Should we continue to honour time honoured practices?

Changes to midwifery clinical practices in Birth Suites at the RWH.

On Friday 2nd June, four senior clinicians spoke about two ‘time honoured’ routine midwifery practices in the birth suites at the RWH and why these were going to be changed. These were cessation of routine weighing of all placentae and routine cord blood collection from all newborn infants.

We heard Dr Lisa Begg, Director of the Clinical Practice Improvement Unit (CPIU), Karen Moffat, Clinical Midwife Consultant, Birth Suites, Dr Jan Pyman, Director Anatomical Pathology RWH, and Dr Helen Savoia, Consultant Haematologist at RWH.

In her introduction, Lisa outlined how the CPIU had undertaken a search for evidence about these two practices. These included research using placental weight as an outcome measure. Themes of the studies include the relationship of placenta weight (PW) to blood pressure, intrauterine growth restriction, congenital malformations, preterm labour, maternal Hb/ferritin status and glycaemic index. This search could not find any balance of evidence in support of these routine practices, nor for that matter, their abolition.

Karen Moffat noted that while there would no longer be a policy of routine weighing of placentae at RWH, it was important to identify which placentae need to be sent for pathology. She spoke of the development of procedure and audit processes which will ensure that appropriate placentae are sent for histopathological examination. Development of this procedure was a team effort by the departments involved and was informed by Kent, A.L., Dahlstrom, J.E. (2006) Placental assessment: Simple techniques to enhance best practice Australian and New Zealand Journal of Obstetrics and Gynaecology 46: 32–37

It is expected that obstetricians continue to identify those placentae which require further investigation pre-natally, and alert the midwifery staff.

The new practice of non routine placental weighing commenced on Monday 2nd June 2006.

Dr Jan Pyman then spoke of the value of placental examination.

Helen’s presentation introduced us to the ‘modern automated laboratory’ where blood testing occurs without human intervention. We saw images of poorly collected (clotted) samples, incomplete pathology request forms, and incorrectly labeled specimen tubes. A new cord collection procedure will be introduced in the near future. Meanwhile, education programs are being conducted for clinical staff while policy development is in the final stages.

Susan Braybrook

Midwife
Coordinator perinatal mortality review RWH

‘Evidence based Clinical Practice is an approach to health care practice in which the clinician is aware of the evidence that bears on her clinical practice, and the strength of that evidence’ (McMaster University).

The relationship between health care and evidence is still a contingent affair. When the British navy introduced citrus juice into sailors’ shipboard diet at the end of the 19th century, the evidence that this was effective in preventing scurvy had been around for 263 years. The evidence that uterine dilation and curettage is not effective in treating uterine bleeding has been available since the 1970s, but it took some time for practice to change and it is still sometimes used inappropriately.

Not at the Women’s of course, where in the decade from 1994, the number of curettes for dysfunctional uterine bleeding fell from 1,274 to just over 100.

In this edition of the Newsletter, we report on changes to a couple of customary practices at the Women’s, routine weighing of placentae and routine cord blood collection. These changes involved a look at the
available evidence, which was not decisive either way, but did not provide a case for our current practice. There was broad, and at times animated, consultation. We used one of our monthly mortality and morbidity review meetings to brief clinical staff on the changes and new protocol and to provide Dr Jan Pyman, Director of Anatomical Services, with an opportunity to give full reign to her passion for examining placentae and what could be learned from this. She had advice to give about how to get the best value from this and stressed that this was a team effort, of which the pathologists were only one member.

Muir Gray described evidence based clinical practice as an approach to decision making in which the clinician uses the best available evidence, in consultation with the patient, to decide on the option that suits the patient best. Managing and providing advice to women and their partners about premature rupture of the membranes travels in an area of clinical uncertainty, but the research into outcomes here at the Women’s, which Neil Everest reported on at a Grand Round, painstakingly aims to create a body of evidence that will provide better information to assist clinicians and women in making decisions.

Mary Draper
Director
Clinical Governance

Should we continue to honour time honoured practices?

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The value of placental examination

Anatomical pathologists have an interest and passion for the placenta and the information that can be gleaned from its examination which is sometimes not shared with the wider clinical community.

Why should the placenta be examined?

- Clarification of causes of adverse pregnancy outcomes
- Identification of conditions that have recurrence risks in subsequent pregnancy
- Understanding of antenatal and intrapartum events that may contribute to neurodevelopmental morbidities (early identification leading to early intervention)
- Assessment of factors that contribute to poor outcome as a factual basis for resolving medicolegal issues
- Zygosity from multiple pregnancy
- Aid the understanding of factors involved in perinatal death (placental abnormalities in 92%, diagnostic of cause of death in 32% or contribute to diagnosis in 16% – Driscoll, 1965)

The clinical information supplied to the pathologist at the time of placental examination is crucial. The placenta acts as a ‘diary’ of pregnancy and forms part of the triad of mother, baby and placenta. The placenta is an easily accessible and significant part of this triad and an important (but not the only) arm in the assessment of pregnancy related problems.

The clinician should undertake a systematic examination of the placenta.

Firstly the cord can tell an important story: knots, thrombosis, entanglement, coiling, narrowing, short cord, velamentous insertion, vasa previa and amnion nodosum. A careful look at the membranes then may provide information about meconium exposure, extrachorial placenta including circumvallation. Systematic examination of the fetal and maternal surfaces of the placenta then follows.

Considerable information may be provided by the macroscopic appearances, but some significant information may only be provided by microscopic examination, such as the presence of chorioamnionitis or fetal thrombotic vasculopathy, significant conditions that may be associated with neurodevelopmental sequelae or sometimes fetal demise. Pathology also plays a role in the determination of zygosity and complications of multiple pregnancy placentae. A small word of caution, however; birth suite staff curious about chorionicity are asked to handle membranes gently and try to preserve membrane integrity as far as it is possible. Pathologic examination may provide information regarding vascular anastomosis in a setting of possible twin to twin transfusion.

The most value from placental examination can be made when clinical questions are posed and the pathological examination can target these clinical queries. In particular, in the event of an adverse perinatal outcome, the information that a pathologist can provide may help clinicians form a significant explanation of the events leading up to the adverse event and provide parents with answers.

In order to provide the most useful clinicopathological information, the examination of the placenta involves TEAM WORK (medical, nursing and laboratory staff), proper handling of the tissue and provision of relevant information. Examination of the placenta may entail the examination in delivery suite by clinical staff or the histopathological examination by hospital pathologists. Examination may include not just histopathology but microbiology and viral studies, karyotyping, molecular studies and more specialized placental research.
Placental lesions are not necessarily the cause of unfavourable outcome and some structural changes may be a consequence of poor fetal condition.

The pathologists requires clinical staff to handle the placenta carefully, being careful to preserve relationships of adherent blood clot, or preserve membrane integrity in placentae from multiple pregnancies. The placenta should be forwarded to the laboratory in the fresh state, allowing special investigations such as karyotyping, microbiology, examination of vascular anastomoses etc. if required.

Careful examination of the placenta provides information on both recent and remote events that may explain antenatal and intrapartum maternal and fetal events.

Several publications have provided suggested practice guidelines for the pathological examination of the placenta, and are listed at the end of this article.

Indications to send the placenta to pathology may be maternal, fetal or placental.

Maternal indications include:
- Systemic disorders with clinical concern for mother or infant (e.g. severe diabetes, hypertension, collagen disease, severe anaemia)
- Premature delivery <34/40
- Peripartum fever and/or infection
- Unexplained 3rd trimester bleeding or excessive bleeding
- Clinical concern for infection
- Severe oligohydramnios
- Unexplained or recurrent pregnancy complications (IUGR, SB, spontaneous abortion)
- Abruption
- Nonelective pregnancy termination
- Thick and/or viscid meconium

Fetal indications include:
- Transfer to NICU
- Stillbirth or perinatal death
- Comprised clinical condition: acidosis, low apgar scores, anaemia, ventilation assistance
- Seizures
- Infection or sepsis
- Hydrops fetalis
- Birth weight <10th percentile
- Multiple gestation
- Major congenital anomalies, dysmorphism, abnormal karyotype

Placental indications include:
- Physical abnormality (e.g. infarct, vascular thrombosis, retrolental haemorrhage, amniotic nodosum, abnormal color or odour)
- Small or large placental size or weight for gestational age
- Umbilical cord lesions (e.g. thrombosis, torsion, true knot, single artery, absence of Wharton’s jelly)
- Long and short cord
- Abnormal cord insertion

Maximum ‘value’ from the examination of the placenta can only be achieved when the request form contains appropriate and adequate clinical information and is clearly signed. It is essential that the doctor who requests the examination is identifiable and available to provide further details.

In a small audit of the request slips accompanying 50 placentae forwarded to the laboratory for examination at the end of 2005, 20 cases did not state the pregnancy gestation. Five of the 50 provided no clinical information at all and three cases were associated with perinatal death but there was no indication on the request slip that consent for autopsy had also been made!

In 2001 Dr Yuen Chan audited 320 placentae sent to pathology in that year. In 2005, there were over 900 placentae examined in pathology.

The importance and value of placental examination is starting to be recognized, and with a concerted team approach, valuable contributions to clinical information and management can be made.

Dr Jan Pyman
Director Anatomical Services, RWH

References
College of American Pathologists guidelines (Arch Pathol Lab Med 1997;121:449-476)
Hargitai et al J Clin Pathol2004;57: 785-792
Khong TY, Broadsheet: a topographical and clinical approach to examination of the placenta (Pathology 2001;33: 174-186)

These are really good sites for further information of placental histology:
www.palpath.com/ MedicalTestPages/placenta2.htm
http://showcase.netins.net/web/placenta/placentaltriage101.htm
http://www-medlib.med.utah.edu/WebPath/PLAHTML/PLACIDX.html
Neonatal outcomes following prolonged membrane rupture occurring before 24 weeks gestation.

Premature rupture of the membranes before 24 weeks’ gestation, in an otherwise normal pregnancy is, in some circumstances a diagnosis that requires the woman and her partner to make a decision based on information and counselling provided by the obstetrician and neonatologist to continue or not continue with the pregnancy. Approximately fifty percent of women will deliver within one week of preterm premature rupture of the membranes (PPROM). The remainder of pregnancies will continue for a varying time, but are unlikely to progress to term. The outcomes of pregnancies affected by PPROM before 24 weeks’ gestation are principally described in the obstetric literature; neonatal management and outcomes of live born infants are poorly described.

There are many risk factors for PPROM, the most important of which are a history of smoking in the index pregnancy and a previous pregnancy complicated by ROM or pre-term labour. PPROM infrequently complicates amniocentesis. Maternal inflammatory conditions such as chorioamnionitis and periodontitis weaken the amniotic membranes, due to the effect of a group of enzymes called matrix metalloproteases. These enzymes are activated by maternal prostaglandins that also promote cervical ripening and myometrial contractions. Placental abruption is also more common in pregnancies affected by PPROM.

Management of PPROM, according to the RWH Clinical Practice Guideline ‘Rupture of the membranes: preterm, premature (PPROM)’ includes administration of erythromycin following a high vaginal swab, with the aim of reducing the effect of inflammation from bacterial colonisation of the uterus and or fetus. The canalicular phase of lung development occurs between weeks 16 and 28 of gestation. This phase is characterised by distal airway subdivision, development of surfactant producing cells and alveoli capable of gas exchange and is dependent upon lung fluid that is secreted and retained in the fetal airways. If membrane rupture occurs, amniotic fluid pressure may fall sufficiently for fetal lung fluid to drain from the primitive airways, interrupting their normal development. The fetal complications of PPROM depend on the gestation at which membrane rupture occurs, the amount of residual liquor, and the interval from membrane rupture to delivery (latent period). Interruption of the canalicular phase has been shown, in postmortem pathological specimens, to lead to pulmonary hypoplasia and pulmonary artery musculature hyperplasia. Traditionally, pulmonary hypoplasia was thought to be a fatal condition. However, some infants may have a non-fatal form of ‘pulmonary hypoplasia’ with early severe hypoxic respiratory failure requiring intensive respiratory support. In addition, the abnormal pulmonary artery vasculature may cause pulmonary hypertension and reduced pulmonary artery flow, which itself can contribute to hypoxia.

Attempts have been made to correlate various antenatal fetal parameters with post-natal respiratory illness severity. Single linear measurements of fetal lung size correlate poorly with post-natal function. A combination of lung length, thoracic and abdominal circumference and middle pulmonary artery flow is reported to be the best predictor of fatal pulmonary hypoplasia. Once membrane rupture has occurred, few therapeutic options exist to alleviate the fetal pulmonary effects of amniotic and lung fluid loss. Two small studies report the use of trans-abdominal amnioinfusion to improve the amniotic fluid volume. Combined, these studies show an overall significant reduction in fatal pulmonary hypoplasia in women where the AFI improved with amnioinfusion. Further evidence from well designed and executed randomised controlled trials is required before amnioinfusion can be recommended in all cases of PPROM. In addition, assessment of important morbidity such as the possibility that amnioinfusion in these circumstances may increase the risk of intrauterine infection has not been adequately addressed.

A literature search of neonatal outcomes from pregnancies affected by PPROM at less than 24 weeks’ gestation was undertaken. Ten cohort studies from 1990-2004 described a total of 330 pregnancies. Reported neonatal survival tended to be higher with advancing gestation of membrane rupture, but was not consistent between the cohorts and there was no overall improvement in survival over this period. The reported neonatal survival ranged from 0-60% for membrane rupture between 15 and 23 weeks gestation.

We report neonatal outcomes from pregnancies managed at RWH complicated by a prolonged latent period (>14 days) of PPROM occurring before 24 weeks’ gestation between 2001 and 2006. Since pregnancies that continue in the presence of PPROM are a source of clinical uncertainty, the duration of membrane rupture was specified for two reasons: the studies described above reported outcomes of pregnancies with any length of latent period, yet animal models demonstrate that it takes at least 5 days of membrane rupture and amniotic fluid loss for pulmonary hypoplasia to begin to evolve. Forty liveborn infants were identified with PPROM at 14 to 23 weeks gestation. Overall survival to discharge was 70%, the majority of deaths occurring in the first 72 hours. All but one of the mothers received a complete course of antenatal steroids.

...23% of infants never required intubation and ventilation...

The degree of lung disease, as determined by the need for respiratory support, varied widely between infants. One of the most interesting findings was that 23% of infants never required intubation and ventilation. Their respiratory management comprised nasal CPAP, oxygen or no respiratory support and all survived. Overall neonatal survival was strongly associated with the need for intubation at any time during the admission. The most severely affected infants required high frequency oscillatory ventilation. In addition, five infants received rescue inhaled nitric oxide within the first 24 hours of life and only two survived. This finding is consistent with results of large randomised trials which have failed to demonstrate improved pulmonary blood flow or outcome in preterm infants treated with inhaled nitric oxide. Early pneumothoraces were common and surviving infants had a high incidence of chronic lung disease.

With respect to other neonatal morbidity, no surviving infants had evidence of retinopathy of prematurity or major grades of intraventricular haemorrhage. Only one infant had continued on page 5...
Profile
Fiona Cullinane

Some clinicians are leaders by virtue of their position within an organisation; others are leaders because of personal attributes, such as the ability to influence others, galvanise involvement and generate change. During her time here at the Women’s and as Director of Birth Suites, Fiona Cullinane has given exemplary leadership in improving clinical practice by virtue of personal attributes and position. Some of the areas where she provided leadership include, not in order of importance:

- Improving the management of postpartum haemorrhage
- Gaining an understanding of the reasons for the current caesarean section rate by applying the Robson classification
- Follow up of the term neonate admitted to SCN and NICU and developing a process for ongoing review and follow-up
- Enhancing the training of the junior medical staff in birth suite procedures through the credentialing processes
- Counselling of women and their families who suffer intrapartum pregnancy loss
- Improving the recording of perinatal deaths, especially around the discussion of the role of autopsy and how to seek informed consent
- The introduction of the normal birth CPG
- Management and support for women undergoing second trimester termination

Throughout her time as Director of Birth Suites, Fiona brought an infectious enthusiasm and passion and a dedication above and beyond any reasonable call to duty. Our challenge is to maintain the momentum that Fiona generated and the legacy that the woman is the centre of our care.

Fiona has taken a period of extended leave and is spending some time in Ireland. We take this opportunity to pay tribute to Fiona’s professionalism, achievements and her commitment to high standards in providing care to women.

Jeremy oats
Clinical Director Women’s Services

Reference

Antibiotic prophylaxis for hysterectomy

The Age newspaper on 1 May reported on data released by the Victorian Nosocomial Infection Surveillance Centre (VicNISS) which found that in 56% of hysterectomies in Victorian hospitals, antibiotic prophylaxis was inadequate. The IMPACT team here at the Women’s collected 12 months of prophylactic antibiotic data in 2003 and 2004 and compliance was 86% (compared with 97% for caesarean section over the same period). Discussions around the management of hysterectomy patients has led to a significant increase in compliance with 94% of women undergoing hysterectomy in the first quarter of 2006 receiving prophylactic antibiotics. In 2003/04 the infection rate in women who did not receive antibiotics was more than twice the rate in those who did, so this is a significant and worthwhile improvement in practice.

Neil Everest
Neonatal Fellow

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limb contractures secondary to oligohydramnios and was delivered following the longest latent period.

Placental histology was available in 38 cases, with 70% showing mild to severe degrees of chorioamnionitis and/or funisitis. Infants delivered in the setting of chorioamnionitis had a higher incidence of chronic lung disease, consistent with current theories of maternal inflammation and adverse neonatal outcome.

We have demonstrated that infants delivered at RWH after a latent period of at least 14 days following PPROM before 24 weeks’ gestation have improved survival compared with the recent literature. This information may assist both clinicians and women making decisions to continue with an affected pregnancy. Further research is required to determine the role of amnioinfusion in pregnancies complicated by PPROM, to identify which fetuses are at greatest risk of pulmonary hypoplasia and/or pulmonary hypertension, and to develop resuscitation and ventilation strategies that improve infant survival and reduce the risk of chronic lung disease.

Neil Everest
Neonatal Fellow

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Daktarin® (miconazole) oral gel warning from pharmaceutical company

On April 3rd 2006, Janssen-Cilag pharmaceutical sent an alert to pharmacists regarding changes to Daktarin® oral gel Product Information stating “Daktarin® (miconazole) oral gel label change – contraindicated under 6 months of age”.

The Royal Women’s Hospital Drug Information Centre contacted Janssen-Cilag regarding this change, and was advised that the reason for change was due to verbal reports of choking and airway obstructions in babies. This was most likely due to the viscosity of the product and the manner in which it was administered.

The Royal Women’s Hospital recommendation

Daktarin® oral gel is an effective treatment for oral thrush in the babies. The gel allows long contact with oral mucosa for maximum therapeutic effect. We are happy to continue recommending this product for babies < 6 month of age as long as clear instructions are given regarding administration to avoid risk of airway obstructions.

The Clinical Practice Guideline on management of thrush in lactation is available on our web and if Daktarin® oral gel is required, the patient is given a handout on how to apply Daktarin® oral gel to the baby’s mouth.

Instructions

• Use the spoon to measure a ¼ teaspoon dose. The spoon should not be used for administering the gel.
• Using a clean finger, apply small amounts of gel at a time to the inside cheeks and over the tongue.
• Apply the gel four times a day after feeds for one week then once a day.

Clinical Practice Guideline


BEss fact sheet for Daktarin® oral gel patient instruction (Breast and nipple thrush) is available from BESS and pharmacy.

For further inquiry contact:
The Royal Women’s Hospital Drug Information Centre
Telephone: 03 9344 2277 or email: drug.information@rwh.org.au
Breastfeeding Education & Support Services (BEss)
Telephone: 93443651 or email: bess@rwh.org.au

References:
1.  Janssen-Cilag. Daktarin (miconazole) oral gel label change. April 3rd 2006 (copy can be obtained from the Pharmacy Department).