



**Fifth Annual Report
Of the
Maternal and Perinatal
Death Review Committee
To the
Chief Coroner
For the Province of Ontario**

September 2009

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This report was prepared by Dr. David Evans, Chairperson of the
Maternal and Perinatal Death Review Committee; and
Ms. Kathy Kerr, Executive Lead – Committee Management.

Introduction

In 2004, Health Canada's *Special Report on Maternal Mortality and Severe Morbidity in Canada* recommended that all provinces establish a specific maternal death review committee to review all maternal deaths. At the time of that recommendation, the Obstetrical Care Review Committee of the Office of the Chief Coroner reviewed only those maternal deaths that were referred by a Regional Supervising Coroner. Such referrals were requests for the assistance of the committee in the investigation of the deaths. Maternal deaths that were not referred to the committee were not reviewed.

The Office of the Chief Coroner for Ontario accepted the recommendations of the Health Canada report. Effective January 1st 2004, the name and terms of reference of the committee were changed to reflect the fact that all maternal deaths occurring in the Province of Ontario would be reported to the committee in addition to the neonatal deaths and stillbirths already referred by the Regional Supervising Coroners. The name of the committee was changed to the Maternal and Perinatal Death Review Committee of the Office of the Chief Coroner.

The committee's function is to provide assistance to coroners in the investigation of: (1) the deaths of all women who died "during pregnancy and following pregnancy in circumstances that could reasonably be attributed to pregnancy" (2) stillborn cases, and (3) neonatal deaths.

The Coroners Act of Ontario directs coroners to investigate deaths to determine who died and when, the cause of death and the circumstances surrounding it. Once these questions are answered, the coroner is directed to consider whether there are recommendations that could be made, either by the coroner or by an inquest jury, to prevent another death in similar circumstances. Findings of legal responsibility or conclusions of law are not permitted.

The Maternal and Perinatal Death Review Committee case reports are prepared for the Office of the Chief Coroner and are therefore governed by the provisions of the Coroners Act, the Vital Statistics Act and the Freedom of Information and Protection of Privacy Act. As a result, each case review included in the annual report is a summary without identifying details. The recommendations made to the Regional Supervising Coroner and relevant organizations and agencies are included with each case.

It is important to acknowledge that these reports relied upon a review of the records. The Coroner/Regional Supervising Coroner conducting the investigation may have received additional information that rendered one or more of the committee's conclusions invalid. Where that fact was made known to the chair of the committee prior to the production of the annual report, the case review was revised to reflect these findings.

Recommendations were made after a careful review of the circumstances of each death. They are not intended to be policy directives and should not be interpreted as such.

This report of the activities and recommendations of the Maternal and Perinatal Death Review Committee is intended to provoke thought and stimulate discussion about obstetrical care and maternal and perinatal deaths in general in the Province of Ontario.

Maternal and Perinatal Death Review Committee

Aims and Objectives

1. To assist coroners in the Province of Ontario to investigate maternal and perinatal deaths and to make recommendations that would prevent similar deaths.
2. To provide expert review of the care provided to women during pregnancy, labour and delivery, and to the care provided to women and newborns in the immediate postpartum period.
3. To provide expert review of the circumstances surrounding all maternal deaths in Ontario, in compliance with the recommendations of the *Special Report on Maternal Mortality and Severe Morbidity in Canada*.
4. To inform doctors, midwives, nurses, institutions providing care to pregnant and postpartum women and newborns, and relevant agencies and Ministries of Government about hazardous practices and products identified during case reviews.
5. To produce an annual report that can be made available to doctors, nurses and midwives providing care to mothers and infants, and hospital departments of Obstetrics, Midwifery, Radiology/Ultrasound, Anaesthesia and Emergency for the purpose of preventing future deaths.

The Maternal and Perinatal Death Review Committee reviews all maternal deaths in Ontario.

The maternal deaths are classified by the following criteria:

Ante-partum - >20 weeks gestation

Intra-partum - during delivery or immediately following delivery

Post-partum - < 42 days

This report does *not* review late maternal deaths occurring >42 days, and does not include them in the statistics for the year.

Methodology

Coroners and Regional Supervising Coroners refer cases to the committee for review. At least one member of the committee reviews the information submitted by the coroner and then presents the case to the rest of the committee. After discussion by the committee, a final case report is written consisting of a summary of events, discussion and recommendations intended to prevent deaths in similar circumstances. The report is sent to the referring Regional Supervising Coroner who conducts further investigation and issues recommendations where warranted.

When a case presents a potential or real conflict of interest for a committee member, a temporary member is named from another centre. Alternatively, the committee reviews that case in the absence of the member with the conflict of interest.

When a case requires expertise from another discipline, an external expert reviews the case, attends the meeting and participates in the discussion and drafting of recommendations, if necessary.

Committee Membership (2008)

Dr. Michael Dunn	Neonatologist (Level 3)
Dr. Karen Fleming	Family Physician (Level 3)
Dr. Robert Gratton	Maternal Foetal Medicine
Dr. Steven Halmo	Obstetrician (Level 2)
Ms. Susan Heideman, R.N.	Clinical Nurse Specialist
Dr. Robert Hutchison	Obstetrician (Level 3)
Dr. Sandra Katsiris	Anesthesiologist
Ms. Michelle Kryzanas, R.M.	Midwife (Rural)
Dr. Catherine MacKinnon	Obstetrician (Level 2)
Dr. Dilipkumar Mehta	Paediatrician (Level 2)
Ms. Linda Moscovitch, R.M.	Midwife (Urban)
Dr. Toby Rose	Forensic Pathologist
Dr. David Evans	Chairperson
Ms. Kathy Kerr	Executive Lead

The Maternal and Perinatal Death Review Committee (MPDRC) would like to acknowledge the valuable contribution of Dr. Karen Acheson, former Chairperson of the MPDRC. Dr. Acheson, Regional Supervising Coroner – Central Region – Guelph Office, was instrumental in expanding the focus and mandate of the committee to include all maternal deaths, as well as referred perinatal and stillborn deaths.

Summary of Cases Reviewed in 2008

This report includes reviews conducted by the Maternal and Perinatal Death Review Committee in 2008. Cases reviewed may involve deaths that occurred in previous years.

Total number of cases reviewed:	30
Total number of deaths reviewed:	30
Total number of recommendations:	46
Number of maternal cases reviewed:	8
Number of recommendations from maternal deaths:	3
Number of neonatal cases reviewed:	12
Number of recommendations from neonatal deaths:	24
Number of stillborn cases reviewed:	10
Number of recommendations from stillborn cases:	20

Case Summaries: Maternal Deaths

Case M-1

Cause of Death:

Undetermined: No anatomic or toxicological cause of death identified.

History:

The deceased was a 32 year old G₃ T₁ A₁ L₁ who was two weeks post partum after delivery of her second child. Her past health history included an uncomplicated pregnancy and term vaginal birth in 2004 and then a spontaneous first trimester miscarriage in 2005 requiring a D & C. She was allergic to Demerol, but otherwise her health history was unremarkable.

Two weeks prior to her death, she was admitted and delivered her second infant at 40 weeks and 3 days' gestation. She was delivered with forceps for a non reassuring foetal heart. She had a vaginal tear that was repaired, but otherwise her delivery and post partum period were unremarkable.

She was having vaginal intercourse with her husband when she complained of decreased sensation in her arms just before becoming unresponsive. Her husband called 911 and began to administer CPR. The EMS arrived six minutes after the call and started resuscitation attempts. This continued until she was transferred to the hospital where she was pronounced dead approximately one hour after her initial arrest. During the resuscitation, she remained asystolic and VSA.

Post mortem:

The post mortem examination was performed and did not find any evidence of abnormality and in particular, no evidence of trauma to relate to the cause of death. Toxicology studies were negative. The police report did not find anything unusual in their investigation.

Discussion:

This 32 year old woman died two weeks post partum of natural causes, exact cause unknown. The most likely cause of death would be cardiac arrhythmia.

This death was unpredictable and unpreventable.

Recommendations:

None

Case M-2**Cause of Death:**

Sepsis and associated DIC, due to Beta Haemolytic Streptococcus group A infection.

History:

The deceased was a 33 year old G₂P₂ who presented to the emergency department of Hospital A, sixteen days after the uncomplicated term vaginal delivery of her second child. She described a two day history of right lower quadrant pain that had gotten worse that evening, constant and made worse by movement. Vaginal speculum examination showed copious cream coloured, but non-foul smelling discharge. There was cervical motion tenderness, more to the right. She was treated with analgesics, antiemetics and antibiotics and instructed to come back the next day for a repeat CBC.

Approximately six hours later, she came back with increasing discomfort and abdominal findings of what appeared to be peritoneal signs and more localized pain into the right lower quadrant. She was hyperventilating and tachycardic, with a low grade temperature of 37.2. She was admitted with broad spectrum antibiotics after consultation with gynaecology. She had transient times of hypotension, corrected with intravenous fluids. Further testing revealed elevated liver function tests including a bilirubin of 60 and hypokalaemia. Because the patient was becoming increasingly ill and the limited resources for investigations at Hospital A, arrangements were made to transfer her to the intensive care unit at Hospital B. She was sent to Hospital B approximately ten and a half hours after she had come for her initial visit in the emergency department, and two and a half hours after she had come back for reassessment.

The patient was accompanied by nursing staff, as well as the emergency physician for this transfer. The diagnosis on transfer was septic shock, possibly secondary to pelvic inflammatory disease. Blood culture taken just before her transfer subsequently grew group A streptococcus with sensitivity to most of the usual antibiotics.

On arrival at Hospital B, the patient was tachycardic with a pulse of 144, low blood pressure of 90/70 and respirations of 24. She was afebrile and had an O₂ saturation of 86% on 50% O₂ by mask. She was awake and alert, but cyanotic with localized tenderness in the right lower quadrant. Her extremities were mottled and her pulses weak.

Investigations revealed a coagulopathy with increased International Normalized Ratio (INR) and partial thromboplastin time (PTT) and Red Blood Cell fragments on a peripheral smear. Imaging revealed mild interstitial pulmonary oedema and pelvic abdominal ultrasound and CT were unremarkable. She was felt at this time to be in septic shock, likely due to a septic source in her pelvic organs, with disseminated intravascular coagulation (DIC) and probably developing Adult Respiratory Distress Syndrome, and acute renal failure. She was aggressively managed with a modified version of broad spectrum antibiotics that would have covered the group A strep. Mechanical ventilation was initiated, fluid support was continued and blood product replacement for her coagulopathy was done. As her condition continued to deteriorate and there was some question as to whether surgical intervention may be helpful, arrangements were made to transfer her to the intensive care unit at Hospital C. She was sent there eleven hours after arriving at Hospital B and twenty one hours after she had originally presented to the emergency department at Hospital A.

Shortly after arrival at the intensive care unit at Hospital C, the patient was assessed by staff and the admitting diagnosis was toxic shock syndrome. Her prognosis was very grave. Gynaecology was consulted and the gynaecologist on call reviewed the patient, but felt surgical intervention would not help and would likely further complicate her case. A colleague in gynaecology oncology was asked for an opinion and he concurred.

Intensive care treatment continued, however the patient continued to deteriorate and after discussion with the family, active care was withdrawn approximately ten hours after admission to Hospital C. The patient died shortly thereafter.

Post Mortem:

Autopsy revealed the findings of acute and chronic salpingitis with focal fibrinopurulent exudates associated with a right peri-tubal congestion/haemorrhage. There was also acute inflammation associated with the episiotomy site. Acute focal right pyelonephritis and patchy acute pneumonia and bilateral pleural effusions. The final cause of death was due to sepsis and associated DIC, due to beta haemolytic strep group A.

Discussion:

This maternal death, two weeks after an uncomplicated delivery, was associated with an acute and unpredictable illness due to group A strep which advanced extremely quickly despite aggressive intensive care treatment. This death was unpredictable, unpreventable and there are no recommendations.

Recommendations:

None

Case M-3

Cause of Death:

Amniotic Fluid Embolism

History:

The deceased was a 38 year old G₃T₀P₀A₂L₀ who was admitted to the hospital for induction of labour at 39 weeks' gestation due to gestational diabetes. Her prenatal history was unremarkable, except for the gestational diabetes diagnosed at 30 weeks and treated with diet only. She was offered, and declined, amniocentesis for advanced maternal age. Weight gain, blood pressures and foetal growth all were within normal limits. She had serial non-stress tests due to the Gestational Diabetes Mellitus which were normal. Past medical history included surgery for a patent ductus arteriosus as a child in China. She was taking Materna and calcium in the pregnancy and had no known drug allergies.

Her labour was induced with a single dose of Prostin gel given vaginally. She was monitored for one hour and discharged with instructions. She returned to the labour room about four hours later with contractions. Artificial rupture of membranes was performed. Her labour was quite rapid with delivery occurring two hours and twenty minutes after re-admission to the labour room and after a twenty minute second stage. She received Nubain intramuscularly 90 minutes prior to delivery.

The birth was a spontaneous vertex vaginal delivery with no complications. A single nuchal cord was noted. She had a second degree tear and blood loss was recorded as 700cc at the time. One percent Xylocaine local anaesthetic was used for the repair. The placenta delivered spontaneously ten minutes after the baby.

The baby, a male, initially had poor respiratory effort and muscle tone. He was given Narcan and responded nicely. The baby weighed 3.06 kg and had Apgar scores of 4 and 9 at 1 and 5 minutes.

At approximately 30 minutes post partum, the nurses noted a heavy vaginal flow. The obstetrician was attending another birth, but ordered an IV bolus. Soon thereafter, the patient had a tonic clonic seizure involving the right side. After this, she was unresponsive. She did not respond to Narcan and never regained consciousness. A second IV was started and Hemabate was given intramuscular (IM) into the uterus by the obstetrician, but the bleeding continued and her blood pressure was falling. At one hour postpartum, her BP was 86/35. The obstetrician summoned help from other physicians in the hospital.

The patient went on to progressively deteriorate with respiratory failure for which she was intubated. This was followed rapidly by a cardiac arrest from which she was resuscitated. Investigations revealed bilateral chest infiltrates on X-ray post intubation, a metabolic acidosis, haemoglobin of 63 and an elevated sugar of 20. She was given adrenaline, sodium bicarbonate and insulin.

Lines were inserted with some difficulties due to her progressively shocky state. Her blood pressure could not be maintained with large doses of Levophed and Dopamine and she began to bleed from her IV sites. She was given blood, fresh frozen plasma (FFP) and vitamin K, platelets and cryoprecipitate were ordered, but she died before they were ready. Further lab tests confirmed the suspected Disseminated Intravascular Coagulopathy (DIC) with platelets of 95, International Normalized Ratio (INR) >10 and partial thromboplastin time (PTT) >200.

The patient continued to bleed from her genital tract and gastrointestinal system. Her pressure could not be maintained. The situation was discussed with her family and she was not given CPR with her next arrest. The patient died five hours and forty minutes after the delivery of her baby.

Post Mortem:

The autopsy revealed the cause of death to be amniotic fluid embolism and associated DIC.

Discussion:

This patient died suddenly from an amniotic fluid embolism. The incidence of this disorder is estimated to be one in 20,000 to 30,000 live births, with a maternal mortality of 30 to 90%. Amniotic fluid embolism may occur in the absence of any risk factors. Most commonly, amniotic fluid embolism occurs during labour and delivery or immediately postpartum.

Recommendations:

None

M-4

Cause of Death:

Amniotic fluid embolism

History:

The deceased was a 36 year old G₂P₁ when she was admitted for induction of labour due to an elevated blood pressure at 39 weeks' gestation.

The pregnancy had been uncomplicated, except for elevated blood pressure. At the first prenatal visit at twelve weeks, blood pressure was 140/100 with no proteinuria. The follow-up visits from 12

until 37 weeks showed pressures from 130-134/80-88. She had routine prenatal investigations, an Integrated Pregnancy Screening Test, oral glucose challenge test and serial ultrasounds which were all within normal limits. Although she had a history of low thyroid, she was not on medications in the pregnancy and Thyroid Stimulating Hormones (TSH) levels were normal. She was also known to have fibroids on her uterus and these were followed with serial ultrasound. She had an elevated Body Mass Index (BMI) of 36.

At 37 weeks' gestation, the patient's blood pressure at the obstetrician's office was 130/100 with 1+ protein. The patient was advised to stop work. An ultrasound, Biological Physical Profile (BPP) and blood work was arranged and came back normal.

The following week, at 38 weeks' gestation, her blood pressure at the obstetrician's office was 160/100 with 1+ protein. She was booked for a Biological Physical Profile (BPP) and lab tests at the hospital for the next day. She was started on Labetalol 200mg orally by mouth, twice a day (po bid) and induction of labour was arranged for three days later.

The following day, the patient came to triage after the ultrasound and complained of feeling shaky. Her pressure was 151/94, there was no protein in her urine and the ultrasound and Biological Physical Profile (BPP) was normal, except for some borderline polyhydramnios. The labour room was noted to be full at that time and she did have an induction time booked for two days later. She was discharged with instruction to return if there were any changes and educated on the symptoms of preeclampsia.

On the day of her booked induction, the patient came to the hospital, but refused to be induced. She had undergone a BPP that day which was 8/8 and she was very anxious about the induction due to her past experience of a long and difficult labour in her first pregnancy. The patient's pressures that day were 142-155/86-95 and her laboratory tests for pre-eclampsia were again all normal. She was seen by two other obstetricians that day as the usual obstetrician was away. Both encouraged induction due to being term with elevated blood pressures. When she refused, she was asked to increase her Labetalol to 200 mg orally by mouth, three times a day (po tid). She agreed to return the following day for reassessment and further discussion about induction.

The patient was reassessed the next day. Her pressures peaked at 156/107, but the BPP and lab tests, were normal. Her cervix was closed, but 40% effaced. She was given Prostin gel, 2 mg vaginally at 1900 hours. Because of poor weather conditions, she remained at the hospital after the gel was inserted. Approximately six hours after gel insertion, she returned to triage with cramps. Her IV was started and Penicillin G was given as she was Group B Streptococcus positive.

About two hours later, she had spontaneous rupture of membranes while still in triage. Her blood pressure was 166/93, the foetal heartbeat was 140 beats per minute (bpm) and she had contractions every two minutes lasting 40-60 seconds. She was moved by wheelchair to a labour room. While the nurse from triage was out of the room providing a report to the labour room nurse, the patient was left sitting on the side of the bed in the labour room with her family. She was noted to have a bowel movement and the family called for help from a passing nurse. This nurse noted that she was breathing short rapid breaths and asked her to lie down on the bed. The patient did not respond and

was helped to bed by the nurse and her family. Cervix exam revealed she had achieved an anterior lip. She continued with rapid grunting breaths and moaning, but was not responding to commands. The nurse called for assistance and summoned the obstetrician from the Caesarean room where he was about to commence a section.

The obstetrician arrived in the patient's room at about fifteen minutes after the rupture of membranes. The obstetrician found the patient's cervix to be fully dilated with contractions and asked for a vacuum to expedite the delivery. It was noted that the patient was struggling and unable to push when the vacuum was applied. It was noted by both the nurse and obstetrician that the patient's lips were turning blue. Oxygen was applied and oxygen saturation monitor was applied showing saturations of 46-60%. A code blue was called and chest compressions were commenced eighteen minutes after rupture of membranes.

The vacuum delivery had failed and forceps were requested by the obstetrician. A baby girl was delivered by low forceps twenty minutes after the membrane rupture. The baby required resuscitation and a code pink was called. Nurses from the Neonatal Intensive Care Unit (NICU) and the paediatrician on call attended the baby. The infant weighed 3.38 kg and had Apgar scores of 1, 4, 5 at 1, 5 and 10 minutes.

A full code was run on the patient with the aid of the emergency doctor, the on-call internist, the on-call anaesthetist, the obstetrical family doctor, the obstetrician, the on-call respiratory therapist and many nurses. The patient never regained cardiac output and was pronounced dead after 25 minutes of cardiopulmonary resuscitation (CPR) and about 45 minutes after rupture of membranes.

Post mortem:

The autopsy revealed the cause of death as amniotic fluid embolism.

Discussion:

This patient died suddenly from an amniotic fluid embolism. The incidence of this disorder is estimated to be one in 20,000 to 30,000 live births, with a maternal mortality of 30 to 90%. Amniotic fluid embolism may occur in the absence of any risk factors. Most commonly, amniotic fluid embolism occurs during labour and delivery or immediately postpartum.

Recommendations:

None

Case M-5

Cause of Death:

Post partum myocarditis.

History:

The deceased was a 35 year old G₃T₁A₁L₁ who died four days post partum after delivery of her second child.

Her past medical history included a term delivery by Caesarean section in 2001. At that time, the infant was affected by a streptococcal infection. The patient subsequently had a first trimester loss. Just four days before she was found deceased, the patient had a Vaginal Birth after Caesarean (VBAC) term delivery of a 4.08 kg male infant. Despite being group B streptococcus negative, she was treated with group B strep prophylaxis with two doses of penicillin during labour because she had an infant with a previous streptococcal infection. She had a temperature elevated to 38.2°C in labour and therefore given an additional dose of Clindamycin. The temperature declined to the normal range within a few hours of labour and remained normal until discharge the following day. There was no indication that she was unwell before labour, or post partum.

A friend was at the deceased's apartment assisting with care of the four year old sibling, as well as the newborn, while the deceased's husband was at work. The friend responded to a "noise" coming from the bedroom where the deceased was. The deceased was found lying in the bedroom on her side, unconscious and unresponsive. A neighbour attended, called 911 and initiated cardio-pulmonary resuscitation (having received CPR instruction) until the emergency personnel were on the scene approximately seven minutes after the call. The Emergency Medical Services attended the scene and found her Vital Signs Absent (VSA) and continued the resuscitation. She was transferred to the hospital where resuscitation efforts were continued to no avail. She was pronounced dead approximately fifteen minutes after arrival. From the time of the initial attempt at resuscitation, she remained VSA with no return of cardiac rhythm.

Post Mortem:

A post mortem examination, including a toxicology study, was done. The only abnormal findings were related to the heart which revealed multiple foci of myocardial myofibril necrosis with inflammation and patchy contraction band necrosis. These findings are compatible with peripartum myocarditis. The mechanism of death was presumed to be acute cardiac dysrhythmia. This is a rare entity in which the myocardium is likely injured by viral infection, a hypersensitivity reaction, or an autoimmune reaction

Discussion

This death was unpredictable, and unpreventable.

Recommendations:

None

References

Miede et al, Circulation 1990;81:922-928.

Case M-6**Cause of Death:**

Stab wounds to neck. Homicide.

History:

The deceased was a 24 year old woman who was involved in an apparent domestic dispute near her apartment on the afternoon of June 24th, 2005. An altercation occurred and she was stabbed in the anterior neck and collapsed on the street. She was found unconscious at 1654 hours and Vital Signs Absent (VSA) when the first Emergency Medical Services (EMS) crew arrived at 1700 hours. She was transferred to the ambulance as a level 1 emergency. She arrived at 1719 hours and was attended by four physicians, three Registered Nurses, two Respiratory Therapists, numerous EMS personnel and police. An endotracheal tube had been inserted by the EMS technicians at the scene. There were no vital signs, no pulse and the pupils were fixed and dilated at 7 mm on arrival to emergency room.

It was determined by direct laryngoscopy, that the tube was not in the trachea, so the endotracheal tube was removed and a re-intubation was carried out. When completed, it was found that the cuffed end came out through the large (approximately 5 to 7 cm) horizontal sharp force injury in the neck, below the larynx, which had virtually transected the trachea. The patient was subsequently re-intubated through the laceration into the distal portion of the trachea and the ventilation stabilized, with good tidal volume. There was evidence of several major vascular structures visible, but not actively haemorrhaging as the patient had no pulse.

The patient had bilateral chest tubes inserted, multiple intravenous lines and was given large volumes of fluid, including five units of packed red cells. Initial attempts were being made to secure and suture large arterial vessels.

A cardiovascular surgeon was consulted and a decision was made to proceed to the operating room for a sternotomy to allow better access to the trachea and large vessel injuries. The procedure started at 1805 hours and the patient continued to have significant, ongoing bleeding and profound hypotension. The ventricle was clearly fibrillating, so the patient was given open cardiac massage with intracardiac adrenaline and ongoing fluid management. During the assessment of the major injuries by the cardiovascular surgeon, the pH was determined to be between 6.6 and 6.7.

At that point, due to the continued haemorrhage, a decision was made to discontinue resuscitation. The patient was pronounced dead at 1818 hours.

Post Mortem:

Post mortem examination revealed multiple and generally minor, stab and incised wounds of the face, neck, left arm, right hand, right leg. The most serious was a stab wound in the front of the neck which almost completely divided the trachea and incised several blood vessels with resulting haemorrhage and aspiration of blood. There are several incised and stab wounds on the left arm and right hand consistent with defensive wounds. A median sternotomy for attempted surgical repair was present.

Other findings included a normal intrauterine pregnancy consistent with 22 weeks' gestation. The internal contents of the uterus were unremarkable with an anterior placenta which itself was unremarkable. The foetus was female and appeared quite unremarkable on external examination. There were no obvious congenital abnormalities or injury.

Forensic toxicology was done and apart from Lidocaine, which was likely used during the resuscitation, there was no evidence of drugs or poisons found.

Discussion:

This 24 year old pregnant female died as a result of a severe sharp force injury of the anterior neck involving the trachea and major vessels leading to haemorrhage and cardio respiratory failure.

She had expedient care and appropriate attempts at resuscitation, but had suffered wounds incompatible with survival.

The deceased was approximately 22 weeks pregnant at the time of her death. It is unclear whether it was known that she was pregnant prior to the assault received in the domestic dispute. The deceased had been the victim of previous abuse by the perpetrator in the past.

Recommendations:

1. Case to be referred to the Domestic Violence Death Review Committee of the Office of the Chief Coroner.

Case M-7

Cause of Death:

Amniotic fluid embolism

History:

The deceased was a 44 year old G₄P₁ with an estimated date of delivery of May 27, 2008. Routine prenatal laboratory investigations were normal. The patient declined genetic testing. Early second trimester ultrasound confirmed her dates. The 20 week ultrasound showed a low-lying placenta and uterine fibroids. Her antenatal course was complicated by gestational diabetes with a Glucose Challenge Test of 11. Blood sugars were subsequently well controlled by diet alone. Ultrasound at 31 weeks indicated polyhydramnios and again the presence of uterine fibroids, the largest measuring 4.6 cm. She was Group B Streptococcus (GBS) positive.

The patient's past obstetrical history included one term pregnancy delivered vaginally of a 3.3 kg female infant in 1994. She had early pregnancy losses in 2000 and 2006. Her past medical history was non-contributory. Induction was scheduled for May 17 at 38 weeks 4 days' gestation.

The patient received two applications of Prostin Gel on May 17, but did not go into labour. She returned to hospital on the morning of May 18 and was found to be having uterine contractions. The cervix was 3 cm dilated and completely effaced. She was given antibiotics for GBS and at 0930 hours, an epidural was placed. A controlled artificial rupture of membranes for a large amount of clear fluid was carried out at 1130 hours. A foetal scalp clip was applied and an Oxytocin infusion was started. She was fully dilated at 1415 hours. During the first stage of labour, there were some variable decelerations. These became more marked during the second stage with expulsive efforts and bleeding noted. Because of these developments, delivery was expedited with vacuum extraction. She was delivered with one pull of a 3.37 kg male infant at 1450 hours. The baby was noted to be pale. Apgars were 5 and 9 at one and five minutes and the baby subsequently did well.

The placenta was delivered at 1455 hours and the bleeding became heavy. The patient was given Misoprostol 300 ug per rectum and vigorous uterine massage for uterine atony. She was also given 5U of Oxytocin push IV and 50U in the IV bag run at 500cc per hour. A Foley catheter was inserted

into the bladder. Bleeding continued and at ten minutes after delivery, she was given Hemabate 250ug IM. Two subsequent doses of Hemabate were administered intramyometrially at 1515 hours and 1530 hours. It was noted that the blood was not clotting. She became tachycardic and hypotensive. A second IV was started at 1520 hours and a Disseminated Intravascular Coagulopathy (DIC) screen was ordered, but because of the appearance of the blood and her vital signs, packed red cells and Fresh Frozen Plasma and platelets were started at 1540 hours without waiting for the results. The CBC report 30 minutes later showed haemoglobin of 106 gm and platelet count of 121,000. The DIC screen done two hours later showed a Partial Thromboplastin Time >200 and International Normalized Ratio >10. At 1610 hours, the patient became unconscious and a “code blue” was called.

The resuscitation team arrived at 1615 hours. Chest compressions were begun at 1620 hours and she was intubated at 1625 hours. Epinephrine was administered via the endotracheal tube at 1627 hours. A femoral venous line was established and further doses of epinephrine were given intravenously. A pulse was re-established. At 1644 hours, her blood pressure was 121/56mm Hg and pulse 133 bpm. She was transferred to the ICU at 1707 hours.

In the ICU, vasopressors were given for circulatory support. Blood work done at 1728 hours showed haemoglobin of 51 gm, platelets 58,000 and International Normalized Ratio > 10. Blood gases showed a metabolic acidosis with pH 6.94, pCO₂ 19, HCO₃ -5 and lactate 13. Packed red cells, platelets, cryoprecipitate and Fresh Frozen Plasma continued to be infused in large amounts. A central line was placed in the right internal jugular vein. During the insertion, the patient demonstrated posturing with deviation of the eyes to the left in a disconjugate fashion suggestive of a possible intracerebral event. It was decided to proceed to hysterectomy to try to control the bleeding.

The patient was transferred to the operating room at 1830 hours and an abdominal hysterectomy was performed. She returned to the ICU at 2008 hours, but continued to bleed. At 2030 hours, the haemoglobin was 80 gm, platelets 24,000, International Normalized Ratio was 1.7, accelerated Partial Thromboplastin Time was 102 and fibrinogen was 1.34. The family was informed of the grave prognosis and a decision was made to continue resuscitation for another hour to try to reverse the DIC. Her condition continued to deteriorate and she arrested at 2135 hours. The patient could not be revived. Efforts were halted at 2142 hours and the patient was pronounced dead.

Post Mortem:

The post mortem revealed evidence of an amniotic fluid embolus and end organ findings that were consistent with shock and DIC. The abdominal hysterectomy site showed no localized complications.

The cause of death was amniotic fluid embolism.

Discussion:

The patient died from amniotic fluid embolism (AFE) causing Disseminated Intravascular Coagulopathy (DIC) and hemorrhagic shock. This is a rare, but often catastrophic obstetrical event, with an incidence of 1 in 20,000 to 1 in 30,000 deliveries and a mortality of 61-86%. Although a number of risk factors, including tumultuous labour, advanced maternal age, grand multiparity, instrumental delivery, abruption and induction of labour have been linked to AFE, it remains unpredictable and unpreventable. Rapid cardiorespiratory collapse occurs at presentation in the majority of cases, but may be preceded by non-specific symptoms including chills, nausea, vomiting and agitation. Haemorrhage as a result of DIC may occur at the same time as the initial cardiorespiratory symptoms or it may follow these symptoms. In this case, the DIC was the major clinical manifestation at the outset. As such, the management is directed toward sustaining the circulation, stopping the bleeding and treating the underlying coagulopathy. In this case, an astute obstetrician identified the coagulopathy clinically and appropriate treatment was initiated without waiting for laboratory confirmation. While intravenous fluids and blood products were being given to support the circulation and treat the coagulopathy, ecbolic agents and uterine massage were administered in an attempt to control the bleeding. Despite these efforts, bleeding continued and hysterectomy was resorted to. A uterine balloon tamponade device, if available in this unit, could be considered to temporarily help control uterine bleeding while trying to reverse the underlying coagulopathy or stabilize the patient prior to surgery. Given the rapidity of onset and the severity of the DIC in this case however, it is unlikely that the use of such a device would have impacted on the outcome.

Recommendations:

1. A central registry should be considered for the reporting of severe maternal and foetal adverse outcomes in association with the use of prostaglandin agents for cervical ripening and induction of labour.
2. Obstetrical units should consider having balloon tamponading devices readily available in the delivery suite for the treatment of post partum haemorrhage due to uterine atony.

References

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Case M-8

Cause of Death:

Invasive bronchopulmonary aspergillosis.

History:

This 17 year old G₁ patient was pregnant with an estimated date of confinement of March 20th, 2006. This date was confirmed by an ultrasound on November 23rd which showed her to be 23 weeks and 3 days pregnant. This information was used for her dating as her menstrual dates were unsure. Prenatal records indicate that she was seen for obstetrical care on December 5th, at 20 weeks, approximately 2 weeks after the 2nd trimester ultrasound scan. The last prenatal visit was on December 20th, at 27 weeks' gestation and she had a normal assessment at that time.

The patient was single, living at home with her mother and two sisters. She was living with friends before becoming ill and coming in for delivery. In the early pregnancy, there was admitted use of the drug Ecstasy. There was some question as to whether the patient had attempted an abortion in the first trimester by taking Tylenol and Ecstasy. The investigating coroner was advised that the patient only took two Tylenols and one Gravol early in the pregnancy, and not excessive medications as had been implied. It is believed that the patient had the full intention of keeping the baby.

The patient was admitted to hospital on January 30th at 34 weeks' gestation. She gave a history of a fever and feeling unwell for several days prior to arrival. She was in labour and ready to deliver when she was admitted. The baby was born by spontaneous vaginal delivery approximately fifteen to twenty minutes later. The infant male had Apgars of 3 and 6 at one and five minutes. Cord blood gases revealed arterial PH of 6.9, a base deficit of -22, and a venous PH of 7 and base deficit of 17. The baby was attended by paediatric staff shortly after birth. As the mother had a febrile

illness, the newborn was treated with antibiotics for the possibility of newborn sepsis. The baby subsequently did well.

On admission, the blood tests revealed a haemoglobin of 90, white cell count of 5.2, neutrophils 1.9, bands 2.3 and platelets 13. The patient's Partial Thromboplastin Time (PTT) was 40 and International Normalized Ratio (INR) 1.4. Platelets remained in this same range as did her PTT and her INR gradually rose to 1.8. Liver function tests revealed an AST of 938 on arrival and 2232 two days later. Total bilirubin was elevated at 159 and increased to 171. Her creatinine remained stable at approximately 150 and her blood pressure remained normal. With this abnormal blood pattern, the patient was presumed as an atypical Haemolysis Elevated Liver enzymes and Low Platelets syndrome (HELLP), or possibly hepatitis or fatty liver.

The patient was tested for herpes, toxoplasmosis, cytomeglo virus, paro virus, rubella, and hepatitis A, B and C. She had aerobic blood cultures, throat swab and urine cultures. All of this testing turned out to be negative.

Telephone discussions took place with a Maternal Foetal Medicine consultant and a local Internal Medicine consult was arranged on day two. It was thought that the patient likely had HELLP syndrome, plus Disseminated Intravascular Coagulopathy (DIC) and a possible sepsis. She was continued on broad spectrum antibiotics which had already been started. She was transferred to the intensive care unit.

The plan at that time was for transfer to a larger hospital if she deteriorated further. Due to her very ill condition, remaining febrile and with significant evidence of liver and renal dysfunction and coagulopathy, she was transferred to the other hospital.

The patient arrived at the hospital later in the day on February 2nd. She was assessed by the obstetrical service that had planned to coordinate her care. Because of her significant liver dysfunction, she was felt to require a hepatology consult with consideration of a possible liver transplant. She was therefore referred to yet another health centre.

On arrival at next health centre on the late afternoon of February 2nd, three days after initial presentation and precipitous vaginal delivery, she was assessed to be dehydrated, jaundiced, malnourished, with tachycardia of 118, hypotensive with blood pressure of 110/43, and febrile. She was alert, awake and oriented. Her chest showed bilateral base consolidation. At this time, her blood work showed a haemoglobin of 90, creatinine of 134 and elevated liver enzymes with AST 1886, ALT 395. The working diagnosis was acute liver failure, possibly due to HELLP syndrome. The plan was to hydrate, attempt stabilization and possibly do a liver biopsy.

The patient's status however, continued to worsen with further elevation of liver enzymes and with respiratory distress. She was transferred to the ICU and required the supportive measures by haematology, hepatology, maternal foetal medicine as well as the Intensive Care Unit staff.

On February 6th, the patient required intubation due to respiratory acidosis and developed acute renal dysfunction and further coagulopathy requiring parenteral nutrition. She remained intubated,

developing pulmonary oedema and bilateral pleural effusion and renal failure which required dialysis. She was treated with multiple blood products for coagulopathy. Neurologically she became more lethargic and drowsy. She was followed up with multiple organ imaging to rule out haematoma or possible liver rupture.

As the patient further developed significant haemodynamic deterioration requiring vasopressors, and without improvement of her coagulopathy, collaboration of the complete liver team, ICU team and transplant team recommended proceeding toward a possible urgent liver transplant.

With a suitable liver available on February 10th, the patient was taken to the OR. During a laparotomy, the liver was found to be oedematous and enlarged with a 10 to 20% fatty infiltration, splenomegaly, and bloody ascities and there was the presence of possible cholecystitis. A liver biopsy was performed and the report was normal liver, with mild portal inflammation.

A Cholecystotomy was performed because of a distended gallbladder. The abdomen was packed and left open because of what had seemed like a compartment syndrome associated with the ascities that had been present. The patient was returned to intensive care to continue the multi system treatment.

Later that evening, there was significant further deterioration with progressive hypoxemia, lactic acidosis, hypotension, and Adult Respiratory Distress Syndrome. The patient died of multi organ failure shortly after midnight on February 11th.

Post Mortem:

The pathology report indicates that the lungs showed pulmonary oedema and pleural effusions. Microscopic examination revealed invasive aspergillosis with aspergillus colonies in the bronchi, lung parenchyma and blood vessels. There was extensive diffuse alveoli damage.

The abdomen revealed serosanguinous ascities and the liver revealed microscopic histologic picture of septic liver. There was no evidence histologically of fatty liver. The kidneys showed histologic features of Acute Tubular Necrosis and a Disseminated Intravascular Coagulopathy like picture.

A review of autopsy and the negative toxicology findings indicates that the cause of death was “invasive bronchopulmonary aspergillosis in a post partum woman with liver failure probably due to HELLP Syndrome.”

Discussion:

This 17 year old gravida 1 patient was unwell for several days prior to coming to hospital at approximately 34 weeks’ gestation when she had a precipitous vaginal delivery. She had early evidence of illness involving her liver, kidneys and haematologic system. She had progressive investigation and medical support and was subsequently transferred to a tertiary centre.

During her hospitalization, her condition continued to deteriorate despite supportive measures which included parental nutrition, ventilation control, dialysis and repeated multiple blood product transfusion. She ultimately developed multi organ failure to the point where she was being considered for a liver transplant, which ultimately was not felt to be indicated. The patient died, despite these efforts on investigation and support, of multi organ failure without definitive predisposing cause.

The patient's death would appear to be due to pulmonary aspergillosis with systemic septic affects. This is an extremely uncommon disorder, particularly in the pregnant population; it is more often seen in an immune compromised patient. This death was due to an illness that was unpredictable, progressive, unrelenting, despite all reasonable medical efforts.

Recommendations:

None

Case Summaries: Neonatal Deaths

Case N-1

Cause of death:

Perinatal asphyxia with severe hypoxic-ischemic encephalopathy.

History:

The mother of the deceased was a 30 year old G₂P₀ with an estimated date of delivery of December 5, 2007. Routine prenatal laboratory investigations, intermediate pregnancy screening and glucose challenge test, were normal. Dating and second trimester ultrasounds were normal. At the last prenatal visit recorded on the Antenatal Record 2 at 37 weeks' gestation, her weight was 111 kg and symphysis-fundal height was 39cm.

Her past medical history included a D&C in 2006 for an incomplete abortion.

She was booked for induction at 40 weeks' gestation for suspected foetal macrosomia. The cervix was closed and the presenting part high.

The mother was given two doses of Prostin on December 5, however she did not go into labour and the cervix remained unfavourable. She was discharged home and returned on December 8. Prostin insertions were carried out at 1000, 1600 and 2200 hours. At 2232 hours, the foetal heart rate was noted as 85 beats per minutes (bpm). Oxygen was given by facemask and an IV was started. At 2238 hours, the foetal heart rate was 95 bpm. The obstetrician was called to attend. The strip was reviewed and orders were given to continue monitoring. At 2240 hours, the foetal heart rate was normal. At 2258 hours, Morphine and Gravol were given for pain and nausea. The foetal heart rate at 2343, 0235, 0354 and 0500 hours was normal. Labour did not ensue, although she was reported as having some contractions at 0235 and 0500 hours.

Electronic external monitoring was started at 0700 hours on December 9. The patient was comfortable and not aware of any contractions. The foetal heart tracing revealed decreased variability and recurrent decelerations. The obstetrician was notified of the tracing at 0800 hours while preparations were being made for Caesarean section. The cervix was examined at 0830 hours and found to be 2 cm dilated with the presenting part at sp-2 to -3.

Poor progression and detection of a non-reassuring foetal heart tracing prompted a decision to deliver the baby by urgent Caesarean section. Under spinal anaesthesia, the patient was delivered of a 3.766 kg male infant on December 9, 2007 at 0933 hours, at 40 weeks' gestation.

The paediatrician was in attendance at the delivery. The baby was limp, dusky and apnoeic. Resuscitative efforts were instituted with airway suction and bag and mask ventilation. The heart rate increased to more than 100 beats per minute, but the infant remained apnoeic. Endotracheal intubation with continued positive pressure ventilation resulted in an improvement in colour and some respiratory effort. At approximately four minutes of age, chest movement became restricted. The endotracheal tube appeared to be blocked and was removed. The infant remained pink with mask oxygen and appeared to have adequate spontaneous respirations, so he was not re-intubated. Apgars were 1, 4 and 7 at 1, 5 and 10 minutes. No cord blood gases were drawn. The baby was transferred to the Special Care Nursery for further management.

According to the nursing notes, the baby was receiving oxygen with bag and mask ventilation at twenty minutes of age. Blood sugar was low at 0.5 mmol/L and an infusion of D10W was commenced after intravenous access was secured. Complete Blood Count (CBC) showed a normal haemoglobin concentration of 221 g/L and an elevated white blood cell count at 49.2. Respirations remained irregular and oxygen saturation was difficult to maintain. According to the nursing notes, follow-up bedside glucose was 1.1 mmol/L.

A severe apnoeic spell occurred at approximately one hour of age and a Code Pink called. The paediatrician on-call attended and the baby was intubated after several attempts. The baby's peripheral perfusion was diminished and a bolus of normal saline was given. Oxygen saturation improved with Intermittent Positive Pressure Ventilation. An umbilical venous catheter was inserted after the peripheral IV line became interstitial. The first blood pressure recording was taken at about one and a half hours of age and was recorded as 72/12 in the left leg. Repeat blood glucose remained low at 1.3 mmol/L. A bolus of normal saline was given for ongoing hypotension and poor perfusion. Ampicillin and Gentamicin were given at 2 ½ - 3 hours of age. No blood cultures were drawn. The baby was placed on a ventilator and a call was placed to the children's hospital transport team to arrange transfer. A capillary blood gas taken at three hours of age indicated pH 7.18, pCO₂ of 53 mmol/L, HCO₃ of 20 mmol/L and base deficit of 8.8 mmol/L.

Documentation of the baby's clinical status is scant, but it appears that he was well-grown and demonstrated no dysmorphic features. He was initially described in the electronic record as lethargic and hypotonic, with poor reactivity on admission to the Neonatal Intensive Care Unit (NICU). Later description at three hours of age indicated some improved reactivity and reflexes. The discharge summary states that, "the baby remained stable until the children's hospital team arrived."

The children's hospital transport team arrived when the baby was three hours of age. They described the baby as "fairly sick" and appearing dusky and "shocky". Blood pressure was initially undetectable, but was subsequently measured at a mean value of 36 mmHg. The umbilical venous catheter was resited and another bolus of normal saline administered. Generalized seizure activity developed when the baby was approximately four hours of age and he was treated with Phenobarb. Venous blood gases revealed ongoing metabolic acidosis with pH of 7.28, pCO₂ of 12.7 mmol/L, HCO₃ of 6.1 mmol/L and base deficit of 21 mmol/L. Serum lactate was elevated at 4.8 mmol/L and increased further to 8.4. Upon discussion with the attending Neonatologist at the children's

hospital and the family, it was decided that treatment with systemic hypothermia for neuroprotection was indicated. The warmer was turned off when the baby was about six hours of age to allow him to become hypothermic. An infusion of dopamine was started with subsequent improvement in blood pressure and perfusion. The baby was transferred to the children's hospital for further management and arrived when he was approximately ten hours of age.

Upon arrival at the children's hospital, the baby was said to be responsive to handling, but hypoactive and somewhat hypertonic. His rectal temperature was 32.8° C. He was ventilated in room air at low rates and pressures. Over the first evening, he exhibited a recurrence of seizure activity and was given additional anticonvulsant treatment with Phenobarb and Dilantin. His perfusion looked poor and vital signs were difficult to maintain, so the attempt at hypothermia was abandoned. He required additional inotropic support and showed evidence of renal dysfunction with poor urine output and elevated serum creatinine.

During his time at the children's hospital, the infant became increasingly neurologically obtunded, although function of other vital organs improved. Neuro-imaging and electrophysiological studies indicated severe central nervous system injury. Upon discussion with the family, the infant was extubated and passed away at three days of age.

Post Mortem:

The post mortem examination revealed the following significant positive findings:

- Perinatal asphyxia, post-resuscitation, with:
 - aspiration of squames with minimal, focal early pneumonia
 - myocardial necrosis, subendocardial, right ventricle
 - hypoxic-ischemic encephalopathy, severe

Death was attributed to perinatal asphyxia with severe hypoxic-ischemic encephalopathy. The pathologist stated that, "it is not possible from the microscopic autopsy findings above to determine with certainty fully hypoxic-ischemic stress on the brain or other organs, such as the heart, occurred in utero, soon after birth or both."

Discussion:

Obstetrical Review

The infant died shortly after birth from hypoxic-ischemic encephalopathy. The post mortem findings were not conclusive as to the timing of the insult. Clinically, antenatal events related to cervical ripening and the attempted induction of labour would be a potential setting for the occurrence of the hypoxic-ischemic insult.

The indication for induction at 40 weeks' gestation was stated as concern that the baby was large for dates. While the baby's birth weight was greater than the 90%ile, concern that the baby is large for dates is generally not considered a valid indication for induction according to Managing Obstetrical

Risk Efficiently. When induction is indicated, cervical ripening is required when the cervix is unfavourable. The use of prostaglandin E2 (Dinoprostone) vaginal gel is one of the modalities approved for cervical ripening and induction of labour. It is available in 1 and 2 mg doses. It is not clear from the medical record which strength was used.

The mother received two applications of Prostin gel on December 5. Foetal heart monitoring was normal according to the nursing notes. As labour did not ensue and there was no significant change in the cervix, it was repeated on December 8. Review of the foetal heart rate tracings for that day indicate a normal tracing after the first application, with little evidence of uterine activity from 1000 hours to approximately 1530 hours. After the second application at 1600 hours, the foetal heart was monitored until 1705 hours. Frequent uterine contractions were evident and the patient was experiencing cramps in her low back. The monitor was reapplied at 1925 hours. Frequent uterine contractions were again evident. The foetal heart was normal during this time. At 2227 hours, the cervix was 2cm dilated and 60% effaced. A third application was inserted and foetal monitoring re-applied. Shortly after the re-application of the monitor, a foetal heart rate deceleration occurred. A bolus of fluid was given IV and O₂ was given by mask. The obstetrician viewed the foetal heart rate tracing and felt it had normalized. The mother was given Morphine and Gravol for pain at 2258 hours. Oxygen was discontinued at 2320 hours. At 2342 hours, toward the end of the tracing, it is not clear whether the periodic changes in the tracing represent accelerations or decelerations. Given this pattern, consideration should have been given to continue monitoring.

Intermittent auscultation was performed at 0235, 0354 and 0500 hours, with the foetal heart in the 140 bpm range. When continuous foetal monitoring was re-instituted at 0700 hours, there were deep and recurring late decelerations. The nursing notes at 0740 hours described the tracing as non-reassuring. Concerns were discussed with the patient and she was asked to be open-minded about a Caesarean section. The obstetrician was not notified until 0810 hours. The decision to move to emergency Caesarean section was made at 0830 hours and the patient moved to the operating room at 0900 hours. Given the ominous nature of the tracing at 0700 hours, more expeditious delivery was warranted. It cannot be determined from this review whether there were other considerations in the delivery or operating room that impacted the delay or whether earlier delivery might have changed the outcome.

Neonatal Review

This infant died after withdrawal of life-sustaining medical therapy following severe hypoxic-ischaemic central nervous system injury. It appears that some hypoxic-ischemic process occurred prior to his birth. At birth, he exhibited depression which required active resuscitation. There was evidence of significant acidosis on the first blood gas.

Seizure activity occurred a few hours after delivery necessitating reintubation, respiratory support and treatment with anticonvulsants. The infant was transferred to the children's hospital for ongoing management. Clinical assessment, Electro Encephalogram and neuro-imaging confirmed the severe neurological injury. After consultation with the parents, withdrawal of life-sustaining medical therapy took place and the infant passed away shortly thereafter at three days of age. Post-mortem examination confirmed the findings of severe hypoxic-ischemic encephalopathy.

The paediatric team at the first hospital were alerted and present at delivery. Initial resuscitation with airway management and assisted ventilation was initiated promptly and effectively. When the endotracheal tube became blocked and was removed, it was felt that a steady respiratory pattern had been established. The infant remained extubated until a severe apnoeic spell took place at one hour of age, necessitating reintubation. Although there is no comment in the notes about abnormal movements in association with this spell, it may have represented a generalized seizure. Blood glucose was documented as being low as early as 20 minutes of age; treatment with intravenous glucose infusion was commenced, but the glucose level was not shown to have improved until several hours of age. No boluses of glucose were given by the team at the first hospital, despite documented low serum glucose on several occasions in a baby with abnormal neurological signs. Vital signs were taken, but no blood pressure measurement is shown until the baby was 1 ½ hours of age. Some blood work was drawn, but there were no cord blood gases and the first blood gas from the baby was not drawn until three hours of age. Documentation by the attending physician is minimal and there is little indication that the hypoxic-ischaemic encephalopathy and/or hypoglycaemia might be factors in the baby's clinical presentation.

The Acute Care Transport team from the children's hospital arrived promptly and assumed responsibility. Intervention with saline and glucose boluses, along with anticonvulsants and cardiotropic agents, was commenced. The team recognized that the infant might be a candidate for systemic hypothermia for neuroprotection and cooling was commenced when the infant was six hours of age. Unfortunately, the clinical condition of the baby did not allow for ongoing hypothermia, although ongoing conventional intensive care was continued.

Care provided by the children's hospital transport team and Neonatal Intensive Care Unit team was of the highest standard. When it was appreciated that the baby had sustained severe, irreversible neurological injury and that his long-term prognosis was grim, the team and family took steps to withdraw life-sustaining therapy. The infant died within a few hours of withdrawal of assisted ventilation.

While it is clear that this infant was compromised at delivery and it is likely that no conventional intervention applied postnatally would have been able to change the outcome, there are several aspects of the post-natal management by the paediatric team at the first hospital that deserve comment. With such a presentation, it would have been standard to draw cord blood gases if possible and if not, to draw a blood gas from the baby as soon as possible after birth. While it is possible that there were other demands at the time, closer personal attention by the attending paediatrician in the hour after delivery would seem to have been indicated. Detection of hypoglycaemia was met with a conservative response, even in the face of encephalopathy and possible seizures. Blood pressure measurements were not taken for some time after birth and it appears that additional measures to provide post-natal support of the baby's circulation may have been helpful. While it is doubtful that a more aggressive response to hypoglycaemia and hypotension would have made little difference to the eventual outcome, it is possible that these post-natal conditions contributed to the central nervous system injury sustained.

Recommendations:

1. Obstetrical care providers are reminded that induction of labour for foetal macrosomia is characterized as an unacceptable indication according to Managing Obstetrical Risk Efficiently (MORE).
2. The labour and delivery unit at _____Hospital should review their drill with regards to the timely management of an abnormal foetal heart rate tracing.
3. Obstetrical care providers are advised that foetal heart rate decelerations occurring during the first hour following the application of a cervical ripening agent, warrants extended monitoring.
4. Obstetrical and paediatric care providers are reminded that cord gases should be obtained at all deliveries. In their absence, an early set of central blood gases from the infant should be obtained when there is significant depression at birth and when active resuscitation is required.
5. Paediatric care providers are reminded of the need to monitor and aggressively treat severe and/or sustained hypoglycaemia in any neonate, especially those exhibiting signs of encephalopathy.
6. Paediatric care providers should routinely obtain central blood pressure measurements in compromised infants in order to apply appropriate interventions when necessary to adequately support the infant's circulation.

Case N-2

Cause of Death:

Intrapartum asphyxia.

History:

The mother of the deceased was a 22 year old G₂P₁ with an estimated date of delivery of September 17th, 2007. This date was based on an ultrasound on March 7th, 2007, showing the foetus to be 12 weeks and 4 days. Her dates were unsure.

The patient's past history included the delivery of a 3.29 kg female infant at 38 weeks' gestation in 2004. She was induced because of hypertension and had an uncomplicated vaginal birth.

In the current pregnancy, the patient was under regular prenatal care from the first trimester. On the first visit, her weight was 136.36 kg (300.6 lbs) and she was 162cm (5 foot 4 inches) in height. Her weight increased to 143.64 kg (316.7 lbs) at 36 weeks' gestation, two days prior to admission. She had elevated blood pressure throughout her pregnancy and was started on Labetalol 100 mg, twice daily, at approximately 14 weeks' gestation. She also used Flovent and Ventolin for asthma. She was diagnosed with gestational diabetes with a 50 gram glucose challenge screen at 28 weeks. She was referred to an endocrinologist, nurse educator and dietician. She subsequently went on insulin until the time of her delivery. She had her last ultrasound on July 26th, 2007 at 32 weeks and 3 days, by her original dating. The average of the measurements was determined to be equivalent to 34 weeks and 2 days, with an abdominal circumference equivalent of 36 weeks and 6 days. The estimated foetal weight at that time was 2.64 kg.

The patient presented to the hospital on August 24th, 2007 at 36 weeks and 4 days. She had a headache for five days. She had gone to the health centre earlier in the day and her blood pressure was elevated at 170/100 and urine was positive for protein. She was advised to go to the hospital.

Her blood pressure in triage was 181/100 and remained the same after two further repeats. She had no proteinuria at that time and her blood work for preeclampsia was normal.

In view of her chronic hypertension and the recent increased and persistent hypertension, and now at 36 weeks and 4 days' gestation, she was admitted and assessed. She was advised that she should have an induction of labour.

On admission, the patient had a normal blood sugar and this remained stable throughout labour under continuous insulin protocol. She was placed on antibiotics for strep prophylaxis and continued on the same dose of Labetalol.

The patient had an unfavourable cervix, so a Foley catheter was placed and left in overnight. Her induction was started with Syntocin in the morning. At the start of the induction at 0915 hours, the cervix was 2 to 3 cms dilated and the vertex was described as being ballottable at spines -3. She had an artificial rupture of membranes for clear fluid in the mid afternoon and an Intra Uterine Pressure Catheter was placed as it was difficult to monitor her contractions. She was fully dilated by 2130 hours. The foetal heart rate remained normal with appropriate reactivity throughout. She began pushing well at 2320 hours, although she had been pushing somewhat ineffectively over the previous two hours. The head was delivered to the perineum. Subsequently, the head was delivered and an attempt was made to deliver the chin. At 2340 hours, the patient was laid flat, a Roberts manoeuvre performed and suprapubic pressure applied. There was an attempt at delivery of the anterior shoulder which was impacted under the symphysis and attempt at rotation of the posterior shoulder, with successful delivery of the posterior arm. Further attempts to deliver the baby were unsuccessful over 6 minutes. The patient was asked to stop pushing and the Zavanelli manoeuvre was performed and returned the head into the pelvis. A foetal scalp clip was applied.

The foetal heart at 2351 hours in the operating room was between 120 and 145 beats per minute. An emergency Caesarean section was carried out under general anaesthetic at 2350 hours and the baby was delivered at 2358 hours. The mother's care, including finishing the Caesarean section and post partum care, were unremarkable.

The neonatal team was available at delivery for initiating resuscitation. The newborn was a male weighing 5.5 kg and with a distended abdomen. Apgars were 0 up to 20 minutes and a heart rate was detected at 24 minutes. It was decided that aggressive treatment be continued, however the prognosis was very guarded from the start. The baby was switched onto high frequency oscillation and admitted to the ICU for ongoing management. The umbilical cord gases at 0006 hours revealed arterial PH of 7.25 and base deficit of 7 and venous PH of 7.23 and base deficit of 7.4. Capillary gases at 0125 hours revealed a PH of 6.92 and a base deficit of 19.

The infant did not have any spontaneous movements, doll's eye, pupil response, gag, suck, babinsky, or response to stimuli. There were occasional gasping respirations from 1.5 hours of age. Of note on an initial examination, the infant had a distended abdomen and an enlarged occipital scalp fluid collection.

The infant developed multi system failure, with significant central nervous system (CNS) and myocardial dysfunction, anuria and significant persistent metabolic acidosis. Neurological consultation confirmed significant CNS depression, likely Sarnat 111, and the prognosis for recovery was felt to be guarded. The abdomen was tapped for approximately 450 ccs of chylous fluid (97% lymphocytes).

At approximately 22 hours of age, the infant had recurrent episodes of hypotension and bradycardia and developed a left tension pneumothorax requiring thoracentesis. Despite aggressive further resuscitation, he again deteriorated at 32 hours of age and at that time, after discussion with the parents; a decision was made to discontinue therapy. The baby was declared deceased at 1032 hours on August 27th, at 34 hours and 34 minutes of age.

Post Mortem:

The autopsy revealed a normally developed male infant of 37 weeks' gestation. There was evidence of macrosomia with a birth weight of 5.5 kg (12.12 lbs) and an autopsy weight of 5.9 kg (13.0 lbs). There was bruising of the arm and neck and a significant scalp haematoma that was felt to be consistent with attempted vaginal delivery. Severe hypoxic ischemic encephalopathy was found, as well as acute subendocardial myocardial necrosis. The placenta was described as being mature and greater than the 90th percentile for weight.

Pathological cause of death was determined to be intrapartum asphyxia, due to shoulder dystocia, due to macrosomia.

Discussion:

The mother of the deceased infant had a previous relatively unremarkable delivery of a normal-sized infant at term, three years before. In this present pregnancy, she had hypertension diagnosed and treated early and which remained quite stable until the time of admission. She already had a high BMI and she developed gestation diabetes for which she had expert assessment and monitoring and remained on insulin treatment until the time of delivery. There are no records available of her blood sugars during the antenatal period; however when she came into the hospital and throughout labour on their diabetic insulin protocol, her blood sugars were normal.

Approximately four weeks prior to admission for delivery, she had an ultrasound showing the infant to be large for dates, with an estimated foetal weight at that time of approximately 2.61 kg (5.75 lbs).

Over the subsequent four weeks, the baby gained approximately 2.9 kg (6.39 lbs), though about 0.45 kg (.99 lbs) would be attributed to the ascities. No further ultrasounds were done prior to the mother's urgent admission at 36 ½ weeks for hypertension and proteinuria. There was no compelling reason to do an ultrasound as continuous monitoring was to be performed. She was almost four weeks pre-term, so the excessive weight gain of the baby and the degree of ascities, would not have been considered or anticipated.

As she was approaching term, the appropriate decision was made to initiate induction due to the elevated blood pressure. She was followed closely and appropriately during labour, with no evidence of concern until delivery of the head.

During the 2nd stage, she was pushing for approximately two hours, although it would appear that there was active pushing with contractions for only about half an hour. She delivered the head spontaneously, and at that point, it was evident that shoulder dystocia was occurring. Appropriate manoeuvres were attempted to relieve the dystocia, but delivery was undoubtedly impeded by the large size of the infant and the dystocia from the distended abdomen.

The efforts at delivery, and subsequently using a Zavanelli manoeuvre and Caesarean section, would have been extremely trying for the staff involved with her care.

Recommendations:

None

Case N-3

Cause of Death:

Craniocerebral trauma with perinatal hypoxic-ischaemic injury.

History:

The mother of the deceased was a 28 year old G₁P₀ with an expected date of delivery of March 14th 2007. She was a non-smoker with no significant previous medical history. Routine prenatal laboratory investigations and the dating and second trimester ultrasounds were all normal. The integrated prenatal screening and her glucose challenge test were also normal.

She began having uterine contractions at 1600 hours on January 30th at 34 weeks' gestation and she presented to the hospital at 2030 hours. The contractions were every five minutes, with no evidence of vaginal bleeding or fluid loss. The cervix was 2-3 cms dilated and the vertex was at spines -1. She was given Penicillin G 5 Million units IV and 12 mgm of Celestone IM and electronic foetal monitoring (EFM) was started. The recording from 2047 to 2130 hours was reassuring.

At 0005 hours, the cervix was 5 cm dilated and she was transferred to the Labour and Delivery room where she saw the obstetrician on call at 0015 hours. The cervix was found to be 8-9 cm dilated, with bulging membranes and an artificial rupture of membranes was carried out for clear fluid. The EFM was restarted at 0021 hours and the tracing showed deep decelerations. At 0030 hours, the cervix was 9 cm dilated and the fluid was blood tinged so oxygen was started and the patient encouraged to push with her contractions. At 0102 the amniotic fluid was heavily blood tinged and there were 2+ clots and the Electronic Foetal Monitoring was non-reassuring. She was taken for an immediate Caesarean section in view of the possible placental abruption and the non reassuring tracings. Under a spinal anaesthetic, the uterine cavity was opened and a fibroid was felt above the head causing difficulty in accessing the head. On request, a charge nurse applied one push from below and following this manoeuvre, the head was extracted delivering a 1.82 kg male infant at 0130 hours.

The baby was limp and depressed at delivery. The paediatrician had been summoned and was in attendance. Bag and mask ventilation was commenced along with 15 seconds of cardiac compressions. The infant responded well with an improvement in heart rate and colour, tone, and spontaneous movement returned. A steady respiratory pattern was established by 3 minutes of age. Apgar scores of 2, 5 and 8 at 1, 5 and 10 minutes were recorded. No cord gases appear to have been drawn. Bruising and swelling in the right parietal area of the head was noted. The baby was transferred to the Special Care Nursery for further management.

Physical examination revealed an appropriately grown infant consistent in appearance with 34 weeks' gestation. Birth weight was 1.82 kg. There were no dysmorphic features. The baby was described as somewhat mottled with poor perfusion and obvious bruising of the face, head and neck. An intravenous line was placed and he was given a bolus of normal saline. Blood cultures were drawn

and antibiotics ordered. Initial Complete Blood Count showed a normal haemoglobin concentration of 207 g/L and platelet count of 157. Capillary blood gas drawn at 3 hours of age revealed a pH of 7.26, pCO₂ of 58 mmHg, pO₂ of 31 mmHg, HCO₃ of 25.4 mmol/L and base deficit of 3.0 mmol/L. The infant was observed over the day and was generally settled, although he was irritable with handling.

At 1730 hours, the infant was reassessed by the paediatrician on call after a repeat CBC revealed a drop in haemoglobin concentration to 129 g/L. Swelling, bruising and tenderness over the right side of the head were again noted. The baby was described as irritable. Because of the history and physical findings, an urgent CT scan was arranged. The scan showed a right parietal bone fracture that was slightly depressed with an overlying scalp haematoma. There was an associated intraparenchymal haematoma in the right parietal lobe of the brain measuring 1.6 x .9 cm and some adjacent subarachnoid blood. A small subdural haematoma at the level of the tentorium was also suspected. There was diffuse decreased attenuation and lateral ventricle effacement compatible with cerebral oedema. Because of these findings and the coincident drop in haemoglobin level, the paediatrician contacted the children's hospital at approximately 1800 hours to arrange for transfer and neurosurgical assessment. Transfer could not be effected right away due to bed availability. The baby remained relatively stable until generalized seizure activity was detected at approximately 2300 hours. The seizures were controlled with the administration of Phenobarb. A repeat haemoglobin showed a further drop to 119 g/L and a transfusion of packed red blood cells was ordered. The transport team arrived at 0130 hours on February 1, 2007 and the baby was transferred to the children's hospital. During transport, the infant had another episode of seizure-like activity and was given an additional dose of Phenobarb.

On arrival at the children's hospital, the infant was breathing regularly in room air with good oxygen saturation and normal vital signs. He was described as very sedated, with minimal spontaneous movement, some abnormal tone and depressed reflexes. The infant had a few apnoeic spells with desaturation that were managed with either stimulation or mask continuous positive airway pressure (CPAP). Bruising and swelling over the right side of the head was again noted and the back of the head was boggy and swollen. Urine output was normal. A repeat CT scan was requested and the infant was sedated, intubated and ventilated for the procedure. Electroencephalogram (EEG) and evoked potentials were ordered. A MRI was performed late in the evening on February 2nd.

The baby's neurological status deteriorated as he developed signs of increased intracranial pressure with bulging fontanelles. He became minimally responsive and dependent upon assisted ventilation. The infant's EEG was abnormal and somatosensory evoked potentials were absent. The repeat CT scan and MRI revealed multiple skull fractures, an undisplaced fracture of the right zygomatic arch, subdural and intraparenchymal haematomas, subarachnoid haemorrhage and diffuse hypoxic-ischaemic encephalopathy. The consultant neurosurgeon did not feel that surgical intervention was indicated and that the baby's prognosis was extremely poor. The Neonatal Intensive Care Unit (NICU) team met with the parents and recommended withdrawal of life-sustaining medical therapy. With their agreement, the infant was extubated on February 4th at 1500 hours. The infant was pronounced dead at 1715 hours.

Post Mortem:

The post-mortem examination revealed the following main findings:

- Multiple skull fractures in the right parietal bone with single fractures in the right frontal and left parietal bones
- Traumatic brain injury with
 - subarachnoid and subdural haemorrhage
 - contusional tears at the cortical grey-white junction
 - diffuse axonal injury
- Hypoxic-ischaemic brain injury, recent.

Death was attributed to “Craniocerebral trauma with perinatal hypoxic-ischaemic injury.”

Discussion:

This infant died after withdrawal of life sustaining medical therapy following the confirmation of severe neurological injury resulting from craniocerebral trauma. Delivery was by emergency Caesarean section following a non-reassuring foetal heart rate pattern and placental abruption. Difficulty extracting the infant was encountered and he was delivered only after several manual manoeuvres were undertaken. It is likely that one of these manoeuvres resulted in the skull fractures and concomitant injury to the underlying brain. The Central Nervous System (CNS) damage appears to have resulted from a combination of direct trauma, haemorrhage, increased intracranial pressure and hypoxic-ischaemic injury.

It is uncertain as to whether some of the CNS injury may have resulted from acute perinatal asphyxia, but it is likely that the head trauma accounts for most of the severe findings. Although there was evidence of an acute placental abruption at delivery and he was depressed immediately after delivery, he had a good response to a brief period of aggressive resuscitation. No cord blood gas results are available, but there was no evidence of significant metabolic acidosis on the first blood gas taken from the baby at three hours of age. There was also no evidence of significant associated end organ damage, despite what appears to have been profound CNS damage.

Care provided in the paediatric units at the first hospital and the children's hospital was of a very high standard. Resuscitation was promptly and skilfully applied. The infant was observed closely at the referring hospital, appropriate investigations were undertaken and treatments applied. When a serious CNS condition was recognized, transfer to the local tertiary NICU was requested and affected. The infant's condition continued to deteriorate and it became clear that he had sustained severe, irreversible neurological injury and that his long-term prognosis was extremely poor. It appears that the baby had sustained significant CNS injury as a result of the traumatic delivery and it is likely that no change to the postnatal management could have improved the outcome.

There appeared to have been some delay in having the infant transferred to the children's hospital as a result of limited bed availability. However, transfer did not appear to be urgent at that time as the infant's condition was relatively stable. As soon as a bed opened up, he was transferred and

assessed. No additional treatment to reduce the CNS injury was offered or felt to be indicated by the children's hospital team. If the transfer had occurred when first requested, it is very unlikely that any difference in approach would have been taken and the outcome would have been unaltered.

Recommendations:

1. The _____ Obstetrical Unit should review its policies regarding monitoring of women in premature labour.
2. Health Care Providers are reminded about drawing cord gases as soon as possible after delivery and especially where the infant appears to be compromised.

N-4

Cause of Death:

Perinatal Asphyxia.

History:

The mother of the deceased was a 29 year old G₂P₁ who was at 30 ½ weeks' gestation when she presented in labour to the hospital.

The mother was healthy with no medical or surgical history and no drug allergies. She was a non-smoker. Her only medication was a maternal vitamin. The mother's first baby was a 3.6 kg female delivered by Caesarean section for failure to progress in labour after post dates induction. She planned a trial of vaginal birth in this pregnancy. She had bleeding at 11 weeks' gestation. Ultrasound documented a twin gestation with demise of one twin. A 'significant chorio-amniotic separation' and particulate material in the amniotic fluid were noted on the 18 week ultrasound. A two vessel cord was also seen and a level 2 ultrasound was performed showing no associated anomalies. Routine prenatal laboratory testing was normal. A glucose challenge test was negative, but her Group B Streptococcus (GBS) screening was positive.

On August 22, 2004 the mother presented to the triage area with increasing contraction activity every three minutes, with good tone. The obstetrician on call assessed her and performed an artificial rupture of membranes at 0235 hours which showed thin meconium stained fluid with cervical dilation of 3 cm, 100% effaced at station spines -2 cm. Admission CBC was normal. She was admitted at 0300 hours and GBS prophylaxis was commenced with IV Ampicillin. External

Electronic Foetal Monitoring (EFM) was normal with baseline 150 beats per minute (bpm) average variability and accelerations noted.

An epidural was requested and was placed at 0421 hours. EFM was normal initially, then became difficult to trace. At 0450 hours the doctor examined the patient, found the cervix to be 5 cm, and placed a fetal scalp clip. EFM over the next 20 minutes showed some decelerations, both late and variable, but moderate variability and normal baseline. From 0510 to 0600 hours there were continued variable decelerations and a marked decrease in baseline variability to minimal 5 bpm.

At 0600 hours, there was a bradycardia to 60 bpm for two minutes. Vaginal exam at this time showed the cervix to be an anterior lip. The variability however improved to normal after the bradycardia. The patient was fully dilated at 0625 hours. For the next 20 minutes, there were frequent variable decelerations, but rapid return to baseline of 150 bpm and moderate variability. Pushing was commenced at 0645 hours. For the first 30 minutes decelerations persisted, but the baseline was maintained at 150 bpm.

At 0715 hours, the scalp clip tracing showed marked artifact and was discontinued and external monitoring restarted. The nursing staff changed at 0730 hours. At 0735 hours, maternal vital signs are recorded as pulse 143 and BP 89/73. No action appears to have been taken to correct this or to assume that the external Doppler was indeed picking up a foetal, rather than maternal, heartbeat.

The nurse's notes state that the obstetrician on call was in to visit at 0750 hours, at which point the patient had been fully dilated for 1 hour 25 minutes and pushing for 1 hour and 5 minutes. The obstetrician does not appear to have examined her at this time and no notes were written. For the next 90 minutes, the external tracing showed a baseline rate which varies greatly from 160 to 110 bpm, with marked variability. There are also many places where the tracing does not show any rate and many places where the rate detected could well be that of the mother. The tracing is technically poor and therefore very difficult to interpret.

At 0910 hours, the nurse's notes state that the obstetrician on call visited, but it does not appear that he examined the patient, or made any notes. At 0920 hours to 0930 hours, the baseline is down to 100-110 and the contractions are not being recorded.

At 0930 hours, the patient requested vacuum delivery after 2 hours and 45 minutes of pushing. The doctor was called and applied the vacuum at 0941 hours. The doctor's notes describe an easy application of the vacuum at spines + 1 in the LOA position and an easy pull, with no pop-offs over two contractions.

At delivery, the baby was pale and limp with no detectable heartbeat. A "Code Pink" was called and resuscitation commenced with bag and mask ventilation. The paediatrician and the rest of the "Code Pink" team arrived when the baby was one minute of age and assumed responsibility for the resuscitation. Cardiac compressions were commenced. Endotracheal intubation was successful and bag ventilation continued. Endotracheal epinephrine was administered and an umbilical venous catheter inserted. Sodium bicarbonate, IV epinephrine and a bolus of normal saline was administered with no response. After 30 minutes of full resuscitation, there had been no restoration

of heartbeat or any spontaneous movements. A spontaneous heartbeat returned shortly after resuscitation was stopped. With this, resuscitative efforts were recommenced and the baby was transferred to the Special Care Nursery for observation and further management. Apgar scores awarded were 0, 0 and 0 at 1, 5 and 10 minutes. Cord arterial blood gas revealed a profound acidosis with pH 6.84, pCO₂ 105 mmHg, HCO₃ 17 mmol/L and a base deficit of 21.2 mmol/L. Cord venous gas was reported as pH 7.27, pCO₂ 43 mmHg and HCO₃ 17 mmol/L, but there is a written notation in the chart that the sample was not drawn until 45 minutes after birth.

The baby's birth weight was estimated to be 3.5 kg (post-mortem weight was 3.14 kg). She exhibited no dysmorphic features. A two-vessel umbilical cord was present. She was connected to a ventilator and a peripheral IV was started, blood culture drawn and antibiotics administered. A call was placed to the NICU and it was decided that transfer of the baby was not indicated at this time due to the poor prognosis. Initial capillary blood gas at approximately 90 minutes of age revealed a profound metabolic acidosis with pH 6.78, pCO₂ 33 mmHg, pO₂ 115 mmHg and HCO₃ 5 mmol/L. Complete Blood Count revealed no anaemia or evidence of overwhelming infection. Her neurological status remained extremely poor with no spontaneous movement and, upon further discussion with clinicians and the family, a decision was reached to withdraw life-sustaining therapy. The baby was extubated and passed away at five hours of age.

Post Mortem:

The post-mortem examination revealed with following main findings:

- Hypoxic-ischaemic injury of brain, adrenal glands and myocardium.

Death was attributed to "Perinatal Asphyxia et Sequelae". It was stated that, "there was no underlying anatomical lesion in the infant or the placental system that is the proximate cause of the perinatal asphyxia."

Discussion:

Due to her previous Caesarean section and epidural anaesthesia, continuous EFM was appropriately used to monitor the mother and her baby while in labour. However, during the second stage of labour, the scalp electrode was lost and not replaced. During the last 90 minutes of her labour, the tracing is technically poor and as a consequence, very difficult to interpret. In retrospective review, it is clear that the tracing was definitely not normal. It was at best, "atypical", but most likely "abnormal". Baseline variability was marked and there was a marked change in baseline rate from 150 bpm to 120 bpm as recorded by the nurse in attendance, but not investigated to assure it was not maternal. This change also does not appear to have been reported to the obstetrician. The obstetrician visited the mother twice during the last two hours of her labour, but does not appear to have examined her, or reviewed the tracing. The obstetrician made no notes until after the birth.

This baby died after withdrawal of ventilatory support following a diagnosis of severe central nervous system injury sustained in the perinatal period. While she was successfully resuscitated after birth, she never regained neurological function. The paediatric team performed effectively and

conducted a skilled resuscitation. When vital signs were restored, but the baby failed to recover neurologically, they provided accurate information to the family and requested support from the neonatal team at the children's hospital who reaffirmed the assessment and collaboratively developed a recommendation to withdraw support. The history, description of physical findings and lab results were sufficiently concerning for the neonatal team to determine that the prognosis for the baby was dismal and to recommend withdrawal without sending a team out to assess.

The cause and timing of the insult are unclear. It is clear that the baby was severely compromised at the time of delivery and no intervention applied postnatally would have been able to change the outcome. While the cord arterial blood gas indicated severe mixed acidosis, the cord venous sample was remarkably normal. There was one notation that the venous sample was taken at least 45 minutes after birth, possibly resulting in some compromise of sample integrity. There could have been a sample mix-up or lab error. Alternatively, such findings can be explained by complete, sudden umbilical cord occlusion. However, there was no notation of a cord prolapse or tight nuchal cord and, other than a two-vessel cord, there was no evidence on post-mortem examination of an "underlying lesion in the infant or placental system and this is the proximate cause of the perinatal asphyxia."

Recommendations:

1. Caregiver's are reminded that an Electro Foetal Monitoring strip, which is inadequate for interpretation, requires further investigation to rule out a maternal trace and application of a scalp clip if the heart tones cannot be reliably recorded with the external monitor.
2. Caregivers are reminded that in the presence of an "abnormal fetal heart rate pattern, usually operative delivery, should be undertaken promptly unless (1) there is clear evidence of normal fetal oxygenation by means of scalp pH assessment or (2) spontaneous delivery is imminent". SOGC 2007: Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline.
3. Caregivers are reminded that when assuming care for a woman in labour, a careful review of the chart should be undertaken to be familiar with the progress to this point and any issues which may have arisen earlier in the labour or in the pregnancy prior to the labour. Reviewing the foetal monitoring strips will allow the identification of baseline heart rate changes and changes in variability.
4. Caregivers are reminded of the importance of documentation of visits, exams and assessments as well as the current and future plan of care for each woman in labour.

N-5

Cause of Death:

Acute perinatal asphyxia associated with acute Staphylococcus Aureus Chorioamnionitis.

History:

The mother of the deceased was a 25 year old G₂P₀ with an estimated date of delivery of February 10, 2008. Her antenatal course was uneventful. She had one previous early pregnancy loss. Her past medical history was otherwise uneventful. Routine prenatal laboratory investigations, ultrasounds and Intermediate Pregnancy Screening, were normal. Findings at her last prenatal visit were normal.

The patient began having contractions at 0430 hours on November 7, 2007. They increased in intensity at 0800 hours. She was admitted to the labour and delivery unit of the hospital at 1126 hours. The cervix was 1-2 cm dilated, 50% effaced and vertex at sp-2. She was given a bolus of Ringer's lactate and started on Ampicillin 1 gm q6h. Celestone was also administered and a Nitropatch was placed as a Tocolytic. Arrangements were made for transfer to a tertiary care centre. However, prior to being transferred, recheck of the cervix found her to be 5cm dilated. The transfer was cancelled. Paediatrics was notified of the impending delivery and the children's hospital transfer team was called.

Due to difficulty in picking up the foetal heart with an external monitor, the membranes were ruptured at 1445 hours and a foetal scalp clip was applied. The amniotic fluid was lightly meconium stained. Deep variable decelerations were apparent and she was moved to the operating room at 1505 hours. An emergency Caesarean section was performed under general anaesthesia. She was delivered of a 0.87 kg male infant with Apgars of 1, 1 and 1 at 1, 5 and 10 minutes at 1526 hours, shortly after the arrival of the transfer team. The umbilical cord was around the neck once. The umbilical arterial cord blood pH was 7.29 and the venous cord blood was 7.31. The baby was intubated, but airway pressures were high and adequate ventilation could not be established. Clinically, sepsis was suspected. Resuscitation efforts were stopped at 15 minutes and the baby expired at 1730 hours.

Post Mortem:

This baby was appropriately grown with no anatomic abnormalities other than bilateral polydactyly of the toes. There was evidence of acute perinatal asphyxia with Grade 2 cerebral germinal matrix haemorrhage.

Post mortem blood culture grew Staphylococcus Aureus. Culture of the lung grew a scant growth of Staphylococcus Aureus. Examination of the lung showed no evidence of pneumonia or meconium. Examination of the placenta showed features of Acute Necrotizing Chorioamnionitis and Acute Funisitis. Placental cultures were negative.

The cause of death was acute perinatal asphyxia associated with acute *Staphylococcus aureus* chorioamnionitis.

Discussion:

This infant died in the setting of prematurity and evidence of chorioamnionitis. Although *Staphylococcus Aureus* is an unusual cause of chorioamnionitis, infection is a common cause of premature labour. There were no clinical signs of infection such as maternal fever or foetal tachycardia. Caesarean section was instead carried out for foetal heart rate decelerations in a timely fashion. The cord gases did not show any evidence of intrapartum asphyxia. During resuscitation, air entry was very poor despite fairly aggressive bagging. Post mortem findings did not indicate the cause of the clinical difficulties establishing ventilation.

Recommendations:

None

Case N-6

Cause of Death:

Septic shock in a premature infant with a Patent Ductus Arteriosus.

History:

The mother of the deceased was a 37 year old G₃P₁ with an expected date of delivery of March 21, 2007. Routine prenatal laboratory investigations were normal. First and second trimester ultrasounds were normal and integrated pregnancy screening was normal. Vaginal cultures for Chlamydia, Gonorrhoea and bacterial Vaginosis were negative and her glucose challenge test was also normal.

The mother's past obstetrical history included a previous miscarriage at 20 weeks (documented in the ambulance report) and premature labour and delivery of a 0.9 kg female infant in 2000. The Coroner's Investigation Statement indicates that the baby was stillborn, but the Antenatal Record I and II and the ambulance report indicate that the child was living. There were no records provided relating to this pregnancy and no further details documented in the Antenatal Record I.

Early on the morning of January 20 at 31 weeks' and 1 day gestation, the mother developed vaginal bleeding. Her husband called 911 at 0148 hours. She was transferred to the hospital and was admitted at 0210 hours. On examination, the foetal heart rate was normal and the cervix was 6 cm dilated with the vertex at spines -1. The delivery summary indicates that the membranes ruptured

spontaneously at 0130 hours. On admission, the mother's temperature was 37.6 and a complete blood count showed a WBC of 9.6. She was given Celestone 12mg IM and Penicillin G 5 million units IV. The Paediatrician on call was notified.

The foetal heart rate was 150-160 beats per minute (bpm) during the first stage of labour. She reached full dilation at 0403 hours. The foetal heart rate was 110-120 bpm during the second stage. She went on to a spontaneous vaginal delivery of a 1.887 kg female infant at 0422 hours. The cord gases were pH 7.37 venous and 7.36 arterial.

The infant was delivered at 0422 hours on January 20, 2007 by spontaneous vaginal delivery at 32 weeks' gestation. Although there had been some vaginal bleeding, the foetal heart tracing had been reassuring and the baby was born in good condition. The paediatrician had been summoned and was in attendance. Apgar scores of 9 at 1 minute and 9 at 5 minutes were awarded. The baby's birth weight was 1.887 kg. Blood was taken, an IV was started and she was started on antibiotics. The infant developed some respiratory distress and was placed on nasal Continuous Positive Airway Pressure (CPAP) with some improvement. She required supplemental oxygen and was not settling, so a transfer to a Special Care Unit at another hospital was organized.

The infant appeared to be suffering from mild respiratory distress syndrome and required supplemental oxygen and (CPAP) until day four, on January 23. By that time, her respiratory distress had improved and she tolerated weaning to room air. She managed to tolerate coming off CPAP, but remained tachypnoeic. During this time, she had been nil per os, receiving Total Parenteral Nutrition as her nutritional source.

By the next day, a systolic murmur was noted and pulses were bounding. The infant was felt to have a patent ductus arteriosus (PDA), but was relatively stable. Blood culture from admission had been negative, so antibiotics were discontinued after five days. The paediatrician was asked to assess the baby because of ongoing tachypnoea and the suspected PDA. A chest X-ray was taken and was described as showing hazy lung fields, but normal heart size. Capillary blood gas showed pH of 7.26, pCO₂ of 51 mmHg, pO₂ of 42 mmHg, HCO₃ of 23 mmol/L and base deficit of 4.6 mmol/L. The infant's complete blood count was normal. The paediatrician concurred with the diagnosis of PDA, but felt that it was not clinically too significant. Nasal CPAP was restarted and a blood gas repeated a few hours later. An echocardiogram was ordered and apparently confirmed the presence of a PDA, but there is no report present on the chart.

By the following day, the infant's condition had not improved and a request was made for transfer to the children's hospital for assessment by a cardiologist and ongoing management. The discharge summary was prepared and transfer ordered, but she was not transferred. Although not stated in the infant's record, the investigating coroner indicated that the children's hospital was unable to accept the baby in transfer.

Over the next three days, the baby continued to exhibit signs of a haemodynamically significant PDA. Since transfer to the children's hospital was problematic, the paediatrician attempted transfer to another institution. Another hospital originally agreed to accept the baby in transfer as she had improved somewhat, but later in the evening of January 29, she deteriorated further with recurrent

apnoeic and bradycardic spells necessitating reinstitution of nasal CPAP. The transfer to that hospital was cancelled and the children's hospital was contacted again. The record states that the “transport team will transfer baby at earliest opportunity.” The infant’s condition stabilized initially, but she had several further spells with desaturation and the paediatrician on call was asked to assess her again. Capillary blood gas was taken and showed a pH of 7.17, pCO₂ of 61 mmHg, pO₂ of 47 mmHg, HCO₃ of 22 mmol/L and base deficit of 6.9 mmol/L. After several additional desaturation spells, the team decided to sedate and intubate the baby. Mechanical ventilation was applied. Repeat blood gas (arterial) showed worsening acidosis with pH of 7.07, pCO₂ of 76 mmHg, pO₂ of 35 mmHg, HCO₃ of 22 mmol/L and base deficit of 9.2 mmol/L. CBC revealed a low haemoglobin concentration of 117 g/L and low white blood cell count of 4.6 with 2.5 neutrophils and 0.6 bands. Blood culture was drawn and antibiotics ordered.

There is no further documentation by the physicians attending the baby from this point onward, so the course of events is not fully clear. However, it appears that the baby steadily deteriorated overnight on January 29-30 with the development of shock and acidosis. There were several manipulations of the endotracheal tube and two reintubations. Blood gases revealed progressive mixed acidosis. At 0247 hours, a call was received from the children's hospital stating that their team would not be able to come for the baby, but that the team from another children's hospital had been despatched. At 0315 hours, the baby was described as pale, jaundiced and mottled. A blood gas was sent that revealed a pH of 6.85, pCO₂ of 64 mmHg, pO₂ of 41 mmHg, HCO₃ of 11 mmol/L and base deficit of 22.7 mmol/L. Heart rate was 116 beats per minute and blood pressure recorded as 63/31. A small bolus of normal saline was given. The transport team from the second children's hospital arrived at 0415 hours and assumed care of the infant. No other notes pertaining to the infant’s care are available on the chart. The infant passed away at 0554 hours. Blood culture taken on the previous evening (January 29) was reported as being positive on gram stain for gram positive cocci (in short chains) and gram negative bacilli. No final identification of the organisms is present on the chart.

Post Mortem

The post-mortem examination revealed the following main findings:

- Blood culture positive for E coli, Enterococcus and coagulase negative Staphylococcus
- Patent ductus arteriosus, 3-4 mm diameter
- Enlarged kidneys with renal congestion
- Marked pulmonary congestion with no evidence of pneumonia

Death was attributed to “Septic shock in a premature infant with a patent ductus arteriosus.”

Discussion:

This infant died at 10 days of age from circulatory collapse, most likely due to nosocomial sepsis. Although she had been born prematurely at 32 weeks' gestation and suffered from respiratory distress syndrome, she had a relatively uncomplicated course for the first week of life. At five days of age, a murmur was noted, accompanied by a minor clinical deterioration. The paediatric team at the delivering hospital suspected a patent ductus arteriosus and tried on several occasions to have the baby transferred to another centre (mainly the children's hospital) for evaluation and ongoing management. It appears that there was considerable pressure on the system at that time and transfer was deferred on several occasions. The infant became more unstable on the evening of January 29 and, in spite of increasing the level of respiratory support and providing antibiotic treatment, she continued on a downhill course until she passed away in the early morning of January 30, 2007. Although the transport team from the second children's hospital arrived prior to the baby's demise, she could not be stabilized and transported to the tertiary centre.

The clinical course leading to the death of this baby is most consistent with septic shock. Mixed bacteria were grown from the blood on post-mortem culture and bacteria were also identified on the gram stain of blood submitted for culture from the baby the night before her death. Although nosocomial sepsis is usually caused by a single organism and mixed growth is usually suggestive of contamination of the sample, the consistent bacterial types recovered pre and post-mortem suggest that these organisms were pathogenic. Confirmation that the pre-mortem blood culture grew exactly the same bacterial types would lend further support to this assertion.

The source of the bacterial invasion is not clear. Mixed organisms such as these would usually come from an enteric source, but there was no evidence of a serious intestinal pathology such as necrotizing enterocolitis. An indwelling intravenous catheter can be responsible, but usually leads to a pure growth of bacteria. There was no pneumonia on autopsy.

The role of the patent ductus arteriosus in this infant's death is unclear. A patent ductus arteriosus can result in ischaemia to vital organs such as the gut as a result of the systemic/pulmonary shunt, but there was no evidence of gut ischaemia in this case. However, the paediatric team tried in vain for several days to have the baby transferred to a tertiary centre for cardiology assessment. If transfer had been affected when first requested, it is possible that a different approach to management and/or different outcome may have resulted.

The paediatric team at the delivering hospital appeared to assess and manage the baby appropriately. They were clearly frustrated by their inability to transfer the baby to one of the local tertiary neonatal centres. It is likely that the baby was considered a relatively non-urgent transfer and was "bumped" by more urgent cases. Although her status became more acute on the evening before her death, there was still some difficulty mobilizing a team to collect her. By the time the team from the second children's hospital arrived, it appears that little could be done to change the outcome. It is unclear whether the shortage of tertiary NICU capacity during this time was due to exceptional circumstances or reflective of a chronic shortage in the system.

The documentation by the paediatric team as reflected in the chart from the delivering hospital is incomplete and difficult to follow. The physician on call who managed this baby on the night of her death wrote only one note. Findings, thought processes and steps taken, cannot be clearly gleaned from the notes. Nursing notes are brief and difficult to follow. Reports of imaging studies are absent. There is no final note from the responsible physician.

In summary, it appears that this baby girl died from septic shock acquired at approximately 10 days of age. She had been born preterm at 32 weeks with Respiratory Distress Syndrome, but had been successfully managed in the Level 2 unit at the delivering hospital when a complication arose in the form of a PDA; the team sought additional advice by requesting transfer to the local tertiary Neonatal Intensive Care Unit. The request could not be accommodated due to capacity problems. The baby then developed what appears to be an unrelated complication in the form of septic shock that eventually led to her death.

This was a high risk pregnancy based on the past history of a pregnancy loss at 20 weeks and premature labour and delivery at 28 weeks. The antenatal records do not indicate any particular concerns related to these risk factors. Given her risk factors, stopping work early, ultrasound assessment of cervical length in the second trimester, other investigations and interventions might have been considered.

It is unclear whether any difference in management would have resulted in a different outcome.

Recommendations:

1. Paediatric caregivers are reminded of the importance of clear documentation of their assessments, impressions and plans in the official record of patients for which they have responsibility.
2. The Acute Care Transport Service at the children's hospital should review its response to the requests for transfer received regarding this patient.
3. The Ministry of Health and Long Term Care urgently review the bed capacity for severely sick neonates needing transfer to neonatal units through out the province.
4. Obstetrical health care providers should discuss risk factors for premature labour and the benefits and risks of interventions that may improve outcome.

Case N-7

Cause of Death:

Foetal Hypoxia.

History:

The mother of the deceased was a 36 year old G₆P₅L₄ with an estimated date of delivery of February 24, 2007. Her antenatal course was uneventful. Ultrasounds at 10 weeks 6 days and 19 weeks 6 days, showed a bicornuate uterus.

The patient's first two babies were delivered vaginally and the last three by Caesarean section. The first pregnancy was a vaginal delivery of a footling breech female infant in 1991. The record seems to indicate that the baby died due to complications at the delivery. The second pregnancy was delivered vaginally of a 3.18 kg male infant at 37 weeks' gestation in 1992. Both of these deliveries occurred in Sudan. The third pregnancy was delivered by Caesarean section for a 2.73 kg male infant in Egypt in 1995. The reason for the Caesarean section appears to be placental abruption. The fourth pregnancy was delivered in Toronto at 37 weeks' gestation for a 2.05 kg male infant in 1998. The fifth pregnancy was also delivered in Toronto at 35 weeks' gestation for a 2.05 kg female infant in 2001.

The patient's past medical history indicates that she had hypothyroidism which was being treated with Eltroxin 0.15mg daily. The patient had undergone a female circumcision. Routine prenatal laboratory investigations were normal. The Antenatal Record 1 does not indicate her HIV status or whether prenatal genetic screening was offered.

Contractions began on January 10 at 1720 hours at 33 weeks 3 days. Membranes ruptured at 1745 hours. The patient was brought to the hospital at 1812 hours. The obstetrician arrived at 1822 hours. The foetal heart was believed to be 130 beats per minute, although it could not be determined whether this was the maternal heart rate. The baby was presenting as a frank breech and was trapped at the introitus. Delivery was affected at 1829 hours by incising the fused labia for a 1.59 kg female infant. Delivery of the placenta was accompanied by blood clots suggestive of an abruption. Apgars were 1 and 0 at 1 and 5 minutes. The baby could not be resuscitated.

Post Mortem:

Post mortem findings were that of an appropriately grown 33-34 week female foetus with no congenital anomalies.

Examination of the placenta revealed marginal insertion of the umbilical cord and a 9.5 cm marginal haematoma.

The cause of death was stated as foetal hypoxia with obstructed labour in association with preterm labour, secondary to marginal placental abruption.

Discussion:

This infant died as a result of intrauterine asphyxia associated with premature labour, placental abruption and outlet obstruction with a breech presentation. Although this was a high risk pregnancy given the patient's past obstetrical history, there were no interventions that could have impacted on the outcome.

Recommendations:

None

Case N-8

Cause of Death:

Perinatal asphyxia and shock secondary to antenatal haemorrhage (possibly Vasa Previa).

History:

The mother of the deceased was a 36 year old G₂P₁ who presented to the hospital on September 8, 2006 with antepartum haemorrhage and foetal distress at 40 weeks' gestation. She had planned to give birth in another hospital, in another city, where she had been receiving care from 34 weeks' gestation as she had hoped to use an epidural anaesthetic in her labour. The mother's pregnancy had been uncomplicated. Her routine antenatal blood work was normal. She had Integrated Prenatal Screening which was negative for Down's syndrome and for spina bifida. She had an ultrasound at 11 weeks for Integrated Pregnancy Screening and dates, and an anatomy scan at 20 weeks which revealed no abnormalities and was negative for placenta praevia. Her first pregnancy was in 2004 when she had a spontaneous vaginal birth of a healthy 3.16 kg girl. The patient was a healthy vegetarian woman, and a non-smoker who did not abuse drugs or alcohol. The only medication she used was Beconase for nasal stuffiness. She had no drug allergies.

The mother experienced vaginal bleeding around 2300 hours on September 8, 2006. She called the hospital and was advised to come to the Delivery Room as soon as possible. She arrived at approximately 2345 hours. The nurses noted large vaginal bleeding and applied a foetal heart monitor at 2353 hours. The foetal heart rate was under 100 beats per minute (bpm) and the nurses notified the obstetrician on call at 2355 hours to come right away. Mild tightening of her uterus was

noted by the nurses and a golf ball sized blood clot was passed and sent to test for foetal haemoglobin. The obstetrician arrived approximately 15 minutes later and found the foetal heart rate was 80 bpm and not recovering. The obstetrician made arrangements for an emergency Caesarean section with the patient and husband's consent. A small gush of lightly meconium stained amniotic fluid was noted as the mother was catheterized for the Caesarean. No further bleeding was noted after her arrival in the Delivery room.

The anaesthetist arrived at 0020 hours and general anaesthesia was given and surgery commenced at 0030 hours. The abdomen was opened through a lower midline incision and a low transverse incision was made in the uterus. It was noted that no amniotic fluid or blood was encountered on entry into the uterus. The baby was delivered at 0034 hours on September 9, and was flat at birth and passed to the nursery staff. The paediatrician arrived seconds after the baby was born. It was noted that the cord was very thin and essentially empty of blood, although a venous cord gas sample was obtained. There was no evidence of abruption. Blood collected from the vaginal flow on admission was reported to contain large amounts of foetal haemoglobin.

The mother made an uneventful recovery and was discharged home on the third day postpartum.

Summary:

This infant was born following an emergency Caesarean section due to severe ante-partum haemorrhage.

Blood clot from vaginal bleeding was screened for foetal haemorrhage and was positive. Apgars were 1, 3, 4 and 6 at 1, 5, 10 and 15 minutes.

At 0034 hours, the baby was described as 'flat' at birth with a HR of 30/min. He was given bag and mask ventilation for 7 min with an increase in HR to 80. At 0041 hours, the infant's mouth was suctioned and he was intubated with #3.5 ETT. The first gasp was noted at eight minutes of age.

At 0100 hours, spontaneous respirations were noted. There was slight improvement in colour, but the BP remained low and capillary refill time was poor. Vital signs on transfer from the birthing area were: Temp 35° C. HR 122, RR 66, BP 41/22 mean 27. (Normal BP 60-74/30-44). Examination revealed no spontaneous movements and no gag reflex. Eyes were open. He was ventilated at a rate of 60. Pressure of 4/5. The infant was bradycardic with HR of 93, O₂ Sat 73% (FiO₂ 100%). Cord Blood Gases were noted to have PH 7.08, pCO₂ 63, pO₂ 56, HCO₃ 18. IPPV was initiated and O₂ saturation improved to 90%. An N/G tube and UV line were inserted and blood glucose, BCC and blood culture were taken.

At 0130 hours, the HR not recorded, but was BP. 41/22 mean 27, O₂ Sat. 98% (100% O₂). The first saline bolus 30 ml was given over six minutes. Blood glucose was 0.6. 6 ml of 25%. Dextrose was given. By 0147 hours BP was 42/26, mean 32, RR 60 and O₂ Sat. was 98%. HR was not recorded. Blood glucose normalized at 4.7 and insertion of U.A. line was unsuccessful. Ampicillin/Gentamicin was given.

At 0210 hours, HR was 122, BP 44/19 mean 2, RR 60 and. O₂ Sat. was 99%. Blood work was done by a laboratory technologist, but the baby did not handle well; O₂ Sat. went down to 81%. The infant was given IPPV and returned to IMV (mechanical ventilation). A #2 bolus of 30 ml of saline given. Blood work taken earlier showed an Hgb of 119. Na+ 140 K+ 7.1. Cl- 110. Total CO₂ of 7.

At 1 hour 56 minutes of age, blood gases were critical: pH 6.8, PCO₂, 77, PO₂ 94, HCO₃ 9. Base deficit of 28.8 mmol. At 0240 hours, a chest X-ray and abdominal x-ray were done. Vital signs were: HR 123, BP 30/18, mean of 21 and O₂ saturation was up to 97%. Upper body was pink. By 0305 hours, the glucose was 4.4. 35 ml of packed cell transfusion was given at the age of 2 hours 41 minutes. The pre transfusion blood pressure was 44/19, mean of 28, and post transfusion was 32/18, mean of 22, showing no significant improvement.

By 0320 hours, the infant's temperature was 34.7 C. A warm blanket was placed under the baby and over the legs. A peripheral IV was inserted.

At 0350 hours of age, the HR was 117, BP 32/18, mean BP of 22, RR 60, O₂ saturation 98%, still showing low blood pressure. Meconium stained fluid was aspirated from the stomach. At 3 hours and 34 minutes of age, a Dopamine infusion was started at 5 microgram/kg/min.

By 0430 hours when the transport team arrived, the infant's temperature was normal at 36.2°C. BP was 47/37, with a mean of 31, and O₂ saturation was 86%. Blood glucose was normal at 3.4. Transport Team arrived. A physical examination showed the infant was neurologically unresponsive. Capillary blood gas again showed critical values of: pH 6.45, pCO₂ 92, pO₂ 124, HCO₃ 6.

By 0547 hours, repeat capillary blood gas showed deterioration in the acidosis and hypercapnia. pH was 6.42, CO₂ 132, pO₂ 38 HCO₃ 8.

During the hours of 0547 and 0700, there were no records from the transport team or the delivering hospital identified in the chart.

At 0700 hours, the infant 'crashed'. HR went down to 30 and the infant went into asystole.

Over a period of 20 minutes, between 0703 and 0723 hours (age 6 hours 25 minutes), the infant received continuous cardiac compressions and IPPV. A total of 7 doses of 1 ml (1:10000) of Epinephrine were given with no response. There was no pulse and the infant was apnoeic. HR was 0, and RR was 0. The infant was pronounced dead at 0723 hours.

Post mortem:

An autopsy was not done. Examination of the placenta showed a normal placenta with three vessel cords inserted close to the edge of the specimen. There is no comment about vessels in the membranes or lacerated vessels.

Discussion:

This baby died as a result of asphyxia and blood loss suffered as a result of antenatal blood loss, possibly from Vasa Previa. Foetal distress was diagnosed on presentation to the Delivery Room between 45 and 60 minutes after the bleeding had begun. The baby was delivered by emergency Caesarean section approximately 49 minutes after the mother presented to the Delivery Room. Surgery was commenced 35 minutes after the obstetrician was initially notified and the nurses identified foetal distress, and 20 minutes after the obstetrician's assessment.

This infant died of cardio-respiratory arrest due to significant ante-partum haemorrhage leading to birth asphyxia. The haemorrhage was just prior to the emergency Caesarian section. The infant was unresponsive at birth with low apgars. There was evidence of cerebral anoxia (HIE). While the resuscitation was appropriate in terms of procedures, there were concerns with the management of the infant's care. These concerns related to the following:

1. Hypotension

The first saline bolus was given appropriately 3 minutes after UV line insertion, but the second saline bolus was not given until after 55 minutes. The blood transfusion was given at 2 hours 45 minutes after the first bolus. During this time, hypotension was well documented in the presence of acidosis.

2. Acidosis.

Severe acidosis was documented on all capillary samples despite of the first saline bolus and Dopamine infusions. There appeared to be a delay in administration of subsequent saline bolus and the blood transfusion. The acidosis did not appear to be addressed adequately.

3. Electrolytes:

Serious Hyperkalemia (K+ of 7.1) was documented, but there is no record of any corrective treatment.

Vasa Previa occurs in approximately 1 in 2500 births and rapid intervention is essential for foetal survival. Although it may be diagnosed antenatally on ultrasound, it is not often identified until blood is noted at the time of membrane rupture. Rarely can foetal vessels be palpated in the membranes at the time of vaginal examination. Diagnosis may be confirmed with the finding of foetal haemoglobin in the vaginal blood, but in the face of foetal distress intervention, should not be delayed for such confirmation. Risk factors include in vitro fertilization, low lying placenta or second trimester placenta previa, marginal or velamentous cord insertion and succenturiate-lobed or bilobed placentas as well as multiple gestations.

Recommendations:

1. Neonatal resuscitation procedure needs to be reviewed by the medical team.
2. Recommendations and treatment documentation by the tertiary care centre should be recorded.

Case N-9

Cause of Death:

Massive, acute pulmonary haemorrhage.

History:

The mother of the deceased was a 31 year old G₂P₁ with an estimated date of delivery of May 11, 2007. Her antenatal course was uneventful. Routine prenatal laboratory investigations, second trimester ultrasound and Integrated Pregnancy Screening were normal. Second trimester Glucose Challenge Test was normal.

The patient's medical history was significant for distal congenital arthrogryphosis and she was referred for genetic counselling. She was advised that the chance of her baby having arthrogryphosis was low, up to 50%. She was advised to have the baby's joints assessed on future ultrasounds. During the second trimester, the patient was to see a paediatrician for recommendations regarding prenatal care and postpartum follow up of the baby, but she cancelled the appointment. Ultrasound examinations, including biophysical profiles done at 35 and 40 weeks, were normal. Group B Streptococcus culture was negative.

The patient's past obstetrical history was a previous term pregnancy delivered of a healthy 2.975 kg female in 2006. Labour was induced at 41 weeks' gestation because of decreased amniotic fluid. Her family history was negative for arthrogryphosis.

Labour commenced spontaneously on May 19 at 40 weeks and 6 days' gestation. She was admitted to the hospital's labour and delivery unit at 0758 hours, under the care of a midwife. The cervix was 7 cm dilated with the vertex at sp-1. The foetal heart was normal.

At 0940 hours, the cervix was an anterior lip and membranes were bulging. ARM was performed at 1020 hours for clear amniotic fluid and she became fully dilated shortly thereafter. Monitoring by intermittent auscultation was normal during the first stage of labour. At 1035, 1045 and 1048 hours, auscultation of the foetal heart rate was attempted. At 1052 hours, the foetal heart was recorded at 130 beats per minute (bpm). The patient went on to spontaneously deliver a 4.085 kg male infant at 1055 hours. The cord was loosely around the shoulder. The baby cried spontaneously. Apgars were 9 and 9 at 1 and 5 minutes. The umbilical cord arterial pH was 7.22.

At approximately 5 minutes of age, the baby became cyanotic. The respiratory rate was 60/min. The paediatrician was called and the midwife gave the baby free flow oxygen. The paediatrician arrived at approximately 11 minutes of age.

Paediatric Summary:

This infant was born following a normal pregnancy and spontaneous vaginal delivery.

The cord gases were pH 7.22, pCO₂ 57, HCO₃ 24, Deficit – 6.3. Examination showed a healthy baby with a head circumference of 38.5 cm and length 56 cm.

The infant was given to the mother for 'skin to skin' contact. Within 5 minutes of age, the infant became cyanosed. He was transferred to an infant warmer and given free-flow oxygen.

The heart rate was 120 bpm and the infant was centrally cyanosed. The respiratory rate was 60/min, tone was satisfactory and he was crying. Continuous Positive Air Pressure was initiated at 6 minutes of age and air entry was documented as bilateral and good. A call was made for the paediatrician. Pulse was noted to be irregular at 8 minutes of age. The Respiratory Technologist arrived at 14 minutes. The infant became bradycardic with an irregular heart rate of 70 bpm. The paediatrician arrived and subsequent management is documented in chronological order:

1112 Baby was intubated (#3.5) at 17 minutes of age. His heart rate was 123. The paediatrician and children's hospital transport team were called. The baby had no spontaneous movements during IV insertion.

1224 The infant was suctioned and reintubated. Poor air entry was again noted and he became bradycardic with a heart rate of 74. Fresh blood was seen coming from the nose and the endotracheal tube.

1130 First dose of epinephrine 1 ml (1:10,000) was given at age 35 minutes. Bradycardia was more severe with a decreasing heart rate to 58 bpm. Management was discussed with children's hospital.

The infant's heart rate remained unstable and more blood was noted in the endotracheal tube.

- 1137 Heart rate was down to 67 and chest compressions were commenced. A repeat dose of epinephrine 0.4 ml (1:10,000) was given intravenously. Blood pressure was unrecordable. Anaesthesia was now present.
- 1142 The heart rate improved to 103 and chest compressions were discontinued.
- 1143 At age 48 minutes, 40 mls of saline bolus was given and the infant was reintubated. HR was maintained at 119. Chest x-ray was done.
- 1146 The infant was placed on a ventilator and the oxygen saturation was only 60% and HR was 122.
- 1202 Umbilical venous line was established and a second bolus of 40 mls of saline was given.
- 1210 The infant had no perfusion to the feet, and respirations were still spontaneous.
- 1214 Umbilical arterial line was inserted and blood work obtained (complete blood count – blood gas and blood culture).
- 1215 Transfer team with neonatal fellow arrived.
- 1217 Positive pressure ventilation was continued, but pulses remained poor. Heart rate was 94 and oxygen saturation was 59%. There was no spontaneous movement.
- 1226 The infant's condition had deteriorated with a critical blood gas: pH less than 6.5, pO₂ 33, pCO₂ more than 130.
- 1227 4th dose of epinephrine 0.4 ml (1:10,000), and 10 mls of NaHCO₃ were given. Chest compressions were restarted with heart rate of 45 bpm. Complete blood count showed an Hgb 207, white cell count of 21.6 and platelets 197 x 10³.
- 1236 The heart rate was up to 129 bpm, but the baby had fixed and dilated pupils. Repeat blood gas was still critical: pH 6.6, pCO₂ 117, pO₂ 87.
- The infant's heart rate continued to drop down to 25 bpm and he became mottled and cyanotic. Chest x-ray showed a normal heart, normal lung volume, but hazy lung fields.
- 1300 At age 2 hrs 5 minutes, the vital signs were: Temp 35.2 C, heart rate 140 bpm, respiratory rate 40, BP was still unrecordable. The infant was maintained on the ventilator with rate of 20, pressures of 18/8 on 100% oxygen.
- In view of extremely poor prognosis, decision to withdraw treatment was discussed and consented by the parents.
- 1340 Baby pronounced dead.

Post Mortem:

Heart: Right ventricle showed moderate hypertrophy and mild dilatation. The left ventricle wall showed a thickness of 0.3 cm (0.5 ± 0.1 cm), which is small for the age. The pulmonary trunk was larger than ascending aorta and the Ductus Arteriosus was widely patent.

Respiratory System: There was marked pulmonary haemorrhage – intra-alveolar, septal and pleural. Pulmonary vessels were variably engorged, but otherwise unremarkable. The rest of the autopsy was normal.

This infant resulted from a normal pregnancy and was delivered vaginally at 40 weeks' gestation, with no complications. He subsequently died of massive, acute pulmonary haemorrhage. There was minimal abnormality of the heart, which did not contribute to his demise.

Discussion:

The pulmonary haemorrhage was idiopathic in this case. The incidence is 1-12 per 1000 live births. In high risk groups, the incidence is as high as 5 per 1000 live births and is often associated with a number of factors. The most common association is with very low birth weight premature infants who have experienced severe perinatal stress or birth asphyxia.

Less commonly, pulmonary haemorrhage can occur in term infants. The cause may be congenital infection, cerebral oedema, hypothermia, haemorrhagic disease of newborn and congenital heart disease with large left to right shunt. Treatment often includes ventilatory support using high positive and end expiratory pressure, transfusion of blood and blood products to correct haemostatic defects. These strategies are often ineffective in preventing poor outcome.

Recommendations:

None

Case N-10

Cause of Death:

Acute Hypoxic Ischemic Encephalopathy.

History:

The mother of the deceased was a 30 year old G₂P₂ with a history of two previous term vaginal deliveries. She was a healthy woman with no significant past or ongoing medical problems. She had not had surgery, except for loop excisions of her cervix for dysplasia. She did report problems with her blood pressure in the previous pregnancy. She had routine laboratory tests, which were normal in early pregnancy. She declined Integrated Pregnancy Screening testing. She was Rh negative and received Rhogam at 32 weeks. Only one ultrasound, at nine weeks, is recorded on the antenatal records and it was consistent with her dates by last menstrual period. She was followed weekly from 34 weeks in the current pregnancy for concerns of increased blood pressure. Her blood pressure in early and mid-pregnancy was normal.

The patient was admitted at 40 weeks and 4 days' gestation for induction of labour due to post dates pregnancy with persistent hypertension with a blood pressure of 155/95. She was given Prepidil gel at approximately 0930 hours. Continuous Electronic Foetal Monitoring (EFM) was continued for 1 hour and showed a baseline of 150 beats per minute (bpm) with moderate variability and accelerations. Initially, there were no contractions, but by 1010 hours, the patient was having uterine contractions every 2 to 3 minutes, which the nurse palpated as mild in intensity. The EFM remained reassuring and was discontinued at 1040 hours. The patient was encouraged to walk.

Intermittent auscultation was implemented and the obstetrician examined, and ruptured, the membranes at 1235 hours. The cervix was 2 cm dilated, 1 cm thick and the presenting part was at spines -2 cm. Copious clear amniotic fluid was noted. Intermittent auscultation was continued every 25 to 30 minutes until 1340 hours when a deceleration was auscultated and EFM commenced again. Variable decelerations were noted and reported to the obstetrician. At 1350 hours, an IV line was started. The tracing continued to show marked decelerations with each contraction to 60 to 90 bpm, with rapid return to baseline and large shoulders on each side of the contraction.

At 1426 hours, the nurse examined the patient and found the cervix unchanged and noted that no cord was palpated. The contractions were every 1.5 to 2 minutes with moderate intensity. The obstetrician was called to see the tracing at 1430 hours. The obstetrician arrived at 1440 hours, noted the large decelerations which were persisting and discussed the option of performing a Caesarean section with the patient. It was agreed to continue the labour. For the next 90 minutes, the decelerations continued with each contraction, but with varying depth and breadth. The baseline rate and variability remained normal. At 1613 hours, the nurse examined the patient and found the cervix to be 3 cm and thin. The obstetrician reviewed the progress at 1635 hours.

Between 1635 and 1715 hours, there were many complicated variable decelerations from 60 to 70 bpm, lasting for 60 seconds, with biphasic shape and slow return to baseline with prolonged secondary acceleration. In addition, there was a decrease in variability of the baseline to minimal, and an increase in the foetal heart baseline rate from 135 to 145 bpm, to 150 to 155 bpm. The obstetrician reviewed the progress at 1715 hours. By 1739 hours, the baseline was 180 bpm. At 1741 hours, the patient was 7 cm dilated and meconium stained fluid was noted for the first time. The patient was asking for pain medication and was given Demerol and Gravol at 1750 hours.

At 1805 hours, a vaginal exam conducted by the nurse revealed cord prolapsing through an 8 cm cervix. The nurse held the baby's head up and called the doctor who arrived immediately and confirmed the cord prolapse. The obstetrician asked for the paediatrician and anaesthetist to be called as well to prepare for Caesarean Section. The anaesthetist was found to be unavailable due to another case and a second anaesthetist was called. By 1810 hours, the patient was now fully dilated and pushing involuntarily. The foetal heart was very difficult to auscultate and felt to be about 60 bpm. Due to the lack of availability of resources for an urgent Caesarean and in consideration of the patient's two previous vaginal deliveries, the obstetrician decided to attempt vaginal delivery. Vacuum was used to expedite the birth vaginally, but abandoned after 10 minutes.

Upon arrival of the anaesthetist at 1822 hours, the patient was moved to the section room and given general anaesthetic. Surgery was commenced at 1828 hours and the baby delivered at 1830 hours. The birth was attended by the Special Care Nursery staff. Although cord blood gases were drawn, they were not tested due to an error in the labelling of the specimens.

The mother's post-operative course was normal to the time of discharge at 26 hour postpartum.

The baby girl was born in poor condition, limp and apnoeic. Because of the meconium stained fluid and poor reactivity, the baby was intubated by the Respiratory Technician and the airways were suctioned. Resuscitation with bag and mask ventilation was commenced and the baby was intubated at three minutes of age when she failed to improve. With positive pressure ventilation, the heart rate increased to over 100 beats per minute. No cardiac compressions or epinephrine were required. Apgar scores awarded were 1, 4 and 5 at 1, 5 and 10 minutes. The paediatrician arrived when the baby was 5-10 minutes of age. A gasp was noticed at 10 minutes of age, but regular respirations had not become established. Positive pressure ventilation was continued and an IV was started. The infant was transferred to the Special Care Nursery for further management.

Physical examination revealed a well grown, term female infant with a birth weight of 3.895 kg. There were no dysmorphic features. She was initially hypotonic and poorly responsive with minimal reflexes. A capillary blood gas drawn at 20 minutes of age revealed severe acidosis with pH 6.97, pCO₂ of 48 mmHg and HCO₃ of 11 mmol/L. At one hour of age, she developed rhythmical movements that were felt to be seizures and was treated with Phenobarb. Blood glucose was normal. Blood culture was drawn and antibiotics commenced. An umbilical venous catheter was inserted. A call was placed to the tertiary centre at the children's hospital. There were no beds available, so arrangements were made to retrieve the baby and transport her to another tertiary children's hospital. The team arrived when the baby was six hours of age. At this point, the infant had tolerated weaning of ventilation and FIO₂ and was breathing on her own. Blood gases had improved. She remained hypotonic with few spontaneous movements. The infant was transferred to another hospital by air ambulance, arriving when she was approximately twelve hours of age.

Upon arrival, the infant remained intubated, but was being ventilated at low settings. She was breathing spontaneously and vital signs were normal. Neurological examination remained very abnormal with hypotonia, depressed primitive reflexes and reactivity, absent gag and increased deep tendon reflexes. Seizure activity was noted again and she was treated with additional Phenobarb and the addition of Dilantin. There was evidence of end organ damage with decreased urine output,

increased serum creatinine and elevated liver enzymes. Subsequent investigations revealed findings consistent with severe hypoxic ischaemic encephalopathy. An electroencephalogram showed poorly organized background activity and electrographic seizures. A Magnetic Resonance Image of the brain performed at 48 hours was markedly abnormal with extensive abnormal diffusion and blurring at the grey and white matter borders. Consultation with the neurologist affirmed the impression of severe Central Nervous System (CNS) injury with a poor prognosis. It was felt that if the infant were to survive, she would be left with severe dyskinetic cerebral palsy. Upon discussion between the parents and the members of the team, withdrawal of life-sustaining medical therapy was felt to be the most appropriate course of action. The baby was extubated on the fifth day of life and passed away five hours later.

Post mortem:

There was evidence of “acute hypoxic-ischemic encephalopathy, severe and widespread, including brainstem and basal ganglia.” There was mild acute bronchopneumonia, severe congestion of the kidneys and autolysis of the right adrenal gland.

Death was attributed to “Acute Hypoxic Ischemic Encephalopathy.”

Discussion:

The mother had a labour that was complicated by atypical foetal heart rate patterns for many hours. It was not until the last two hours of her labour that there were reassuring factors, including normal baseline rate, variability and rapid return to baseline of the foetal heart after decelerations. There were however, some profound and prolonged decelerations that would indicate the potential need for urgent Caesarean Section at any time. Unfortunately, when the need for an emergency Caesarean arose, the anaesthetist on call was unavailable. Although it appears that a second call system for anaesthesia exists in this hospital, it does not appear that this doctor was notified in advance of potential problems in the labour room. The paediatrician on call was also not immediately available when the emergency occurred.

At 1613 hours, 90 minutes prior to the cord prolapse, the obstetrician assessed the patient's progress. A nurse's exam 20 minutes prior showed little progress at 3 cm, despite frequent contractions and four hours after Artificial Rupture of Membranes. The tracing showed repeated complicated variable decelerations and decreasing baseline variability and increasing baseline rate. In light of the slow progress and lack of imminent vaginal delivery, a Caesarean should have been reconsidered at that time. If delivery was not implemented, scalp gas sampling should have been immediately considered. For the last 45 minutes of the labour prior to identification of the cord prolapse, the EFM trace is abnormal with minimal variability and increased baseline, at times to 180 bpm, and recurrent complicated variable decelerations. Also at this time, meconium was first noted. It is not clear if the doctor was aware of these changes. It is possible that urgent Caesarean at that time, or even reassessment by the obstetrician and implementation of intrauterine resuscitation or measures to slow the contractions during the last 90 minutes, could have allowed a better outcome.

The decision to attempt vaginal birth and to use a vacuum extractor in such a situation is unusual, but given the description of the patient's urge to push after full dilation, it does seem reasonable. Unfortunately, the baby did not deliver expeditiously. The ability to comment further on this and other decisions is hampered by a lack of charting by the obstetrician on call. There are no notes, except by the nurses written contemporaneously during the labour. The dictated note by the obstetrician after the birth does not give insight into the doctor's interpretation of the EFM tracing, or the progress of labour, or the plan for after each reassessment. During the final bradycardia, the obstetrician's dictated note explains well the decision to attempt vaginal birth. However, the details of manoeuvres to expedite the delivery are missing, including the station and position of the foetal head and how, or if, the vacuum was applied, and if it was, how much traction, over how many contractions, was it used.

Cord blood gases analysis is recommended for all births by most guidelines and they can be important in guiding the resuscitation of the baby and in understanding the outcomes. In this case, although the samples were taken and sent to the lab, they were not analysed due to a labelling error. Unfortunately, the team was not notified of this problem and by the time it was discovered, the sample was too old for analysis.

Management of the infant by the nursery team at the birth hospital and the intensive care team at the tertiary hospital was of the highest standard. Although the paediatrician was not immediately available at the delivery, the Special Care Nurse and Respiratory Technician applied appropriate manoeuvres promptly and skillfully. When the paediatrician arrived a few minutes later, ongoing assessment and management was completely appropriate. Full support was provided until investigations could be performed that confirmed the presence of severe hypoxic-ischemic CNS injury. It appears that the baby was severely compromised at the time of delivery and that no conventional post-natal therapy could have improved the outcome.

Recommendations:

1. The _____ hospital should review the policy for notifying the on call anaesthetist and paediatrician of potential emergencies in the labour room.
2. The _____ hospital should review with the nursing staff and the obstetrician, the interpretation of EFM strips and protocols for intrauterine foetal resuscitation.
3. The _____ hospital and the obstetrician should consider more detailed documentation of the labour and birth by either an improved written labour birth summary, use of progress notes or dictation of procedures such as failed vacuum birth.
4. The _____ hospital should review the labelling of specimens and the protocol for notification of such errors.

Case N-11

Cause of Death:

Severe hypoxic-ischaemic encephalopathy.

History:

The mother of the deceased was a 26 year old G₁P₀ with an estimated date of delivery of September 21st 2006. Her antenatal course was uncomplicated with normal serology, antenatal screening and normal ultrasounds. Her only risk factor was a history of rare outbreaks of genital herpes. She presented to the hospital at 37 weeks' gestation on August 31st, with a 24-hour history of intermitted diarrhea and cramping. She was observed and then sent home as contractions settled and the cervix was unchanged. She returned later that day with increased contractions, but now the cervix was 3 cm dilated and fully effaced at sp-3. She was admitted at 2200 hours. She was monitored by intermittent auscultation, until she requested an epidural which was placed at 0300 hours on September 1st 2006. She then progressed to full dilatation at 0342 hours. The doctor was contacted to come in by the nurse. The doctor attended at 0400 hours and performed an artificial rupture of membranes (ARM) for clear fluid. The patient pushed for several contractions, but it was elected to expedite delivery with a vacuum as late decelerations were noted on the foetal monitoring. The vacuum was applied at 0415 hours and the baby's head was brought down to the perineum where the vacuum was removed and a left mediolateral episiotomy was made under local anesthesia. The baby delivered at 0422 hours. The paediatrician had been summoned just prior to delivery.

The baby was born in poor condition, limp with no detectable heart rate. Birth weight was 3176 g. Resuscitative efforts were instituted immediately by the nurse with bag and mask ventilation. A heart rate of approximately 100 beats per minute was detected at one minute of age and the baby's colour improved. The paediatrician arrived and took charge of the resuscitation when the baby was just over one minute of age. There were no respiratory efforts, so an oral endotracheal tube was passed without difficulty and intermittent positive pressure ventilation was continued. An intravenous was inserted and 10% Dextrose infused. The first gasp was noticed at about 20 minutes of age. The Apgar scores awarded were 2, 4 and 4 at 1, 5 and 10 minutes of age. The cord arterial blood gases revealed severe acidosis with pH 6.68; pCO₂ of 97 mm Hg; HCO₃ of 11 mmol/L and base deficit of 33 mmol/L. The baby was transferred to the Special Care Nursery for further management.

The baby was supported with assisted ventilation and received infusions of normal saline and sodium bicarbonate. Cultures were taken and antibiotics administered. The initial complete blood count was normal, while the blood gases revealed an ongoing severe metabolic acidosis that gradually corrected. Seizure activity was detected at just over one hour of age and Phenobarb was administered. A call was placed to the local tertiary neonatal intensive care unit (NICU) and the

transport team was dispatched. The team arrived at 0640 hours when the baby was just over two hours of age. The seizures continued and a dose of Ativan and an infusion of Dilantin were given. The parents were approached regarding consent for the ICE trial - a randomized trial of systemic hypothermia for the treatment of Hypoxic Ischaemic Encephalopathy. Consent was obtained and the baby was randomized to the normothermic group. Transport to NICU was undertaken. The baby arrived at the NICU at just over six hours of age.

On admission to the NICU, the baby's vital signs were normal, but he was neurologically obtunded. He exhibited few spontaneous movements other than tremors, had absent reflexes and was ventilator-dependent. An electroencephalogram (EEG) done shortly after admission was described as showing "no brain activity". He was deemed to have a hypoxic-ischaemic encephalopathy, Sarnat Stage III.

The baby's ongoing clinical course indicated severe neurological injury with minimal recovery demonstrated. Neuroimaging and EEG supported a diagnosis of severe hypoxic-ischaemic encephalopathy. The paediatric neurology service concurred, as did the full neonatal team. The baby's prognosis was deemed to be extremely poor and the parents were counselled that withdrawal of life-sustaining medical therapy was the most appropriate course of action. With their agreement, the endotracheal tube was removed on September 13, 2006 just after noon. The baby passed away at 1930 hours. The coroner was informed and an autopsy was not felt to be necessary.

Discussion:

The mother of the deceased had an uncomplicated antenatal course and presented in labor at 37½ weeks' gestation. She was monitored by intermittent auscultation initially and this was interpreted by nursing staff as reassuring. She received an epidural at 0300 hours on September 1st, 2006 and the monitoring from approximately 0320 hours onwards was non-reassuring. According to the nursing notes, the doctor was notified at 0340 hours and attended at 0402 hours. An artificial rupture of membranes was undertaken and pushing began, but a decision to expedite delivery was not made until 0415 hours. The doctor recognized the non-reassuring tracing, and a vacuum-assisted delivery was performed within 13 minutes of arrival. It is unclear if earlier intervention would have altered the outcome.

The baby boy died after withdrawal of life-sustaining medical therapy following severe hypoxic-ischaemic central nervous system injury. It appears that this injury occurred prior to his birth as he exhibited depression at birth, severe acidosis, end organ damage and encephalopathy. He was well-managed by the neonatal team. The parents agreed to the withdrawal of life-sustaining medical therapy after the clinical assessment, EEG and neuroimaging confirmed the severe neurological injury. The infant died at 13 days of age.

The paediatric team responsible for resuscitation and stabilization initiated appropriate measures promptly and effectively. Ventilation and circulation were quickly established and effectively supported until transfer to the local Level 3 NICU could be affected. Seizures were promptly detected and appropriately treated. The NICU team recognized that the baby was a candidate for inclusion in a randomized trial of systemic hypothermia for neuroprotection; he was, however,

randomized to the normothermic arm of the trial. Care provided by the transport team and NICU team was of the highest standard and the parents were fully informed and involved. When it was appreciated that the infant had sustained severe, irreversible neurological injury and that his long-term prognosis was grim, the team and family took steps to withdraw life-sustaining therapy. He died within a few hours of the withdrawal of assisted ventilation. The baby was severely compromised by the time he was delivered and no conventional intervention applied postnatally would have been able to change the outcome.

Although hypothermia for neuroprotection of term infants with severe hypoxic-ischaemic encephalopathy has been the subject of intensive investigation over the past few years, it has not been universally accepted as something that should be routinely offered to families with an affected newborn. Most trials have suggested improved outcome for the group of infants treated with hypothermia, but it is not universally protective and many infants with Hypoxic Ischaemic Encephalopathy will do poorly despite the use of total body or head cooling. At the time of this baby's birth, the hospital was participating in the ICE Trial - a multicentre international randomized trial designed to examine this question. The evidence in favour of the efficacy and safety of hypothermia is accumulating and many centres are beginning to offer this therapy to babies with Hypoxic Ischaemic Encephalopathy under their care. However, the effectiveness in any individual infant cannot be accurately predicted and it is likely that, even if hypothermia had been applied, this baby's outcome would have been the same.

Recommendations:

None

N-12

Cause of Death:

Perinatal Hypoxic-ischaemic Encephalopathy.

History:

The mother of the deceased was a 26 year old G₁P₀ with an estimated date of delivery of February 27, 2007. The mother was married to her first cousin. An ultrasound at 6 weeks showed a bicornuate uterus. Routine antenatal laboratory investigations and second trimester ultrasound were normal. A Glucose Challenge Test and Group B Streptococcus culture were negative.

The patient's past medical health was unremarkable and her antenatal course was uneventful.

On March 5, 2007 at 40 weeks 6 days, cervical ripening with prostaglandin gel was carried out in preparation for induction the following day. The cervix was a fingertip dilated.

The patient was admitted to the hospital's labour and delivery unit on March 6 at 1100 hours with mild contractions. The cervix was 2 cm dilated and 80% effaced with the vertex at spines -1. An artificial rupture of membranes was performed at 1215 hours and Oxytocin started at 1430 hours. The Oxytocin was discontinued at 1518 hours because of foetal heart decelerations. An epidural was placed at 1615 hours. At 1637 hours, the cervix was 3-4 cm dilated. The tracing was reviewed by the obstetrician at 1720 hours. It was noted that there had been variable decelerations, some of which were prolonged. Presently, there was minimal variability, but no decelerations. The decision was made to restart the Oxytocin and the patient was advised that if the baby didn't tolerate the contractions, delivery by Caesarean section would be necessary.

The foetal tracing was reviewed by the obstetrician at 1930 hours and the labour was allowed to continue. At 2220 hours, variable decelerations were noted to have a late component. The patient was re-positioned and given oxygen. At 2215 hours, the Oxytocin was decreased. The obstetrician was informed.

At 2230 hours, the patient was fully dilated. The vertex was at spines -1. At midnight, the foetal heart dropped to 100 beats per minute (bpm) with good recovery to baseline. The obstetrician was informed and the Oxytocin was stopped.

At 0017 hours, the foetal heart dropped to 100 bpm for two minutes, but recovered to baseline. Decelerations continued and the obstetrician was informed at 0035 hours. The obstetrician was attending delivery of twins at the time.

At 0048 hours, the patient was preparing to push. Decelerations to 30-90 bpm, lasting 90s were occurring after contractions. The obstetrician attended at 0103 hours. It was determined that the delivery should be expedited given the foetal heart tracing. On examination, the presenting part was at spines +1. Manual rotation was carried out from OP to LOT and the presenting part came down to spines +2 with pushing. At 0108 hours, a mid-station vacuum was applied. After the third push with traction, a pop-off occurred and there was no descent. The patient was taken for emergency Caesarean section. The foetal heart was lost during preparation.

The Caesarean section was started at 0120 hours under anaesthesia, although the epidural had not been topped up and the anaesthetist was not initially in attendance. The patient was delivered of a 3.55 kg female infant through thick meconium.

The infant was born at 41 weeks' gestation on March 7, 2007 at 0125 hours by emergency Caesarean section. There had been a period of foetal bradycardia prompting an attempt at vacuum assisted vaginal delivery that was unsuccessful. The foetal heart rate was not detectable in the period immediately preceding delivery. The paediatrician had been summoned and was present to receive the infant. The baby was born in poor condition, limp and with no detectable heart rate. She was covered in thick meconium. Apgars were 0, 0 and 3 at 1, 5 and 10 minutes. Cord gases were

obtained, but could not be analyzed due to the sample being inadequate. The baby was intubated immediately and the airway suctioned for recovery of a large amount of thick meconium. The procedure was repeated, the baby re-intubated and bag ventilation commenced. It was difficult to achieve effective ventilation even with high bagging pressures and the heart rate remained undetectable. Cardiac compressions were commenced and high-pressure bagging continued. A dose of endotracheal epinephrine was administered followed by two additional doses when there was no response. A saline lavage of the upper airways eventually resulted in an improvement in compliance and adequate chest excursion with bagging. A heart rate of approximately 60 bpm was detected at 6 minute of age and continued to climb to over 100 bpm. Cardiac compressions were discontinued at this point. The baby's colour slowly improved, although no spontaneous movements were present at this time.

The baby was transferred to the Special Care Nursery for further management. An intravenous catheter was placed and a bolus of normal saline infused. An umbilical venous catheter was inserted and a second bolus of saline given. A dose of surfactant was given via the endotracheal tube and there was a further improvement in lung compliance.

The baby was placed on a ventilator. Examination revealed a normally grown female infant with no dysmorphic features. She was completely unresponsive to stimuli and exhibited no spontaneous movements. Additional circulatory support was provided by infusions of normal saline and a dopamine drip. Cultures were taken and antibiotics administered. Initial Complete Blood Count was normal. An arterial blood gases taken at approximately 90 minutes of age revealed pH 7.05, pCO₂ of 48 mmHg, pO₂ of 153 mmHg, HCO₃ of 13 mmol/L and base deficit of 19 mmol/L. Spontaneous respiration became established and it was possible to wean the ventilator settings. Seizure activity was detected at approximately one hour of age and Phenobarbital was administered. Blood pressure normalized and the dopamine was discontinued. A call was placed to the children's hospital to request transfer for ongoing management. The transport team arrived at 0325 hours, when the baby was three hours of age. The parents were approached regarding consent for the ICE trial - a randomized trial of systemic hypothermia for the treatment of Hypoxic Ischaemic Encephalopathy. Consent was obtained and the baby was randomized to the normothermic group. Transport to the Neonatal Intensive Care Unit (NICU) was undertaken and the infant arrived at just over seven hours of age.

On admission to the NICU, the infant's vital signs were normal, but she was neurologically obtunded. She exhibited limited reactivity and had no purposeful movements. She was hyper-reflexic and had tremulous movements that appeared to be stimulus-sensitive. A bedside Electroencephalogram (EEG) monitor was placed and showed severely abnormal background activity, no Sleep-Wake Cycling and recurrent short bursts of seizure activity. Additional Phenobarbital was given to control the seizures. She was felt to be exhibiting moderate-severe hypoxic-ischaemic encephalopathy and her overall prognosis was described as looking grim. Additional investigations to assess the degree of central nervous system injury were ordered.

The infant's ongoing clinical course indicated severe neurological injury with minimal recovery demonstrated. Neuroimaging and electrophysiological studies supported a diagnosis of severe hypoxic-ischaemic encephalopathy. There was evidence of end-organ injury with renal impairment

and elevated liver enzymes, although her general medical condition was fairly stable. Nevertheless, the neurological outlook was deemed to be extremely poor and the parents were counselled that withdrawal of life-sustaining medical therapy was the most appropriate course of action. With their agreement, the endotracheal tube was removed on March 10, 2007 at 1650 hours. The baby continued to breathe spontaneously until the morning of March 11, at which time respirations ceased and she was pronounced dead at 0840 hours.

Post Mortem:

The post-mortem examination revealed the following findings:

- Diffuse hypoxic-ischaemic injury, brain and spinal cord, recent
- Periventricular leukomalacia, recent
- Cardiomyopathy, with
 - Biventricular hypertrophy, marked, right >> left
 - Endocardial fibroelastosis, biventricular
 - Cardiomegaly, mild
 - “Pediatrix screen for Acylcarnitine profile; CAH 17-0HP and Galactose- (Gal and Gal-1-P) were negative
- Post-mortem bacterial and viral cultures – negative for pathogenic organisms

Death was attributed to “Perinatal Hypoxic-ischaemic Encephalopathy (HIE), Contributory Factor: Congenital Cardiomyopathy of Undetermined Aetiology.”

Discussion:

This infant died at four days of age from complications of perinatal hypoxic-ischaemic encephalopathy. Labour had been induced at 41 weeks because of post dates. At the time of cervical ripening on March 5, the foetal heart tracing was normal (previously termed “reactive”). The following day, continuous electronic foetal monitoring was commenced at the start of the induction. During the labour, concerns had been raised on a number of occasions about the occurrence of foetal heart rate decelerations, some of which resulted in the discontinuation of the Oxytocin. The obstetrician was appropriately informed. The obstetrician reviewed the tracing at 1720 hours and indicated that if the baby did not tolerate resumption of the Oxytocin, delivery by Caesarean section would have to be considered. The tracing thereafter was normal (previously “reassuring”) until approximately 2000 hours after which the tracing was atypical (previously “non-reassuring”) with no accelerations, decreased variability and occasional decelerations. Deeper and more prolonged decelerations occurred subsequently but were not consistent. The baseline heart rate increased during this time from 140’s to 150-160’s. At some point during these developments, the obstetrician became involved with the delivery of twins. When she subsequently became available, it was recognized that emergent delivery was indicated. A clinical judgment was made that vaginal delivery by vacuum extraction was feasible and therefore the quickest way to effect delivery. Unfortunately, vacuum extraction was unsuccessful, resulting in the need for Caesarean section. Although carried out expeditiously, this inevitably resulted in further delay in delivery. It cannot be determined if successful vaginal delivery at 0108 hours would have impacted the outcome.

This infant died after withdrawal of life sustaining medical therapy following severe hypoxic-ischaemic central nervous system injury. The injury occurred sometime prior to her birth as she exhibited depression at birth, severe acidosis on the first blood gas, end organ damage and encephalopathy. She was well managed by the neonatal teams involved in her care. The parents agreed to the withdrawal of life-sustaining medical therapy once ongoing clinical assessment, EEG and neuroimaging confirmed the severe neurological injury. The infant died at four days of age.

The paediatric team at the hospital responsible for resuscitation and stabilization initiated appropriate measures promptly and effectively. The airway was cleared, effective ventilation established and circulation supported such that transfer to the local Level 3 neonatal intensive care unit (NICU) at the children's hospital could be affected. Seizures were promptly detected and appropriately treated. The children's hospital team recognized that the infant was a candidate for inclusion in a randomized trial of systemic hypothermia for neuroprotection. The infant was, however, randomized to the normothermic arm of the trial. Care provided by the children's hospital transport team and NICU team was of the highest standard and the parents were fully informed and involved. When it was appreciated that the baby had sustained severe, irreversible neurological injury and that her long-term prognosis was grim, the team and family took steps to withdraw life-sustaining therapy. The infant died within a few hours of the withdrawal of assisted ventilation. The baby was severely compromised by the time she was delivered and no conventional intervention applied postnatally would have been able to change the outcome.

Although hypothermia for neuroprotection of term infants with severe hypoxic-ischaemic encephalopathy has been the subject of intensive investigation over the past few years, it has not been universally accepted as something that should be routinely offered to families with an affected newborn. Most trials have suggested improved outcome for the group of infants treated with hypothermia, but it is not universally protective and many infants with HIE will do poorly despite the use of total body or head cooling. At the time of this infant's birth, the children's hospital was participating in the ICE Trial, a multi-centre international randomized trial designed to examine this therapy. The evidence in favour of the efficacy and safety of hypothermia is accumulating and many centres are beginning to offer this therapy to babies with HIE under their care. However, the effectiveness in any individual infant cannot be accurately predicted and it is likely that, even if hypothermia had been applied, this infant's outcome would have been the same.

It is difficult to explain the cause of the congenital cardiomyopathy detected at the time of autopsy and the possible contribution to the baby's death. There was no evidence of disturbed in-utero growth, development or function and the baby appeared normally grown and without dysmorphic features after birth. The baby's clinical course was most consistent with severe perinatal hypoxic-ischaemic central nervous system injury and there was no significant functional cardiac disorder recognized postnatally. After resuscitation, cardiac function was quite normal with good blood pressure and tissue perfusion. If a congenital cardiomyopathy were to have contributed to the perinatal hypoxic-ischaemic central nervous system injury, one would have expected to see evidence of either prenatal or postnatal congestive heart failure, and this was not seen in this case.

Endocardial Fibroelastosis is a pathological term that describes changes to the Endocardium that can arise from a number of primary abnormalities. When present at birth, it is often felt to arise from accumulated damage that begins in early gestation. Live born infants with this condition often suffer from congestive cardiac failure that presents in early infancy. A genetic defect, viral infection, congenital cardiac abnormality or maternal autoimmune disease may be the underlying cause. In the current case, no potentially causative infectious agents were identified and metabolic screens were negative. However, the consanguinity of the parents suggests that there may have been a genetic basis for this abnormality. Although not clearly associated with the unfortunate outcome in this case, the baby may have suffered from cardiac insufficiency if she had survived. Investigation of the family by the Genetics/Metabolic and Cardiology services at the children's hospital might shed some light on the cause of this abnormality and provide counselling to the family as to the risk in future pregnancies.

Recommendations:

1. Obstetrical care providers are reminded that non-reassuring foetal heart rate tracings necessitate vigilant reassessment.
2. This family should be referred to the Genetics/Metabolic and Cardiology Services at the children's hospital for investigation of the possible cause of their baby's cardiomyopathy.

Case Summaries: Stillbirths

Case S-1

Cause of Death:

Stillbirth in a post dates pregnancy (43 weeks' gestation).

History:

The mother of the deceased was a 36 year old G₃P₁ with a last menstrual period of November 27, 2006. Her periods were regular q30 days, giving an estimated date of delivery of September 6, 2007. An ultrasound on April 17 was consistent with 22 weeks, 1 day giving an estimated date of delivery of August 20. Her antenatal care was shared between her family doctor and an obstetrician. The estimated date of delivery given on the family doctor's Antenatal Record I was October 4 and on the obstetrician's record as September 6. The dates given on the Antenatal Record II's of both physicians' entries correspond with the estimated date of delivery of September 6. At the last recorded visit with the family doctor on August 31, the gestational age was recorded as 39 weeks. The last recorded prenatal visit recorded with the obstetrician was on September 5 and the gestational age given was 39 weeks, 6days. At that visit, the Symphysis Fundal Height (SFH) was 38 cm, and the cervix was 1 cm dilated and long. A biophysical profile for post dates was ordered for September 12.

Her prenatal laboratory investigations were normal. Second trimester glucose challenge test was elevated. The 2 hour 75gm oral glucose tolerance test was normal. Genetic screening testing was not done.

Her past medical history was unremarkable. Her past obstetrical history is one previous term pregnancy delivered vaginally at 40 weeks for a 3.63 kg female infant.

The mother presented to the labour and delivery unit of the hospital on September 9 (40 weeks, 3 days?) at 0740 hours with some vaginal bleeding. No blood was seen, but she was having contractions q15m. She noted that foetal movement had been less than usual. She was placed on an external monitor at 0800 hours. At 0855 hours, a sterile speculum examination by the resident revealed small clots. The foetal heart rate tracing showed decreased variability. The cervix was 4 cm dilated. The plan at 1105 hours was to admit the patient for induction of labour by artificial rupture of membranes and Oxytocin. Before the induction could be started, a late deceleration occurred at 1210 hours. The decision was made to proceed to an emergency Caesarean section.

At the time the Caesarean section was called, the anaesthetist on-call was finishing a case in the operating room. He asked that the second on-call anaesthetist be called. The page was not returned and as a result, the first on-call anaesthetist was asked to attend as soon as possible. The first on-call anaesthetist arrived in the Caesarean section room at 1300 hours, 15 minutes after first being paged, anticipating that the patient had already had an epidural placed. Instead, the patient had been positioned on the table for placement of a spinal. The attempt at placement of the spinal anaesthetic was unsuccessful. Because of the urgency of the situation, it was elected to proceed directly to a general anaesthetic at 1313 hours. She was delivered of a stillborn male infant weighing 4.47 kg through thick meconium at 1318 hours. The cord was loosely around the neck once. The baby was noted to have long fingernails and some peeling skin. Apgars were 0 and 0 at 1 and 5 minutes. The baby was suctioned below the vocal cords for thick meconium. The baby did not respond to resuscitation and was pronounced dead at 20 minutes of age. Cord gases showed an arterial pH of 7.05 and venous pH of 7.15.

Post Mortem:

At autopsy, the baby was noted to be well developed and symmetrical. There were no congenital or anatomic abnormalities. The skin was heavily stained by meconium and examination of the lungs revealed extensive meconium aspiration.

The placenta was also heavily meconium stained. There was a 1.5 cm pale area on the placenta representing a small placental infarction. Microscopic examination showed mature villi with extensive syncytial knots.

The cause of death was stated as stillbirth in a post dates pregnancy (43 weeks gestation).

Discussion:

This infant died as a result of intrauterine asphyxia with the presumed mechanism being acute placental insufficiency associated with being post dates.

The record indicates a significant degree of uncertainty as to the gestational age at the time of admission. Eventually, it was concluded that the mother was in fact 43 weeks at the time of presentation based upon the ultrasound done at 22 weeks' gestation. She herself told her caregivers at the time of admission, that her due date was October 4. Her menstrual dates were quite certain however, and she subsequently recalled informing her doctor at an early prenatal visit that she was almost certain that the date of conception was December 15. This historical data is consistent with an estimated date of delivery of September 6, which is the date on the obstetrician's antenatal records. The estimated date of delivery established by the obstetrician was justifiable considering that the normal variation for dating a pregnancy by ultrasound at 22 weeks is +/- 2 weeks. A dating ultrasound in the first trimester would have been the most accurate method for determining the estimated date of delivery. The Society of Obstetricians and Gynaecologists of Canada Clinical Practice Guideline No. 135 from October 2003, states that first trimester ultrasound for dating is not recommended when the Last Menstrual Period and physical exam are concordant. Managing

Obstetrical Risk Efficiently acknowledges that ultrasound at 8-12 weeks is an effective strategy to reduce the incidence of post date pregnancy.

Review of the foetal heart tracing from the time of admission reveals an essentially flat tracing with the baseline in the 130's and no accelerations. Although the tracing was recognized as being non-reassuring, it was not acted upon until around 1210 hours, approximately four and a half hours after admission. With the cervix 4 cm dilated, consideration should have been given to an artificial rupture of membranes and foetal scalp clip earlier that morning. The finding of thick meconium in the setting of a non-reassuring tracing may have prompted a much earlier Caesarean section. Once an emergency Caesarean section was called, it was another hour before the baby was delivered. It cannot be determined from this review whether Caesarean section earlier that morning would have changed the outcome.

Recommendations:

1. Obstetrical care providers, both doctors and nurses at the _____, should ensure that all caregivers can interpret foetal heart rate monitoring and know appropriate and timely responses to an atypical/abnormal foetal heart rate tracing.

Case S-2

Cause of Death:

Acute intra-partum asphyxia associated with acute bronchopneumonia due to ascending E. coli infection.

History:

The mother of the deceased was a 34 year old G₂P₁ with an estimated date of delivery of April 6, 2007. Ultrasound at 11 weeks 5 days showed a 5 cm fundal fibroid. A routine second trimester ultrasound showed bilateral foetal hydronephrosis. This was followed up with ultrasounds at 22 weeks, 27 weeks, 35 weeks and 40 weeks showing no significant progression and normal amniotic fluid volume. The 40 week ultrasound showed normal foetal growth with no evidence of macrosomia.

Routine antenatal laboratory investigations were normal. Integrated Pregnancy Screening was negative. A second trimester Glucose Challenge Test was abnormal, but a two hour Oral Glucose Tolerance test was normal. Group B Streptococcus culture was negative.

The patient had one previous term pregnancy in 1999 delivered vaginally at 36 weeks 6 days of a 2.86 kg female infant. The pregnancy and delivery were uncomplicated. Her past medical history was unremarkable.

The patient's pre-pregnancy weight was 77.1 kg (169.9 lbs) and she gained 16.7 kg (36.8 lbs) during the pregnancy.

The patient was admitted to the hospital at 2100 hours on April 1, 2007 with premature rupture of membranes at 39 weeks' gestation. At the time of admission, rupture of membranes was confirmed for clear fluid. The cervix was thick, posterior and a finger-tip dilated. The foetal heart was normal. She was not having any contractions. To allow for spontaneous labour, and because the unit was busy, induction was delayed until 1300 hours on April 2. She subsequently became uncomfortable with her contractions and an epidural was placed at 1540 hours. Three variable decelerations occurred around 1915 hours down to 60 beats per minute (bpm) and lasting 20 seconds. The foetal heart recovered and was then normal. At 1940 hours, the cervix was 7-8 cm dilated and 100% effaced. She remained afebrile and the amniotic fluid was clear. There was another episode of variable decelerations around 2030 hours which subsequently resolved. She was fully dilated at 2130 hours and began pushing at 2150 hours. Deep variable decelerations occurred during the second stage of labour. The obstetrician on call was informed at 2155 hours, at which time she was busy doing a delivery. She advised that the Oxytocin be stopped and the patient be placed on her side.

The obstetrician attended at 2215 hours and the vertex delivered shortly thereafter. A shoulder dystocia ensued. The patient was placed in the McRobert's position. The anterior shoulder could not be reached, but with traction in the posterior armpit of the posterior shoulder, descent was obtained such that the anterior shoulder could be reached and the shoulders were released. The interval from delivery of the head to delivery of the shoulders was less than two minutes. She was delivered at 2224 hours of a female infant weighing 4.08 kg. The baby was flat at delivery. Apgars were 0, 0 and 0 at 1, 5 and 10 minutes. The cord was around the neck once. Cord arterial gases showed a pH of 7.02, pCO₂ 79, bicarbonate 22 and base excess -10.8. Resuscitation was begun immediately, but there was no response and the baby was pronounced dead at 2254 hours.

Post Mortem:

Significant findings at autopsy were foetal macrosomia and acute focal bronchopneumonia of the left lower lobe. Cultures from a gastric swab and lung were positive for E. coli. Blood cultures were negative.

There was a non-displaced fracture of the left clavicle.

The cause of death was stated as acute intra-partum asphyxia associated with acute bronchopneumonia due to ascending E. coli infection. Macrosomia with cardiac hypertrophy were contributory factors.

Discussion:

This infant died as a result of acute perinatal asphyxia. Potential risk factors for asphyxia include chorioamnionitis secondary to prolonged rupture of membranes, umbilical cord compression secondary to a nuchal cord and shoulder dystocia. Although *E. coli* was cultured from a gastric swab and lung culture, there was no intrapartum evidence of infection such as maternal fever or foetal tachycardia. Although variable decelerations occurred on occasion during the labour consistent with cord compression, these resolved and overall the tracing was not concerning until just before delivery. Although a significant shoulder dystocia occurred as is evident by the special manoeuvres required to affect delivery and the finding of a fractured clavicle, it was managed appropriately and the delay in the delivery of the shoulders would not normally result in a significant neonatal asphyxia. The post mortem findings of macrosomia and cardiac hypertrophy were in contrast to the clinical findings of a normal oral glucose tolerance test and normal growth parameters on serial ultrasounds.

In summary, there were a number of risk factors present that individually, do not appear to be sufficient to explain the outcome in this case. This raises the possibility that the outcome may have been a result of a combination of these factors.

Recommendations:

None

Case S-3

Cause of Death:

Intrapartum death of uncertain aetiology.

History:

The mother of the deceased was a 33 year old G₂P₀ with an Estimated Date of Delivery of February 8, 2007. Routine antenatal laboratory investigations were normal. Integrated prenatal screening was positive (increased nuchal translucency). Amniocentesis was declined. The second trimester ultrasound was normal. The patient was Rh negative and received Rh immune globulin at 28 weeks. A glucose challenge test was normal. The Group B Streptococcus culture was negative.

The patient had a first trimester spontaneous abortion in 2005. She was otherwise well and her antenatal course was uneventful. She smoked two cigarettes a day.

The membranes ruptured spontaneously at 0045 hours on January 22, 2007 at 37 ½ weeks' gestation. She was seen in triage at 0215 hours. Speculum examination confirmed ruptured membranes with clear fluid. The foetal heart was normal. She was having occasional tightenings. The cervix was long and closed.

The patient was admitted to Labour and Delivery at 1354 hours for Oxytocin induction. The cervix was unchanged and the foetal heart was normal. At 1715 hours, she was 2 cm dilated and having regular contractions. An epidural was started at 1805 hours. At 2130 hours, she was 4 cm dilated with the vertex at spines -2. At 2345 hours, she was 8-9 cm dilated and went on to full dilation at 0042 hours on January 23. The foetal heart tracing was normal throughout the first stage of labour although from 0010 hours, there were no further accelerations. At 0042 hours, the foetal heart rate suddenly dropped to 60-70 beats per minute and did not recover. A vaginal examination was negative for a cord prolapse. The obstetrician was notified and attended at 0048 hours. The vacuum extractor was applied and she was delivered at 0054 hours of a 3.325 kg female infant. The cord was around the neck once and the baby appeared very pale. The infant's Apgar scores were 0 and 0 at 1 and 5 minutes. A code pink was called. Chest compressions were started and the baby was intubated. Epinephrine was given twice via the endotracheal tube, with no response. An umbilical line was inserted. At 10 minutes, the Neonatal Intensive Care Unit team at the children's hospital was consulted and advised that with no heart rate from the beginning, and by ten minutes of resuscitation, the code should be stopped. A third dose of epinephrine was administered with again, no response. The resuscitation was stopped at 30 minutes.

The cord pH was 6.91. A Kleihauer was negative. A haemoglobin could not be obtained.

Post Mortem:

There were no congenital anomalies or anatomical findings to explain the death.

Examination of the placenta determined a discrepancy between the weight and degree of villous maturation with the former being appropriate for late third trimester and the latter for the mid third trimester. It was commented that this can be seen with maternal diabetes, however the second trimester Glucose Challenge Test was normal and there were no other clinical features to suggest this diagnosis. There was a small basal intervillous thrombus. There was no specific comment made as to the presence or absence of an umbilical cord thrombus.

The cause of death was given as intrapartum death of uncertain aetiology, however umbilical cord compression could not be ruled out.

Discussion:

This infant was stillborn following a reassuring course of labour up until twelve minutes before delivery when a sudden foetal bradycardia occurred. Delivery was carried out expeditiously. The

sudden development of the bradycardia was suggestive of acute cord compression. There was no evidence of a prolapsed cord. There was a nuchal cord at delivery, but it was not tight and there had been no preceding variable foetal heart rate decelerations as is typically seen with cord compression. The presence or absence of an acute umbilical cord thrombosis was not commented on in the pathology report.

The death is classed as a stillbirth as the infant fits the definition of a stillbirth as defined in the Vital Statistics Act:

“Stillbirth” means the complete expulsion or extraction from its mother of a product of conception either after the twentieth week of pregnancy or after the product of conception has attained the weight of 500 grams or more, and where after such expulsion or extraction there is no breathing, beating of the heart, pulsation of the umbilical cord or movement of voluntary muscle.

Recommendations:

None

Case S-4

Cause of Death:

No anatomical cause of death.

History:

The mother of the deceased was a 29 year old G₃P₂ with an expected date of delivery of June 15th, 2007. Her past obstetrical history was two previous pregnancies. The first was delivered in 1999 at 37 weeks of a 3.31 kg female infant by low forceps after a twelve hour labour. The second pregnancy was delivered in 2004 at 38 ½ weeks of a 3.3 kg male infant spontaneously after a four hour labour. Her past medical history was unremarkable.

Routine prenatal laboratory investigations and second trimester ultrasound were normal and the Group B Streptococcus culture was negative. A glucose challenge test (GCT) at 26 weeks was abnormal. A subsequent Oral Glucose Tolerance Test was diagnostic for gestational diabetes. She was started on a diabetic diet and blood sugar monitoring. She was followed at the Diabetic Health Clinic. Her midwife consulted an obstetrician for shared antenatal care with the midwives attending the birth. Blood sugars were well controlled. Weekly Non Stress Tests were normal and biophysical profiles on May 8th at 34 weeks and May 22nd at 36 weeks scored 8/8. Growth parameters showed an

increased abdominal circumference in keeping with macrosomia. Amniotic Fluid Indices were normal, but the Symphysis Fundal Heights were large for dates during the last six weeks of the pregnancy. Her pre-pregnant Body Mass Index was approximately 35. The plan was to induce labour on June 13th at 39 weeks 5 days.

The patient went into spontaneous labour on June 7th at 38 weeks 6 days' gestation. On admission to hospital at 1600 hours, the cervix was 3-4 cm dilated and her contractions were moderate. The foetal heart rate (FHR) was normal. An epidural was placed at 2120 hours and membranes ruptured spontaneously for clear fluid at 2040 hours. She was fully dilated at 2350 hours. The FHR had been normal during the first stage of labour.

The FHR was documented as 125 bpm with reported good variability. After twenty minutes of pushing, it was noted that the FHR was tachycardic to 180 beats per minute (bpm) with resolution in the left lateral position and the baseline 110 bpm with accelerations to 165 bpm. At 0036 hours, a scalp clip was applied as there were decelerations to 80 bpm with reported good variability and recovery. Two clips were applied as a good tracing could not be obtained. A nurse was called to the room and an obstetrician and paediatrician paged as the baby was bradycardic down to 40. An episiotomy was performed and at 0047 hours, a stillborn female weighing 4.23 kg was delivered. A "code pink" was called, however resuscitation was not possible and the baby was pronounced at 0113 hours. The cord gases were pH <6.8, pO₂ 17, pCO₂ 101.

A manual removal of the placenta was performed in the operating room under a general anaesthetic. The mothers' subsequent recovery was normal.

Post Mortem:

There was foetal macrosomia with the foetal heart and liver weights greater than expected for gestational age. The placenta was normal, although the foetal/placental weight ratio was increased for gestational age.

Tests for an inborn error of amino acid, organic acid and fatty acid metabolism were negative.

There was no anatomical cause of death.

Discussion:

Obstetrics

This infant was stillborn with an arterial cord pH of <6.8. Review of the foetal heart rate tracing indicates that it became atypical (formerly non-reassuring), just prior to full dilation. The tracing became abnormal and remained so from approximately midnight until delivery at 0047 hours. The progress notes first suggest some concern with the tracing at 0020 hours. An entry "decel to?" was crossed out, then went on to describe a baseline of 110 bpm with accelerations to 155-165 bpm. Review of the tracing however, indicates the baseline was elevated and erratic with deep decelerations to 110 bpm. A foetal scalp clip was applied at 0036 hours because of uncertainty of the

foetal heart rate decelerating to 80-90 bpm. It was at this point that the tracing was interpreted as being concerning and delivery was expedited by performing an episiotomy. Although the tracing had been abnormal for some 30-40 minutes prior to delivery, it cannot be determined that earlier intervention would have changed the outcome.

Midwifery

The Midwives appropriately consulted and referred the mother to the diabetic clinic. The plan was to monitor the baby and induce at term. The mother's gestational diabetes was controlled by diet alone and therefore the midwives could continue to provide care as per College of Midwives of Ontario guidelines.

The FHR was documented throughout the labour as being reassuring. This did not appear to be the case however:

- There is no mention of accelerations being present from 1830 hours until 2230 hours. The criteria for reassuring foetal heart tones include the presence of accelerations within an 80 minute period.
- At midnight, the midwife noted that the heart rate was 125 bpm with good variability. The strip does not however reflect this. The baseline had in fact dropped from 130-140 at 2340 hours to 90-105 bpm with accelerations during contractions to 130-140 bpm.
- There is also evidence of marked variability. According to the foetal monitoring guidelines, the baseline heart rate is determined once the contraction is over, for a period of two minutes. If there is not a steady heart rate for two minutes, the baseline is not determined. This baby was exhibiting late decelerations during the second stage of labour. This, along with a marked drop in baseline, was not reassuring. This pattern was followed by a period of tachycardia which is concerning following a period of decelerations. At 0030 hours, the decelerations were following contractions with slow recovery. This pattern is an indication of possible foetal compromise.
- Throughout the second stage of labour, the contractions were every 70-90 seconds with virtually no rest period. Hypertonicity of the uterus does not allow the baby to recover after each contraction and may lead to hypoxia.

During the second stage, it became difficult to reliably trace the foetal heart rate and the midwife was concerned. At 0036 hours, a foetal scalp clip was applied because of foetal heart rate decelerations. There was some uncertainty as to whether the scalp clip was working properly as the heart rate was very low. A second clip was applied approximately 90 secs later. The foetal heart was 80-90 bpm, decelerating down to 40 bpm. The obstetrician and paediatrician were called. The mother went on to spontaneously deliver at 0047 hours over an appropriately performed mediolateral episiotomy, a 4.43 kg stillborn female. There was a loose nuchal cord once around.

A “code pink” was called. The baby could not be resuscitated and was pronounced at 0113 hours. Cord gases showed an arterial pH of <6.8, venous pH 7.04. The total period of time from the first signs of foetal compromise until birth, was about forty minutes

It is possible that earlier intervention might have prevented this unfortunate outcome.

Recommendations:

1. The obstetrical care providers in this case should ensure an understanding of the interpretation electronic foetal monitoring for establishing foetal well-being in labour.
2. Obstetrical care providers are reminded that for non reassuring foetal heart sounds, a timely consultation with an obstetrician is recommended in the College of Midwives Guidelines.
3. The obstetrical care providers in this case should ensure an understanding of the interpretation electronic foetal monitoring for establishing foetal well-being in labour.

Case S-5

Cause of Death:

Intrapartum asphyxia.

History:

The mother of the deceased infant was a 27 year old G₃T₁L₁ with an Expected Date of Delivery of October 15th, 2006. Her past medical history included a first trimester pregnancy loss in 2003 and an uncomplicated pregnancy and term vaginal birth in 2004. Her health was otherwise unremarkable and she had regular prenatal care with her last obstetrical visit on August 22nd, 2006 at 32 weeks and 2 days. At that time, her clinical examination was normal and the foetus was in a breech presentation, which had been confirmed by an ultrasound at approximately 30 weeks’ gestation.

She presented to Hospital A on the evening of September 1st, 2006 at 33 weeks and 5 days with a history of passing a small amount of red blood per vagina. There is no record in the history of cramps or contractions at that time, or predisposing causes to the spotting. Foetal heart monitoring record was normal. Biophysical profile was performed and she received a score of 8/8, though there is no formal record of the test. The cervix was examined and found to be closed. The patient was advised to return if increased vaginal loss, regular contractions, or decreased foetal movement.

She was again seen at the hospital on the following evening with “cramps Q4 to 5 minutes.” She described having vaginal spotting the night before and again that afternoon. She was examined and found to have a cervix that was long, firm, anterior and with a closed internal os. A urine test indicated leukocytes 3+ and blood 2+. A diagnosis of a urinary tract infection was made and the patient was given Macrobid. She was discharged, advised to return if there were ruptured membranes, decreased foetal movement, bleeding or contractions. The clinical note is written by a post graduate year 1 (PGY1) resident. It is indicated on the form that it was discussed with a physician, but it is not clear whether an attending physician saw the patient.

On the early morning of September 3rd, 2006 her pain was becoming worse and she called the Obstetrics’ Department of Hospital A at 0330 hours. According to the coroner’s investigation statement, she was told this was normal for a urinary infection and was instructed to take Tylenol. Two hours later, her water broke and the ambulance was called. When the ambulance arrived, she was on the floor in the bathroom with a foetal foot presenting.

With a plan to go to Hospital A, the ambulance had to stop thirteen minutes into the trip due to delivery of the foetus up to the shoulder, with the head still not delivered. There were no signs of life in the foetus. The mother’s condition remained stable until they arrived at Hospital B where they diverted for care.

The infant was delivered fully in the emergency department at 0542 hours, fifteen minutes after delivery of the body. A physician in the emergency department delivered the head. The infant, weighing 1.97 kg was flaccid with Apgar of 0. The baby was intubated and Cardio Pulmonary Resuscitation was initiated. No vital signs were detected and the Apgar remained 0 at 10 minutes at which time resuscitation was stopped.

Post Mortem:

The post mortem examination revealed marginal placental haematoma and infarction consistent with marginal abruption (approximately 15 % of the placental volume), as well as findings consistent with intrapartum asphyxia.

Discussion:

This infant was stillborn at 34 weeks’ gestation after a premature labour and delivery, likely precipitated by an abruption of the placenta. The delivery was further complicated as the baby was in the breech presentation, had a precipitous labour and foetal head could not be delivered by a caregiver who was not skilled in a breech delivery. It is not clear whether the foetus had significant compromise even at the time of the delivery of the body, as the ambulance attendant’s notes indicated that the foetus was limp with no palpable pulse in the umbilical cord.

The symptoms of vaginal spotting and cramping in the antenatal period as this patient experienced, are commonly seen. The diagnosis of a urinary tract infection was made with a dipstick of the urine, a process that is common in clinical practice today. The subsequent urine culture had no significant growth. To initiate treatment for a possible urinary infection was appropriate. However, making this

diagnosis did not resolve the question of the vaginal spotting that the patient had presented with and undoubtedly affected the advice given by the caregivers at Hospital A on the early morning of September 3rd, 2006 when the patient called in with increasing cramps.

Because of this, the onset of premature labour was not considered and the patient was not advised to come urgently to the hospital. The fact that between the two visits to Hospital A and then this final call in the early morning of September 3rd, 2006, several different people were involved, there was likely a lack of appreciation of the progressive nature of the symptoms which started with vaginal spotting, increased to cramps and persistent spotting, then further increased to more painful cramps or contractions.

The paramedics would appear to have done an appropriate job in trying to care for this patient; they diverted to another hospital to assist in her care and the subsequent delivery. The paramedics would not be expected to be skilled in the delivery of a breech and it is not realistic to consider that ambulance attendants or paramedics should develop these skills. It would be an extremely uncommon situation where paramedics would use these skills and it is becoming increasingly uncommon for even physicians to develop these skills.

Recommendations:

1. Obstetrical caregivers are reminded of the often subtle nature of the onset and symptoms of premature labour.
2. _____ should review its supervision of junior house staff regarding out patient assessments.

Case S-6

Cause of Death:

Intrauterine asphyxia.

History:

The mother of the deceased was a 24 year old G₃P₂ with an estimated date of delivery of June 23, 2007. Routine prenatal laboratory investigations and second trimester ultrasound were normal. Intermediate Pregnancy Screening was accepted, but was not done. The patient denied using illicit drugs and smoked 3-4 cigarettes a day.

Her past obstetrical history was two term pregnancies: a 4.01 kg male in 2001 and a 3.86 kg male in 2004 were delivered vaginally. Both pregnancies were complicated by pre-eclampsia, possibly drug related. She had a history of codeine abuse.

During this pregnancy, the patient attended the prescribed antenatal visits, except between March 28 and May 3, during which time she had been hospitalized. Weight gain was appropriate and blood pressure was normal.

The patient was admitted to Hospital A in early April with post-coital bleeding, abdominal and pelvic pain. She was given steroids and Rhesus immune globulin. Investigations, including surgical consultation and Magnetic Resonance Imaging (MRI) of the abdomen and pelvis, were negative. Blood pressure was intermittently mildly elevated. She developed new onset headache with associated visual disturbances. The laboratory investigations for pre-eclampsia were normal and she was subsequently discharged home.

On April 10, the patient was re-admitted with complaints of blacking out on three occasions, and falling. She was also having persistent headaches. A neurological consultation was obtained and an MRI venogram was ordered. During this admission, family members expressed concerns about possible codeine abuse. Consultation was obtained with maternal-foetal medicine and a social worker and a methadone program was offered. The implications of her codeine use for herself and her pregnancy were discussed. She left the hospital before the MRI could be done. She was given a prescription for codeine until arrangements could be made through her regular obstetrician to undergo withdrawal under medical supervision.

On her last prenatal visit on May 3 at 33 weeks' gestation, the Symphysis Fundal Height was 33 cm. An ultrasound for foetal growth was ordered on May 4 and it showed the estimated foetal weight to be at the 8th percentile. A referral was made to the high risk unit at the Hospital B on May 11. She did not keep the appointment.

Paramedics and police brought the mother to the labour and delivery unit of Hospital C at 2130 hours on May 30. A male living in the house had called police because of her unusual behaviour, and on arrival in the delivery suite, she was combative and screaming. On assessment, she was found to be in advanced labour. The foetal heart could not be detected and assessment was extremely difficult due to the mother's combative behaviour. She went on to spontaneously deliver a stillborn male infant weighing 2.4 kg at 2152 hours. Meconium was noted at the time of delivery. Clinically, there were no signs of abruption. The umbilical cord venous pH was 6.98. The baby could not be resuscitated.

Following delivery, the mother was transferred to the Intensive Care Unit and treated for a possible acetaminophen overdose. She subsequently admitted to having taken 150 Tylenol #1's that morning and approximately 75 Gravol tablets. Her acetaminophen level was 194 umol/L (N 66-125).

Post Mortem:

Findings were consistent with intrauterine asphyxia. The death to delivery interval was determined to be >6 hours to <24 hours. Placental findings of increased nucleated red blood cells, villous oedema, chorioangiosis and meconium pigments in the membranes were supportive of the occurrence of hypoxic-ischemic episode(s) in utero.

Toxicology studies showed a foetal blood codeine level <0.25mg/ml, which is in, or below, the therapeutic range. The Diphenhydramine level was 1.5mg/ml, which is greater than the therapeutic range.

Discussion:

This infant was stillborn due to intrauterine asphyxia. The mother had a history of illicit drug use for several years, but it had only been identified during the third trimester of this pregnancy. She had been specifically asked about this at her first prenatal visit, but had denied it. Studies show that most parturients with a history of drug abuse deny it when asked. Risk factors suggesting substance abuse in pregnancy include lack of prenatal care, history of premature labour and cigarette smoking.

The post mortem report suggests that there had possibly been more than one episode of hypoxic-ischemic events during the pregnancy, but a defining terminal event such as placental abruption, was not identified. Maternal drug dependence has been identified as a risk factor for intrauterine foetal death independent of placental abruption¹. The mechanism by which intrauterine foetal death ensues, whether directly related to the drugs or other variables such as medical or psychosocial comorbidities, has not been studied. Acetaminophen toxicity in the mother can also cause hepatic necrosis in the foetus, but the post mortem examination showed no evidence of foetal liver damage. If a history of drug abuse can be identified, these patients need referral to a drug addiction program and intense foetal monitoring during the pregnancy. In this case, when the drug use became apparent, referral was made to a high risk obstetrical unit, but apparently the appointment was not kept.

Recommendations:

1. Obstetrical care providers are reminded that a high index of suspicion for drug abuse in pregnancy combined with a non-judgmental questioning of every parturient is necessary.
2. Obstetrical care providers are advised that drug use in pregnancy is a risk factor for intrauterine foetal death independent of placental abruption and warrants increased foetal surveillance.

References

McDonald S D, Vermeulen M J, Joel G R. Risk of Fetal Death Associated With Maternal Drug Dependence and Placental Abruption: A Population-Based Study JOGC 2007;29(7):556-559

Case S-7

Cause of Death:

Asphyxia due to uterine rupture.

History:

The mother of the deceased was a 33 year old G₂P₁ with an estimated date of delivery of October 2, 2007. Her antenatal course was uneventful. Routine prenatal laboratory investigations and glucose challenge test were normal.

Her first pregnancy was delivered of a 3.87 kg male infant in 2004 after a six hour labour. Her past medical history was unremarkable.

The mother's membranes ruptured spontaneously on October 5 at 0250 hours. At 0945 hours, the cervix was 2-3cm dilated and the presenting part was high. She was not in good labour. Oxytocin augmentation was commenced.

Labour became established by 1100 hours. The cervix was 4 cm dilated and the vertex was at sp-2. Thin meconium was noted at 1245 hours. An epidural was placed at 1315 hours with good effect. The Oxytocin infusion was at 12mU/min. Contractions were q1-3 minutes and moderate in intensity.

At 1330 hours, the cervix was 5-6 cm dilated.

At 1345 hours, the baseline foetal heart rate was 160 bpm with decreased variability and what were described as variable decelerations down to 130-140 bpm.

At 1415 hours, contractions were q2-3 minutes and moderate intensity. The Oxytocin infusion rate was increased to 14mU/min.

At 1445 hours, the cervix was 6 cm dilated. The Oxytocin infusion was increased to 16mU/min. The variable decelerations continued. Contractions became strong with a frequency of every 1.5 to 2.5 minutes. At 1525 hours, she was examined by the obstetrician and found to be 6 cm dilated. She was fully dilated at 1630 hours and started pushing at 1645 hours.

The obstetrician was called at 1720 hours because of more severe variable decelerations. At 1730 hours, the vertex was at spines + 3 in the LOA position. Because of concerns with the foetal heart tracing, vacuum extraction was attempted, but was abandoned after two pop-offs and no descent. At 1747 hours, forceps were attempted, but were abandoned as a proper application could not be achieved. The decision was then made to proceed with Caesarean section and the Oxytocin was turned off. A foetal scalp clip was applied and the foetal heart rate was in the 100's until delivery. The operating room team was involved with a major case and a second team had to be called in. The Caesarean section was carried out at 1821 hours. The patient was delivered of a 4.41 kg male infant with Apgars of 0, 0 and 0 at 1, 5 and 10 minutes. The cord was around the neck once. A uterine rupture into the right broad ligament was noted on taking down the vesico-uterine peritoneum. The baby could not be resuscitated.

Post Mortem:

An autopsy was not performed.

The placenta, membranes and cord were all normal.

The cause of death was certified as "Asphyxia due to uterine rupture."

Discussion:

This infant was stillborn as a result of antepartum asphyxia.

The Coroner's Investigation Statement indicates that the asphyxia occurred as a result of the uterine rupture. While this may have played a role, the rupture as described, was an unexpected finding and it did not appear to disrupt the placenta or cause a cord compression. The mother was haemodynamically stable and the operative report does not suggest that there was extensive bleeding from rupture into the uterine artery such that uterine blood flow might have been compromised.

Review of the intrapartum foetal heart tracing indicates the occurrence decelerations beginning at 1345 hours and continuing throughout the rest of the labour. They were described as being variable in nature. Starting around 1450 hours, shortly after the Oxytocin rate was increased to 16mU/min, the tracing became atypical with a late component and a slow rise back to the baseline from the decelerations occurring with most contractions. The obstetrician was not notified of any concerns with the tracing until 1720 hours. At that point, it was apparent that delivery needed to be expedited. Unfortunately, vaginal delivery first with attempted vacuum extraction, and then with forceps, was not successful. Further delay in delivery occurred because the second operating room team had to be called in and this did not occur until after the failed vaginal operations. Throughout this time, there was a sustained foetal bradycardia. It cannot be determined from the clinical course or the foetal heart tracing, when the uterine rupture may have occurred.

Recommendations:

1. Obstetrical care providers in this case should ensure that they are able to interpret foetal heart rate monitoring and know appropriate and timely responses to an atypical/abnormal tracing.

Case S-8**Cause of Death:**

Exsanguination.

History:

The mother of the deceased was a 22 year old T₁P₀A₂L₁ who was followed by her family physician during pregnancy. Her obstetrical risk factors included smoking, two early pregnancy losses and an earlier pregnancy that required the induction of labour at 37 weeks for severe intrauterine growth restriction. The birth weight of that infant was 2.43 kg.

The patient had a dating ultrasound and an early second trimester ultrasound at fifteen weeks that showed the placenta to be anterior. It cannot be determined if she had a repeat morphology ultrasound. An ultrasound at 30 weeks' gestation had documented normal foetal growth, normal umbilical artery doppler and biophysical profile of 8 out of 8.

On September 8, 2006 at 31 weeks' gestation, the mother was involved in a motor vehicle collision where her car was T-boned by another vehicle. The ambulance record indicated that the collision was a high speed impact to the passenger side with "about one foot impaction" of the door. The airbags deployed. It was not clear whether the mother may have lost consciousness at the scene. She was extracted from the vehicle by the fire department that arrived ten minutes after the collision.

The time from initial assessment of the paramedics, to the report at the triage, was just under an hour. The patient was haemodynamically stable. The transfer time was attributed to the fact that the patient had to be extracted and the attendants waited for a second crew for the husband and one year old child who were also in the vehicle.

The mother was triaged in the Emergency Department at 1835 hours. The nursing notes indicated that she complained of back pain, right-sided rib pain and suprapubic pain. Her abdomen was soft and non-tender. Hospital staff were aware that she was seven months' pregnant and the patient complained of contractions. On arrival, the patient was assessed by the Emergency Room Resident.

Her Glasgow coma scale was normal and she was haemodynamically stable. Initial assessment revealed only minor abrasions/contusions.

The Gynecology staff and Resident were present and examined the patient at 1845 hours. A bedside ultrasound was completed and the foetal heart rate was 120 beats per minute. Nursing notes indicate that the baby was “moving on ultrasound and that there was no evidence of bleeding on ultrasound.” Presumably this was verbalized by the gynecology staff. Trauma blood work was sent at 1855 hours and included a Kleihauer-Betke test.

The mother continued to complain of back pain. The pain was relieved somewhat when she was removed from the straight board. She required morphine for pain at 1918, 1949 and 2055 hours and Gravol for nausea. She was appropriately wedged with support under her right hip. She received oxygen and crystalloid - two and half litres of fluid over the next two hours.

At 2015 hours, the patient was unable to void, so she was catheterized and gross blood was found in her urine. At 2020 hours, an ultrasound was arranged because of the back pain and gross haematuria. The emergency physician requested continuous foetal monitoring. The nursing note indicates the delivery room was notified and at 2050 hours, the delivery nurse arrived to assess the foetal heart. At 2102 hours, it is documented that the nurse was unable to find the foetal heart. The doctor was notified and instructed that the patient be sent to ultrasound. The ultrasound does not show any abdominal pathology, but confirms foetal demise. The patient arrived back in the emergency room at 2130 hours and then was transferred to the delivery room at 2140 hours.

The patient's Kleihauer-Betke was positive, indicating 50 mls of foetal red blood cells in the maternal circulation. From the records, it is difficult to tell when, and to whom, this information was reported, but there is one lab slip from September 8th at 2153 hours indicating a positive Kleihauer-Betke.

The Obstetrics admission was completed by a PGY2 resident at 2215 hours. At 2315 hours the patient was assessed by the gynecologist and foetal demise was confirmed by ultrasound. The patient requested an induction process be started and she required two doses of Cervidil before she had a spontaneous delivery on the 10th of September 2006 at 0130 hours. Placenta weighing 211 grams was delivered spontaneously and was intact.

Placental pathology:

1. Small retroplacental, intradecidual and pardecidual recent hemorrhages with early ischemic changes.
2. Mild acute chorionitis

There is a comment in the pathology report that “the absence of a large retroplacental hemorrhage does not rule significant acute or subacute abruptio placenta. Although the subchorionic, retroplacental and decidual hemorrhages are mild in the available sections, some of these hemorrhagic foci are associated with early ischemic changes in the adjacent villi and were

probably larger at some point causing compression of the adjacent villous tissue. These hemorrhages were likely due to the trauma incurred during the motor vehicle accident.”

Discussion:

The mother of the deceased was involved in a motor vehicle collision at 31 weeks’ gestation. She was a belted passenger in a car that was T-boned at an intersection. The physical damage to the vehicle suggested a significant impact. The air bags were deployed and the patient required extraction from the vehicle. She was stable at the scene and in the emergency room. She was assessed promptly and carefully and no significant injury was identified. The Ob/Gyn team assessed her promptly and a bedside ultrasound was reassuring. Continuous foetal monitoring was ordered at 2020 hours. The Delivery Room nurse arrived at 2050 hours and was not able to find a foetal heart.

The Kleihauer-Betke later reported a significant foetal-maternal hemorrhage and the baby died some time during the 1 hour and 55 minutes the foetus was not monitored. As the placental injury/abruption likely occurred at, or shortly after the collision, it’s not certain if continuous foetal monitoring once the patient was assessed to be stable would have altered the outcome.

The risk of foetal death with trauma is significant when there is direct foeto-placental injury, maternal shock, pelvic fracture, maternal head injury, or hypoxia. In cases of blunt trauma during pregnancy, placenta abruption and uterine rupture may occur. These two conditions are often life-threatening to both mother and foetus. A third catastrophic event is a placental tear or "fracture" that can result in foetal hemorrhage and exsanguination, either into the amniotic sac or as foeto-maternal hemorrhage.

A small amount of foetal-maternal bleeding (less than 15 mls in 90 percent of cases) has been described in up to a third of trauma cases (Goodwin and Breen, 1990; Pearlman et al, 1990). Massive foetal-maternal hemorrhage may coexist with traumatic abruption, but more often it is not associated with classic signs of placental abruption.

Primary goals in abdominal trauma are evaluation and stabilization of maternal injuries. Following emergency resuscitation, evaluation is continued for fractures, internal injuries, bleeding sites, as well as placental, uterine, and foetal injuries. Because placental injury / abruption usually develops early following trauma, foetal monitoring is begun as soon as the mother is stabilized. The duration that post-trauma monitoring should be performed is not precisely known. While important in diagnosis and quantification of foetal-maternal hemorrhage, the Kleihauer-Betke is not helpful in the acute management. It is a manual haematologic test that cannot be completed rapidly. Flow cytometry is not available in many centers. Continuous foetal monitoring and ultrasound (determination of gestational age, detection of foetal heart, localization of placenta, biophysical profile, detection of foetal anemia) are the most important monitoring to ensure fetal well-being (William’s Obstetrics, 22nd Edition, 2005)

Recommendations:

1. Obstetrical care providers are reminded of the importance of continuous foetal heart rate assessment in the determination of foetal well-being following blunt abdominal trauma.
2. Obstetrical care providers are reminded that significant foetal-maternal hemorrhage can occur associated with maternal trauma, even without abruption of the placenta.
3. Results for tests to determine foetal-maternal hemorrhage should be requested as an urgent priority in cases of maternal trauma or other cases of increased risk.
4. Obstetrical and Emergency Care providers should review the SOGC “Guidelines for the Management of a Pregnant Trauma Patient” currently being drafted.

References

Goodwin TM, Breen MT. Am J Obstet Gynecol. 1990 Mar;162(3):665-71.

Pearlman MD, Tintinalli JE, Lorenz RP. N Engl J Med. 1990 Dec 6;323(23):1609-13.

Case S-9

Cause of Death:

Abruptio of placenta.

History:

The mother of the deceased was a 32 year old G₃P₀ with a dichorionic twin pregnancy conceived through artificial insemination and an expected date of delivery of December 6, 2007. She had two earlier pregnancy losses. The only risk factor recorded on the antenatal record is a history of proteinuria which led to a referral to a specialist a number of years ago. There is no urinary protein recorded on her antenatal II.

A nuchal translucency ultrasound completed at 12 weeks showed normal nuchal measurements for both twins (1.2 mm) and the risk of Down's syndrome 1 in 3500 based on age and the nuchal

translucency). The AFP testing was normal for a twin pregnancy. She was referred to the hospital's prenatal diagnostic service on July 27, 2007 after concerns with the foetal anatomy were found on the morphology ultrasound. The ultrasound showed twin A had no abnormalities and growth consistent with 21 weeks 7 days. Twin B had multiple abnormalities: short femurs, small abdominal circumference, absent nasal bone, a ventricular septal defect and a Dandy Walker variant. The increased risk of aneuploidy, the risks of prenatal diagnosis, and the risks of selective termination were discussed. An amniocentesis was done and the interphase FISH for chromosome 21, 13, 18, X, Y were all normal. The final chromosome results showed a mosaic trisomy 10 (4 colonies Trisomy 10, 16 colonies normal) and she was referred to Maternal Foetal Medicine and subsequently to Genetics at another hospital.

The couple was counseled in relation to the outcome of the Trisomy 10 mosaic and their options. They elected to proceed with selective termination of twin B at 32 – 34 weeks. The late gestational age was recommended to reduce the risks of prematurity should a complication from the procedure occur. The case was discussed at the Ethics Review Committee and the selective termination approved.

The mother was admitted and a selective termination was performed on October 6, 2007 at 31 weeks 2 days' gestation. She was given Betamethasone on October 1st and October 2, 2007. She was admitted overnight because of uterine activity and covered with Adalat and Clindamycin. Biophysical profile of twin A was normal prior to discharge.

The mother was seen at hospital on October 11. She reported having contractions and these were felt to be Braxton-Hicks. The baby was active, but she had noticed a decrease in activity. An ultrasound examination showed twin A in breech presentation. Biophysical profile score was 8/8 and the umbilical artery Doppler examinations were normal. She was discharged with a foetal kick count sheet and an appointment in two weeks. Although she was given the option of delivery in the hospital closer to home, they chose to continue to receive their care and delivery at the larger hospital.

At 0230 hours on October 14 at 32 weeks' gestation, membranes ruptured and the patient started to bleed. She was taken by ambulance to the local hospital at 0305 hours. The foetal heart could not be detected. The uterus was felt to be tense and there was vaginal bleeding consistent with placental abruption. Ultrasound by the obstetrician on call at 0335 hours did not show foetal heart activity. The radiologist on call was called at 0345 hours and repeat ultrasound at 0450 hours suggested the presence of a faint heart beat. Laboratory investigations showed no coagulation abnormalities. The obstetrician consulted the obstetrician on call at the larger hospital and the decision was made to proceed with an emergency Caesarean section. Under general anesthesia, the patient was delivered of a 1.635 kg female stillborn infant by breech extraction at 0536 hours. Stillborn twin B was delivered as a vertex. Attempted resuscitation of twin A was halted at 0545 hours.

Post Mortem:

Autopsies were not performed.

The pathology confirmed a dichorionic/diamniotic separate twin placenta. There was velamentous insertion of one cord into a placenta weighing 254 grams whereas the other placenta weighed 406 grams and on sectioning, revealed blood clot within the substance occupying more than half of the placenta. Microscopic section revealed acute chorioamnionitis, intervillous thrombus and infarct. It is unclear which placenta is A or B. The gross description of one of the placentas (again not sure which) reveals “blood clot within the substance, almost occupying more than half of the placenta.” There is no reference to this in the final pathology.

A Kleihauer test was ordered on October 15, but there was no report of the result documented on the chart. The report was found and stated:

“Kleihauer - Result - Within normal limit. The result is within normal limits with the amount of Feto-maternal Hemorrhage (FMH) estimated to be no more than 10 mL.”

The investigating coroner indicates in the narrative that Baby A “appeared normal” and “looked at the placenta and could see where the abruption occurred.” It was decided that no autopsy was required. No comment of Baby B is made. The conclusion was “the death resulted solely of the abruption and that it was in no way related to the previous procedure.”

Discussion:

This is a tragic case of IUI dichorionic twins discordant for foetal anomalies. Twin B had multiple anomalies and after a number of prenatal investigations was found to have a mosaic trisomy 10. After counseling with respect to the prognosis, this couple decided to have a selective termination at 32 weeks' gestation. The specific risks are not documented in the hospital charts. Eight days following the procedure, the patient presented with premature rupture of the membranes and what seems to fit clinically with a severe placental abruption leading to foetal demise. The conclusions of this case are compromised by the lack of autopsy results and confusing placental pathology. There is no comment on the morphology of twin B and confirmation of the ultrasound findings or genetic diagnosis reached prenatally could not be made. Similarly, no autopsy results on twin A makes the diagnosis of abruption and subsequent demise more, or less, likely. It cannot be determined if the chorioamnionitis is primarily in the membranes of twin A (subsequent to SROM or even leading to it) or twin B (potential procedure related infection). The conclusions reached by the investigating coroner could be substantiated with more complete information from autopsies and coroners pathologic assessment.

Recommendations:

1. Coroners are reminded of the importance of placental pathology and autopsy findings in the investigation of perinatal mortality.

Case S-10

Cause of death:

Unascertained. No post mortem.

History:

The mother of the deceased was a 40 year old G₂P₀ with an expected date of delivery of May 2, 2008. This was an IVF pregnancy with embryo transfer on August 8, 2007. Initially there were twins, but subsequent ultrasound showed only a singleton pregnancy. Routine prenatal laboratory investigations, ultrasounds and genetic screening were normal. The patient's haemoglobin was 105 gm and a haemoglobin electrophoresis was normal. On February 4, at 27 weeks 3 days, her haemoglobin was 95gm and Glucose Challenge Test was elevated at 8.3. There was no indication from the records provided that she was prescribed iron supplementation or a follow up glucose tolerance test. She was Group B Streptococcus negative. Her past medical history was non-contributory.

The patient's past obstetrical history included one very early spontaneous abortion in 2006. Her antenatal course was uncomplicated. She was booked for induction of labour on May 7 at 40 weeks 5 days.

The patient was admitted to the hospital at 1645 hours on May 7, 2008. A Non Stress Test was normal. The cervix was a finger tip dilated. Cervidil was inserted at 1710 hours. Subsequent monitoring of the foetal heart rate over the next two hours was normal and the monitor was removed as per protocol. On assessment at 2200 hours, she was not having any contractions and the foetal heart was normal.

At 0400 hours, she began to bleed per vagina. On assessment at 0420 hours, she was found to be having contractions every 4-5 minutes, lasting 40 seconds and moderate on palpation. The foetal heart rate was 90-100 beats per minute (bpm) and the toco tracing did not indicate uterine hyperstimulation. The cervix was a finger tip dilated and clots were noted. The Cervidil was removed and the obstetrician was notified at home at 0430 hours. The foetal heart dropped down to 85-95bpm and preparations were made for Caesarean section. The nursery was notified. The

obstetrician arrived at 0457 hours. A spinal anaesthetic was placed at 0510 hours and the patient was delivered of a stillborn male infant weighing 3.195 kg at 0532 hours. The cord was loosely around the neck two times. The baby could not be resuscitated. Cord gases were not obtained.

In the operative report, the obstetrician indicated that “there was perhaps at the very most, a marginal abruption”.

Post Mortem:

A post mortem examination was not performed.

The examination of the placenta revealed focal intervillous fibrin deposition and calcification with no evidence of placental infarcts or abruption. A maternal Kleihauer was not done.

Discussion:

This infant was stillborn twelve hours after the insertion of Cervidil for the induction of labour. Clinically, the presentation was suggestive of placental abruption, but the findings at the time of Caesarean section and the placental pathology report did not confirm this. A Kleihauer test to rule out a foeto-maternal bleed was not done. An autopsy may have provided more information as to the cause of death.

The indication for induction was postdates. Although the World Health Organization’s definition of postdates is 42 weeks and beyond, in 1997, the Society of Obstetricians and Gynaecologists of Canada (SOGC) recommended that induction of labour should be offered at 41-42 weeks. At the time of Cervidil insertion, the patient was 40 weeks and 5 days. A more recent SOGC guideline for the management of pregnancy at 41 and 42 weeks’ gestation recommends that each obstetrical department establish guidelines dependent on local resources for scheduling labour induction¹. It cannot be determined from this review whether such factors as local resources impacted on the decision to induce labour before 41 weeks in this case.

Cervical ripening is recommended when labour induction is indicated and the cervix is unfavourable to lower the risk of failed induction and Caesarean section. Monitoring following the administration of vaginal prostaglandin preparations has not been standardized. A SOGC clinical practice guideline in 2001 indicated that most studies suggest monitoring 30 minutes to 2 hours after the administration of prostaglandin gel and to continue monitoring if regular uterine contractions are noted. Randomized trials comparing prostaglandin gel with controlled-release prostaglandin (Cervidil) showed a higher rate of excessive uterine activity with the controlled-release preparation. The American College of Obstetricians and Gynaecologists recommends continuous electronic monitoring for the duration of insertion.² The SOGC has not made any recommendations, although the 2001 guideline concluded that there was not sufficient data in randomized trials to make strong recommendations for, or against, monitoring. As such, the monitoring following the insertion of Cervidil in this case cannot be criticized.

Recommendations:

1. Obstetrical care providers are reminded of the guidelines for induction of labour in postdates pregnancies.
2. Obstetrical care providers are reminded of the recommendations for obtaining cord gases.
3. Obstetrical care providers are reminded to obtain a maternal Kleihauer test in the investigation of stillbirths.
4. The SOGC should consider issuing guidelines for monitoring after the use of vaginal prostaglandins for cervical ripening and induction of labour.
5. In the absence of further data from randomized controlled trials, consideration should be given to the establishment of a central registry with the Health Protection Branch for adverse obstetrical outcomes related to the use of vaginal prostaglandin preparations.

References

1. Guidelines for the Management of Pregnancy at 41+0 to 42+0 Weeks. J Obstet Gynaecol Can 2008;30(9):800-810.
2. Induction of Labour at Term SOGC Clinical Practice Guideline No. 107, August 2001.

Summary of Recommendations

Maternal Deaths

	Cause of Death	Recommendation
M1	Undetermined. No anatomic or toxicological cause of death.	None
M2	Sepsis and associated DIC, due to beta haemolytic strep group A.	None
M3	Amniotic Fluid Embolism	None
M4	Amniotic fluid embolism	None
M5	Post partum myocarditis	None
M6	Stab wounds to neck. Homicide.	1. Case to be referred to the Domestic Violence Death Review Committee of the Office of the Chief Coroner.
M7	Amniotic fluid embolism	1. A central registry should be considered for the reporting of severe maternal and foetal adverse outcomes in association with the use of prostaglandin agents for cervical ripening and induction of labour. 2. Obstetrical units should consider having balloon tamponading devices readily available in the delivery suite for the treatment of post partum haemorrhage due to uterine atony.
M8	Invasive bronchopulmonary aspergillosis	None

Neonatal Deaths

	Cause of Death	Recommendation
N1	Perinatal asphyxia with severe hypoxic-ischemic encephalopathy.	1. Obstetrical care providers are reminded that induction of labour for foetal macrosomia is characterized as an unacceptable indication according to Managing Obstetrical Risk Efficiently (MORE). 2. The labour and delivery unit at _____Hospital should review their drill with regards to the timely management of an abnormal foetal heart rate tracing. 3. Obstetrical care providers are advised that foetal heart rate decelerations occurring during the first hour following the application of a cervical ripening agent, warrants extended monitoring. 4. Obstetrical and paediatric care providers are reminded that cord gases should be obtained at all deliveries. In their absence, an early set of central blood gases from the infant should be obtained when there is significant depression at birth and when active resuscitation is required. 5. Paediatric care providers are reminded of the need to monitor and aggressively treat severe and/or sustained hypoglycaemia in any neonate, especially those exhibiting signs of encephalopathy. 6. Paediatric care providers should routinely obtain central blood pressure measurements in compromised infants in order to apply appropriate interventions when necessary to adequately support the infant's circulation.
N2	Intrapartum asphyxia.	None

	Cause of Death	Recommendation
N3	Craniocerebral trauma with perinatal hypoxic-ischaemic injury.	1. The _____ Obstetrical Unit should review its policies regarding monitoring of women in premature labour. 2. Health Care Providers are reminded about drawing cord gases as soon as possible after delivery and especially where the infant appears to be compromised.
N4	Perinatal Asphyxia.	1. Caregivers are reminded that an EFM strip, which is inadequate for interpretation, requires further investigation to rule out a maternal trace and application of a scalp clip if the heart tones cannot be reliably recorded with the external monitor. 2. Caregivers are reminded that in the presence of an "abnormal fetal heart rate pattern, usually operative delivery, should be undertaken promptly unless (1) there is clear evidence of normal fetal oxygenation by means of scalp pH assessment or (2) spontaneous delivery is imminent". SOGC 2007: Fetal Health Surveillance: Antepartum and Intrapartum Consensus Guideline. 3. Caregivers are reminded that when assuming care for a woman in labour, a careful review of the chart should be undertaken to be familiar with the progress to this point and any issues which may have arisen earlier in the labour or in the pregnancy prior to the labour. Reviewing the foetal monitoring strips will allow the identification of baseline heart rate changes and changes in variability. 4. Caregivers are reminded of the importance of documentation of visits, exams and assessments as well as the current and future plan of care for each woman in labour.

	Cause of Death	Recommendation
N5	Acute perinatal asphyxia associated with acute Staphylococcus aureus chorioamnionitis.	None
N6	Septic shock in a premature infant with a patent ductus arteriosus.	1. Paediatric caregivers are reminded of the importance of clear documentation of their assessments, impressions and plans in the official record of patients for which they have responsibility. 2. The Acute Care Transport Service at the children's hospital should review its response to the requests for transfer received regarding this patient. 3. The Ministry of Health and Long Term Care urgently review the bed capacity for severely sick neonates needing transfer to neonatal units through out the province. 4. Obstetrical health care providers should discuss risk factors for premature labour and the benefits and risks of interventions that may improve outcome.
N7	Foetal Hypoxia.	None
N8	Perinatal asphyxia and shock secondary to antenatal haemorrhage (possibly Vasa Previa).	1. Neonatal resuscitation procedure needs to be reviewed by the medical team. 2. Recommendations and treatment documentation by the tertiary care centre should be recorded.
N9	Massive, acute pulmonary haemorrhage.	None

	Cause of Death	Recommendation
N10	Acute Hypoxic Ischemic Encephalopathy.	1. The _____ hospital should review the policy for notifying the on call anaesthetist and paediatrician of potential emergencies in the labour room. 2. The _____ hospital should review with the nursing staff and the obstetrician, the interpretation of EFM strips and protocols for intrauterine foetal resuscitation. 3. The _____ hospital and the obstetrician, should consider more detailed documentation of the labour and birth by either an improved written labour birth summary, use of progress notes or dictation of procedures such as failed vacuum birth. 4. The _____ hospital should review the labelling of specimens and the protocol for notification of such errors.
N11	Severe hypoxic-ischaemic encephalopathy.	None
N12	Perinatal Hypoxic-ischaemic Encephalopathy.	1. Obstetrical care providers are reminded that non-reassuring foetal heart rate tracings necessitate vigilant reassessment. 2. This family should be referred to the Genetics/Metabolic and Cardiology Services at the children's hospital for investigation of the possible cause of their baby's cardiomyopathy.

Stillbirths

	Cause of Death	Recommendation
S1	Stillbirth in a post dates pregnancy (43 weeks gestation).	1. Obstetrical care providers, both doctors and nurses at the _____, should ensure that all caregivers can interpret foetal heart rate monitoring and know appropriate and timely responses to an atypical/abnormal foetal heart rate tracing.
S2	Acute intra-partum asphyxia associated with acute bronchopneumonia due to ascending E. coli infection.	None
S3	Intrapartum death of uncertain aetiology.	None
S4	No anatomical cause of death.	1. The obstetrical care providers in this case should ensure an understanding of the interpretation electronic foetal monitoring for establishing foetal well-being in labour. 2. Obstetrical care providers are reminded that for non reassuring foetal heart sounds, a timely consultation with an obstetrician is recommended in the College of Midwives Guidelines. 3. The obstetrical care providers in this case should ensure an understanding of the interpretation electronic foetal monitoring for establishing foetal well-being in labour.
S5	Intrapartum asphyxia.	1. Obstetrical caregivers are reminded of the often subtle nature of the onset and symptoms of premature labour. 2. _____ should review its supervision of junior house staff regarding out patient assessments.

	Cause of Death	Recommendation
S6	Intrauterine asphyxia.	1. Obstetrical care providers are reminded that a high index of suspicion for drug abuse in pregnancy combined with a non-judgmental questioning of every parturient is necessary. 2. Obstetrical care providers are advised that drug use in pregnancy is a risk factor for intrauterine foetal death independent of placental abruption and warrants increased foetal surveillance.
S7	Asphyxia due to uterine rupture.	1. Obstetrical care providers in this case should ensure that they are able to interpret foetal heart rate monitoring and know appropriate and timely responses to an atypical/abnormal tracing.
S8	Exsanguination.	1. Obstetrical care providers are reminded of the importance of continuous foetal heart rate assessment in the determination of foetal well-being following blunt abdominal trauma. 2. Obstetrical care providers are reminded that significant foetal-maternal hemorrhage can occur associated with maternal trauma, even without abruption of the placenta. 3. Results for tests to determine foetal-maternal hemorrhage should be requested as an urgent priority in cases of maternal trauma or other cases of increased risk. 4. Obstetrical and Emergency Care providers should review the SOGC "Guidelines for the Management of a Pregnant Trauma Patient" currently being drafted.

	Cause of Death	Recommendation
S9	Abruption of placenta.	1. Coroners are reminded of the importance of placental pathology and autopsy findings in the investigation of perinatal mortality.
S10	Unascertained. No post mortem.	1. Obstetrical care providers are reminded of the guidelines for induction of labour in postdates pregnancies. 2. Obstetrical care providers are reminded of the recommendations for obtaining cord gases. 3. Obstetrical care providers are reminded to obtain a maternal Kleihauer test in the investigation of stillbirths. 4. The SOGC should consider issuing guidelines for monitoring after the use of vaginal prostaglandins for cervical ripening and induction of labour. 5. In the absence of further data from randomized controlled trials, consideration should be given to the establishment of a central registry with the Health Protection Branch for adverse obstetrical outcomes related to the use of vaginal prostaglandin preparations.

Glossary of Terms

AFE	amniotic fluid embolism
AFP	alpha -fetoprotein
ANA	antinuclear antibody
APTT	activated partial thromboplastin time
ARM	artificial rupture of membranes
AST	Aspartate aminotransferase
AVM	arteriovenous malformation
BID	two times a day
BMI	body mass index
BP	blood pressure
BPP	biophysical profile
CBC	complete blood count
CK	Creatine Kinase
CNS	central nervous system
CPR	cardio pulmonary resuscitation
CT (CAT)	computerized axial tomography
CXR	chest x-ray
DIC	disseminated intravascular coagulation
ECG	electrocardiogram
EDD	estimated date of delivery
EFW	estimated foetal weight
EMR	electronic medical record
ETT	endotracheal tube
FFP	fresh frozen plasma
GAS	Group A streptococcus
GBS	group B streptococcus
GCT	glucose challenge test
GSC	Glasgow Coma Scale
HCG	human chorionic gonadotropin
HIV	Human Immunodeficiency Virus
ICH	intracerebral haemorrhage
ICU	intensive care unit
INR	International Normalization Ratio
IPS	integrated perinatal screening
IUGR	intra uterine growth retardation
LDH	Lactate dehydrogenase
MCAD	medium chain acyl CoA dehydrogenase

MFM	maternal foetal medicine
MRI	magnetic resonance imaging
NST	non-stress test
OGTT	oral glucose tolerance test
OR	operating room
PIH	pregnancy induced hypertension
PPROM	pre-term premature rupture of membranes
PPV	positive pressure ventilation
PTT	partial thromboplastin time
QID	four times a day
RR	respiratory rate
SC	serum creatinine
SFH	symphysis fundal height
SLE	systemic lupus erythematosus
SOGC	Society of Obstetricians and Gynaecologists of Canada
STSS	Streptococcal Toxic Shock Syndrome
TID	three times a day
TSS	Toxic Shock Syndrome
V/Q	ventilation-perfusion
VSA	vital signs absent
VSD	ventricular septal defect
WBC	white blood cell
WNL	within normal limits