Trimester Bleeding and Obstetric Catastrophes

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I. Hemorrhagic Catastrophes

A. Antepartum

1. Placenta previa
   a. Incidence: 1/200 live births
   b. Risk factors include:
      (1) Age
      (2) Parity
      (3) Multiple gestation
   c. Classification
      (1) Central
      (2) Partial
      (3) Marginal
      (4) Low-lying
   d. Diagnosis made on:
      (1) Signs
      (2) Symptoms
      (3) Ultrasound
   e. Management is based on prolonging pregnancy to lung maturity

2. Vasa previa
   a. Definition: fetal vessels cross cervical os
   b. May result in fetal hemorrhage
   c. Diagnosed by clinical suspicion
   d. Immediate delivery

3. Abdominal pregnancy
   a. Incidence: 1/7,000 deliveries
   b. Signs:
      (1) Amenorrhea
      (2) Abdominal pain
      (3) Fetal malpresentation
      (4) Poor response to oxytocin
   c. Cornerstone of management is surgery

B. Intrapartum

1. Abruptio placentae
   a. Incidence: between 1/50 and 1/200 live births
   b. Risk factors include:
      (1) Hypertension
      (2) Increasing parity
      (3) Previous abruption
      (4) Smoking
      (5) Trauma
      (6) Vascular disease
   c. Signs
      (1) Uterine irritability
      (2) Tenderness

Hemorrhagic catastrophes during the antepartum period. Placenta previa has an incidence of about 1 in 200. Risk factors include increasing age, parity, multiple gestation and previous cesarean section. If you have had one placenta previa, your risk of having it happen again is about 4-8%.

The diagnosis of placenta previa is made by painless vaginal bleeding. We should remember to look for it when we see fetal malpresentations since that happens in about 30% of the cases as opposed to the 2-3% which is the background incidence of malpresentation at term. The diagnosis is made with ultrasound almost exclusively.

We can classify placenta previa depending upon the relationship between those two structures, between the placenta and the cervix. When the cervix is completely covered, complete placenta previa, partially encroaching on the cervical os, partial placenta previa and marginal when the edge of the placenta approaches the cervical os. The only time that I would consider diagnosing a low-lying placenta is during an anomaly screen or a growth scan at 20 weeks or 24 weeks in an asymptomatic patient where the placenta looks low.

Principles of management include maintaining the pregnancy to fetal lung maturity if and when that is possible. Bed rest has been used for quite a long time and is felt to be beneficial although that has never been studied. Replace blood lost and keep blood on hold. Steroids have been for a long time, but we have really turned around about tocolytics a lot in the last 10 years. Now, I think mostly as a result of our trust in our fetal assessment, people are much more likely to give tocolytics to patients that are bleeding if our two usual criteria are met. That is a healthy, hemodynamically stable mother and a fetus in whom we can demonstrate well being. Monitoring fetal well being, paying attention to our second patient and delivery by cesarean section.

Velamentous cord insertion is when vessels cross course in amniotic fluid unprotected by Wharton's jelly. This occurs in about 1% of singleton pregnancies and 7% of multiple pregnancies where it usually doesn't cause a problem. Here is the blood vessels getting to the placenta and Wharton's jelly stops out here. Usually that really doesn't cause any difficulty except to increase likelihood of tearing the cord when we are delivering the placenta. But if these velamentous vessels do cross the cervical os, which they may, the vessels may be easily ruptured during rupture of membranes, either during amniotomy or spontaneously, and that may result in fatal fetal hemorrhage.

Diagnosis and management is to identify patients at risk. That is patients with multiple gestations. Palpation of membranes prior to amniotomy. The APT test has been described for a long time, although I don't particularly find it clinically useful. We know that sodium hydroxide lyses fetal blood into homogenous pink color to the solution whereas it doesn't lyse adult blood and we know that bleeding from placenta previa and placental abruption is maternal blood, at least initially, and should not be lysed by sodium hydroxide. I think that kind of takes a long time in a clinical setting and this really should be a clinical diagnosis and the management is immediate delivery.

Abdominal pregnancy. How many people have had the fortune or misfortune to see an abdominal pregnancy? Quite a few. I have seen three. Things kind of tend to come in bunches and several years ago I had three within a two year span. I haven't had any for awhile so maybe it is time to have one again. The incidence is about 1 in 7,000. The perinatal mortality from the fetal standpoint is about 80%. It probably results usually from rupture of a tubal pregnancy with a secondary implantation somewhere in the pelvis usually.

Diagnosis is actually a difficult diagnosis sometimes and I do have to admit that I have missed one of these on initial examination and didn't make the diagnosis until the next day. But situations where we should give consideration to the diagnosis would be in a patient with abdominal pain and amenorrhea. Fetal malpresentation again can be associated - almost always associated. A closed uneffaced cervix as well as a poor myometrial response to oxytocin.

The diagnosis itself is made by failing to visualize the chorionic plate on ultrasound.
C. Postpartum

2. Uterine rupture
   a. Incidence: 1/1,000 to 1/2,500 deliveries
   b. Classification
      (1) Traumatic
      (2) Spontaneous
         (a) Previous uterine surgery
         (b) No previous surgery
   c. Clinical manifestations
      (1) Abdominal pain
      (2) Cessation of uterine contraction
      (3) Vaginal bleeding
      (4) Fetal distress
   d. Management

   3. Placenta accreta
      a. Incidence: 1/2,000 deliveries
      b. Definition
      c. Etiology: defective endometrium
         (1) Lower uterine segment
         (2) Uterine diverticulum
         (3) Previous radiation
         (4) Myomectomy scars
      d. Management is hysterectomy

   3. Uterine inversion
      a. Incidence: 1/2,000 deliveries
      b. Risk factors include
         (1) Uterine relaxation
         (2) Inappropriate management of the third stage
         (3) Fundal implantation of placenta

   Management. Allowing the pregnancy to continue does risk intraperitoneal hemorrhage so we should proceed to surgery shortly after diagnosis and many people would consider that if the mother is stable that we wait long enough to do a bowel preparation, give antibiotics and keep blood available. I do want to mention that of the three that I have seen, one of them actually carried to near term and ended up with a live born baby which is very unusual. We generally suggest leaving the placenta intact unless the structure to which it is attached could be conceivably ligated such as an ovary for example. But usually it is on the pelvic side wall or the fundus of the uterus or bowel, it should be left in place unless it is interfering with that structure.

   Hemorrhagic catastrophes during the intrapartum period. Abruptio placentae is initiated by hemorrhage into the decidua basalis followed by formation of a decidual hematoma and then separation, compression and destruction of the placenta ultimately. Then we have the mandatory ultrasound picture of a large abruption here. The way that I put this to the residents is that we should consider that ultrasound can reinforce the diagnosis and can even make the diagnosis but certainly cannot rule out the diagnosis of placental abruption since that diagnosis can't always be made depending on the position of the placenta, the amount of bleeding, how much clot is actually present, the size of the patient, etc.

   The incidence of placental abruption really varies depending on how it is counted. If we count just abruption during the first stage of labor, the incidence is about 1 in 200. However, if you include abruptions during the second stage of labor, the incidence is 1 in 50. Complete placental separation occurs much less commonly. About 1 in 500. If you have had one placental abruption, the risk is 6-15% in various studies and about 25%, maybe 30%, if it has occurred twice.

   Risk factors include maternal hypertension, with or without preeclampsia, increasing parity, cocaine use, previous abruption, smoking, trauma and maternal vascular disease such as lupus.

   Signs and symptoms include uterine irritability, generalized uterine tenderness, absence of uterine relaxation between contractions and vaginal bleeding, generally although not always. In the classic description, not always the case, is port wine colored non clotting blood because of utilization and consumption of the clotting factors.

   Management of placental abruption includes frequent vital signs, fetal monitoring, CVP monitoring, occasionally blood transfusion and we should be alert of the need for that, monitor for DIC and delivery. I do need to mention that many people and we also have been recognizing much smaller degrees or much less clinically significant abruptions. I think there is an increasing appreciation that many patients that we termed in idiopathic preterm labor in the past do have some placental etiology and possibly a low grade placental abruption as a contribution to their preterm labor.

   Uterine rupture. A topic that has come under a lot of scrutiny and a lot of discussion in our literature lately as I am sure most everyone knows because of the recent, you could say, discussions in our literature about how we are counseling our patients to have VBACs. The incidence of uterine rupture is 1 in 1,000 to 1 in 2,500 approximately. If one has had a previous low transverse C-section, the incidence of rupture is about 0.3%. In a previous classical C-section, about 1-2%. How many people here have experienced a uterine rupture? Nearly everyone.

   Maternal mortality is about 1% although smaller in the large California study. The fetal mortality is still significant and that is 10-15%. Classification of uterine rupture really depends upon preexisting factors. It can be traumatic or not, and spontaneous can occur associated or not associated with previous uterine surgery. Spontaneous rupture of the uterus can occur associated with previous uterine surgery such as cesarean section, a myomectomy that entered the uterine cavity and a previous curettage. Patients that have not had surgery risk factors are congenital anomalies of the uterus, cornual pregnancy and placenta percreta.
II. Mechanical Catastrophes

A. Labor and delivery

1. Shoulder dystocia
   a. Incidence: 0.15-0.38% of all deliveries
   b. Risk factors
      (1) Macrosomia
      (2) Prolonged second stage
      (3) Midpelvic delivery
      (4) Multiparity
      (5) History of previous macrosomia
   c. Complications
      (1) Fetal
      (2) Maternal
   d. Management

2. Cord prolapse
   a. Incidence: 1/200 deliveries
   b. Classification
      (1) Cord presentation
      (2) Occult prolapse
      (3) Complete prolapse
   c. Management requires immediate delivery

B. Fetal anomalies

1. Hydrocephaly
2. Sacral coccygeal teratoma
3. Amniotic band syndrome

C. Multiple gestation

1. Interlocking twins
2. Monoamniotic twins
3. Conjoined twins

III. Traumatic Catastrophes

A. Blunt trauma

1. Most common type of injury
2. Usually secondary to auto accidents
3. May result in uterine rupture
4. Amniotic fluid protects fetus

B. Penetrating trauma

1. Incidence increasing worldwide
2. Enlarged uterus is protective to mother by displacement of viscera

C. Major burns

1. Definition: 10% of surface area
2. Complications of burns
   a. Electrolyte disturbances

Traumatic rupture of the uterus can occur sometimes from things that we do such as uterine curet or sound, manual removal of the placenta, violence also. Oxytocin administration. Forceps, breech extraction or internal podalic version. Fundal pressure - a procedure that I think should be abandoned in contemporary obstetrics - and neglected CPD.

Signs and symptoms of uterine rupture include tearing abdominal pain, cessation of uterine contractions, vaginal bleeding and fetal distress as well as the turtle ascent of a previously low presenting part. Of these, the collaborative study that came out of California about five years ago showed us some surprising information. We always had used kind of the top three of these as being the kind of textbook presentation of a patient with uterine rupture. It ends up that the thing that was present most consistently in their large series was fetal distress as a predictor or as a harbinger of the impending uterine rupture or maybe not even impending but present.

Indications for hysterectomy. We have a patient that has a uterine rupture. Hopefully, we are able to intervene in time to have a reasonable fetal outcome and then we have to make the decision whether to try to repair the ruptured uterus or do a hysterectomy at the time. Here are some suggestions about times to consider hysterectomy. That would be transverse tears involving the bladder, multiple lacerations of the uterus, extensive rupture of a classical scar, any involvement of the cervix or vagina and probably the most important, the presence of infection.

The third stage of labor is always a time of anxiety which no obstetrician ever wholly outlives and 25% of the time, uterine atony is present without identifiable risk factors. But there are risk factors 75% of the time and here they are. Chorioamnionitis. The prolonged dystocic labor, general anesthesia, grand multiparity, a low placental implantation, a large uterus such as from multiple gestation, hydramnios and macrosomia, a precipitous labor, preeclampsia probably because of magnesium administration, as well as a prolonged protracted labor.

Clinical approach. I am sure everybody has some idea how to approach this. I would suggest that this is something we should all have kind of in our minds at all times because eventually this is going to happen everyone practicing obstetrics and I am sure it has happened to most everybody to some extent already. I would suggest going about it in a stepwise fashion. Manual exploration with curettage if it is felt that there are retained products within the uterus. Bimanual compression or bimanual massage while oxytocin administration is increased. Ergotrate is a reasonable next step, I think. Keeping in mind that Ergotrate should not be administered intravenously because of the association with pituitary necrosis and should be given IM and actually can be given right into the uterus which is a fine way to give it. Prostaglandin F2 alpha can be given IM, IV or also, again, right into the uterus which I find to be pretty effective. That is marketed under the trade name of Hemabate.

At this time, surgery if uterine atony is still present we should give consideration to preparing for surgery and the choices for surgery are hypogastric artery ligation versus hysterectomy. I think the trend over the last several years, because of surgical experience perhaps, has been kind of towards hysterectomy. Selective arterial embolization I mentioned for completeness. There have been a couple of small studies looking at selective arterial embolization as being pretty effective in the management. I don't think that is something that is readily available to most of us but it is a possibility.

Placenta accreta on the other hand with placental implantation problems occurs about 1 in 2,000 deliveries. We can divide placenta accreta depending upon how veraciously the placenta is attacking the uterus. Placenta accreta vera where the villi contact the myometrium but do not invade, placenta increta where the villi invade the myometrium and placenta percreta where the villi penetrate the myometrium and sometimes approach or even traverse the serosa.

The etiology of placenta accreta is worth keeping in mind and that is implantation into an area of defective endometrium such as the lower uterine
b. Hypovolemia and hypotension

c. Pulmonary problems

d. Infection

e. Inadequate nutrition

3. Pregnancy outcome

a. Maternal

(1) 25% mortality if burn 50% of surface area

(2) 100% mortality if burn 80% or greater of surface area

(3) Gestational age does not influence survival

b. Fetal

(1) Premature labor is very common

(2) Stillbirth: 75%, if burn equals 30% of surface area

(3) Largest burn survived by mother and fetus: 58%

4. Management of burns during pregnancy

a. Standard burn therapy

b. Daily evaluation for fetal well-being

c. Burns <30%: good prognosis

d. Burns >50%: consider delivery

e. C-section not contraindicated

D. Perimortem C-section

1. General guidelines

a. Determine potential for survival

b. Fetus alive prior to procedure

c. Classical C-section

d. Maintain CPR

e. Place surgical kit near any critically ill obstetrical patient

f. Do not compromise mother by intervening too early

IV. Catastrophic Medical Complications of Pregnancy

A. Anaphylactoid syndrome of pregnancy (amniotic fluid embolism)

1. Occurrence

a. 1/20,000 pregnancies

b. 10% of maternal deaths

c. 61% maternal mortality

d. 24% maternal neurologic deficit

e. 61% fetal mortality if intrapartum

2. Diagnosis:

a. Acute hypotension or cardiac arrest

b. Acute hypoxia

c. Coagulopathy

d. Labor, delivery, or dilation and evacuation

e. No pre-existing conditions

3. Characteristics/risk factors

a. No correlation with maternal age, gravidity, gestational age, Pitocin, duration of labor, or presence of meconium

b. 67% male fetuses

Management of placenta accreta. Conservative therapy risks sepsis and hemorrhage. The procedure of choice in diagnosed placenta accreta is hysterectomy and not hypogastric artery ligation because in this situation ligating the hypogastric artery really will not change the hemodynamic picture or the situation of the uterus. This is a patient that we had with placenta accreta and I think you can visualize the cervix here. Here is the fetal aspect. The fetal side of the placenta and here is the uterine surface of the placenta really growing entirely through the left cornu of the pregnancy. Looking with ultrasound a couple of times and evaluating the clinical situation, the patient had a dropping hemoglobin and hematocrit, some vague abdominal symptoms but really nothing else.

A surgeon thought that she might have an atypical presentation of appendicitis. I probably should have let him do the surgery but we didn't think that really was the case. Finally we did recognize some blood in the abdomen at time of uterine ultrasound and then made this diagnosis. We questioned the patient at length about her history while we were trying to figure out exactly what her problem might be. She promised that she had had no previous surgeries and no previous pregnancies on multiple questioning and then we took her to surgery for this problem, gave her multiple blood transfusions and delivered the 28 week baby which did survive and do well fortuitously. When she was recovering and finally got off the ventilator, she did then recall that she had had a late pregnancy termination with a possible perforation of the uterus. Just for interest we got some records and they did feel that they had perforated the uterus at the time and had kept her over for observation and such.

Uterine inversion has an incidence of about 1 in 2,000. Significant mortality secondary to hemorrhagic shock. Some people would suggest that the shock is sometimes out of proportion to the hemorrhage itself possibly because of the inversion of the viscus.

Risk factors for uterine inversion include uterine relaxation such as from general anesthesia or magnesium administration, inappropriate management of the third stage of labor - so we can always blame the residents or the medical students for pulling on the umbilical cord while they are pushing on the fundus, a fundal implantation of the placenta which we generally don't have the luxury of knowing at the time of delivery and a precipitous labor. Perhaps the placenta is not ready to be separated in that situation.

Management of uterine inversion. Repositioning immediately if possible because the arterial blood supply is probably still intact and still pumping blood into the inverted uterus and edema can worsen and make it more difficult to turn inside out. Laparotomy, I have never had to do but I am told that that can be beneficial most of the time with traction on the round ligaments which are pictured here. People have also described putting a heavy ligature in the uterine fundus at the time of the laparotomy and pulling up on that while they are pulling up on the round ligaments and perhaps even having someone push vaginally at the same time.

Mechanical catastrophes during the labor and delivery time period. I think if we polled a group of obstetricians about the things that made them most nervous, they would probably say entrapment of the aftercoming head in a breech delivery, for people that do breech deliveries, and probably shoulder dystocia. Despite the fact that it is something that gets a lot of attention in the literature and people write about risk factors in diabetic and nondiabetic patients, etc and it is something we rightfully are concerned about and worried about, it really accounts for a pretty small incidence. It is about 0.15-.38% of all deliveries.


Complications from the fetal aspect include fractures of the humerus or
C. Peripartum cardiomyopathy
d. Clinical and hemodynamic presentation similar to anaphylaxis and septic shock

4. Treatment
a. Expedient diagnosis and treatment
b. Hemodynamic support: central monitoring
c. Respiratory support: intubation
d. Treatment of coagulopathy: component replacement

B. Diabetic proliferative retinopathy
1. Pathophysiology
2. Effect of pregnancy on disease process
3. Guidelines for therapy
   a. Background retinopathy is not a contraindication to pregnancy
   b. Patients require frequent examinations
c. Photocoagulation may be protective
d. Treat aggressively
e. Termination of pregnancy is rarely necessary

C. Peripartum cardiomyopathy
1. Incidence: 1/4,000 deliveries in the United States
   a. Maternal mortality: 25-30%
   b. Tends to recur in subsequent pregnancies
2. Criteria for diagnosis
   a. Presents in last month of pregnancy or first 6 months postpartum
   b. No other causes of heart failure
   c. Absence of prior heart disease
3. Risk factors for peripartum cardiomyopathy
   a. Multiparity
   b. African American descent
   c. Poor nutrition
d. Multiple gestation
e. Tropical climate
4. Management of peripartum cardiomyopathy
   a. Standard therapy for heart failure
   b. Immunosuppressive therapy
c. Allow future pregnancies only in select cases
d. Good candidates for heart transplant

clavicle, brachial plexus injury because of the traction that we put sometimes when we are faced with that difficult situation. Asphyxia. Maternal complications include extensive vaginal lacerations from trying to get that baby delivered, suburethral tears and uterine rupture. The uterine rupture is again from fundal pressure which, should be abandoned and is not helpful at all in shoulder dystocia.

Management of shoulder dystocia. I think, like uterine atony, we should have some stepwise approach in mind. I think a reasonable thing to begin with is extension of the episiotomy down to and probably through the rectum. I am sure everyone has had experience at repairing 4th degree lacerations and that shouldn’t keep us from delivery. We would hate to have soft tissue contribute to the outcome in this situation.

Suprapubic pressure. Again, not fundal pressure but suprapubic pressure to try to dislodge that anterior shoulder or rotation of the posterior shoulder. The thing that I personally have found most useful is the McRobert’s maneuver or the forced flexion of the maternal thighs up onto the abdomen and chest in a very forcible way. Delivery of the posterior arm has also been described. Deliberate fracture of a clavicle as well as Zavanelli maneuver. Has anyone here done a Zavanelli maneuver? A couple of people want to own up to it. I am still waiting for my first one. Actually, I will be very happy if I never have to or get to do a Zavanelli maneuver. The Zavanelli maneuver, remember, is when some portion of the baby is delivered and the baby is pushed back up into the uterus and then delivered by Cesarean section. Dr. Zavanelli reported this several years ago. He reported very good results in his first few cases. Other people have reported good results and then there have been some series showing some not so good results.

Prevention is something that we should always keep in mind. There have been several series lately looking at if we can really prevent shoulder dystocia and the disappointing use of ultrasound in helping us predict shoulder dystocia. We know that the bigger the baby is the more likely it is to have shoulder dystocia, particularly in patients of diabetic mothers because of how they have grown. By actually predicting it, we are just not very good at it. Many people suggest that a cutoff of 4,500 grams is reasonable in a nondiabetic patient and 4,000 or perhaps 4,200 grams in the diabetic patient. I think we should also think about looking at the head to body ratio. I think that 0.90 is a reasonable cutoff.

Umbilical cord prolapse I am sure everyone has seen. Incidence is 1 in 200. Fairly common. Perinatal mortality is 20-30% unfortunately. Cord presentation. We can divide umbilical prolapse into three categories and that is cord presentation where the umbilical cord lies below the presenting part with membranes intact. That is an ultrasound diagnosis generally, although I suppose it could be palpated. Occult prolapse which is pretty common, where the umbilical cord lies alongside the presenting part and may or may not be palpable. Complete cord prolapse. That is the patient that we all picture when we think of it, getting up to go to the bathroom with the umbilical cord dangling between her legs, the cord protruding through ruptured membranes in the cervix.

Risk factor really is anything that would allow the cord to slip by the presenting part such as an abnormal presentation. Cord prolapse occurs in about 10% of breech, prematurity because the cervix frequently dilates before the head gets low into the maternal pelvis, multiple gestation because of the high incidence of malpresentation, in hydramnios also the cervix tends to dilate before the presenting part gets low in the pelvis, CPD for the same reason, grand multiparity and pelvic tumors kind of speak for themselves. Long cord, low lying placenta, amniotomy itself and displacement of the fetal head also.

Management of cord prolapse includes vaginal examination to assist in making the diagnosis. Evaluation for possible vaginal delivery which is sometimes feasible. Knee-chest or Trendelenburg position. I kind of like knee- chest myself. Manual elevation of presenting part. Delivery by Cesarean section if it can’t be accomplished vaginally. It has been said in the past that if the cord is pulsating, the baby is alive and if the cord can’t be felt to pulsate that the...
An ultrasound examination was performed and the liver was found to be outside the body, the lower portion of the body was not visualized, legs weren’t visualized, nor to grow. This is the entire extent of this umbilical cord. This patient happened to present to us at 34 weeks of gestation with intractable preterm labor. In our evaluation, Amniotic band syndrome is, as you probably know, where the amnion can stick to some portion of the fetus cord or placenta and cause very minor defects such as doing ultrasound in an anomaly screen would pick up this finding.

or any type, can have representation of all three cell types and this one just happened to have multiple feet. Each of the blue slips of paper is behind a foot. For some reason, it grew that way.

Dr. Zavanelli first reported the Zavanelli maneuver in a situation such as this where there was a large sacral coccyegeal teratoma that was undiagnosed and unknown in a baby that delivered through and to the shoulders and could not be delivered further. That was his first case presentation of delivery. My suggestion is that hopefully doing ultrasound in an anomaly screen would pick up this finding.

Sacral coccyegeal teratoma can be very small, can be intra-abdominal, can be anterior to the spine, can also be such as this. It can be posterior to the spine or down in the region of the sacrum. They can be very small and insignificant or they can be large and life-threatening. In this situation, this baby died from hydrops which is a common cause of death in babies with large sacral coccyegeal teratomas. A teratoma, if it is a sacral coccyegeal teratoma, just like any teratoma, whether it is ovarian or any type, can have representation of all three cell types and this one just happened to have multiple feet. Each of the blue slips of paper is behind a foot. For some reason, it grew that way.

Amniotic band syndrome is, as you probably know, where the amnion can stick to some portion of the fetus cord or placenta and cause very minor defects such as amputation of a small part of the fetal anatomy like a finger or toe. Or it can cause very severe abnormalities such as body stalk abnormality, which this child unfortunately had, or something called short cord sequence where the baby can be actually tethered to the placenta and the umbilical cord never has the opportunity to grow. This is the extent of this umbilical cord. This patient happened to present to us at 18 weeks of gestation with intractable preterm labor. In our evaluation, an ultrasound examination was performed and the liver was found to be outside the body, the lower portion of the body was not visualized, legs weren't visualized, nor was the distal spine and this patient was counseled that this probably was not compatible with life and she chose not to have any heroic measures performed. As we told her was a possibility, the very short umbilical cord did tear during the course of the delivery and while certainly that wasn't a terrific outcome, at least we were able to know that ahead of time and offer her the option of nonintervention and the baby died during labor as we thought it might perhaps.

Mechanical catastrophes secondary to multiple gestation. The one that comes immediately to mind is interlocking twins. Pretty uncommon. Incidence of 1 in 1,000 of twins and remember that twins occur about 1 in 80 so pretty uncommon finding. However, in situations where we have a breech-cephalic set of twins, we should expect the interlocking twins about 1% of the time. 1 in 87. In a few series retrospective studies, for some reason 75% of the patients that had interlocking twins were primigravida. We have no idea why that it is. If vaginal delivery is attempted, the mortality rate is about 50%.

Mmonoamniotic twins. Something we should always rule out whenever we have twins. It should be ruled out sonographically. It occurs in about 3% of twins and it occurs embryologically associated with monozygotic twinning occurring after day 8 but before day 13. The mortality rate is 50-75% and the mortality is generally secondary to cord entanglement. The analogy that I use to explain it to my patients is if you take two big old dogs and you tie them up to a tree with a rope and go away and come back a few hours later, they are certainly not going to be as they were. The ropes will be all intertwined with the tree as well as with each other and that is the situation we really have to think about in monoamniotic twins.

Early delivery by C-section is generally recommended and what we have done as well. In fact, we hospitalize the patients at 24-26 weeks and test them doing nonstress tests twice daily. Since the nonstress test is to assess placental function, that is not really our question here, but it can help us in predicting worsening cord entanglement by worsening variable decelerations. We do that twice daily beginning at viability and then deliver the patient at 32-34 weeks by cesarean section. We have been very, very fortunate. In the last five years, we have not lost one of these that made it to viability. We have had some losses that occurred prior to viability.

Phenobarbital or other means of fetal sedation has been suggested and has not been statistically shown to be beneficial but I think it is a good idea to give consideration to. This is a set of monoamniotic twins that unfortunately didn’t make it to viability. You can see the incredible degree of intertwining of the umbilical cords of these two monoamniotic twins.

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Conjoined twins. How many people have seen conjoined twins? Just a couple. I have seen two so I ought to be set for awhile. The incidence is 1 in 200 to 1 in 400 of monozygotic twins for an overall incidence of about 1 in 50,000 births. It occurs with monozygotic twinning after day 18. It can be suspected on ultrasound or x-ray examination. The mortality rate is dependent on the type and the nature of the anastomosis. This is a set of thoracopagus conjoined twins joined at the chest that we had. Unfortunately they shared a heart. The surgeon was present at the delivery and prepared to evaluate the babies for separation but there was so much entanglement and abnormalities of the great vessels of the heart as well as the upper GI tract that the babies succumbed before we could give consideration to that more fully.

Traumatic catastrophes during pregnancy. Just like when someone is not pregnant, when a woman is pregnant she can sustain some form of trauma and that happens to about 7% of women sometimes during pregnancy. Trauma accounts for about 25% of nonobstetrical maternal deaths.
Blunt trauma is the most common, usually secondary to automobile accidents. It is something that we see pretty frequently and it may result in uterine rupture or placental abruption. The fetus is protected by amniotic fluid so actual trauma to the fetus is very uncommon although it has been reported.

Penetrating trauma is increasing worldwide. The enlarged uterus is protective to the mother because of displacement of the viscera. There is no need for surgical exploration probably. If the fetus is dead, if the entrance wound is below the fundus of the uterus, if a bullet can be localized radiographically within the uterus, if the maternal vitals are stable and if there is no other sign of visceral injuries such as blood in the bladder or GI tract. This was a near term male fetus who was unfortunate enough to absorb a gunshot wound to the chest when the mother was assaulted.

Major burns during pregnancy. Major burns are defined the same during pregnancy as they are outside of pregnancy and that is a full thickness burn of at least 10% of the surface area of the body. Burns happen to be the third largest cause of accidental death in the United States. Four percent of women with burns happen to be pregnant.

Complications include the same things that they do when one is not pregnant. That includes electrolyte disturbances, hypovolemia and hypotension, pulmonary problems due to inhaled irritants, infection and inadequate nutrition due to increased metabolic demands.

Specific to pregnancy from the maternal standpoint, there is about a 25% mortality if the burn is 50% or greater. There is a 100% mortality if the burn is 80% or greater. Gestational age does not appear to influence maternal survival. From the fetal standpoint, premature labor is very common in the first five days. Stillbirth is extremely high - 75% - and with significant burns 30% or greater probably because of acidosis metabolic changes, it is thought. The largest burn that was survived by mother and fetus was 58%.

Management includes multidisciplinary care with standard burn therapy. Daily evaluation of fetal well being because of the very high stillbirth rate. It the burn is 30% or less, the prognosis is pretty good. If it is 50% or greater, I think consideration should be given to elective delivery. C-section is not contraindicated and probably does not change the overall course of the maternal outcome.

Perimortem C-section. How many people have done a perimortem C-section? Several people. I have done one and that is entirely enough. I hope never to do another one. There have been about 500 reported successful cases. The fetal survival unfortunately is only about 15%.

General guidelines should be to determine the potential for survival prior to the procedure. In 1997, we consider that to be greater than 23 completed weeks. The fetus should be known to be alive prior to the procedure, hopefully. A classical procedure is performed. We should maintain CPR during the pregnancy, a surgical kit should be kept near any critically ill obstetric patient and do not compromise the mother by too early intervention.

It seems like we kind of go back and forth about that topic. Sometimes we talk about not intervening too early because of the medical/legal risk. The only lawsuits I have ever seen associated with it were because of the thought that someone may have intervened a bit too early. Lawsuits are very uncommon in this circumstance actually. Although, on the other hand there have been several reports by Steve Clark and some others that write about maternal pathophysiology that perhaps sometimes that can even be helpful to the mother’s resuscitative efforts by increasing the bolus of blood that we get from the contracting uterus and by taking the pressure of the uterus off of the compression of the inferior vena cava. I think that really has to be a judgment call between the obstetrician involved and the rest of the resuscitation team.

This is other medical complications occurring. Amniotic fluid embolism is something that we always have to think about. It is very uncommon. It occurs about 1 in 20,000 and yet while it is so uncommon it does account for about 10% of maternal deaths. That is just how lethal it is. It used to be said that if you didn’t die from it, it wasn’t an amniotic fluid embolism. I think that is less true than it used to be probably because of the degree of sophistication of our intensive care units and hopefully because of the enthusiasm and repetitive for making the diagnosis. But it still does have about a 61% maternal mortality. Not great but it beats 100% and a 24% maternal neurologic deficit for an overall intact mother that comes out of an amniotic fluid embolism alive and intact of about 15%. The fetal mortality is easy to remember and that is the same as the maternal mortality and that is about 60% if it occurs during the intrapartum period.

We’ve known from 20 or 30 years ago when somebody did some autopsies and reported that they found fetal squames in the pulmonary trunk in patients that died from this certain spectrum of findings. They found those fetal squames in a large number of those patients and said, this is probably from amniotic fluid getting into the maternal circulation and causing hypotension and cardiac arrest.”

A few years ago there was another study that said “We are not sure that those two things are necessarily related and a group of patients was allowed to have right heart catheterization. Routine normal patients with normal risk factors had catheterization of the right heart during labor. I am not sure how much they paid the patients to allow that. Not something I would volunteer for but they demonstrated fetal squames in about 50% of the patients, fetal cells present in maternal circulation in a large number of patients that went home two days later and did great after their cardiac catheterization was removed.

Maybe it doesn't have anything to do with amniotic fluid. Maybe it doesn't have anything to do with fetal cells and maternal circulation and that probably is true. Dr. Clark, he has national registry so if you have any cases you really should consider contacting him and see if they fit his criteria. He said that the patients that he is including in the registry, the first report was in 1995 and I am sure he will update that from time to time. The patients to be included should have acute hypotension or cardiac arrest, acute hypoxia, coagulopathy. It should occur during the labor and delivery period or during D&E and there should be no preexisting conditions such as pulmonary or cardiac disease.

These were all risk factors for amniotic fluid embolism or anaphylactoid syndrome of pregnancy as I think we ought to consider calling it now. That is that it happens more to older gravidas, it is associated with black patients with higher gravidity, higher gestational age, it is associated with Pitocin, duration of labor and the presence of meconium.

The only thing that was found to be significant in that series was that 67% of the time they were male fetuses and 41% of the patients had a history of drug allergy or a previous atopic response really contributing to the thought that the basis of the disease probably is immunologic at least to some extent. Clinical presentation is secondary to anaphylaxis and septic shock and hence the suggestion for the name.
Treatment really is the same as we have tried to do in the past and that is expedient diagnosis and treatment. Hemodynamic support with central monitoring as quickly as possible, respiratory support almost always requiring intubation and treatment of the present coagulopathy with component replacement.

Diabetic retinopathy might be an indication for termination of pregnancy in some patients. The damage to sight occurs because of damage to endothelial cell junctions which promotes leakage of plasma then leading to macular edema. There is also increased platelet adhesiveness. We know that is present in normal regular pregnancies but that can cause thrombosis of retinal capillaries in patients that already have neovascular disease. Retinal hypoxia causes the neovascularization and then on top of the platelet adhesiveness it can cause obliteration of those vessels and a rapid deterioration of the disorder.

Areas represent neovascularization with growth toward us of capillaries in the posterior chamber and here is blurring of the disk margin. Dibble et al has fortunately looked at this question and said that if there was no retinopathy prior to pregnancy, there was no progression to neovascular disease. If background retinopathy was present with steel wool hemorrhages or flame hemorrhages, 16% of those patients did progress to proliferative disease. In patients that had untreated proliferative retinopathy mostly looked at by retrospective analysis, 86% of those patients did progress to severe disease, severe proliferative neovascular disease during the pregnancy. However, if those same patients could be treated prior to pregnancy or early in pregnancy with laser coagulation, there was only a 17% incidence of proliferation of the disease to worsening retinopathies.

Background retinopathy is not a contraindication to pregnancy necessarily. Frequent examinations by an ophthalmologist is certainly very helpful in diabetic patients and mandatory. I think, in contemporary care. Photocoagulation before pregnancy is protective. Aggressive treatment during pregnancy may prevent further deterioration and we should really consider termination only for patients unresponsive to photocoagulation.

Peripartum cardiomyopathy. I have seen only one of these. The incidence is about 1 in 4,000 and I was surprised when I did this to find out how high an incidence that is. Maternal mortality is 25-50%. A significant incidence. It does tend to recur in subsequent pregnancies. Risk factors for peripartum cardiomyopathy include multiparity and higher order multiparity, African American descent, poor nutrition, multiple gestation and tropical climate.

Criteria for diagnosis of peripartum cardiomyopathy are that it has to present in the last month of pregnancy or the first six months postpartum, there cannot be other identifiable causes of heart failure and there cannot be prior heart disease present in that patient. Management includes really the same as any cardiomyopathy and that should be digoxin, diuretics and decreased sodium to decrease afterload, vasodilator therapy really for the same reason, anticoagulation, immunosuppression if endomyocardial biopsy does demonstrate an inflammatory myocarditis and we should really consider future pregnancies only in select patients. That is patients that have a normal sized heart with normal function six months after the occurrence. Heart transplant for patients that have progressive disease. Just like any cardiomyopathy, it really is the only cure for that progressive disease.