthesia. (Anectine is not reversible, but in most patients will wear off within 3-5 minutes, allowing resumption of spontaneous breathing. The sleep drug Pentothal would also wear off in the same time frame, although it's effect may be prolonged by the Fentanyl given just prior; the Fentanyl is also a respiratory depressant which might slow the patient's return to spontaneous breathing.)

Mr. M. noted that he was trying to awaken the patient and that she developed laryngospasms, a dangerous condition which may be associated with airway manipulation, in which the vocal cords spasm closed, thus blocking the airway. Mr. M. documented that he treated this with Anectine, at a dose of 100mg (10-20mg is the usual dose of Anectine for laryngospasms).

Meanwhile at 12:47 a.m., it was noted by use of a special stethoscope, that the baby's heart rate had dropped from 130 to 80 to 60 (indicating probable lack of oxygen, which would be consistent with loss of the mother's airway for the delivery of oxygen to the tissues, including the baby.) Dr. Pearce proceeded rapidly with C-section delivery of the baby (as the patient remained unresponsive) while more intubation attempts were being made. A live male was delivered at 12:50 a.m. and passed to the pediatrician. The time from the surgical incision to delivery of the infant was 1 ½ minutes. Initially the baby had an apgar score of 1 (out of 10) then 9 after 5 minutes. [The Apgar scoring system is an objective evaluation of the infant at birth, assessing heart rate, respiratory effort, muscle tone, reflexes, and color.] The Apgar score of 1 at birth indicated the infant was in severe distress. (Baby Craft will be discussed again later).

At 12:55 a.m., Mr. M. documented that the spasm of the larynx had broken and he was trying to wake the patient up. By now her oxygen saturation had fallen to 70. Apparently, using the mask and bag had now become unsuccessful. An Emergency Room physician, Dr. L., arrived in the operating room to provide assistance at about 12:50 a.m.. At 12:55 a.m., PER (don't know what this abbreviation means) was tried without success.

At 1:00 a.m., another 100mg Anectine was given and Dr. L. attempted three times to intubate the patient without any success. At this time her oxygen saturation had fallen to 35, indicating failure

to get oxygen into her lungs. Despite attempts to obtain an airway by intubation, the mask ventilation was now ineffective, probably due to repeated use of the laryngoscope. The airway was "lost".

At 1:06 a.m. L. L., CRNA (chief anesthetist) attempted twice to intubate the patient and was unable to ventilate her with the bag and mask. A tracheostomy was suggested and Dr. N., the general surgeon on call, was notified at home. At 1:07 Mrs. C.'s heart stopped in a "flat-line" pattern called asystole. CPR chest compressions were started, although she still didn't have an open airway. [For CPR to be effective, an adequate airway and oxygenation of the patient's blood are essential along with the chest compressions.]

Between 1:10 and 1:20 a.m., several unsuccessful attempts at a cricothyrotomy were made by Mr. M. and Ms. L.. This life saving procedure may be done quickly by inserting a large IV needle through the cricothyroid membrane into the neck, allowing a high-flow of oxygen to be administered into the trachea and lungs, providing oxygenation until a tracheostomy can be done. It is a stop-gap technique which can buy precious time in an airway emergency.

At 1:15 a.m., Mr. M. noted that the patient's pupils were fixed and dilated.

At 1:30 a.m., Dr. N. arrived and quickly started to do a tracheostomy by making an incision into the front of the neck. A size 8 tracheostomy tube was attempted to be inserted directly into the trachea. This tube was too large for passage, and a size 6 tube was placed by 1:45 a.m., the patient's lungs now were able to receive oxygen, almost an hour after the loss of the airway. It was sutured and secured with cloth ties around the neck. CPR and Advanced Cardiac Life Support (ACLS) were still in progress.

Arterial blood gas (ABG) studies drawn to assess her oxygenation at 1:30 a.m. showed severe hypoxia, acidosis and hypercarbia:

- pH 7.028 (severe acidosis; normal= 7.35-7.45)
- pO2 12 (Severe hypoxia; normal = 80-100)
- pCO2 77.9 (Severe hypercarbia; normal = 35-45; slightly lower in pregnancy)

These values are consistent with failure to move oxygen into the lungs and carbon dioxide out of the lungs.

ACLS medications and techniques were used, although an open airway was not obtained. Atropine was given initially for a drop in heart rate to 35 (which was consistent with both hypoxia and a second dose of Anectine; both can cause profound bradycardia and asystole) . Epinephrine was also given every 3-5 minutes; this medication increases the contractility of the heart, increases the blood pressure (if present). These medications are the only treatments for asystole, although use of a pacemaker may be tried early in the course of an arrest.

Mrs. C.'s heart was "shocked" several times with the defibrillator, although there was no documentation of a rhythm such as ventricular fibrillation or ventricular tachycardia which might respond to defibrillation. "Shocking" the heart which is in asystole probably makes the heart more resistant to resuscitation.

The main focus in treating asystole is seeking the cause and correctly it, while the A-B-C's of CPR (Airway, Breathing, Circulation) are being done to sustain life. Causes of asystole include:

- Hypoxia (lack of oxygen to the tissues)
- Hypokalemia (low potassium)
- Hyperkalemia (high potassium)
- Hypothermia (low body temperature)
- Acidosis

- Drug overdose with certain medications

Advanced cardiac life support was performed until 2:32 a.m., when Mrs. C. was pronounced dead by Dr. P.. She had no pulse, no sign of cardiac activity, and no respirations.

An autopsy was performed by Dr. D. J. on 3/15/94. The cause of death was listed as hypoxic cardiac arrest. The pertinent findings included: marked congestion or swelling of the blood vessels in the main branches of the lungs, and marked pulmonary edema (fluid in the lungs) with bleeding. "Death in this case was caused by hypoxia which was likely due to inadequate oxygenation while under anesthesia during cesarean section." Upon examination the larynx and trachea were patent with no evidence of structural or congenital abnormality. "However, the mucosa lining the main branches of the lungs and larynx was significantly edematous and congested, a factor which may have impaired attempts to intubate this patient. Laryngeal edema occurs during pregnancy. However, in this particular case, some of the edema may have been secondary to attempts at intubation. Also most likely secondary to resuscitative measures were the pulmonary edema and hemorrhage, and slight congestion of the spleen and liver."

Baby C. had no spontaneous respirations at birth. A breathing tube had to be placed into his small trachea for a short time and given oxygen with a bag. Since any medication given to the mother also passes to the baby through the placenta, Narcan was given to reverse the sedative given to Mrs. C., and the baby started to breath on his own. The breathing tube was removed and he was transferred to the nursery in stable condition.

The plan on admission to the nursery was to make sure he didn't have any infection and to monitor and treat his low blood sugar. His initial newborn assessment was unremarkable except for a laceration to the right side of his forehead, dusky color and a weak cry. The oxygenation of his

blood without any supplemental oxygen was 100%. A finger stick was done to determine Baby C.'s blood sugar and was found to be 40. The lab was in to draw blood from his little veins, but they were unsuccessful. His blood had to be obtained by Dr. D.. A repeat finger stick still read 40. Dr. Diansio performed a spinal tap or lumbar puncture on Baby C. This requires inserting a large, long needle into the baby's tiny spine, to measure the pressure and obtain fluid for specimens and cultures to see if there is any infection. A long IV needle was inserted into Baby C.'s little veins to administer a concentrated dextrose solution. After he started to receive this fluid, a repeat finger stick showed that his blood sugar had increased to 60. When Baby C. was stabilized, Mr. C. requested that he be transferred to MCV for further care.