

Magazine Vol 12 No 2 Winter 2010

Lightening The third trimester of pregnancy

The Royal Australian and New Zealand College of Obstetricians and Gynaecologists



Available online at: www.ranzcog.edu.au/publications/oandg

O&G Magazine Advisory Group

Prof Caroline de Costa Council Rep, QLD Dr Sarah Tout Council Rep, New Zealand A/Prof Stephen Robson Fellows Rep, ACT Dr John Schibeci Diplomates Rep, NSW Dr Brett Daniels Trainee Rep, TAS

O&G Magazine Editors

Penelope Griffiths Julia Serafin Peter White Rachel Corkery

Designer and Production Editor

Rachel Corkery

Editorial Communications

O&G Magazine Advisory Group, RANZCOG 254-260 Albert Street EAST MELBOURNE, VIC 3002 Australia (t) +61 3 9417 1699 (f) +61 3 9419 0672 (e) ranzcog@ranzcog.edu.au

Advertising Sales

Bill Minnis Director Minnis Communications (t) +61 3 9824 5241 (f) +61 3 9824 5247 (e) info@minniscomms.com.au

Printer

Fineline Printing Australia Pty Ltd (t) +61 3 8791 4200 (f) +61 3 8971 4277

O&G Magazine authorised by Dr Peter White © 2010 The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG). All rights reserved. No part of this publication may be reproduced or copied in any form or by any means without the written permission of the publisher. The submission of articles, news items and letters is encouraged.

> For further information about contributing to O&G Magazine visit: www.ranzcog.edu.au/publications/oandg

The statements and opinions expressed in articles, letters and advertisements in $O \dot{\sigma} G$ Magazine are those of the authors and, unless specifically stated, are not necessarily the views of the RANZCOG.

Although all advertising material is expected to conform to ethical and legal standards, acceptance does not imply endorsement by the College.

ISSN 1442-5319

Cover image ©Shutterstock_Kirill Vorobyev

Lightening: The third trimester of pregnancy

- 13 Editorial: Lightening John Schibeci
- 14 Metformin in pregnancy Anna McLean and Paul Howat
- 15 Imaging placenta accreta Rajeev Jyoti and Meiri Robertson
- 19 Perineal preparation Ann Yates
- 20 Management of prolonged pregnancy yesterday, today and tomorrow **David Bailey**
- 22 Attempting vaginal birth after a previous caesarean section Stephen Robson
- 26 Infections in the third trimester of pregnancy Jen Kok and Lyn Gilbert
- 28 Gene-environment interactions and the developmental origins of health and disease Craig Pennell
- 31 Substance abuse in pregnancy Joanne Ludlow
- 35 Management of PPROM in the third trimester Sarah Buchanan and Jonathan Morris

Women's Health

- 38 Magnesium sulphate in women at risk of preterm birth for neuroprotection of the fetus
 Susan Walker
- 42 Diagnosis and management of benign vulval dermatological disorders Catherine Drummond
- 51 Journal Club Brett Daniels
- 52 Surgical Safety Checklist (World Health Organisation)
- 54 *Q&a:* Utero-vaginal prolapse and the use of a ring pessaryLynsey Hayward
- 55 Gynaecological Management Update: Emergency contraception Caroline Harvey
- 60 Improving outcomes for women with gynaecological cancer Cancer Australia and RANZCOG
- 61 Supporting women living with gynaecological cancers Cancer Australia

RANZCOG Regional Committees

New Zealand Dr John Tait Chair

Ur John Iait Chair Kate Bell Executive Officer Level 3, Alan Burns Insurances House 69 Boulcott Street/PO Box 10 611 WELLINGTON, NEW ZEALAND +64 4 472 4608 (f) +64 4 472 4609 (f) kate.bell@ranzcog.org.nz (e)

Australian Capital Territory

Dr Andrew Foote Chair Deakin Gynaecology Centre 39 Grey Street DEAKIN, ACT 2600 +61 2 6273 3102 (t) +61 2 6273 3002 (f) muttons@dynamite.com.au (e)

New South Wales

Professor Alec Welsh Chair Lee Dawson Executive Officer Suite 4, Level 5, 69 Christie Street ST LEONARDS, NSW 2065 +61 2 9436 1688 (t) +61 2 9436 4166 (f) admin@ranzcog.nsw.edu.au (e)

Queensland

Dr Paul Howat Chair Lee-Anne Harris Executive Officer Unit 22, Level 3, 17 Bowen Bridge Road HERSTON, QLD 4006 +61 7 3252 3073 (t) +61 7 3257 2370 (f) Iharris@ranzcog.edu.au (e)

South Australia/Northern Territory

Dr Preeti Khillan Chair Tania Back Executive Officer 1-54 Palmer Place/PO Box 767 NORTH ADELAIDE, SA 5000 +61 8 8267 4377 (t) +61 8 8267 5700 (f) ranzcog.sa.nt@internode.on.net (e)

Tasmania

Dr Stephen Raymond Chair Hobart Urogynae & Incontinence Clinic 4/44 Argyle Street HOBART, TAS 7008 +61 3 6223 1596 (t) +61 3 6223 5281 (t) rfullert@tassie.net.au (e)

Victoria

Dr Elizabeth Uren Chair Fran Watson Executive Officer 8 Latrobe Street MELBOURNE, VIC 3000 +61 3 9663 5606 (t) +61 3 9662 3908 (f) vsc@ranzcog.edu.au (e)

Western Australia

Dr Tamara Walters Chair Janet Davidson Executive Officer Level 1, 44 Kings Park Road WEST PERTH, WA 6005/PO Box 6258 EAST PERTH, WA 6892 +61 8 9322 1051 (†) +61 8 6263 4432 (†) ranzcogwa@westnet.com.au (e)

The Royal Australian and New Zealand College of Obstetricians

and Gynaecologists College House 254-260 Albert Street EAST MELBOURNE, VIC 3002 +61 3 9417 1699 (t) +61 3 9417 0672 (f) ranzcog@ranzcog.edu.au (e)

> President Dr Ted Weaver Vice Presidents Prof Michael Permezel Dr Rupert Sherwood Dr Digby Ngan Kee Honorary Secretary Dr Gino Pecoraro Honorary Treasurer Dr Bernadette White Chief Executive Officer Dr Peter White

www.ranzcog.edu.au (w)

62 Letter to the Editor: Kjelland's rotational forceps Peter Monks

- 63 A life-changing experience in Ethiopia Maurice Lichter
- 66 Fostering links with Fiji Kirsten Connan and Daniel Jolley
- 89 Lifetime Achievement Award (ASCCP) Malcolm Coppleson Louise Farrell
- 90 Sydney IVF donates ultrasound machine to Papua New Guinea Glen Mola

Medico-legal

58 Obligations when dealing with patients under 18 years of age Sonia Grover

The College

- 5 From the President Ted Weaver
- 9 From the CEO Peter White
- 45 Meetings Calendar Winter 2010
- 68 A Launceston odyssey reflections on a SOLS experience Wah Hin Lee
- 70 College Statements Update March 2010 Michael Permezel
- 80 Council Meeting Report March 2010 Penelope Griffiths
- 84 RANZCOG Research Foundation Scholarship Recipients John Newnham and David Healy
- 90 RANZCOG Women's Health Award 2009
- 92 News from the Historical Collections
- **93** Obituaries
- 94 Staff News
- 94 News from the Frank Forster Library



From the President



Dr Ted Weaver President

he winter edition of O&G Magazine's theme is 'Lightening: The third trimester of pregnancy'. Lightening is one of those delightful old terms, like attitude, denominator and station, that older consultants like to use to bamboozle medical students and junior doctors on ward rounds. It harks back to the era when obstetrics was, as it still is, a hands-on specialty, albeit with a changed set of clinical tools. This issue deals with common third trimester problems that we encounter frequently in practice, such as management of diabetes, the management of post dates pregnancy and counselling before

an attempt at a vaginal birth after a previous caesarean section. Having just come from managing a 44-year-old obese woman, who was post dates, through labour, I can attest to the timeliness of these articles. I would like to thank all the authors of this edition of $O \not \sim G$ *Magazine* for their invaluable contributions.

Since the last issue of $O \not \subset G$ Magazine, there have been many areas of intense activity for Executive and Council and for College House staff. Some of these activities are highlighted below.

Release of NHMRC Guidance Document for Collaborative Care

The National Health and Medical Research Council (NHMRC) were tasked by the Australian Federal Government to develop a guidance document about collaborative maternity care, to underpin the legislated maternity changes due to start in Australia in November 2010. The guidance was developed by a working party of approximately 20 health professionals from different areas of the maternity workforce. The group was chaired by Professor Chris Baggoley, an emergency medicine specialist, who is the current Chief Executive of the Australian Commission on Safety and Quality in Health Care (ACSQHC). There were two obstetricians on the group, myself and Professor Alec Welsh; one GP obstetrician, Dr Ruth Stewart; two representatives from the Australian College of Midwives, one midwife in private practice and one midwife from Belmont Hospital in Newcastle; two Maternity Coalition consumers; and a midwifery professor from Brisbane, who has extensive experience in rural and remote health, and in indigenous health.

The group developed a document which was then taken to a key stakeholder's forum in Canberra in December 2009 and the document further developed to its present state. It has been sent to all Australian Fellows practising in obstetrics and NHMRC have had a group travelling to different states seeking useful feedback. RANZCOG will send a formal response to NHMRC.

Merging of RANZCOG and ACM Maternity Referral Guidelines

Side by side with this process is a process to merge RANZCOG and the Australian College of Midwives (ACM) Referral Guidelines, to produce a document that will underpin the proposed collaborative maternity reforms scheduled to commence in November of this year. RANZCOG has agreed with ACM to convene a small writing group which will work through the guidelines and produce a merged document for discussion. ACM requested that there be consumer representation on this group and RANZCOG agreed, on the proviso they were selected through a robust process and could clearly be seen to be independent consumer representatives. Concerns with the two consumers ultimately selected was expressed to NHMRC and, at the time of writing, the College is heartened by the understanding that the process will now involve the small writing group of RANZCOG and ACM representatives proposed initially, with consumer and other relevant stakeholder input gathered once that small group has produced a draft set of merged guidelines. I have committed RANZCOG to ensuring the merge does happen, but by a process that is fair and transparent.

The Australian Federal Government is very eager for the Referral Guidelines to be merged and see them as a way of being able to audit quality of practice, and possibly use them to make decisions on whether eligible midwives would be indemnified for their practice.

Another parallel process going on is the development of a safety and quality framework around the midwives, who have been granted exemption under the National Registration and Accreditation Scheme (NRAS) for the next two years. RANZCOG has been involved in discussions about this, with Professor Jeremy Oats and his team, and have insisted that matters such as vaginal birth after caesarean section (VBAC) at home must not be allowed under the framework. This group is also eager to see the release of the Referral Guidelines mentioned above, which will pronounce on place of birth, defining such things as when intrapartum care starts, what 'failure to progress' is, what the appropriate lengths of the different stages of labour are, etc.

National Registration

RANZCOG has received advice from the National Medical Board that they need to populate the specialist database and have requested the College to provide information from our databases to do this. We have sought legal advice and will progress this as appropriate. The new board is due to start functioning on 1 July 2010 and will greatly simplify registration. There will also be a better, more streamlined process for dealing with complaints to medical boards about doctors.

Practice Profile

Analysis of the practice profile has been completed and is now in the practice profile section of the RANZCOG website. It has two new questions. Fellows can look at the results of the profile so far, which is interesting, and complete the other two questions. It will be a useful data set when the proportion of Fellows completing it gets as close to 100 per cent as possible, which will help in workforce planning and in the planning of content for the training program.

PROMPT

It was announced at the RANZCOG 2010 Annual Scientific Meeting in Adelaide that the Victorian Managed Insurance Authority (VMIA) has secured the Australian licence for the PRactical Obstetric MultiProfessional Training course, appropriately named PROMPT, and have gifted it to RANZCOG. This is an exciting development and one that could be developed into a RANZCOG program which could be rolled out in Australia and New Zealand, recognising that PROMPT has already started in New Zealand, under Dr Martin Sowter's capable direction. Dr Malcolm Barnett from Box Hill in Victoria is chairing a pilot group, and progress and outcomes of the project will have an evaluation at the end of 2010. Following that, if it is satisfactory, there will be an effort made to 'train the

Continued on page 6.

trainers', and the project developed in different states, along the lines of the Fetal Surveillance Education Program (FSEP). If delivered well, PROMPT should improve training and safety in maternity care, and fit in well with the collaborative maternity care models that are currently mooted.

RANZCOG 2010 Annual Scientific Meeting

The RANZCOG 2010 ASM ran from 21 to 24 March 2010 and was held at the Adelaide Convention Centre in South Australia. The theme of the meeting, *'It's not all black and white'*, encapsulated well the dilemma we face often as clinicians when trying to decide the best management for our patients, as well as reflecting the Adelaide Zoo's proud acquisition of two pandas. A highlight of the meeting was a talk given by the chief vet for the pandas.

The meeting was very enjoyable and well-subscribed, with over 600 registrants. The Diplomates' Day was oversubscribed and the workshops the day before the conference were fully booked. Overall, it was a very successful meeting, both scientifically and socially. I would like to pay tribute to Dr Chris Hughes and his organising team for the great job they did, and to Associate Professor Martin Oehler, Dr Jodie Dodd and the rest of the scientific team, for the scientific program which was exemplary in its interest and breadth.

Professor Alastair MacLennan delivered the Arthur Wilson Oration and gave a superb talk, much enjoyed by all.

The next RANZCOG ASM will be held in Melbourne in October 2011, so please mark it in your diaries.

GP Procedural Training Support Program

The Australian Federal Government is seeking to address structural and financial barriers that impinge on the capacity of individual doctors to increase their skills, and have provided financial support for GPs to improve their skills in obstetrics and anaesthetics, if they work in remote and rural locations. The program will provide support for 110 GPs over four years, with an aliquot of A\$40,000 for each GP. The program is open to GPs who have commenced procedural training from July 2009 and is also open to DRANZCOG holders.

RANZCOG will be responsible for:

- Undertaking a selection process
- Promoting the program
- Financial support and entering into a fund-holding agreement with each GP
- Monitoring progress
- Monitoring DRANZCOG Advanced completion and notifying the department.

The program will be monitored by the General Practice Obstetrics Advisory Committee (GPOAC) and College House staff.

Accreditation of Practices Providing Diagnostic Imaging

The Australian Federal Government has set up the Diagnostic Imaging Accreditation Scheme. The legislation for this was passed in June 2007. Under the scheme, mandatory accreditation of providers of diagnostic imaging would be linked to the payment of Medicare benefits for radiology and non-radiology services.

When this was first mooted, the College had meetings with the Department of Health and Ageing, pointing out that the standard of accreditation for a radiology practice, or a practice dedicated to diagnostic imaging, such as obstetric ultrasound, should be different from that which applied to practitioners providing nonreferred ultrasound in obstetrics and gynaecology in private rooms. One of the reasons for this was because practitioners in obstetrics and gynaecology often use ultrasound in their rooms, as a clinical adjunct, to answer a specific clinical question, for example: 'What is the presentation of a particular fetus?' Used in this way, it provides a cost-effective alternative to referred ultrasound. Also, it was thought that the compliance and accreditation requirements for referred services, for things such as image capture and storage, reporting and notification of adverse findings, and medico-legal considerations, were vastly different when compared to a practice doing non-referred scans. RANZCOG was of the view that future accreditation requirements could be met through the College's Continuing Professional Development program.

The Government, though, has tended to ignore that advice and has deemed that all providers of non-referred scans, if they intend to access Medicare benefits, must be registered for accreditation by 1 July 2010. I have written to the Fellowship about this, informing them of their need to register for the scheme before 1 July 2010, if they wish to charge Medicare for non-referred scans.

Health Workforce New Zealand

The College has written and submitted an excellent document to Health Workforce New Zealand. This document will be crucial in helping the New Zealand Government plan their future workforce in obstetrics and gynaecology. The document highlights a number of areas of concern the College has about the obstetric and gynaecological workforce in New Zealand. These relate to the inequity of distribution of Fellows, the lack of subspecialists in different areas of practice, an undue reliance on overseastrained specialists and the almost complete demise of the general practitioner obstetrician.

Seventh RANZCOG Council

Nominations will be called soon for Fellows to nominate for election to RANZCOG Council. This will be a ground-breaking Council, as it will be the first to operate under the recently approved new governance structure and will work with the Executive Board. I would encourage you strongly to nominate for election, as it is important that the College's Fellows keep coming forward to give their time 'pro bono' in the running of the College. Involvement in Council can be immensely rewarding professionally, intellectually and socially. I would suggest you give serious thought as to how you, wherever you are in your professional life, could contribute to RANZCOG, your professional body.

Finally, I wish to congratulate Dr Rupert Sherwood on his successful election as the next RANZCOG President and wish him well during his term in office. As mentioned above, it will be an interesting time, with an Executive Board working with Council and with Council meetings adopting a different format.

From the CEO



Dr Peter White Chief Executive Officer

n his introduction to this edition of *O&G Magazine*, Dr John Schibeci speaks of the derivation of 'lightening' and its implication of things becoming 'less heavy', yet, paradoxically, in obstetrics also being the portent of events of major significance that represent the culmination of the gestation process. For those who follow developments in the environment in which you practice and in which the College functions, the suggestion at the moment would probably be that, on balance, there is no clear onset of 'lightening' on either side of the Tasman, with all activity currently in

train continuing to produce an environment of relatively uncertain, yet no doubt exciting, outcomes in the way medicine in general and this specialty is taught and delivered.

As I write this column, Winter in Melbourne is clearly on the way. Temperatures are becoming more reflective of that season, three of the scheduled six meetings of the College Executive for the year have been completed and the activities of the year are in full swing. This applies both inside and outside the College. For example, a successful RANZCOG Annual Scientific Meeting has been run in Adelaide by Dr Chris Hughes and his team and, by the time you are reading this magazine, members registered in Australia should have received correspondence from the Medical Board of Australia (MBA) in relation to arrangements for the National Registration and Accreditation Scheme (NRAS). The correspondence urges medical practitioners to ensure their details with current State and Territory medical boards (and communicated in the letter from the MBA) are correct, and to advise the Australian Health Practitioner Regulation Agency (AHPRA) of incorrect information. The College has been asked to reinforce this request in order to aid all who are impacted by this change to transition to the new scheme as efficiently as possible. That said, at the time of writing, it was understood that three States, Western Australia, South Australia and Tasmania, had not passed the necessary legislation involved to enable the scheme to operate fully by 1 July 2010. However, the scheme will proceed and it behoves potential registrants of the new scheme to ensure that the Board possesses accurate information about them.

Also in regard to NRAS, the College is in the process of formulating responses to the MBA relating to standards for two registration categories contained within the most recent discussion paper distributed by the Board. That is, limited registration for teaching or research and limited registration in the public interest, in order to ensure as much as possible that both members and the public are well-served by the new scheme.

As I indicated in the Autumn edition of *O&G Magazine*, information relating to NRAS may be found on the AHPRA website (www. ahpra.gov.au) with a link to MBA (www.medicalboard.gov.au) also available on this website. Among other information on the websites, of interest to College members practising in Australia may be the Registration Standard relating to Continuing Professional Development that was approved by the Australian Health Workforce Ministerial Council on 31 March 2010 (access at: www. medicalboard.gov.au/documents/Continuing%20professional%20 development.pdf).

The Standard indicates that 'CPD must include a range of activities to meet individual learning needs including practicebased reflective elements, such as clinical audit, peer-review, or performance appraisal, as well as participation in activities to enhance knowledge such as courses, conferences and online learning', and that 'CPD programs of medical colleges accredited by the Australian Medical Council (AMC) meet these requirements'. Further, it indicates that 'Members or fellows of medical colleges accredited by the AMC can only choose a self-directed program of CPD if that program meets the standards for CPD set by their college'. In short, as a specialist college accredited by the AMC, participation in the RANZCOG CPD program will enable the CPD requirement for RANZCOG Fellows under NRAS to be satisfied.

College members will have by now most likely received their annual subscription notices from the College for the 2010- 2011 financial year, with increases held to the relevant Australian Consumer Price Increase (CPI). The subscription notices will have been accompanied by a letter from the College President indicating, among other things, the need for College Fellows to complete a Renewal of Fellowship Declaration. This is the second year in which completion of this declaration has been a requirement for the annual renewal of the College Fellowship and represents a sign from the College of the importance with which it views its role in maintenance of standards, both from the perspective of the Fellowship and the public, as well as representing another aspect of risk management from the perspective of the College as an accountable organisation. The work and the task of running the College is increasingly becoming a more complex undertaking and the assistance of Fellows in completing and returning the completed declaration is greatly appreciated. As with the non-payment of subscriptions by the due date, reminders are sent periodically to Fellows who do not return the declarations and it gives no one associated with the process any joy or satisfaction to have to write to Fellows, who for one reason or another, have not completed the requirement by the required time following three reminders that their Fellowship is to be removed. Based on experience, refinement of the declaration from last year's version has been undertaken at College House and the declaration is this year also available for completion online. As always, College staff are able to assist any Fellow requiring assistance in relation to the completion of the declaration.

Also available for completion online is the College Workforce Survey for Fellows, with a new survey for Diplomates to be launched in July. Both surveys represent increasing efforts by the College to gather meaningful data in order to inform increasing debate and activity in relation to maternity workforce. The College needs such information in order to be able to contribute meaningfully from an evidence base to the deliberations that will be undertaken in this area with the advent of bodies such as and Health Workforce New Zealand. I would echo the sentiments of the President in seeking all members to complete the relevant workforce survey and, thus, contribute to the solution of the O and G component of the workforce debate.

As always, within the College, a range of activities continue in relation to what is generally considered our core business; education and training. The work of the Conjoint Committee for the Diploma of Obstetrics and Gynaecology (CCDOG) is moving forward as the body that now oversees the DRANZCOG and DRANZCOG Advanced qualifications, and includes development of revised curricula for both qualifications. The work of the FRANZCOG Training Program Review Working Party and the Overseas Trained Specialist Assessment Working Party is gearing up, and the trial of a CPD program that aligns CPD more closely to the RANZCOG curriculum is progressing.

Much activity is being undertaken in regard to the Specialist Training Program initiative of the Commonwealth Government and there is also significant effort and resources being committed in relation to the development of online educational materials to support all categories of College members and trainees. The College is very aware of the need to ensure that it continually examines and refines its education offerings and the ways in which they are delivered, to ensure that it remains at the forefront of its sector in this regard. The work that is undertaken can be appreciated in a 'snapshot' manner every year when an Annual Report is produced for the purpose of the AMC accreditation process. The work that is reported is a testament to the commitment of College members and staff in that 'partnership' that I have written and spoken about on many occasions. To move forward in that partnership, the organisation continually needs contributions from members and I would once again implore anyone who thinks they may be able to contribute in some way to the work of the College to do so.

As I indicated in the Autumn edition of *O&G Magazine*, all involved with College affairs were very aware of the impending election at the then upcoming March meeting of Council for the next College President. I echo the current President, Dr Ted Weaver, in congratulating Dr Rupert Sherwood on his election to that role, one that will see him lead not only the Seventh RANZCOG Council, but also the inaugural RANZCOG Board, following overwhelming support from the Fellowship for a change in the governance arrangements underpinning the organisation. The remaining positions on the Board will be decided at the July 2010 meeting of Council and the Board and new Council members will take office from the College Annual General Meeting in November 2010.

As anticipated, the year is moving swiftly and the College is paying attention to activity on many fronts. The work of the current Council and Executive under Dr Weaver will continue unabated for the remainder of their period of Office, when once again the cycle of renewal and progress will take place. Some may experience a 'lightening', however, the College and the profession continues to be aware of the need for careful observation, monitoring and judicious intervention to guide a safe outcome.



Lightening



A corollary to the recent edition of $O \mathscr{C} G Magazine$, 'Obstetrics: art or science?'¹, is that obstetrics is replete and at the same time enriched by its own archaic terminology. The term 'lightening' is one such quaint term, yet it quintessentially symbolises the third trimester in the same way that the second trimester was branded by 'quickening' in the corresponding edition of $O \mathscr{C} G$ Magazine.²

Dr John Schibeci Dranzcog

'Lightening' for the Latin scholars is the gerund derived from the verb 'to lighten', which means 'to make less heavy'. So applied to obstetrics, lightening means

'...the sensation, experienced by many women late in pregnancy when the head of the fetus enters the pelvis, of a reduction in pressure on the diaphragm, making it easier to breathe.'³ I have been unable to determine the origin of its use in midwifery and obstetrics, but I do remember in the 'olden days' before our practices were dictated to by ultrasonography, emblazoned on the antenatal cards were the terms 'quickening' and 'lightening'. In the attached boxes we could affix the relevant dates, which helped to estimate the dates of confinement for our more forgetful patients.

It has always been taught that in the primigravida lightening occurs at 36 weeks, which we all know from our practices is not true. Weeks and colleagues⁴ suggested that in the majority of primigravidae, the fetal head does not engage between 36 and 38 weeks, but more importantly, that in 80 per cent of patients, the engagement-delivery interval was less than 14 days.

'Lightening', however, is truly an oxymoron, for in the expectant mother, it heralds an increase in burden in many other ways from pelvic and back discomfort to the mother of all burdens – labour. Likewise, for the obstetrician, the third trimester is the 'business end' of the pregnancy and with it comes more problems than in the rest of the pregnancy – there is certainly no lightening of burden here. To ease the load, we have assembled a number of very interesting and challenging articles from diverse areas such as metformin use in gestational diabetes; the perennial problem of the management of PPROM; the caesarean scar; and the prolonged pregnancy. As always, we would like to thank all authors for their generous donation of their time and words of wisdom.

- 1. O&G Magazine, Vol 11 No 4 Summer 2009.
- 2. OćrG Magazine, Vol 11 No 2 Winter 2009.
- 3. Collins English Dictionary. 2003.
- Weeks ARL, et al. British Journal of Obstetrics and Gynaecology 1975; Volume 82 (1) p7-11.

Metformin in pregnancy

Dr Anna McLean

FRACP Endocrinologist Cairns Base Hospital

Dr Paul Howat

FRANZCOG Director of O and G **Cairns Base Hospital** It seems a logical treatment option, but it does cross the placenta and there have been concerns about possible adverse fetal outcomes. The mainstay of management of GDM remains diet, exercise and insulin therapy. However, metformin is a valid addition/alternative offer some advantages in terms of

to insulin therapy and may offer some advantages in terms of avoiding maternal weight gain and hypoglycaemia.

Metformin acts by reducing insulin resistance through various mechanisms. These include a reduction in hepatic glucose production, decreased absorption of glucose and increased peripheral uptake and utilisation of glucose. Serious side effects are rare. Common side effects include abdominal bloating, diarrhoea, indigestion and nausea. However, metformin is generally well tolerated.

The first prospective randomised controlled trial, *Metformin Versus Insulin for the Treatment of Diabetes in Pregnancy* published in 2008¹, showed that metformin was not associated with increased perinatal complications compared to insulin. This study included 751 women with gestational diabetes and used doses of up to 2500mg daily. Metformin was found to be safe for mother and infant and follow-up studies in the offspring are awaited. Metformin had the obvious advantage of being in tablet form rather than injections and women preferred metformin over insulin. In the last few years, the use of metformin in pregnancy has become more widespread with positive outcomes.²

The Australasian Diabetes in Pregnancy Society currently does not recommend the use of metformin in pregnancy. However, their position statement is up for review later in 2010. British guidelines do include metformin as an adjunct or alternative to insulin.³ We believe that there is a definite role for metformin in the management of gestational diabetes and the following scenarios are provided as examples.

A 32-year-old woman with polycystic ovarian syndrome (PCOS) and impaired glucose tolerance has been trying to conceive for three years. She recently started metformin 500mg bd and then finds out she is six weeks pregnant. She wonders what to do now with this medication.

- Metformin has been extensively studied in the PCOS population, where there is a wealth of safety data showing no increased teratogenic effects.⁴
- Metformin decreases the rate of miscarriage in PCOS.
- Metformin in PCOS reduces insulin resistance and can return the patient to ovulatory status.
- Good blood sugar control in early pregnancy is vital in order to reduce the risk of fetal anomaly.
- In this case, metformin should be continued.

A 25-year-old woman is screened for gestational diabetes at 28 weeks gestation and her two-hour blood sugar level (BSL) is 10mmol/L. She monitors her BSLs for one week and they are all slightly over target. She is advised to take insulin, however, is very

14 O&G Magazine

Metformin is a biguanide oral hypoglycaemic agent whose place in the management of type II diabetes mellitus is well established. Its use for type II diabetes in pregnancy and gestational diabetes (GDM) has been controversial.

reluctant to do so as she has a needle phobia. She asks if there is any alternative. Is metformin a reasonable option?

- Metformin is a reasonable option if the patient refuses insulin.
- The patient must be counselled that up to 50 per cent of women will require insulin as well to achieve good control.¹
- The patient can be reassured that there is now data showing that metformin is safe in pregnancy.¹
- The only contraindication for use of metformin is presence of fetal growth restriction.²

A 38-year-old type 2 diabetic woman falls pregnant unexpectedly. She is taking metformin 1g bd, perindopril 5mg daily, atorvastatin 40mg daily and gliclazide MR 60mg daily. Her GP tells her to stop taking all of her medication immediately. Is this appropriate?

- Metformin should be continued while awaiting specialist review.⁵
- It is important that any change in therapy occurs without deterioration in glycaemic control in early pregnancy. Stopping metformin may result in greater teratogenic risk by exposing the fetus to high blood sugar levels.⁵
- ACE inhibitors/statins/sulphonylureas should be ceased.

A 29-year-old woman with type 2 diabetes and obesity has been taking insulin and metformin during pregnancy and delivers a healthy baby boy at 38 weeks. She wants to breastfeed but is concerned about the risk of breastfeeding with metformin.

- The dose of metformin in breast milk is much less than the usual ten per cent level of concern and women can be reassured that it is unlikely to have any effect on their baby.^{6,7}
- In particular, because of the mechanism of action of metformin, there is no risk of hypoglycaemia as opposed to drugs which stimulate insulin secretion, such as sulphonylureas.
- Maintenance of normal blood sugar while breastfeeding is important to reduce the risk of obesity in the child.

A 32-year-old woman has gestational diabetes and is told that this means she will have to be induced at 38 weeks if she needs insulin. Her sugars are high, the baby is macrosomic and her insulin requirements are now several hundred units a day despite maximum efforts.

- Metformin reduces insulin resistance and may help to control this patient's blood sugar and reduce her insulin requirements.
- Induction prior to 38 weeks may be necessary in this patient.

Although not a first-line drug, metformin has a definite role to play in the management of gestational and type II diabetes in pregnancy.

- Rowan JA, Hague WM, Gao W, et al. Metformin versus insulin for the treatment of gestational diabetes. N Engl J Med. 2008; 358:2003-15.
- Hyer SL, Balani J, Johnson A, Shehata H. Metformin treatment for gestational diabetes. *British Journal of Diabetes & Vascular Disease* 2009; 9; 220-225.
- 3. National Institute for Health and Clinical Excellence Diabetes in Pregnancy: full guideline. London: NICE, 2008.
- Palomba S, Falbo A, Zullo F, Orio F. Evidence-based and Potential Benefits of Metformin in Polycystic Ovary Syndrome: A Comprehensive Review. Endocrine Reviews 2009; 30(1):1-50.
- 5. Simmons D, Walters B, Rowan JA, McIntyre HD. Metformin therapy and diabetes in pregnancy. *MJA* 2004; 180: 462-464.
- Hague WM. Metformin in pregnancy and lactation. Aust Prescr. 2007; 30:68-9.
- Glatstein MM, Djokanovic N, Garcia-Bournissen F, Finkelstein Y, Koren G. Use of hypoglycemic drugs during lactation. *Canadian Family Physician* 2009; 55:371-373.

Imaging placenta accreta

Dr Rajeev Jyoti

FRANZCR Consultant Radiologist **Canberra Hospital**

Dr Meiri Robertson

Fetal Medicine Unit Canberra Hospital In normal placentation, the trophoblast does not invade deeper than the deciduas. Invasion of part or

all of the placenta, through the deciduas into the myometrium and beyond, is termed morbidly adherent placentation and is divided into three subtypes: placenta accreta is trophoblast invasion into the superficial layer of the myometrium; placenta increta is extension of trophoblast invasion deep into the uterine myometrium¹; and placenta percreta is trophoblastic invasion through the entire myometrium to, and sometimes beyond, the uterine serosa.¹

The risk of placenta accreta increases significantly with consecutive caesarean sections.² The complications of placenta accreta are significant and include a risk of maternal death due to severe haemorrhage, as well as damage to the uterus, bladder and bowel. These complications can be managed with prior knowledge of the underlying condition, allowing for appropriate resource allocation at the time of surgery. This may include provision of blood products, interventional radiology, appropriate surgical and anaesthetic cover, as well as intensive care facilities.²

Our ability to diagnose placenta accreta has changed over the last decade and, like most things in medicine, a high index of suspicion and experience has increased our chance to make a correct diagnosis. Ultrasound, MRI or a combination of these modalities are nowadays utilised depending on the availability of expertise and equipment. This article will explore the features of placenta accreta on ultrasound and MRI. Patients at risk of placenta accreta include those with a history of previous operative delivery; previous gynaecological procedures that disrupt the integrity of the uterus (such as curettage and myomectomy); and retained placenta. Advanced maternal age is also an independent risk factor.³

It is important for both the sonographer and sonologist to ask a patient about previous uterine surgery and also document relevant history in the report. The classical features of placenta accreta/increta/percreta at the morphology ultrasound are summarised in Table 1.^{4,5,6}

Table 1. Features of placenta accreta/increta/percreta at ultrasound

Method	Feature
2D grey scale	Loss of placental-myometrial interface Placental lacunae Placenta bulging into the bladder
Colour Doppler	Increased amount of blood vessels Turbulent blood flow
3D power Doppler ultrasound	Colour flow mapping of newly formed vessels and lacunae

A number of studies looking at all these factors have been published, some with conflicting evidence. Interestingly, a recent study suggests that if multiple features of placenta accreta are

The changing landscape of obstetric care in Australia and elsewhere in the world has brought new challenges to our practice. As well as the changing demographics of women who become pregnant, the steady rise in the rate of caesarean sections means we will encounter more complex forms of placentation, including placenta praevia and placenta accreta.

present, the risk of a false positive is far less likely. We should therefore include assessment and documentation of all the features mentioned in Table $1.^7\,$

With improved equipment and insight, this diagnosis may also be possible in early first trimester (as early as seven weeks). A retrospective study considered early features of placenta accreta to be a low-lying gestational sac and myometrial thinning of the uterine scar area.⁸ In two of the six reported cases, there was early fetal demise. Subsequent curettage lead to very heavy bleeding requiring a hysterectomy in both cases. It is also important to remember that a morbidly adherent placenta can also occur without any risk factors. We should take care to look at every placenta at morphology study to allow for appropriate referral to a tertiary care centre in case of suspicious findings.

Figure 1a.

Figure 1b.



2D and colour Doppler imaging showing loss of placental-myometrial interface, lacunae and increased blood vessels. Placenta bulging into the bladder.



3D power Doppler: Newly formed blood vessels and lacunae.

Continued on page 17.

Figure 2a.

Figure 2b.



Coronal blade image showing placenta accreta at bladder placental interface.

Placental MRI is now frequently utilised in diagnosing placenta accreta. It complements ultrasound and Doppler evaluation in cases suspected of placenta accreta. With improvement in Fast T2 imaging, sensitivity and specificity of MRI now reaches that of ultrasound with Doppler.⁹ In our institution, we acquire True Fast T2 images (True FISP and Blade sequences) in three planes through the placenta (Avanto, Seimens, Germany). A loss of placentalmyometrium interface with signal alteration (see Figures 2a and 2b) suggests abnormal placental adhesion. A wide field of view is an added advantage of MRI over ultrasound. It provides a complete view of placenta with relationship to adjoining structures.

Sagital True FISP image showing placenta accreta at bladder/

placental interface (arrow).

The presence of fetal and placental cells in maternal circulation in placenta accreta is a novel approach under research. A study by Muira and colleagues has suggested that higher levels of placental mRNA are present in the maternal circulation in the subgroup of patients at risk of placenta percreta.¹⁰ It might assist us in future to decide on the most appropriate procedure for the individual patient.

- 1. Ozcan T, Presman EK. Imaging of the placenta. *Ultrasound Clin. 3* 2008; 13-22.
- Welsh A, Ellwood D, Carter J, Peduto A, Vedelago J, Bennett M. Opinion: Integration of diagnostic and management perspectives for placenta accreta. ANZJOG 2009; 49 (6) 578-87.
- Miller DA, Chollet JA, Goodwin TM. Clinical risk factors for placenta previa-placenta acreta. *Am J Obstet Gynecol.* 1997;177:210-214.
 Chomstock CH. Antenatal diagnosis of placenta accreta: a review
- 4. Chomstock CH. Antenatal diagnosis of placenta accreta: a review. *Ultrasound Onstet Gynecol.* 2005; 26:89-96.
- Yang JI, Lim YK, kim HS, Chang KH, Lee JP, Ryu HS. Sonographic findings of placental lacunae and the prediction of adherent placenta in women with placenta totalis and prior caesarean section. *Ultrasound Obstet Gynecol.* 2006; 28: 178-182.
- Wong HS, Cheung YK, Strand I, Carryer p, Parker s, Tait J, Pringle KC. Specific Specific sonographic features of placenta accreta:tissue interface disruption on gray-scale imaging and evidence of vessel crossing interface-disruption sites on Doppler imaging. Ultrasound Onstet Gynecol. 2007; 29:239-241.
- Shih JC, Palacios Jaraguemada JM, Su YN, Shyu MK, Lin CH, Lin SY, Lee CN. The role of three-dimensional power Doppler in the antenatal diagnosis of placenta accreta: comparison with gray-scale and colour Doppler techniques. *Ultrasound Obstet Gynecol.* 2009; Feb:33(2): 193-203.
- Comstock CH. Antenatal diagnosis of placenta accreta: a review. Ultrasound Obstet Gynecol. 2005; 89-96.

- Masselli, et al. MR Imaging in the evaluation of placental adhesive disorders: correlation with color Doppler ultrasound. Eur Radiol. 2008; 18: 1292-1299.
- Miura S, Yamasaki K, Yoshida A, Yoshiura k, Nakayama D, Niikawa N, Mazuzaki H. Increased level of cell-free placenta mRNA in a subgroup of placenta previa that needs hysterectomy. *Prenat Diagnosis*. 2008; Sep 28(9):805-9.

Prevention of perineal tears



Ann Yates Midwife

Preparing a woman for labour and birth is a fundamental part of antenatal care. For many women, the prospect of damage to their genital tract is of concern and some fear they will never function the same again.

Part of the miracle of birth is the amazing ability of the vagina and perineum to expand and for the baby to navigate the birth canal unaided. The fact that most of us survived this right of passage to give birth again is often no consolation to a first time mum and inevitably the midwife is asked to provide helpful advice and reassurance before the event.

The following points list my personal

repertoire of method of prevention for perineal trauma from over 33 years of midwifery practice. Some points are common sense, some points have been confirmed through research to be good practice and some are just comforting for the woman at the time:

- 1. Good nutrition and health equals strong elastic tissue and rapid healing postpartum. This needs to be discussed antenatally and women require good information to make healthy food choices.
- Sore, irritated, swollen perineal tissue is not likely to be as pliable, can tear easily and heals badly. Hygiene basics need discussion antenatally – advise to avoid perfumed soaps and sprays and sometimes panty liners can cause irritation. Comfortable cotton underwear should be encouraged. Ensure timely follow-up on vaginal swabs and treatment of yeast or other infections.
- 3. Perineal massage has been taught in childbirth education for the past 40 years and remains controversial. The concept of stretching of the perineum by placing two thumbs into the introitus and gradually stretching the perineum open and out is thought to be beneficial. Some midwives recommend evening primrose or unscented almond oil for this. Unfortunately, it is physically challenging in later stages of pregnancy and is generally taught as a couple's activity. Evidence on perineal massage is light, other than being shown to be effective for nulliparous women after 34 weeks gestation.¹
- 4. A dilating balloon device is currently being promoted to increase vaginal elasticity antenatally, however, I am challenged to see the benefits of dilating a woman's vagina to 10cm without actually being in labour and giving birth. The vaginal/perineal tissue does this particularly well when there is gradual dilation in labour, with sufficient expulsive urge, support and encouragement.
- 5. Management of the second stage of labour is critical to preserving the integrity of the perineum. We should observe the resting (wait and be thankful) or passive phase of second stage labour and wait for physiological urge to push occurs. NICE guidelines² suggest approximately an hour for this to happen and in this time the doctor or midwife should not be guiding or coaching.

There is a significant trend towards poorer perineal outcomes when directed valsalva pushing is used³, with evidence that pushing on command and valsalva/breath-holding contributes to pelvic floor damage, fetal distress, exhaustion and perineal tears. A meta-analysis of randomised controlled trials showed that spontaneous vaginal birth reduced the incidence of perineal trauma.⁴ There is no clear evidence or consensus about guarding the perineum in the second stage of labour, maternal position in labour (apart from avoidance of lithotomy), or perineal massage in the second stage of labour. ^{5,6,7} In my experience, few women elect to lie on their backs during the second stage and describe lithotomy as the most painful position to be in. However, women will instinctively assume a birthing position that works for them.

A comforting midwifery practice is to place warm packs over the perineum to help relieve the burning sensation during crowning. Provide gentle support of haemorrhoids with a warm cloth. There is a traditional midwifery saying: 'Never EVER take your eyes off the perineum to avoid sudden rapid uncontrolled birth'.

6. For some women, the possibility of defecating during active second stage inhibits their efforts. Ina May Gaskin describes this as 'shit shock' and encourages women to open their mouth, drop their jaw and try to keep their lips loose and open. She advocates humour to help women to relax and close interaction between midwife and the woman throughout with gentle coaxing and quiet, peaceful talking to encourage slow birth of the head after crowning.

The most effective preventative for perineal trauma in my experience is the trusting relationship between mother and midwife developed through pregnancy. Discussion occurs about what happens in the second stage and how the midwife will provide encouragement and support to get through this overwhelming and sometimes frightening experience.

- Labrecque M, Eason E, Marcoux S, et al. RCT Trial of prevention perineal trauma by perineal massage during pregnancy. *American Journal of Obstetrics and Gynaecology* 1999; 180(3): 593-600.
- National Institute for Health and Clinical Excellence (NICE 2007). Care of Healthy Women and their Babies During Childbirth; Clinical Guideline 55 – Intrapartum Care.
- Bosomworth A, Bettany-Saltikov J. Just take a deep breath A review to compare the effects of spontaneous versus directed valsalva pushing in the 2nd stage of labour on maternal and fetal wellbeing. *Midwifery Digest* 2006; 16:2 p157.
- Eason E, Labrecque M, Wells G, et al. Preventing perineal trauma during childbirth: a systematic review. Obstetrics and Gynaecology 2000; 95(3)464-471.
- Beckmann MM, Garratt AJ. Antenatal perineal massage for reducing perineal trauma (protocol). Cochrane Database of Systematic Reviews 2005; (1) Oxford.
- 6. Enkin M, Keirse MJ (eds). *Effective Care in Pregnancy and Childbirth* 1999; Vol 2. Oxford University Press.
- Stamp G, Kruzins G, Crowther C. Perineal massage in labour and prevention of perineal trauma: randomised controlled trial. *British Medical Journal* (clinical research edition 2001; 322(7297):1277-1280.
- 8. Pairman, Pincombe, Thorogood & Tracy. *Midwifery Preparation for Practice*. 2006 Elsevier Australia.
- Mayes M, Winship J, Sweet B ed, Tiran D ed. Mayes' Midwifery: A Textbook for Midwives 12th edition. 1998. University of Surrey, UK.

Management of prolonged pregnancy

Yesterday, today and tomorrow



Dr David Bailey FRANZCOG

Until the mid-20th century, obstetricians in developed countries were preoccupied with the prevention of maternal death. As maternal mortality rates declined in the second half of the 20th century, attention turned to the welfare of the unborn and newborn child.

In the early 1940s, Dugald Baird reported that the perinatal mortality rate for nulliparous women delivering after 42 weeks gestation in Aberdeen, Scotland, was a staggering one in eight, due to a combination of antenatal and intrapartum asphyxia, meconium aspiration and birth trauma. He was among the first obstetricians to advocate induction of labour for prolonged pregnancy and more liberal use of

caesarean section. These views were supported by the British birth survey in 1958, which showed a two-fold increase in stillbirth for pregnancies delivering at 42 weeks, compared to 40 weeks gestation.¹ From the 1960s onwards, it became common practice to induce labour before 42 weeks gestation. Randomised controlled trials showed a policy of induction at 41 to 42 weeks gestation was associated with reduced perinatal morbidity and mortality without a significantly increased rate of caesarean section.²

Current practice

In most developed countries, prolonged pregnancy is now managed by planned delivery. The most recent evidence-based guideline on induction of labour from the UK National Institute for Health and Clinical Excellence (NICE) recommends offering induction any time from 41 weeks, provided resources allow.³ Prior to formal induction, membrane sweeping is commonly practised, as this reduces the need for 'post-dates' induction.⁴ Most women with a previous caesarean section have repeat caesarean delivery if the pregnancy becomes prolonged, as induction is associated with an increased risk of uterine rupture.

There is considerable variation in practice. Women with induced labour are likely to need more support, analgesia and monitoring than women in spontaneous labour, so most units limit the number of inductions. Local custom and resources largely dictate the gestation at which 'routine' delivery is offered.

Gestational age is calculated inconsistently. Estimated date of delivery (EDD) is most reliably predicted by ultrasound measurement of crown-rump length (CRL) at eight to 12 weeks or biometry at 14 to 20 weeks.⁵ However, clinicians often use an EDD based on the last menstrual period (LMP) or CRL measured before eight weeks or after 12 weeks, both of which are less reliable.

Women who do not have a scan until late in pregnancy pose a particular problem. Estimating gestational age is unreliable and women may be offered induction on the basis of an uncertain EDD, or serial monitoring while awaiting spontaneous labour. It is not known which of these strategies is more effective.

Problems with current practice

In most cases, the purpose of labour induction is to reduce the risk of stillbirth. Unfortunately, a policy of routine 'post-dates induction' has little impact on the overall term stillbirth rate, as most stillbirths happen before 41 weeks gestation and the pregnancies at greatest risk are often not identified. Many factors, other than gestational age, are associated with an increased risk of stillbirth (see Table 1). Women with these risk factors may be offered earlier induction, but this is not applied in a systematic or consistent way. Conversely, some women have a very low risk of stillbirth despite prolonged pregnancy and may benefit little from induction. For example, while the stillbirth rate for nulliparous women increases exponentially between 39 and 42 weeks, the increase in the rate for multiparous women is much less marked (see Figure 1).^{6,7}

Table 1. Risk factors associated with unexpected term stillbirth

Maternal factors
Nulliparity
Increased maternal age
Increased maternal weight and BMI
Maternal disease (for example, diabetes, hypertension, renal disease, thrombophilia)
Previous caesarean section
Assisted conception
Fetal factors
Fetal growth restriction
Multiple pregnancy
Increased gestational age

It is also unclear what the stillbirth rate would be in prolonged pregnancy with modern maternity care. Most of the randomised trials of induction for prolonged pregnancy were conducted 20 to 40 years ago. Some women undoubtedly benefit from induction at 41 to 42 weeks (or earlier), while others are at very low risk of stillbirth and may be worse off with induction. The question is: can we identify who benefits most from delivery?

The current situation with regard to fetal risk assessment at term resembles Down's syndrome (DS) screening as it was 30 years ago. Invasive testing was offered on the basis of maternal age and most DS pregnancies went undetected. With the introduction of serum screening and ultrasound assessment, it is now possible to detect a much higher proportion of DS pregnancies without increasing invasive testing rates. Our future goal should be to reduce unexpected term stillbirths by a policy of risk assessment and selective delivery, while minimising the harm caused by unnecessary interventions.

'In the future, we need to develop more effective strategies to reduce perinatal morbidity at term by selective delivery based on overall risk, not on gestational age alone.'

The future

We need to devise customised assessment tools so that we can estimate the stillbirth risk for individual pregnancies. Factors assessed would include maternal age, parity, ethnic group, past history, co-morbidity and estimated fetal size. Women would be offered delivery at a gestation where the estimated stillbirth risk exceeds an agreed threshold. As a result, an overweight nulliparous woman might be delivered at 39 to 40 weeks gestation; an uncomplicated multiparous woman might be advised against induction at any gestation. This strategy would have the potential to reduce term stillbirths without an increase in interventions.

There will be many challenges to developing a risk assessment strategy to reduce perinatal morbidity and mortality. Factors such as age, parity and co-morbidity are not independent variables and risk ratios cannot simply be multiplied, as is done for DS screening. We also need to debate the level of risk that would justify delivery and the level of maternal morbidity that would be acceptable in order to reduce stillbirths – particularly as induction before 41 weeks is likely to be associated with an increased caesarean section risk. What is appropriate for one community may not be appropriate for others.

Conclusion

Prolonged pregnancy is a common indication for planned delivery. However, in the future we need to develop more effective strategies to reduce perinatal morbidity at term by selective delivery based on overall risk, not on gestational age alone.

References

- Butler NR, Bonham DG. Perinatal Mortality: The First Report of the 1958 British Perinatal Mortality Survey. E&S Livingstone Ltd, London 1963.
- Gülmezoglu AM, Crowther CA, Middleton P. Induction of labour for improving birth outcomes for women at or beyond term. *Cochrane Database of Systematic Reviews* 2006, Issue 4. Art. No.: CD004945. DOI:10.1002/14651858.CD004945.pub2.
- National Institute for Health and Clinical Excellence (UK). CG 70 Induction of Labour: Clinical Guideline. July 2008.
- Boulvain M, Stan CM, Irion O. Membrane sweeping for induction of labour. Cochrane Database of Systematic Reviews 2005, Issue 1. Art. No.: CD000451. DOI: 10.1002/14651858.CD000451.pub2.
- Bottomley C, Bourne T. Dating and growth in the first trimester. Best Practice & Research Clinical Obstetrics & Gynaecology 2009; 23: 439-52.
- Hilder L, Sairam S, Thilaganathan B. Influence of parity on fetal mortality in prolonged pregnancy. *European Journal of Obstetrics & Gynaecology and Reproductive Biology* 2007; 132: 167-70.
- Ingemarsson I, Kallen K. Stillbirths and rate of neonatal deaths in 76 761 post-term pregnancies in Sweden 1982-1991: a register study. *Acta Obst Gynaecol Scand.* 1997; 76: 658-62.

Figure 1.

Stillbirth rate by week of gestation. North-East London 1989-92.6



Attempting vaginal birth after a previous caesarean section

How should we counsel women and their families?



A/Prof Stephen Robson FRANZCOG

From my time as a registrar in the early 1990s, I remember a morbidity and mortality meeting where we discovered that the caesarean section rate for our hospital had reached 25 per cent during the preceding month.

At this, the Professor stood up and announced that he was truly shocked by the figure. After a moment, though, he added that he couldn't decide whether he was shocked because the proportion was too high, or because it was too low.

These days, many people still profess shock that almost

one baby in three is delivered by caesarean section: 'There is widespread public and professional concern about the increasing proportion of births by caesarean section.' But should this rise really have the power to surprise? Powerful demographic forces are reshaping our communities. It is now becoming clear that these irresistible changes are likely to influence birth for years to come.

In the first place, obesity is being described as an epidemic and is a well-known independent risk factor for caesarean birth.^{2,3} Secondly, the age at first birth continues to rise across the developed world. There is now evidence that increasing age appears to affect uterine function in labour, making older women more prone to dystocias necessitating operative delivery.⁴ Thus, as the proportion of women in older age groups labouring for the first time increases, so must the rate of caesarean section increase.

Thirdly, and I think most sadly, obstetricians are abandoning complex vaginal births (such as breech deliveries, forceps rotations and many twin births) and resorting to caesarean delivery in these circumstances. A great deal of the summer 2009 edition of O c G Magazine was devoted to this topic.

Fortunately, there is no suggestion that rates of neonatal morbidity or mortality are increasing in hospital-based birth. Indeed, this is a very safe time to be born. Similarly, rates of maternal morbidity and mortality are low too. The confidence we should be able to inspire in women and their families – that they and their babies are very safe in modern Australia and New Zealand – should build a lot of goodwill toward obstetricians. In theory, anyway.

Accepting that the rate of primary caesarean section is increasing and knowing that the majority of women will have more than one baby, it is inevitable that obstetricians will have to work out how to deliver the next baby. Should a woman attempt vaginal birth after her previous caesarean section (VBAC)? This will obviously involve a negotiation with the woman, her partner and possibly their family. Attempts to work through the voluminous literature about VBAC are challenging. The Royal College of Obstetricians and Gynaecologists (RCOG) *Green-top Guideline* is boldly pro-VBAC, yet it concedes that: '...new evidence is emerging that VBAC may not be as safe as originally thought'.¹ Other publications that address women's perceptions about VBAC commonly include statements such as:

'Informed choice is the key to effective women-centred care. Women must have access to non-biased, evidence-based information in order to engage in a collaborative partnership of equals with midwives and obstetricians.'⁵

As we might expect, the *Cochrane* review, 'Planned elective repeat caesarean section versus planned vaginal birth for women with a previous caesarean birth', puts it like this:

> 'Planned elective repeat caesarean section and planned vaginal birth after caesarean section for women with a prior caesarean section are both associated with benefits and harms. Evidence for these care practices is drawn from non-randomised studies, associated with potential bias. Any results and conclusions must therefore be interpreted with caution.'⁶

Caution should be the watchword when contemplating a vaginal delivery after a previous caesarean delivery. I would like to approach the issue of counselling around the decision for VBAC from the perspective of patient autonomy. 'Patient autonomy' is simply the right of our patients to make their own decisions about treatment. Doctors (and others) may educate the patient about important relevant issues, but ultimately, the patient must decide. It is a nonsense to promote a 'collaborative partnership of equals with midwives and obstetricians'⁵ if the woman is then hectored into accepting a trial of VBAC against her wishes.

The principle of patient autonomy is different from that of evidencebased medicine (EBM). Sackett and colleagues describe EBM as: '... the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients.'⁷ The expectation of EBM is that the doctor assimilates the available 'evidence' and formulates a plan for the patient's management. As we now know, the evidence base informing VBAC is somewhat patchy and prone to bias.

With respect to interventions such as caesarean section, patient autonomy is intimately bound with the principles that underpin 'informed consent'. These issues have been eruditely reviewed by Patrick and colleagues in a recent issue of the *Mayo Clinic Proceedings* and I would like to summarise their review here.⁸ Whenever there is a choice between medical treatments (in this case, trying for a vaginal birth or just having an elective caesarean section), the doctor responsible for the patient's care must discuss the potential benefits and risks of each treatment. This discussion needs to be presented in such a way that the patient can understand it. Serious risks, such as death for example, or permanent incapacity resulting from the treatment, must be discussed even if the probability of them occurring is low. Patrick and colleagues stress that, '...the magnitude of the risks and their frequency should receive special emphasis...and the probability of a good outcome with the proposed strategy'.⁸

'The principles of patient autonomy and informed consent mean that women and their families should be informed that their individual risks vary, depending on age, stature, reason for the previous caesarean delivery, size of the baby and whether they have had a vaginal birth prior.'

Such principles are also promoted by RANZCOG in College Statement *C-Gen 2: Guidelines for Consent and the Provision of Information Regarding Proposed Treatment.*¹⁵ Using these principles as a template, the information that women and their partners should receive to assist in their decision-making becomes obvious. Comparing an attempt at vaginal birth with a planned elective caesarean section, they must be given information about the likely success rate of each treatment option and the likelihood of serious adverse outcomes (death of the baby being the most important) or permanent impairments (hysterectomy, long-term injury to the mother or baby). All of this must be presented in a way that is easily comprehensible.

How, exactly, should we define 'success' in a trial of VBAC? Obviously, an elective caesarean section is almost guaranteed to lead to delivery of the baby. The best available data from Australia in 2006 suggest that attempted VBAC results in vaginal birth in 53 per cent of attempts.⁹ As well, those data also reveal that only 35 per cent of women attempt a VBAC. These figures differ markedly from those in the RCOG *Green-top Guideline*, where rates of vaginal delivery greater than 70 per cent are described.¹ To put things in perspective for Australian women, then, it should be stated that slightly more than one third of women attempt vaginal birth after a caesarean section and slightly more than half of those attempts result in a vaginal delivery. However, this overall metric obviously does not take into account the varied circumstances of each individual woman.

'Successful' and 'failed' are pejorative terms. Women and their families may well feel that setting out to try for a vaginal birth, but undergoing emergency caesarean section with the delivery of a healthy baby, is indeed successful. Others may resent a perception of being forced to try at all. If we adopt a definition of 'success' that the mother and baby are healthy and the family are satisfied, no matter what happens, then it ought to be possible to make almost every delivery after a previous caesarean section 'successful'.

Trying to pick who is likely to achieve a vaginal birth is not easy to do. Special circumstances, such as preterm birth, multiple

pregnancy and two or more previous caesarean sections, are beyond the scope of this article. Intrapartum management protocols are a different matter entirely, deserving of a full article. The overwhelmingly most common setting is a woman and her family in an uncomplicated term pregnancy, contemplating a plan for their next birth. This situation confronts almost all obstetricians all the time.

Large studies suggest that, for women who have no previous vaginal births before their caesarean section and in whom the caesarean was performed for a dystocia, the chance of vaginal delivery is considerably less than 50 per cent.^{10,11,12,13} Other negative predictors include a birthweight of 4kg or more, increased maternal age, maternal overweight and obesity, and short stature. Thus, women who should be told that if they have no past vaginal birth and their previous caesarean was for 'obstructed labour,' the chances of a successful VBAC are less than even.

What are the risks to mother and baby of the two approaches? The risk of neonatal death in VBAC is 12.9/10,000, compared to only 1.1/10,000 in repeat elective caesarean section. Similarly, the risk of hypoxic-ischaemic encephalopathy (HIE) is 8/10,000 in VBAC but essentially zero in planned caesarean section.^{10,11,12,13} Women should be told that, although the numbers are small, the risk of the baby dying or having HIE is about ten times higher after VBAC than with planned repeat caesarean section.

Much of the important morbidity to mother and baby flows from uterine rupture. Smith and colleagues found that the group of women at risk of unsuccessful VBAC (no previous vaginal birth, large baby, short woman, being the main common adverse predictors) are also the group at highest risk of uterine rupture and its catastrophic sequelae.¹³ In fact, this group faced a risk of almost one in one hundred of uterine rupture. Uterine rupture is bad. A recent large study from Scandinavia reported the odds ratio for neonatal death in uterine rupture at more than 65.¹⁴

There are a small number of circumstances when VBAC is clearly contraindicated. Classical caesarean section or complex uterine injury at the time of the first caesarean section are examples of this.¹ Obviously, it is important to be familiar with the circumstances and conduct of the previous surgery before proffering an opinion. There are other considerations too – attempted VBAC slightly increases the requirement for blood transfusion (by one per cent), but also slightly reduces the risk of respiratory morbidity (from two to three per cent to three to four per cent) depending on the gestation.¹

'If the woman plans for an attempt at VBAC, those responsible for her care must have confidence that the resources necessary for management of a catastrophic complication are readily available.'

Where does this information leave us? The principles of patient autonomy and informed consent mean that women and their families should be informed that their individual risks vary, depending on age, stature, reason for the previous caesarean delivery, size of the baby and whether they have had a vaginal birth prior. This allows risks to be presented in a more bespoke way. Women who are young, have had a vaginal delivery prior to their caesarean section and have a baby predicted to be less than 4kg, would have the highest chance of a safe, successful vaginal delivery. The remainder of women, especially those who have not had a vaginal birth and those whose primary caesarean section involved obstruction, may face a less than even chance of having the baby vaginally, and also have the greatest risk of uterine rupture and its ugly consequences.

An important issue that often remains ignored in discussions of VBAC is the issue of access to emergency resources if the attempted VBAC goes awry. Uterine rupture can place enormous demands on the clinician, operating theatres, anaesthetists, paediatricians and all those involved in managing catastrophic complications. If the woman plans for an attempt at VBAC, those responsible for her care must have confidence that the resources necessary for management of a catastrophic complication are readily available. This should surely be brought to the attention of women making decisions about both mode of birth and place of birth.

There is no 'one size fits all' solution to counselling around VBAC. Women and their families need to be aware of the probability of achieving a safe vaginal birth in their individual circumstances and what the risks of a VBAC are for themselves and their babies when things go wrong. Principles of patient autonomy should then lead us to respect the decision and support the plan.

- 1. RCOG. Birth after previous caesarean birth. *Green-top Guideline No* 45. February, 2007. [accessible at www.rcog.org.au]
- Yakin J, Toner RW, Goldfarb N. Obesity management interventions: a review of the evidence. *Popul Health Manag.* 2009; 12: 305-16.
- 3. Wax JR. Risks and management of obesity in pregnancy: current controversies. *Curr Opin Obstet Gynecol.* 2009; 21: 117-23
- 4. Smith GC, Cordeaux Y, White IR, *et al.* The effect of delaying childbirth on primary caesarean section rates. *PLoS Med.* 2008; 5: e144.
- Meddings F, Phipps FM, Haith-Cooper M, Haigh J. Vaginal birth after caesarean section (VBAC): exploring women's perceptions. J Clin Nurs. 2007; 16: 160-7.
- Dodd JM, Crowther CA, Huertas E, Guide JM, Horey D. Planned elective repeat caesarean section versus planned vaginal birth for women with a previous caesarean birth. *Cochrane Database Systematic Rev.* 2004; 4: CD004224.
- Sackett DL, Rosenberg WMC, Gray JAM, et al. Evidence based medicine: what it is and what it isn't. Lancet 1996; 312: 71-2.
- Patrick TJ, Carson GV, Allen MC, Paterick TE. Medical informed consent: general considerations for physicians. *Mayo Clin Proc.* 2008; 83: 313-9.
- Homer CS, Johnston R, Foureur MJ. Birth after caesarean section: changes over a nine-year period in one Australian state. *Midwifery* 2009; September 19 [Epub ahead of print].
- Smith GC, Pell JP, Cameron AD, Dobbie R. Risk of perinatal death associated with labor after previous caesarean delivery in uncomplicated term pregnancies. *JAMA* 2002; 287: 2684-90.
- Landon MB, Hauth JC, Leveno KJ, et al. Maternal and perinatal outcomes associated with a trial of labour after prior caesarean delivery. N Engl J Med. 2004; 351: 2581-9.
- Cahill AG, Stamilio DM, Odibo AO, et al. Is vaginal birth after caesarean or elective repeat caesarean safer in women with a prior vaginal delivery? Am J Obstet Gynecol. 2006; 195: 1143-7.
- 13. Smith GC, White IR, Pell JP, Dobbie R. Predicting cesarean section and uterine rupture among women attempting vaginal birth after prior cesarean section. *PLoS Med.* 2005; 2: e252.
- Kaczmarczyk M, Sparen P, Terry P, Cnattingius S. Risk factors for uterine rupture and neonatal consequences of uterine rupture: a populationbased study of successive pregnancies in Sweden. *BJOG* 2007; 114: 1208-14.
- 15. RANZCOG Statement. C-Gen 2: Guidelines for Consent and the Provision of Information Regarding Proposed Treatment. Access at: www.ranzcog.edu.au/publications/statements/C-gen2.pdf.

Infections in the third trimester of pregnancy

Dr Jen Kok and Prof Lyn Gilbert

Centre for Infectious Diseases and Microbiology Laboratory Services Westmead Hospital, NSW

Absence of evidence is not evidence of absence, but why the absence of evidence when evidence should be present?

(Adaptation of a quote by Dr Carl Sagan.)

Despite the advent of evidence-based medicine, controversy can exist when there is a paucity of evidence to guide practice, or when there is conflicting evidence. Further confusion arises when guidelines from different resources recommend different practices. Within infectious diseases, controversies exist in areas including screening, prophylaxis, treatment and/or vaccination of pregnant women against pathogens such as group B streptococcus (GBS), influenza, herpes simplex virus (HSV), varicella zoster virus (VZV) and human immunodeficiency virus (HIV).

The perinatal management of GBS remains controversial, as debate continues over the optimal approach (universal versus risk-based screening) and the efficacy of intrapartum antibiotic prophylaxis (IAP) in preventing GBS infection. Retrospective data suggest that screening confers a lower risk of early onset GBS infection (EOGBS) compared to a risk-based approach (relative risk 0.46).¹⁵ Although the adoption of universal screening and IAP did reduce the incidence of EOGBS, one study showed that 61.4 per cent of term infants with EOGBS were born to mothers who had negative screen results²⁰, highlighting the need to develop other preventive strategies such as vaccination. Furthermore, the largest meta-analysis examining the use of IAP to date demonstrated that although IAP use in women colonised with GBS reduced EOGBS, it did not significantly reduce the incidence of all cause mortality or morbidity from GBS infection.¹¹ Unfortunately, the studies included in this meta-analysis were either of poor quality (lack of placebo or changing statistical methods post-hoc to demonstrate significance) or had significant biases (exclusion of infants whose mothers developed intrapartum fever or imbalance of study participants). At present, due to inconclusive and conflicting evidence, RANZCOG recommends either screening or a risk-based approach and IAP to prevent EOGBS.¹⁸

Pregnant women are at increased risk of developing severe influenza infection, with increased mortality rates and obstetric complications, including preterm labour and spontaneous miscarriages.^{5,7} During the 2009 Australasian influenza season, 9.1 per cent of all patients (or 17.6 per cent of women) admitted to intensive care units (ICU) with pandemic (H1N1) 2009 influenza were pregnant.¹⁷ A four-fold increase in the risk of ICU admission or death was also noted when the institution of oseltamivir was delayed beyond 48 hours after initial presentation.⁹ Despite the benefits of oseltamivir (a category B1 medication), only 50 to 81 per cent ^{5,7,9} of hospitalised pregnant women with pandemic influenza received oseltamivir. The safety of oseltamivir is still in question, given the limited information about its safety and efficacy in pregnancy¹³, and it is only recommended by the manufacturer if the potential benefit justifies the potential risk to the fetus. Nevertheless, the dose of oseltamivir administered in some hospitalised patients was twice the normal recommended dose.⁹

Australian guidelines recommend trivalent seasonal influenza vaccination for all pregnant women who will be in the second or third trimester of pregnancy during the influenza season.¹ The monovalent influenza A pandemic (H1N1) 2009 vaccine was also recommended for pregnant women when it became available in September 2009 and is included in the 2010 seasonal vaccine. The influenza vaccine protects pregnant women in their third trimester against febrile respiratory illnesses and reduces laboratoryconfirmed cases of influenza in infants of vaccinated mothers.²² No significant long-term side effects (teratogenic, carcinogenic or neurologic) have been shown in pregnant women receiving the influenza vaccine during the first trimester¹⁰ and would not be expected from a killed vaccine. However, the uptake of the vaccine in pregnant women, even in the later stages of pregnancy, is universally poor despite evidence of significantly increased morbidity from influenza infection.⁵

Although antiviral therapy is recommended for primary genital herpes simplex virus (HSV) infection at any stage of pregnancy, the risk of HSV infection in neonates born to mothers with primary genital HSV infection who seroconvert before delivery is very low, in contrast to the high (up to 50 per cent) risk of neonatal infection when mothers acquire HSV infection just prior to delivery.² This suggests that antiviral therapy for primary HSV infection occurring well before the onset of labour should be based on maternal clinical indications, bearing in mind that maternal fever and toxicity can precipitate the onset of premature labour. In pregnant women with recurrent genital herpes, aciclovir or valaciclovir use during the last four weeks of pregnancy reduces maternal recurrences, viral shedding and rates of caesarean delivery, but not neonatal HSV infection.⁶ Aciclovir (category B3 medication) use is still not recommended in pregnancy unless potential benefits clearly outweigh the potential risk to the fetus⁴, despite extensive data showing no increased risk of teratogenicity.¹⁶ Interestingly, the evidence for caesarean delivery to protect against neonatal infection, which is recommended for primary HSV infection and where active lesions are present in pregnant women with preexisting infection, was only available long after it became standard practice.³

Pregnant women without a definite history of varicella should be tested for varicella IgG antenatally or following an exposure to chicken pox in order to guide further management. Antenatal screening with postnatal vaccination has been shown to be more cost-effective than using zoster immunoglobulin (ZIG) for seronegative women.¹⁹ A previous seroprevalence study showed that 86 per cent and 96 per cent of pregnant women with no history or an unclear history of previous VZV infection respectively were seropositive when tested.⁸ However, the use of serology is not recommended to document seroconversion in patients who have previously received adequate varicella vaccination, as current methods of antibody detection using enzyme immunoassays are insensitive in reliably detecting vaccine-induced antibodies.¹⁴ Post-exposure prophylaxis with ZIG aims to prevent or attenuate maternal varicella, which can be severe or even life-threatening, especially in the third trimester. There is little evidence that it affects congenital varicella syndrome, following maternal VZV exposure in the first or second trimester, unless it prevents, rather than reduces the severity of maternal infection. ZIG is also given to prevent neonatal varicella following maternal exposure in the perinatal period. The optimal timing for ZIG administration is the first 48 to 72 hours after exposure, although it may modify severity of disease, up to ten days post exposure.¹ The use of ZIG in pregnant women who have been previously vaccinated but with undetectable antibody levels (if tested) is highly contentious, although previously reported breakthrough varicella infections following vaccination would support its use. For pregnant women not given ZIG, aciclovir may be considered as post-exposure prophylaxis, although evidence for this strategy is lacking.

'Until conclusive evidence can be obtained to guide clinical practice, management is likely to be based on "expert" opinion, which remains a concoction of intuition, anecdotes and experience.'

The goal of antiretroviral therapy in HIV-infected pregnant women is to achieve maximal viral suppression, in order to prevent motherto-child transmission of HIV. Although zidovudine is generally recommended as part of the antiretroviral regimen (unless severe toxicity occurs or resistance is documented), patients already on antiretroviral therapy are usually continued on their pre-existing regimens (with the exception of efavirenz during the first trimester due to its potential teratogenicity) provided their HIV infection is well controlled. Controversially, zidovudine monotherapy has been suggested for antiretroviral naïve pregnant-women who have HIV RNA levels below 1000 copies/ml, to limit unnecessary antiretroviral exposure, as the short and long-term fetal effects of many antiretroviral drugs remain unknown. However, combination antiretroviral therapy reduces perinatal transmission more than zidovudine alone.¹² In addition, there were no short-term differences in CD4 T-cell counts, viral load or disease progression in antiretroviral naïve patients (with CD4 T-cell counts greater than 350 cells/mm³) who continued or stopped antiretroviral therapy that was commenced during pregnancy to prevent mother-to-child transmission.21

In conclusion, many questions and issues remain unanswered and unresolved in the management of infectious diseases in the third trimester. Until conclusive evidence can be obtained to guide clinical practice, management is likely to be based on 'expert' opinion, which remains a concoction of intuition, anecdotes and experience.

- Australian Government Department of Health and Aging. 2008. The Australian Immunisation Handbook 9th Edition 2008 (Accessed March 5, 2010 at: www.health.gov.au/internet/immunise/publishing.nsf/ Content/Handbook-specialrisk232).
- Brown ZA, Selke S, Zeh J, Kopelman J, Maslow A, Ashley RL, Watts DH, Berry S, Herd M and Corey L. The acquisition of herpes simplex virus during pregnancy. N Engl J Med. 1997; 337:509-515.
- Brown ZA, Wald A, Morrow RA, Selke S, Zeh J and Corey L. Effects of serologic status and Cesarean delivery on transmission rates of herpes simplex virus from mother to infant. JAMA. 2003; 289:203-209.
- GlaxoSmithKline. 2006. ZOVIRAX[™] (Aciclovir) Product Information (Accessed March 25, 2010 at: www.gsk.com.au/resources.ashx/ prescriptionmedicinesproductschilddataproinfo/819/FileName/48 48CEB0CDECBA9085768E3FE5A2244F6C/PI ZoviraxIV.pdf).
- Hewagama S, Walker SP, Stuart RL, Gordon C, Johnson P, Friedman N, O'Reilly M, Cheng A and Giles M. 2009 H1N1 Influenza A and pregnancy outcomes in Victoria, Australia. *Clin Inf Dis.* 2010; 50:686-690.
- Hollier LM and Wendel GD. Third trimester antiviral prophylaxis for preventing maternal genital herpes simplex virus (HSV) recurrences and neonatal infection. *Cochrane Database Syst Rev.* 2008. 2:1-20.
- Jamieson D, Honein M, Rasmussen S, et al. H1N1 2009 influenza virus infection during pregnancy in the USA. *Lancet.* 2009; 374:451-458.
- Karunajeewa H, Siebert D, Hammond R, Garland S and Kelly H. Seroprevalence of varicella zoster virus, parvovirus B19 and Toxoplasma gondii in a Melbourne obstetric population: Implications for management. Aust N Z J Obstet Gynaecol. 2001; 41:23-28.
- Louie J, Acosta M, Jamieson D and Honein M. Severe 2009 H1N1 Influenza in pregnant and postpartum women in California. N Engl J Med. 2010; 362:27-35.
- Mak T, Mangtani P, Leese J, Watson J and Pfeifer D. Influenza vaccination in pregnancy: current evidence and selected national policies. *Lancet Infect Dis.* 2008; 8:44-52.
- Ohlsson A and Shah V. Intrapartum antibiotics for known maternal Group B streptococcal colonization. *Cochrane Database Syst Rev.* 2009; 3:1-35.
- Perinatal HIV Guidelines Working Group. 2009. Public Health Service Task Force Recommendations for Use of Antiretroviral Drugs in Pregnant HIV-Infected Women for Maternal Health and Interventions to Reduce Perinatal HIV Transmission in the United States (Accessed March 15, 2010 at: http://aidsinfo.nih.gov/ContentFiles/PerinatalGL. pdf).
- Roche Products Pty Ltd. 2009. TAMIFLU® (oseltamivir) Product Information (Accessed March 23, 2010 at: www.roche-australia.com/ downloads/tamiflu-pi.cfm?action=get).
- Sauerbrei A and Wutzler P. Herpes simplex and varicella-zoster virus infections during pregnancy: current concepts of prevention, diagnosis and therapy. Part 2: Varicella-zoster virus infections. *Med Microbiol Immunol.* 2007; 196:95-102.
- Schrag S, Zell E, Lynfield R, Roome A, Arnold K, Craig A, Harrison L, Reingold A, Stefonek K, Smith G, Gamble M and Schuchat A. A population-based comparison of strategies to prevent early-onset group B streptococcal disease in neonates. *N Engl J Med.* 2002; 347:233-239.
- Stone K, Reiff-Eldridge R, White A, Cordero J, Brown Z, Alexander E and Andrews E. Pregnancy outcomes following systemic prenatal acyclovir exposure: Conclusions from the international acyclovir pregnancy registry, 1984-1999. *Birth Defects Res A Clin Mol Teratol.* 2004; 70:201-207.
- The ANZIC Influenza Investigators. Critical Care Services and 2009 H1N1 Influenza in Australia and New Zealand. N Engl J Med. 2009; 361:1925-1934.
- The Royal Australian and New Zealand College of Obstetricians and Gynaecologists. 2009. Screening and Treatment of Group B Streptococcus in Pregnancy (Accessed March 8, 2010 at: www. ranzcog.edu.au/publications/statements/C-obs19.pdf).
- Troughton J, Crealey G, Crawford V and Coyle P. Management of varicella contacts in pregnancy: VZIG or vaccination? J Clin Virol. 2009; 46:345-348.
- Van Dyke M, Phares C, Lynfield R, Thomas A, et al. Evaluation of universal antenatal screening for group B streptococcus. N Engl J Med. 2009; 360:2626-2636.
- 21. Watts D, Lu M, Thompson B, Tuomala R, *et al.* Treatment interruption after pregnancy: effects on disease progression and laboratory findings. *Infect Dis Obstet Gynecol.* 2009:456717.
- Zaman K, Roy E, Arifeen S, Rahman M, et al. Effectiveness of maternal influenza immunization in mothers and infants. N Engl J Med. 2008; 359:1555-1564.

Gene-environment interactions and the developmental origins of health and disease

A/Prof Craig Pennell FRANZCOG CMFM

Recent studies have clearly established an inverse relationship between suboptimal antenatal and postnatal environments and the development of adult diseases, including the metabolic syndrome (obesity, atherogenic dyslipidemia, hypertension, insulin resistance); type 2 diabetes; cardiovascular disease; neurologic disorders; and mental illness.^{1,2,3,4}

These observations have been confirmed in multiple human populations^{1,5} and in numerous animal studies across multiple species^{4,6,7,8}. It is thus clear that the environment of mother, baby and child is a key contributor to diseases and conditions that account for approximately one third of the global burden of disease, affecting both developed and developing countries. Although adverse antenatal and postnatal environments increase the risk of particular adult diseases, not all individuals exposed to these environments develop these conditions suggesting that an individual's genotype may contribute to the eventual outcome.^{9,10,11,12} It has therefore been hypothesised that interactions between an individual's genotype and the environment underlie the developmental origins of health and disease (DOHaD). See Figure 1.

In 1988, retrospective epidemiological studies in British cohorts reported an inverse relationship between birth size and rates of mortality from coronary heart disease across the normal birth weight range.^{2,3} Subsequently, similar relationships were discovered for other components of the metabolic syndrome, including hypertension, stroke, insulin resistance, type 2 diabetes and dyslipidemia.⁴ Over the next decade, numerous studies were performed in different populations, both in the developed and developing world, confirming this relationship.^{1,5} These studies led to the 'fetal origins' hypothesis that suggested that events in utero which reduced fetal growth permanently altered the structure and

physiology of the offspring such that the risk of heart disease and diabetes in later life was increased.

Animal models have provided the most direct evidence to support the DOHaD thesis.⁴ Programming has been demonstrated in sheep¹³, guinea pigs⁶, pigs, mice and rats^{7,14} and can be induced by: 1) prenatal under-nutrition (in total caloric or protein dietary content); 2) unbalanced nutrition (for example, maternal highlard diet); 3) impairing utero-placental perfusion; or 4) maternal exposure to synthetic glucocorticoids.⁴ Animal studies have demonstrated another important concept, namely, that the fetus need not be manipulated for the relationships between birth size and cardiovascular and metabolic outcomes to be observed.^{4,15,16} The environmental cue can occur in early or late pregnancy, or within the peri-conceptual period.¹⁷ Thus, the term 'fetal origins' has now been replaced by 'developmental origins'.

The most frequent misunderstanding in the DOHaD hypothesis has been the role of birth size. Birth size is simply a crude surrogate reflecting the interactions between the fetal environment and the fetal genome.⁴ There is a growing body of evidence from both animal^{7,18-22} and human studies^{4,23,24} that environmental cues during pregnancy need not alter birth weight to alter long-term outcomes. For example, in the Dutch winter famine, women who ate less than 800 calories a day during the first trimester gave birth to normalsized infants who later became obese.^{23,24} Programming is not a

process confined to the extremes in fetal growth, but rather one that accompanies the adaptations that every fetus makes to its environment, including subtle variations in growth.⁴ Indeed, these relationships exist across the normal birth weight range. Whilst initial studies of the DOHaD focused on events that occurred before birth, more detailed analysis of human cohorts^{25,26,27} and animal studies⁷ have demonstrated interactions between antenatal and postnatal environment. Those who had the most adverse intrauterine environment and the fastest weight gain after birth were shown to be at greatest risk for programming of the metabolic syndrome. Further, human and animal studies have shown clear differences between males and females, not only in the response to adverse antenatal and postnatal environments, but also in the mechanisms that underlie programming.^{28,29,30}

Although a clear relationship exists between adverse antenatal and postnatal environments and the risk of adult disease, there is growing



evidence that genetic polymorphisms modulate this relationship. This is illustrated by the influence of polymorphisms in the peroxisome proliferator-activated receptor (PPAR)-γ2 gene on the relationship between size at birth and adult diseases including: 1) insulin sensitivity and metabolism^{9,31}; 2) hypertension¹²; 3) obesity¹¹ and 4) dyslipidemia¹⁰. The well-known association between small body size at birth and insulin resistance⁹ and/or hypertesion¹² was seen only in individuals with the high-risk Pro12Pro allele. Similarly, polymorphisms in the glucocorticoid receptor (GR), a key control element in the hypothalamic pituitary adrenal axis, have been implicated in determining obesity^{32,33,34}, hypertension³⁵, hypercholesterolemia³⁵ and responses to psychosocial stress in adults³³. Thus complex interactions between genes and the environment modulate developmental programming of adult disease.

Over the last 18 months, in conjunction with international collaborators, we have completed the first analyses investigating the association of the fat mass and obesity (FTO) gene with antenatal and postnatal growth trajectories. We have shown that the AA genotype of the SNP rs9939609 in the FTO gene (which has been found to predispose to diabetes through an effect on BMI in adults) was associated with symmetric intrauterine growth restriction beginning as early as 28 weeks gestation. The same polymorphism in FTO was independently associated with increased body mass index (BMI) in males (but not females) at eight, 14 and 17 years of age in multivariate analyses adjusted for birth weight, smoking, socio-economic status, nutrition and activity. Breastfeeding was associated with reduced BMI in childhood/adolescence, in males but not females, independent of FTO genotype. In both sexes, increased weight gain over the first year was associated with higher BMI in adolescence. The obesogenic allele (AA) in FTO was also associated with greater weight gain in the first year compared to non-obesogenic allele (TT). Gene-environment interactions were identified between FTO and measures of nutrition. The impact of weight gain in the first year of life on BMI at eight, 14 and 17 years of age was greater in AA than TT genotypes. Further, the reduction in BMI at 8 and 14 years of age in males associated with breastfeeding was greater in AA than TT genotypes. There was a significant interaction between FTO genotype and diet at 17 years of age in females, with an even greater reduction in BMI seen in AA than TT genotypes in those with a prudent/healthy diet. We have found evidence of gene-environment interactions between childhood nutrition and the FTO gene acting on measures of childhood obesity. These findings highlight the complex pathways underlying the development of obesity and raise the possibility of reducing the prevalence of this condition by targeted interventions in those at greatest genetic risk.

We have also identified a number of SNPs within key candidate genes (leptin, leptin receptor, adiponectin, CRP, IGF-2BP, insulin receptor, mineralocorticoid receptor and others) with associations with antenatal growth trajectories, biometric measures at birth, postnatal growth trajectories, insulin resistance at 14 years of age and non-alcoholic fatty liver disease at 16 years of age. Moreover, we have identified a number of gene-environment interactions between some of these SNPs and measures of nutrition (breastfeeding, healthy/prudent dietary pattern) suggesting the possibility of potential interventions to limit the impact of some of the SNPs associated with increased risk of obesity, insulin resistance and non-alcoholic fatty liver disease.

Epigenetics refers to covalent modification of DNA and core histones that regulate gene activity without altering the nucleotide sequence of DNA. There is growing evidence that the methylation status of genomically imprinted genes can be altered with consequences for subsequent organ growth and function.^{36,37} Importantly, the epigenetic lability of imprinted genes is not limited to the pre-implantation period and includes the early postnatal period. Recent studies have demonstrated that retrotransposons are elements within the genome that may also be epigenetically labile to early nutrition.^{38,39} The application of epigenetic approaches and the determination of imprinted or non-imprinted genes and transposon insertion sites that are targets for early nutritional effects on epigenetic gene regulation are important new areas of investigation⁴⁰ which have great potential to uncover important mechanisms underlying DOHaD.

Identification at birth of genetic signatures that enhance the risk of coronary heart disease, stroke, diabetes, obesity, neurologic disorders or mental illness will provide opportunities to develop lifestyle or medical intervention strategies aimed at preventing these adverse outcomes. The potential to impact on global disease burden and the opportunity to establish healthy life-long trajectories for children is immense.

References

- 1. Barker DJ. *Mothers, babies and health in later life*. Edinburgh: Churchill Livingstone, 1998.
- Barker DJ, Osmond C. Low birth weight and hypertension. BMJ 1988; 297:134-5.
- Barker DJ, Winter PD, Osmond C, Margetts B, Simmonds SJ. Weight in infancy and death from ischaemic heart disease. *Lancet* 1989; 2:577-80.
- Gluckman PD, Hanson MA. Developmental origins of disease paradigm: a mechanistic and evolutionary perspective. *Pediatr Res.* 2004; 56:311-7.
- Osmond C, Barker DJ. Fetal, infant, and childhood growth are predictors of coronary heart disease, diabetes, and hypertension in adult men and women. *Environ Health Perspect.* 2000; 108 Suppl 3:545-53.
- Matthews SG. Early programming of the hypothalamo-pituitaryadrenal axis. Trends Endocrinol Metab. 2002; 13:373-80.
- 7. Bertram CE, Hanson MA. Animal models and programming of the metabolic syndrome. *Br Med Bull*. 2001; 60:103-21.
- 8. Newnham JP. Is prenatal glucocorticoid administration another origin of adult disease? *Clin Exp Pharmacol Physiol.* 2001; 28:957-61.
- Eriksson JG, Lindi V, Uusitupa M, et al. The effects of the Pro12Ala polymorphism of the peroxisome proliferator-activated receptorgamma2 gene on insulin sensitivity and insulin metabolism interact with size at birth. *Diabetes* 2002; 51:2321-4.
- Eriksson J, Lindi V, Uusitupa M, et al. The effects of the Pro12Ala polymorphism of the PPARgamma-2 gene on lipid metabolism interact with body size at birth. *Clin Genet*. 2003; 64:366-70.
- 11. Pihlajamaki J, Vanhala M, Vanhala P, Laakso M. The Pro12Ala polymorphism of the PPAR gamma 2 gene regulates weight from birth to adulthood. *Obes Res.* 2004; 12:187-90.
- Yliharsila H, Eriksson JG, Forsen T, et al. Interactions between peroxisome proliferator-activated receptor-gamma 2 gene polymorphisms and size at birth on blood pressure and the use of antihypertensive medication. J Hypertens. 2004; 22:1283-7.
- Moss TJ, Sloboda DM, Gurrin LC, Harding R, Challis JR, Newnham JP. Programming effects in sheep of prenatal growth restriction and glucocorticoid exposure. *Am J Physiol Regul Integr Comp Physiol*. 2001; 281:R960-70.
- Matthews SG, Owen D, Banjanin S, Andrews MH. Glucocorticoids, hypothalamo-pituitary-adrenal (HPA) development and life after birth. *Endocr Res.* 2002; 28:709-18.
- Kind KL, Simonetta G, Clifton PM, Robinson JS, Owens JA. Effect of maternal feed restriction on blood pressure in the adult guinea pig. *Exp Physiol.* 2002; 87:469-77.
- Kind KL, Clifton PM, Grant PA, et al. Effect of maternal feed restriction during pregnancy on glucose tolerance in the adult guinea pig. Am J Physiol Regul Integr Comp Physiol. 2003; 284:R140-52.
- Kwong WY, Wild AE, Roberts P, Willis AC, Fleming TP. Maternal undernutrition during the preimplantation period of rat development causes blastocyst abnormalities and programming of postnatal hypertension. *Development* 2000; 127:4195-202.

List of references continued on page 30.

- Oliver MH, Breier BH, Gluckman PD, Harding JE. Birth weight rather than maternal nutrition influences glucose tolerance, blood pressure, and IGF-I levels in sheep. *Pediatr Res.* 2002; 52:516-24.
- Hanson M. Birth weight and the fetal origins of adult disease. *Pediatr Res.* 2002; 52:473-4.
- Bertram CE, Hanson MA. Prenatal programming of postnatal endocrine responses by glucocorticoids. *Reproduction* 2002; 124:459-67.
- Hoet JJ, Hanson MA. Intrauterine nutrition: its importance during critical periods for cardiovascular and endocrine development. J Physiol. 1999; 514 (Pt 3):617-27.
- Lingas RI, Matthews SG. A short period of maternal nutrient restriction in late gestation modifies pituitary-adrenal function in adult guinea pig offspring. *Neuroendocrinology* 2001; 73:302-11.
- Ravelli GP, Stein ZA, Susser MW. Obesity in young men after famine exposure in utero and early infancy. N Engl J Med. 1976;295:349-53.
- Ravelli AC, van Der Meulen JH, Osmond C, Barker DJ, Bleker OP. Obesity at the age of 50 y in men and women exposed to famine prenatally. *Am J Clin Nutr.* 1999;70:811-6.
- Eriksson JG, Forsen T, Tuomilehto J, Winter PD, Osmond C, Barker DJ. Catch-up growth in childhood and death from coronary heart disease: longitudinal study. *BMJ* 1999;318:427-31.
- Burke V, Beilin LJ, Simmer K, et al. Predictors of body mass index and associations with cardiovascular risk factors in Australian children: a prospective cohort study. Int J Obes Relat Metab Disord. 2004.
- Burke V, Beilin LJ, Blake KV, et al. Indicators of fetal growth do not independently predict blood pressure in 8-year-old Australians: a prospective cohort study. *Hypertension* 2004;43:208-13.
- McCormick CM, Smythe JW, Sharma S, Meaney MJ. Sex-specific effects of prenatal stress on hypothalamic-pituitary-adrenal responses to stress and brain glucocorticoid receptor density in adult rats. *Brain Res Dev Brain Res.* 1995;84:55-61.
- Dean F, Matthews SG. Maternal dexamethasone treatment in late gestation alters glucocorticoid and mineralocorticoid receptor mRNA in the fetal guinea pig brain. *Brain Res.* 1999;846:253-9.
- Andrews MH, Matthews SG. Programming of the hypothalamopituitary-adrenal axis: serotonergic involvement. Stress 2004;7:15-27.
- Laakso M. Gene variants, insulin resistance, and dyslipidaemia. Curr Opin Lipidol. 2004;15:115-20.
- van Rossum EF, Voorhoeve PG, te Velde SJ, et al. The ER22/23EK polymorphism in the glucocorticoid receptor gene is associated with a beneficial body composition and muscle strength in young adults. J Clin Endocrinol Metab. 2004;89:4004-9.
- Wust S, Van Rossum EF, Federenko IS, Koper JW, Kumsta R, Hellhammer DH. Common polymorphisms in the glucocorticoid receptor gene are associated with adrenocortical responses to psychosocial stress. J Clin Endocrinol Metab. 2004;89:565-73.
- Rosmond R. Association studies of genetic polymorphisms in central obesity: a critical review. Int J Obes Relat Metab Disord. 2003; 27:1141-51.
- Di Blasio AM, van Rossum EF, Maestrini S, et al. The relation between two polymorphisms in the glucocorticoid receptor gene and body mass index, blood pressure and cholesterol in obese patients. Clin Endocrinol. (Oxf) 2003;59:68-74.
- Young LE, Fernandes K, McEvoy TG, et al. Epigenetic change in IGF2R is associated with fetal overgrowth after sheep embryo culture. Nat Genet. 2001;27:153-4.
- Waterland RA, Jirtle RL. Early nutrition, epigenetic changes at transposons and imprinted genes, and enhanced susceptibility to adult chronic diseases. *Nutrition* 2004;20:63-8.
- Wolff GL, Kodell RL, Moore SR, Cooney CA. Maternal epigenetics and methyl supplements affect agouti gene expression in Avy/a mice. *Faseb J.* 1998;12:949-57.
- Waterland RA, Jirtle RL. Transposable elements: targets for early nutritional effects on epigenetic gene regulation. *Mol Cell Biol.* 2003; 23:5293-300.
- McMillen IC, Robinson JS. Developmental origins of the metabolic syndrome: prediction, plasticity, and programming. *Physiol Rev.* 2005; 85:571-633.

Substance abuse in pregnancy

Dr Joanne LudlowSubstance abuse has been with us since antiquity. It is epitomised in the picture
below of 'Gin Lane', issued in 1751 by English artist William Hogarth in support of
the Gin Act, to depict the evils of drinking gin as opposed to the merits of beer.

The combination of 'Beer Street' and 'Gin Lane' showed those on Beer Street as happy and healthy and those in Gin Lane as living in poverty, depicting infanticide, starvation, madness, decay and suicide. The free market economy of the day leaves the occupants of Beer Street as prosperous, while making those of Gin Lane poorer. This is similar to today where the tobacco companies are growing rich whilst tobacco smokers are overrepresented amongst the poorest in our society. Gin Lane also depicts some of the more harrowing aspects of substance addiction which are still seen in some cases today – prostitution and drug use being of more importance than child-rearing. These are clearly extreme examples. The prints were published in the *London Evening Post* between 14 and 16 February 1751 with the intention of 'shocking the lower classes into reforming'.^{1,2}

On a per capita basis, there was more narcotic abuse in the late 19th century than in the late 20th century.³ Substance use and addiction is a worldwide phenomenon and affects the whole social spectrum. While there is heterogeneity in the drug scene from between locations, the clinical management and controversies are remarkably similar.

According to the 2004 National Drug Strategy Household Survey⁴, alcohol use decreased during pregnancy and/or breastfeeding from 85 per cent to 47 per cent. Illicit drug use decreased from 17 per cent to six per cent and smoking decreased from 22 per cent to 20 per cent. The highly addictive nature of cigarette smoking is reflected in the minimal reduction in smoking between the pregnant and non-pregnant state.



Determining whether a drug is teratogenic or has caused some form of problem for the mother and her baby is complicated by the interaction with environmental factors. Clearly, the amount of drug consumed per day has some influence. Polydrug use is common, hence teasing out all individual contributory drugs and environmental factors is extremely difficult.

There is a paucity of robust level 1/11 evidence in this field. Many recommendations have been based on case reports and case series from the 1970s which have been passed on through the reference cascade. Many of these case reports, case series, etc, would not be published in 2010 as they would not survive the rigour of the peer review process. However, it is not possible to perform high quality double-blind randomised trials with pregnant drug-using women and drug-exposed neonates due to ethical considerations. For instance, randomising pregnancies to receive stratified dose/ frequencies of a particular licit or illicit drug and measuring outcomes is not ethically possible nor desirable! However, cohort studies both retrospective and prospective are affected by multiple confounders.

While some women with substance addiction are chaotic and only present in labour having had no antenatal care, many pregnant women with an opioid addiction are stabilised on methadone, hold down steady jobs, manage a family and engage in antenatal care.

National guidelines

In June 2006, the National Clinical Guidelines for the Management of Drug Use During Pregnancy, Birth and the Early Development Years of the Newborn commissioned by the Ministerial Council on Drug Strategy under the Cost Shared Funding Model were published.⁵ These guidelines can be accessed from the New South Wales Health website (www.health.nsw.gov.au/pubs/2006/ncg_ druguse.html).

This publication was a collaborative work between midwives, nurses, obstetricians, general practitioners, neonatologists, psychiatrists, psychologists, drug and alcohol physicians, social workers, researchers, a policy analyst and an international expert from the United States, Dr Karol Kaltenbach.

Medical problems

Medical problems include infectious diseases; poor nutrition; malabsorption; poor dentition; dermatological problems such as cellulitis and dermatitis artefacta; cardiovascular problems such as bacterial endocarditis and valvular heart disease; respiratory morbidity such as pneumonia; and neurological problems such as cerebrovascular accident (CVA) from emboli, Wernicke's encephalopathy (alcohol) and peripheral neuropathy (inhalants).

Psychosocial problems

Women whose pregnancies are affected by a substance addiction are often complicated by and/or associated with poverty, homelessness, social isolation, a chaotic lifestyle, prostitution, criminal activity, family disruption, violence and trauma. 60 to 80 per cent of these women have psychiatric co-morbidity⁶ such as depression, anxiety disorder, mania, dysphoria and drug overdoses. A reported history of physical, psychological, emotional and sexual abuse is common. Subsequently, high levels of low self-worth, self-harm and eating disorders are reported amongst this group of women. They are more likely to have an unplanned pregnancy, be unemployed, be Indigenous, to have not completed their schooling and to be incarcerated.

Obstetric and perinatal complications

The reported increased obstetric complications include increased rates of miscarriage, perinatal and infant mortality, anaemia, abruption, preterm delivery and low birth weight. These women need more pharmacological analgesia during labour/delivery and in the postpartum period following caesarean section. Babies exposed to licit and illicit substances in utero are more likely to require admission to the special care nursery, to experience Neonatal abstinence syndrome (NAS) and to be medicated. These babies are more likely to be removed from their mothers by the local child protection authorities, to die from sudden infant death syndrome (SIDS) and to be developmentally delayed.

Teratogenicity

There is a three per cent background rate of congenital abnormalities. To ascribe teratogenicity to a drug death, an observed above expected rate of a particular abnormality or series of abnormalities, growth restriction or functional disorders would need to be seen amongst the offspring of women using this drug exclusively. This relationship should be demonstrated to be doserelated and the fetus should be exposed during a critical period of development and is affected by an interaction of genetic and environmental factors.⁷

Detection of drug use and patterns of use in pregnancy

Detection of drug dependence by history and examination is the cornerstone of management of these complex pregnancies. This allows referral to the appropriate antenatal clinic, stabilisation of the patient onto legal alternatives, management of the medical and obstetric complications and provision of psychosocial support.

Most obstetric units use a history of self-reporting, detailed interview and a screening questionnaire. Objective screening has been suggested, however, should this be universal or targeted? Which biological sample should be examined and which metabolite should be measured? There are ethical, legal and financial considerations of such an approach. There are certain clinical manifestations and psychosocial clues, for example, women who present in preterm labour having had no antenatal care, unkempt, needle tract marks, etc.

Many drug-addicted women who become pregnant are motivated to cease their habit and engage in healthcare. The marker for success in these cases is whether their partner also uses drugs. The women whose partners are also motivated to stop their drug use have a much higher likelihood of remaining drug free.

The HITS study⁶ showed that women reduced their drug use during pregnancy from the first trimester to 36 weeks gestation. However, by six months postpartum, their drug use was back to prepregnancy levels. Perhaps this is because the incentive (not exposing their fetus to drugs) has gone. We need to work out effective interventions to prevent this relapse.

Engagement and retention in care

Maternal and child wellbeing is enhanced by trust between patient and health professional. Continuity of caregiver, assurance of confidentiality, being treated with respect, allaying patient fears about being stigmatised and refraining from displaying patronising attitudes contribute to successful engagement of the patient in ongoing antenatal care. However, poor treatment, the sheer array of professionals seen in a multidisciplinary setting, fears that their baby will be apprehended, that they cannot cope and sometimes inconsistency of information, do not encourage patients to reattend. The importance of always thoroughly investigating increasing analgesia requirements was demonstrated to me once in a postpartum patient. Her increasing requests for analgesia for severe lower back pain were being interpreted as drug-seeking behaviour. She was subsequently diagnosed (after discharge from the maternity hospital) with a sacroiliac joint abscess!

Obstetric care

Most tertiary obstetric units in Australia and New Zealand run a multidisciplinary antenatal clinic for women with a chemical dependency in pregnancy staffed by midwives, obstetricians, drug and alcohol clinicians, a psychologist/psychiatrist, a social worker and a dietician. Both neonatal and developmental paediatricians are involved in planning neonatal and community care post-delivery depending on each individual patient's circumstances. Whilst the majority of patients have regular visits to the hospital antenatal clinic, they are encouraged to maintain links with their local GP during their pregnancy. Some patients obtain their methadone from their GP. Some women prefer to use someone other than their GP as their prescriber if they are embarrassed about their drug dependence and do not want the GP they may have had from childhood and are familiar with their family to know. Indigenous women may prefer to use their local Aboriginal medical services. Whether home visiting in the antenatal and or postnatal period is of value is controversial.

Specific symptoms and conditions which present in this population are anaemia from deficiencies of iron, folate and/or vitamin B or as a result of chronic disease; constipation from the large doses of methadone required for maintenance in pregnancy; nausea and vomiting; skin conditions such as cellulitis; dental problems; and infections (sexually transmitted, respiratory, cardiac and idiosyncratic infections).

There can sometimes be overlap between drug-related effects and obstetric complications causing confusion in diagnosis. I was involved in the care of a patient who presented, having had no antenatal care, with mildly elevated blood pressure, proteinuria, low platelets, mildly elevated liver function tests (LFTs) and was diagnosed with HELLP syndrome and treated accordingly. Her intravenous opioid addiction had gone undetected for 18 hours until she began to withdraw and was found injecting herself in the ward bathroom. Postpartum she developed subacute bacterial endocarditis. In retrospect, it became clear that her blood pressure was most likely mildly elevated whilst being in labour. Her LFTs were elevated and platelets were low due to the developing infection. Subsequently, she underwent a mitral valve replacement.

Nausea and vomiting

Nausea and vomiting can be particularly intractable requiring hospitalisation, intravenous fluids, 5HT3 antagonists such as ondansetron and consideration of split dosage of methadone to reduce the serum peaks. Obviously, other causes of vomiting need to be excluded (for example, gastroenteritis, urinary tract infections, preeclampsia, etc). Prochlorperazine and metoclopromide are used as first-line, although prolonged use should be avoided due to the risk of extra pyramidal side effects, especially in younger women. Supplementation with pyridoxine and thiamine is recommended.

Addressing the psychosocial situation

This involves management of any psychiatric co-morbidity, assisting with accommodation and appropriate housing for a nursing mother, baby plus or minus partner, help with budgeting and benefits. If domestic violence has been elicited, moving the woman to a safe environment is paramount. Antenatal reporting to the local child protection agency in the setting of domestic violence, chaotic mental health situations and hazardous drug use may allow the social work team to address these problems thereby increasing the chances of the woman keeping her baby.

Hepatitis C

Hepatitis C is common amongst ever-intravenous drug users in the

eastern states of Australia (90 per cent) compared with the rate in Western Australia (45 per cent).⁸ The detection of seropositivity mandates quantification of hepatitis C RNA and viral load and referral to gastroenterology for consideration of interferon therapy once the pregnancy and puerperium are over. A study is about to commence in New South Wales to evaluate the incidence and mechanisms of vertical transmission of hepatitis C.

Labour and delivery

It is essential that methadone is given on time. Non-compliance by clinicians is cruel and unprofessional and only causes withdrawal symptoms in the patient. I have seen this occur. Unfortunately, the person who failed to administer the methadone had gone home and the person on the next shift then had to manage the patient's opiate withdrawal. The high levels of smokers in this cohort (greater than 90 per cent) is commensurate with high prevalence of intrauterine growth restriction (IUGR) and placental insufficiency. In some cases, the placenta may not have the reserve for labour. Whilst reduced fetal heart rate variability is a common feature in women on opioids or other central nervous system (CNS) depressants, decelerations are always suspicious or pathological. Meconium-stained liquor and abruption are more common in this group of women. They also require more analgesia for labour and delivery. Nitrous oxide and regional anaesthesia are more appropriate choices of pain relief than opiates. Venous access is often extremely difficult to obtain.

Postpartum

Unless there are concerns about neonatal abstinence syndrome (NAS) the mother and baby should be together. Exceptions include planned apprehension to child protection services. A further tailoring of methadone is usually required. Co-therapies such as benzodiazepines may also need modification. Contraception and advice on pregnancy spacing should be provided. Comprehensive discharge planning which commences antenatally should be finalised. Ideally, there should be a seamless connection from the hospital into the community so that the patient knows where to access help with mother-crafting and other matters. Discharge from hospital should not be at the weekend. The patient should be aware of any planned follow-up appointments for her and her baby. There is an urgent need for more mother and baby units in Australia, so that women with drug addiction and co-related mental health problems can be monitored and supervised in the nursing of their babies, to try and reduce the current level of mother and infant separations.

Stabilisation to legal alternatives

Methadone is the recommended therapy of choice for opiate addiction.⁵ Withdrawal has a high relapse rate. Enough methadone needs to be given to prevent cravings, manage withdrawal symptoms and remove the euphoric effects of heroin. Stabilisation on methadone improves antenatal attendance, gestational age and birth weight at delivery. The trade-off is NAS. The daily dose varies in pregnancy depending on the amount of placental metabolism of the methadone and degree of protein-binding of the methadone. Withdrawal is not recommended due to the high relapse rate⁹ and the wide troughs and peaks to which the fetus is then exposed. Withdrawal should be discouraged but accommodated if the patient requests this, but only in the second trimester.

Buprenorphine, a partial opioid agonist and partial antagonist, is being used increasingly in pregnancy and several studies have shown no adverse effects in comparison to methadone.¹⁰

Naltrexone implants offer some hope for the future, as they would minimise NAS and have been successfully used in some cases (clinical experience and anecdotal evidence). However, far more work in the animal model and non-pregnant state need to be performed to establish safety and pharmacokinetics before its use can be recommended in the management of opioid dependence in pregnancy. Polydrug use should be discouraged and women told of the high risk of IUGR and prematurity where methadone as well as street drugs are used.⁶

Effect of difference drugs in pregnancy

Alcohol is socially acceptable, legal and the most commonly used substance. However, it is potently teratogenic in moderate amounts and may have subtle cognitive effects in the third trimester. It causes a spectrum of disorders from fetal alcohol syndrome (FAS)¹¹ to fetal alcohol spectrum disorders (FASD) to fetal alcohol effects (FAE). FAS is characterised by distinct facial anomalies, pre and postnatal growth restriction and poor cognitive function and behavioural problems.^{11,12} The 'safe' level of alcohol consumption in pregnancy is not known although episodic bingeing is worse than chronic low level consumption. Abstinence is not recommended as this would cause panic and unnecessary termination of pregnancy if women had an alcoholic binge one night out when they were unaware that they were pregnant. Surely one glass of champagne to toast a wedding at 28 weeks is not going to have significant long-term sequelae.

Amphetamines are often used with other drugs or replace opioids when the latter are not available. They have not been proven to be teratogenic, but are associated with pre term delivery low birth weight for gestational age and high levels of depression after prolonged use. It can mimic obstetric complications such as preeclampsia in the third trimester. The most potent of this family of drug is 'ice', which causes profound behavioural problems and in some cases fully blown psychoses and in some a deep depression in the 'hangover' phase. Ecstasy, the dance party drug, is the most commonly used of this class of drug and its use is usually stopped once the women knows she is pregnant. Despite its bad press and sporadic deaths, ecstasy causes fewer admissions to hospital emergency departments than alcohol use.

There is conflicting data about the effects of **benzodiazepines** on the human embryo and fetus. Pooled data from case controlled studies increased incidence of oral clefts.¹³ But meta analysis data from cohort studies¹³ has shown no such association. The fetus exhibits benzodiazepine receptors by 15 weeks gestation. Abrupt cessation of any benzodiazepine should be avoided. The concomitant use of benzodiazepines with opioids in pregnancy increases the likelihood and the severity of NAS.

Cocaine is a potent vasoconstrictor. It affects dopamine receptors, produces a feeling of euphoria, is highly lipid soluble and alters opioid receptor densities. Cocaine increases the incidence of placental abruption and infarcts; IUGR; intrauterine hypoxia; preterm labour and delivery; fetal intraventricular haemorrhage; cerebral infarction; neonatal necrotizing enterocolitis; and maternal cardiac effects such as arrhythmias, myocardial infarction and cerebrovascular events. It is estimated to be used by two to three per cent women of childbearing age. 'Cocaine arrests up' headlines the Wentworth Courier, the local free advertising magazine of the affluent eastern suburbs of Sydney, this week.¹⁴ This substance is mainly, but not exclusively, used at 'the high end of town'. However, it is one of the most teratogenic (together with alcohol). How many members of the medical profession use both of these drugs, yet show scant respect for the more vulnerable and disadvantaged in our society, who for various reasons, find themselves in the grip of substance addiction? Isn't a double standard being perpetuated?

Marijuana is an effective treatment for nausea and vomiting, according to many of our patients. The National Institute on Drug Abuse (NIDA) USA 1998 estimated that 2.8 per cent of women used marijuana in the first trimester.¹⁵ There have been few human studies on its effects in pregnancy. The reports on its effects on birth weight for gestational age have been conflicting. It has been reported that long-term use causes schizophrenia and it is postulated that it may have behavioural, developmental and cognitive effects on the

developing fetus. This is difficult to measure given the interaction with the home and parenting environment.

Heroin is not teratogenic per se. Most of the poor outcomes are due to a combination of lack of antenatal care, poor diet, injection of other substances used to cut the street drug and the environmental milieu. It is often adulterated and causes unstable peaks and troughs in maternal and fetal blood. Heroin rapidly crosses the placenta.

Tobacco is the major preventable cause of poor pregnancy outcome. Twenty per cent of all pregnant women⁴ and more than 90 per cent in this population smoke during pregnancy (personal observation). Most of the obstetric and perinatal complications seen amongst intravenous drug users are probably due to concomitant smoking. Miscarriage; IUGR; preterm delivery; fetal death in utero (FDIU); abruption; neonatal death (NND); sudden infant death (SIDS); behavioural problems; and lung and respiratory abnormalities, are all increased compared to the general obstetric population.¹⁶ Smoking doubles the incidence of IUGR.¹⁷ There is a demonstrable dose-response relationship even after controlling for age, parity, maternal weight gain, prepregnancy BMI, GA, socioeconomic factors and ethnicity.^{18,19} Babies of smokers are 200 to 300g lighter on average than babies of non-smokers.²⁰

Babies of smokers demonstrate altered critical autonomic reflexes²¹, unstable breathing, impaired arousal and catecholamine biosynthesis, increased susceptibility to stress, sudden death and disease in later life. These children are then more likely to take up smoking and become nicotine dependent. ^{22,23,24}

All of these findings may be mediated via nicotine receptors.²⁵ So it is possible that the use of nicotine preparations in cessation of smoking may not negate some of these effects, even though it will improve the mother's long-term health if she does stop smoking preterm delivery.

Despite this knowledge and the current television advertising campaign, one in five women continue to smoke during pregnancy, indicating the highly addictive nature of nicotine rather than character flaws in our patients. Recent legislation, changes in packaging and increasing the price of cigarettes is unlikely to change this significantly. Smoking is the only pleasure for some of our most disadvantaged patients. It is their way of de-stressing. Whilst we have to continue to advocate non-smoking, we could perhaps be more understanding of why this particular group of women do not find it easy to concur. This is especially true of those who have ceased their heroin and marijuana habit. In doing a practice station for MRANZCOG oral examination candidates last year, I played the role of a patient who was a heavy smoker. After eight consultations, I (a non-smoker) became quite agitated about being repeatedly told to stop smoking! We may come across as being superior, smug and lacking any understanding of their circumstances. Sadly, smoking is now being replaced by obesity as the major health problem - few in this cohort of patients have a weight problem. As a population, we are stopping smoking and we are getting fatter - surely the two are related!!

Discussion points

Management of pregnancies complicated by substance abuse provides an opportunity for women and their babies to improve their health outcomes. Antenatal care does make a large difference to this cohort of women. My view is that all drugs should be legalised to bring the 'industry' above ground and remove the attraction to this area by the criminal element in our society. Doses and drug content would be known. The drugs would be cheaper, negating the requirement for risky activities to fund their habit. They would be more likely to engage in antenatal care and be open about the extent of their drug use and less likely to end up in jail. Safe injecting rooms would reduce their exposure to blood-borne viruses. We need seamless transition between hospital and community. Then mother and baby units may help more babies to stay with their mothers.

- Paulson R. Hogarth: Art and Politics, 1750-64 Vol 3. Lutterworth Press. 1993. ISBN 0718828755.
- 2. Uglow J. *Hogarth: A life and a world*. New York: Farrar Straus Giroux. 1997. ISBN 0374528519).
- Courtwright D. Addicts who survived: an oral history of narcotic use in America 1923-1965. Knoxville: University of Tennessee Press, 1989:24.
- 4. Australian Institute of Health and Welfare (2005). *Statistics on drug use in Australia in 2004*. AIHW Canberra, p63.
- National clinical guidelines for the management of drug use during pregnancy, birth and the early developmental years of the newborn. Commissioned by the Ministerial Council on Drug Strategy under the Cost Shared Funding Model. NSW Department of Health. June 2006.
- Bartu A, Sharp J, Ludlow J, Doherty D. Postnatal home visiting for illicit drug-using mothers and their infants: A randomised controlled trial. ANZJOG 2006; 46: 419-426.
- Kalter H. Teratology on the 20th century: environmental causes of congenital malformations in humans and how they were established. *Neurotoxicology and Teratology* 2003; 25(2): 131-282.
- Richardson R, Bolisetty S, Ingall C. The profile of substance-using mothers and their newborns at a regional rural hospital in New South Wales. ANZJOG 2001;41;415-419.
- Luty J, Nikolau V, Bearn J. Is opiate detoxification unsafe in pregnancy? Journal of Substance Abuse Treatment 2003; 24:363-367.
- Kakko J, Heilig M, Sarman I. Buprenorphine and methadone treatment of opiate dependence during pregnancy: comparison of fetal growth and neonatal outcomes in two consecutive case series. *Drug and Alcohol Dependence* 2008; 96(1-2);69-78.
- Lemoine P, Harousseau H, Borteyru J and Menoet J. Children of alcoholic parents; anomalies observed in 127 cases. *Quest Medicale* 1968; 21; 476-482.
- 12. Jones K, Smith D. Recognition of fetal alcohol syndrome in early infancy. *Lancet* 1973; 2(7836); 999-1001.
- Dolovich L, Addis A, Vaillancourt J, Power J, Einarson T. Benzodiazeoine use in pregnancy and major malformations or oral cleft: meta-analysis of cohort and case control studies. *BMJ* 1998; 317 (7162): 839-43, Sep 26.
- 14. Wentworth Courier, Sydney, Australia. Wednesday 28th April 2010.
- 15. NIDA 1998: National Household Survey on Drug Abuse. Washington DC: Government Printing Office.
- Malloy M, Kleinman J, Land G, Schramm W. The association of maternal smoking with age and cause of infant death. *American Journal of Epidemiology* 1988; 128(1)p46-55.
- Haug K, Irgens L, Skjaerven R, Markestad T, Baste V, Schreuder P. Maternal smoking and birthweight: Effect modification of period, maternal age and paternal smoking. *Acta Obstetricia et Gynecologica Scandinavica* 2000; 79(6)p485-489.
- Abel E. Smoking and pregnancy. Journal of Psychoactive Drugs 1984; 16(4):327-38, 1984 Oct-Dec.
- Werler M, Pober B, Holmes L. Smoking and pregnancy. *Teratology* 1985 Dec; 32(3):473-81.
- Hebel J, Fox N, Sexton M. Dose-response of birth weight to various measures of maternal smoking during pregnancy. *Journal of Clinical Epidemiology* 1988; 41(5):483-9.
- Nachmanoff B, Panigrahy A, Filiano J, Mandell F, et al. Brainstem 3H-nicotine receptor binding in the sudden infant death syndrome. Journal of Neuropathology & Experimental Neurology 1998 Nov; 57(11):1018-25.
- Shenassa E, McCaffery J, Swan G, et al. Intergenerational transmission of tobacco use and dependence: a transdisciplinary perspective. [Review] [164 refs] Nicotine & Tobacco Research 2003 Dec; 5 Suppl 1:S55-69.
- Abreu-Villaca Y, Seidler F, Slotkin T. Does prenatal nicotine exposure sensitize the brain to nicotine-induced neurotoxicity in adolescence? *Neuropsychopharmacology* 2004; 29(8)p1440-1450.
- Abreu-Villaca Y, Seidler F, Tate C, Cousins M, Slotkin T. Prenatal nicotine exposure alters the response to nicotine administration in adolescence: Effects on cholinergic systems during exposure and withdrawal. Neuropsychopharmacology 2004; 29(5)p879-890.
- Cohen G, Roux J, Grailhe R, et al. Perinatal exposure to nicotine causes deficits associated with a loss of nicotinic receptor function. Proceedings of the National Academy of Sciences of the United States of America. 102(10):3817-21, 2005 Mar 8.

Management of preterm prelabour rupture of the membranes in the third trimester

Dr Sarah Buchanan RANZCOG Trainee

Preterm prelabour rupture of the membranes (PPROM) occurs when there is rupture of the membranes prior to term and prior to the onset of labour. PPROM complicates pregnancy for one per cent to two per cent of all women and is associated with 30 per cent to 40 per cent of preterm births.^{1,2,3}

Prof Jonathan Morris FRANZCOG

The optimal management of such pregnancies is uncertain. This article reviews the evidence for various aspects of the management of PPROM.

Hospitalisation

At the time of suspected PPROM, women should be advised to present to hospital. The diagnosis of PPROM should be confirmed by a careful history and examination, including a sterile speculum examination. Digital vaginal examinations are best avoided. Following PPROM, the duration of latency is inversely related to gestational age at membrane rupture.

'There is currently insufficient evidence to guide the safety of the management of PPROM in the home.'

The management of women with PPROM and a viable pregnancy has traditionally been as an inpatient. However, there is little evidence on which to base this decision. There has only been one randomised trial evaluating the safety of outpatient versus inpatient management of women with PPROM.⁴ This trial included 55 women with PPROM who were randomly assigned to either expectant management at home or expectant management in hospital. There were no significant differences in maternal or neonatal outcomes between groups, but the home group had lower maternal costs. However, this was a small study with insufficient power to assess differences in possible serious maternal and neonatal morbidity between the groups. There is therefore currently insufficient evidence to guide the safety of the management of PPROM in the home.

Use of tocolysis

Tocolysis to prolong pregnancy, in order to obtain short-term benefits for the mother and the fetus, has been demonstrated in women at risk of preterm birth with intact membranes.⁵ However, the use of tocolysis in the setting of PPROM is controversial. A number of clinical trials have found that while tocolytics may prolong pregnancy for a short period, there is no benefit with respect to improving perinatal outcome in the setting of PPROM.^{6,7}

However, as tocolytics themselves do prolong pregnancy for a short duration, their use should be considered in the setting of PPROM at less than 34 weeks gestation for 48 hours if there is no evidence of clinical chorioamnionitis or complications, which would necessitate delivery, for the fetus to receive the maximal benefits of corticosteroid administration. Tocolysis is also appropriate to enable transfer to a tertiary centre.

Use of corticosteroids

The use of antenatal corticosteroids has been demonstrated to reduce the incidence of neonatal respiratory distress, intraventricular haemorrhage and neonatal death in the preterm neonate.⁸ These beneficial effects of corticosteroids also apply to women with PPROM.^{9,10,11} Corticosteroids should be administered to women at less than 34 weeks gestation.

Use of antibiotics

The short-term benefits of prophylactic antibiotics in the setting of PPROM has been well established.¹² In the ORACLE I trial, women with PPROM were randomised either to treatment with erythromycin 250mg every six hours, or with co-amoxiclav, or both or placebo. Women randomised to the erythromycin aroup had an increased latency between PPROM and delivery, a reduction in neonatal death, chronic lung disease and major cerebral abnormality on ultrasound. Co-amoxiclav demonstrated a similar benefit at prolonging pregnancy and reducing maternal infection, however, these babies had an increased incidence of necrotising enterocolitis.¹³ Based on this evidence, women presenting with PPROM should receive a ten-day course of erythromycin 250mg every six hours according to the protocols used in the ORACLE trial. However, recent follow-up data at seven years for the participants in the ORACLE I trial demonstrated no long-term benefit of such an antibiotic regimen.¹⁴

In addition, the benefits of intrapartum chemoprophylaxis for group-B streptococcus have been well demonstrated.^{15,16,17} For this, intrapartum chemoprophylaxis intravenous penicillin should be used in labour.

Monitoring of women with PPROM who are managed expectantly

Clinical chorioamnionitis is present in one to two per cent of women with PPROM and subsequently develops in three to eight per cent.¹⁸ The incidence of clinical infection increases with decreasing gestational age at which PPROM occurs.¹⁹ Intrauterine infection predisposes the fetus to white matter injury and subsequent adverse neurodevelopmental problems such as cerebral palsy.^{20,21,22,23} Therefore, in monitoring women with PPROM who are managed expectantly, it would be important to identify chorioamnionitis and expedite delivery to minimise complications for the mother and baby.

A number of markers have been looked at in an attempt to predict chorioamnionitis. None of these markers have shown promise. It would be ideal to have a marker that had both a high sensitivity and specificity for chorioamnionitis. C-reactive protein (CRP) has been proposed as such a marker. However, its sensitivity of 54 per cent and specificity of 56 per cent preclude its routine use for prediction of chorioamnionitis.¹⁸

Timing of delivery

The management of PPROM is dependent upon the gestation at which rupture of the membranes occurs. The health benefits for the fetus in continuing a pregnancy after PPROM may be considerable, particularly in the early third trimester. However, there is currently no consensus as to the optimal management of PPROM in women in whom the fetus is relatively mature, at gestations near to term. The aim of care for women with PPROM is to maximise the benefits of further fetal maturity, while avoiding the potential harms of remaining in utero.

For women with PPROM at less than 34 weeks gestation, there is some consensus that unless there are factors that necessitate delivery, the management should be a conservative approach. Delivery would be expedited if complications were found in monitoring of the mother and fetus.

These women with PPROM prior to 34 weeks are best cared for in a hospital with facilities capable of providing neonatal resuscitation and neonatal intensive care, should they require emergency delivery.

'There is no evidence on the optimal timing of delivery for women with PPROM at gestations greater than 34 weeks.'

There is currently no evidence to provide guidance as to when pregnancies complicated with PPROM at greater than 34 weeks gestation should be delivered. We recently performed a *Cochrane* review to assess the evidence for this.²⁴ We included seven trials (690 women) in the review. These trials were performed in the United States from 1977 to 1994 and included women with PPROM between 26 and 36 weeks of gestation. These trials were variable as to their inclusion criteria and also with respect to their co-interventions. A number of these trials did not incorporate what has now become accepted best practice for the management of PPROM, such as the routine use of prophylactic antibiotics. In addition, these trials differed as to how they defined early delivery, with early delivery occurring anywhere from 24 to 72 hours after PPROM had occurred.

There was no difference in the primary outcomes of neonatal sepsis (risk ratio [RR] 1.33, 95% confidence interval [CI] 0.72 to 2.47) or respiratory distress (RR 0.98, 95% CI 0.74 to 1.29). Early delivery did increase the incidence of caesarean section (RR 1.51, 95% CI 1.08 to 2.10).

In assessing secondary outcomes, there was no difference in the overall perinatal mortality (RR 0.98, 95% Cl 0.41 to 2.36), intrauterine deaths (RR 0.26, 95% Cl 0.04 to 1.52) or neonatal deaths (RR 1.59, 95% Cl 0.61 to 4.16) when comparing early delivery with expectant management. There was no significant difference in measures of neonatal morbidity, including cerebroventricular haemorrhage (RR 1.90 95% Cl 0.52 to 6.92), necrotising enterocolitis (RR 0.58, 95% Cl 0.08 to 4.08), or duration of neonatal hospitalisation (mean difference [MD] -0.33 days, 95% Cl -1.06 to 0.40 days). In assessing maternal outcomes, we found that early delivery increased endometritis (RR 2.32, 95% Cl 1.33 to 4.07), but that early delivery had no effect on chorioamnionitis (RR 0.44, 95% Cl 0.17 to 1.14). There was a significant reduction of early delivery on the duration of maternal hospital stay (MD -1.13 days, 95% Cl -1.75 to -0.51 days). If the delivery was planned for less than 24 hours after PPROM, there was a decrease in the rate of chorioamnionitis and a shorter hospital stay for the mother. 24

The PPROMT (preterm prelabour rupture of the membranes close to term) study is currently being undertaken to address this clinically important decision as to the timing of delivery in women with PPROM. In this randomised controlled trial, women with PPROM between 34 and 36+6 weeks gestation are randomised either to early delivery, ideally within 24 hours of PPROM, or to expectant management. The primary outcome of this trial is the incidence of neonatal sepsis. Currently, 801 of the required 1812 women have been randomised to PPROMT.

Conclusion

In assessing the evidence, a best practice approach to the management of women with PPROM in the third trimester would include a number of interventions. This would include inpatient monitoring, the use of corticosteroids at gestations less than 34 weeks and the routine use of antibiotics. These antibiotics would include intrapartum treatment with penicillin for group-B streptococcus and prophylactic treatment with erythromycin.

There is some consensus that the management of women with PPROM prior to 34 weeks should be conservative. However, there is no evidence on the optimal timing of delivery for women with PPROM at gestations greater than 34 weeks. The PPROMT trial is currently recruiting and will provide evidence on this important clinical question.

- 1. Arias F, Tomich P. Etiology and outcome of low birthweight and preterm infants. *Obstet Gynecol Clin North Am.* 1982; 338:663-70.
- Lee T, Silver H. Etiology and epidemiology of preterm premature rupture of the membranes. *Clinics in Perinatology*. 2001;28(4):721-34.
- Mercer BM. Preterm premature rupture of the membranes: current approaches to evaluation and management. Obstet Gynecol Clin North Am. 2005 Sep;32(3):411-28.
- Carlan SJ, O'Brien WF, Parsons MT, Lense JJ. Preterm premature rupture of membranes: a randomized study of home versus hospital management. *Obstet Gynecol.* 1993 Jan;81(1):61-4.
- King J, Flenady V, Papatsonis D, Dekker G, Carbonne B. Calcium channel blockers for inhibiting preterm labour. *Cochrane Database Syst Rev.* [Art. No.: CD002255. DOI: 10.1002/14651858. CD002255.]. 2003(1).
- Christensen KK, Ingemarsson I, Leideman T, Solum T, Svenningsen N. Effect of ritodrine on labor after premature rupture of the membranes. Obstet Gynecol. 1980 Feb;55(2):187-90.
- Levy DL, Warsof SL. Oral ritodrine and preterm premature rupture of membranes. *Obstet Gynecol.* 1985 Nov;66(5):621-3.
- Roberts D, Dalziel S. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. *Cochrane Database Syst Rev.* 2006(3).
- Harding JE, Pang J, Knight DB, Liggins GC. Do antenatal corticosteroids help in the setting of preterm rupture of membranes? *Am J Obstet Gynecol.* 2001 Jan;184(2):131-9.
- Lewis DF, Brody K, Edwards MS, Brouillette RM, Burlison S, London SN. Preterm premature ruptured membranes: a randomized trial of steroids after treatment with antibiotics. *Obstet Gynecol.* 1996;88(5):801-5.
- Roberts D, Dalziel S. Antenatal corticosteroids for accelerating fetal lung maturation for women at risk of preterm birth. *Cochrane Database Syst Rev.* [Art. No.: CD004454. DOI: 10.1002/14651858. CD004454.pub2]. 2006(Issue 3).
- Kenyon S, Boulvain M, Neilson J. Antibiotics for preterm rupture of membranes. *Cochrane Database Syst Rev.* 2003(2).
- Kenyon SL, Taylor DJ, Tarnow-Mordi W, Group OC. Broad-spectrum antibiotics for preterm, prelabour rupture of fetal membranes: the ORACLE I randomised trial. ORACLE Collaborative Group.[Erratum appears in Lancet 2001 Jul 14;358(9276):156]. *Lancet*. 2001 Mar 31;357(9261):979-88.

Lightening: The third trimester of pregnancy

- Kenyon S, Pike K, Jones D, Brocklehurst P, Marlow N, Salt A, *et al.* Childhood outcomes after prescription of antibiotics to pregnant women with preterm rupture of the membranes: 7-year follow-up of the ORACLE I trial. *Lancet.* 2008;372:1310-8.
- Schrag S, Gorwitz R, Fultz-Butts K, Schuchat A. Prevention of perinatal group B streptococcal disease. Revised guidelines from CDC. MMWR Recomm Rep. 2002 Aug 16;51(RR-11):1-22.
- Schrag S, Zell E, Lynfield R. A population-based comparison of strategies to prevent early-onset group B streptococcal disease in neonates. *New England Journal of Medicine*. 2002;347:233-9.
- Gibbs RS, Schrag S, Schuchat A. Perinatal infections due to group B streptococci. Obstet Gynecol. 2004 Nov;104(5 Pt 1):1062-76.
- Yoon BH, Jun JK, Park KH, Syn HC, Gomez R, Romero R. Serum C-reactive protein, white blood cell count, and amniotic fluid white blood cell count in women with preterm premature rupture of membranes. *Obstet Gynecol.* 1996 Dec;88(6):1034-40.
- Hillier S, Martius J, Krohn M, Kiviat N, Holmes K, Escenback D. A case-control study of chorioamnionitic infection and histologic chorioamnionitis in prematurity. *New England Journal of Medicine*. 1988;1988(319):972-8.
- Yoon B, Park C, Chaiworapongsa T. Intrauterine infection and the development of cerebral palsy. BJOG. 2003 Apr;110 Supp 20:124-7.
- 21. Wu YW. Systematic review of chorioamnionitis and cerebral palsy. Ment Retard Dev Disabil Res Rev. 2002;8(1):25-9.
- Wu YW, Colford JM, Jr. Chorioamnionitis as a risk factor for cerebral palsy: A meta-analysis. JAMA. 2000 Sep 20;284(11):1417-24.
- Wu YW, Escobar GJ, Grether JK, Croen LA, Greene JD, Newman TB. Chorioamnionitis and cerebral palsy in term and near-term infants. JAMA. 2003 Nov 26;290(20):2677-84.
- Buchanan S, Crowther C, KM. L, P. M, Morris J. Planned early birth versus expectant management for women with preterm prelabour rupture of membranes prior to 37 weeks' gestation for improving pregnancy outcome. *Cochrane Database of Systematic Reviews*. [Art. No.: CD004735. DOI: 10.1002/14651858.CD004735.pub3.]. 2010(Issue 3).

Vol 12 No 2 Winter 2010 37

Magnesium sulphate in women at risk of preterm birth for neuroprotection of the fetus

A/Prof Susan Walker FRANZCOG CMFM

Cerebral palsy and preterm birth

Cerebral palsy (CP) is a group of disorders characterised by motor and/or postural dysfunction of a non-progressive nature commonly associated with cognitive impairment. The prevalence of CP is 2/1000 live births¹, with principal obstetric risk factors being preterm birth (particularly less than 34/40) and very low birth weight (less than 1500g).² Obstetric contributors include chorioamnionitis, antepartum haemorrhage, complications of multiple pregnancy, placental insufficiency and, less commonly, perinatal asphyxia. Neonatal intraventricular haemorrhage and periventricular leucomalacia are risk factors for CP and are inversely associated with gestational age. CP is a continuing major disability and carries a huge emotional and financial cost to the patient, their family and the wider community. The cost of CP to the Australian community is A\$3.87 billion per annum.³ To date, there have been limited antenatal strategies to prevent this devastating outcome, but encouraging results from several large and well-designed trials^{4,5,6,7,8} and subsequent meta-analyses^{9,10,11} confirm that administration of magnesium sulphate improves the neurodevelopmental future of fetuses destined to deliver preterm.

Magnesium sulphate: How does it work?

The exact mechanism of fetal neuroprotection is not fully understood, but the following purported pathways have been supported by animal data and have biological credibility:

- Magnesium sulphate reduces neuronal injury by 'down regulation' of excitatory stimuli. Damaged neurons are sensitive to the excitatory neurotransmitter glutamate, but the blocking of N-methyl-D-aspartate (NMDA) receptors by magnesium prevents the influx of calcium that causes cell death.¹²
- 2. The vasoactive properties of magnesium may result in increased cerebral blood flow due to cerebral vasodilatation, thus minimising hypoxic-ischaemic damage.
- In an inflammatory model of preterm birth inducing proinflammatory cytokines, magnesium sulphate has been shown to prevent neuronal injury.¹³
- 4. Magnesium may have anti-apoptotic (programmed cell death) effects, thus directly reducing neuronal loss.¹⁴

Given the varied pathophysiologies associated with preterm birth, it is likely that one or more of these mechanisms may be responsible for neuroprotection in any given clinical circumstance.

The current state of the evidence

As early as 1992, Kuban first reported that administration of magnesium sulphate was associated with a reduction in intraventricular haemorrhage (IVH) from 18.9 per cent to 4.4 per cent in babies under 1500g.¹⁵ The case control study of Nelson and colleagues confirmed a reduction in CP (odds ratio 0.14) among infants under 1500g if the mother had received magnesium sulphate in labour.¹⁶ These observations, together with the animal data referred to above, were the basis of a series of prospective interventional trials to evaluate the impact of magnesium sulphate on CP.

The five trials examining the impact of magnesium sulphate on CP have been the subject of a *Cochrane* review¹⁷ and three metaanalyses in the last 12 months.^{9,10,11} Four of these trials were given specifically with 'neuroprotective intent'^{4,5,6,7} and one⁸ was not. The individual trial characteristics are summarised in Table 1. The *Cochrane* and three other systematic reviews have all come to similar conclusions and so the data from the *Cochrane* review is presented here.¹⁷

The objectives of the *Cochrane* review were to assess the efficacy and safety of magnesium sulphate as a fetal neuroprotective agent for women at risk of preterm birth. The review encompassed all five trials, thereby involving 6145 pregnancies in which women at risk of preterm birth at less than 34 weeks gestation were randomised to either magnesium sulphate or placebo. A further subgroup analysis was performed involving those trials which had used magnesium sulphate for 'neuroprotection intention only'. This involved four trials and 4446 children, having excluded the MAGPIE Trial and a subset of patients receiving magnesium sulphate for tocolysis in MagNET.

i. Paediatric outcomes

The primary outcome (paediatric) was the composite outcome of death, severe neurological impairment (cerebral palsy, neurodevelopmental delay, blindness and deafness), or both.

1. Mortality

Antenatal magnesium sulphate had no overall significant effect on mortality (fetal, neonatal and later) (RR 1.04; 95% CI 0.92-1.17). This was an important negative finding since concerns were raised from one of the earlier trials that the reduction in CP may have been achieved at the expense of increased death rates in the magnesium sulphate group. The finding was unaltered when confined to those where magnesium sulphate was given specifically for neuroprotective intent.

2. Cerebral palsy

Magnesium sulphate significantly reduced the risk of cerebral palsy (RR 0.68; 95% CI 0.54-0.87) and this remained significant when only the four trials in which magnesium with neuroprotective intent were considered (RR 0.71; 95% CI 0.55-0.91). A similar magnitude of risk reduction was seen for moderate to severe cerebral palsy (RR 0.64; 95% CI 0.44-0.92) and substantial gross motor dysfunction (RR 0.61; 95% CI 0.44-0.85).

Combined mortality and neurological outcomes

Although there was no significant effect for the composite outcome when all five trials were included (RR 0.94; 95% CI 0.78-1.12), there was a significant reduction among those given magnesium with neuroprotective intent (RR 0.85; 95% CI 0.74-0.98).

How do these numbers translate into absolute risk reduction?

The 'numbers needed to treat' (NNT) will rise in parallel with increasing gestational age, given the reducing incidence of cerebral palsy with advancing gestation. In these trials, the absolute risk of cerebral palsy was 3.7 per cent among those treated and 5.4 per cent in those receiving the placebo. This relative risk reduction (31 per cent) translates to an absolute risk reduction of 1.7 per cent and therefore the number of women needed to treat to benefit one baby was 63. Among those infants delivered at less than 28 weeks gestation, where the background incidence of cerebral palsy is much higher, the NNT was only 29. It should be noted that both of these NNTs compare favourably with the approximately 70 women with preeclampsia who need to be treated to prevent one eclamptic fit.¹⁸

ii. Maternal outcomes

There were no significant differences observed for the major maternal outcomes of death (RR 1.25; 95% CI 0.51-3.07), cardiac arrest (RR 0.34; 95% CI 0.04-3.26) or respiratory arrest (RR 1.02; 95% CI 0.06-16.25). Significantly more women ceased therapy in the magnesium group (RR 3.26; 95% CI 2.46-3.51). Of secondary maternal outcomes, magnesium therapy was associated with significantly more hypotension (RR 1.51; 95% CI 1.09-2.09) and tachycardia (RR 1.53, 95% CI 1.03-2.29). There were no differences seen in rates of maternal respiratory depression, postpartum haemorrhage or caesarean delivery.

National guideline for magnesium sulphate for neuroprotection

In response to these findings, the Antenatal Magnesium Sulphate for Neuroprotection Guideline Development Panel convened at the end of 2009. The panel has now released the National Clinical Practice Guidelines for Antenatal Magnesium Sulphate Prior to Preterm Birth for Neuroprotection of the Fetus, Infant and Child.³ These guidelines can be accessed at: www.adelaide.edu.au/arch/ antenatalMagnesium_SulphateGuidelines.pdf.

The summary of the recommendations are provided in Table 2 on page 40. In essence, where delivery is planned or definitely expected within 24 hours at less than 30 weeks gestation, it is recommended that magnesium sulphate be administered for neuroprotection of the fetus or neonate. Despite the strength of these recommendations, there remain some practical uncertainties. The 'good practice points' accompanying these recommendations attempt to address some of the common dilemmas:

a. Why stop at 30 weeks given that some of the trials continued up to 34 weeks?

Although many of the studies addressed neuroprotection up to and including 34 weeks gestation, the decision to recommend 30 weeks was to some extent a pragmatic one. It was acknowledged that there are significant resource constraints in many maternity care settings. In addition, a more liberal upper limit of gestation would increase the absolute numbers of pregnant women who would be administered magnesium sulphate under

the guideline recommendations, and also an increased number needed to treat to prevent each case of cerebral palsy. Further trials to guide gestational age limits for magnesium sulphate are being considered and will hopefully provide better information in this area.

Continued on page 40.

Table 1.

Trial characteristics of the five randomised controlled trials contributing to the meta-analysis of Doyle et al.

				-			
Study	Gestation	Inclusion	Exclusion	Women	Fetuses	Regimen	Follow-up
Mittendorf ⁴ (MagNET)*	25-33 weeks	Singleton or twins, preterm labour.	Non-reassuring fetal status, infection or preeclampsia	149	165	4g load only (neuroprotection arm)	18 months
Crowther⁵ (ACTOMgSO4)*	< 30 weeks	Singleton or twins. Delivery expected within 24 hours.	Second stage of labour, received magnesium sulphate this pregnancy.	1062	1255	4g load; 1g/hour maintenance	24 months
Marret ⁶ (PREMAG)*	< 33 weeks	Singleton, twins, triplets. Delivery expected within 24 hours.	Fetal abnormality, emergency caesarean delivery, contraindication to magnesium.	573	688	4g load only	24 months
Rouse ⁷ (BEAM)*	24-31 weeks	Singleton or twins. High risk for preterm delivery, preterm PROM, preterm labour (4-8cm), indicated delivery.	Delivery anticipated < 2 hours, major abnormality, PROM < 22 weeks, hypertension, preeclampsia.	2241	2444	6g load; 2g/hour maintenance	24 months
Duley ⁸ (MAGPIE)	All gestation	Preeclampsia and uncertainty whether magnesium sulphate indicated.	Myasthenia, hepatic coma, Cl to magnesium.	1544	1593	4g load; 1g/hour maintenance, OR 5g IM four-hourly	18 months

*Magnesium sulphate was given in the first four of these trials with specific neuroprotective intent, although MagNET had both a neuroprotective and tocolytic arm.

#MAGPIE was designed to prevent eclampsia, not for neuroprotection, but data was provided from MAGPIE regarding women at less than 37 weeks to contribute to the meta-analysis.

b. What is the minimum necessary time of exposure for the fetus to receive benefit?

It is unclear how much time the fetus needs exposure to magnesium sulphate to receive benefit. Magnesium freely crosses the placenta and can be detected in fetal serum within one hour of maternal administration and in the amniotic fluid within three hours.¹⁹ From the proposed mechanisms of action outlined above, it would seem that several hours would be optimal and the guideline pragmatically recommends that magnesium ideally be given for four hours prior to planned or anticipated preterm birth. Nevertheless, there will be many occasions where delivery is expected **within** four hours. Under these circumstances, magnesium should still be given as there is still likely to be benefit.

c. What about urgent delivery?

Where there is maternal or fetal compromise necessitating urgent delivery, delivery should **not** be deferred for administration of magnesium sulphate.

d. Should magnesium be repeated if delivery does not occur and there is a further episode where delivery under 30 weeks appears imminent?

Although immediate repeat doses are not recommended, it is left to the clinician's discretion whether to administer a further course when a recurrent presentation occurs, indicating that preterm birth is again planned or expected at less than 30 weeks.

e. Many of these patients will also be receiving nifedipine for tocolysis. Is it safe to give both?

Despite the potential for interaction between magnesium and nifedipine in terms of neuromuscular blockade and hypotension, this is rarely reported in practice. Nevertheless, maternal observations as recommended in individual units' protocols for magnesium sulphate should be adhered to. If hypotension develops, both the magnesium sulphate and nifedipine should be ceased and medical review instituted.

What's next?

Despite the strength of the data presented for magnesium sulphate in fetal neuroprotection, there remain significant gaps in our knowledge and future research priorities have been indentified in the guideline. Some clinicians remain sceptical regarding the validity of the findings (given the heterogeneity of the trials [see Table 1] and the lack of statistical significance for their apriori primary outcome [death or disability]) and point to the need for further confirmatory data. This is a huge undertaking. For example, a further randomised controlled trial to confirm a reduction in moderate to severe cerebral palsy (assuming an incidence of CP of 3.5 per cent, 30 per cent risk reduction with magnesium sulphate and 80 per cent power, two-tailed alpha 0.05) would require 8000 women at less than 32 weeks gestation and 100 per cent follow-up. Enrolment of 2241 mothers in the trial of Rouse *et al* took ten years and cost US\$25 million!²⁰ This highlights the difficulty in adequate powering for a trial with rare but important outcomes.

Nevertheless, individual patient meta-analysis from the existing trials may give further insights into subgroups most likely to benefit and optimal administration regimens. Long-term follow-up of the trial participants is essential to ensure that findings at two years of age translate into improvements in long-term health outcomes in later childhood. Audit of implementation of this guideline is imperative and should be considered at a national level. Basic scientific research into the origins of preterm brain injury continues to inform future clinical avenues for fetal neuroprotection.

Conclusion

Cerebral palsy is a major contributor to childhood morbidity, for which there is no known cure. As such, efforts must continue to focus on primary prevention. Significant gaps in our knowledge undoubtedly remain, but currently available evidence suggests that administration of magnesium sulphate prior to anticipated preterm birth minimises the risk of cerebral palsy among surviving infants. The American Congress of Obstetricians and Gynaecologists (ACOG) Committee opinion released in March 2010 concurs²¹ and recommends that clinicians electing to use magnesium sulphate for fetal neuroprotection develop guidelines surrounding its' use, informed by the recent large trials.

The Antenatal Magnesium Sulphate for Neuroprotection Guideline Development Panel goes one step further. There is now a readily accessible document summarising the research thus far and an evidence-based clinical practice guideline to assist clinicians and institutions with implementation. Uptake of this guideline will be further enhanced by the already widespread knowledge and experience with magnesium sulphate in the setting of preeclampsia and awareness of the monitoring necessary to ensure maternal and fetal safety. In the future, individual patient meta-analysis of the existing trials may better inform us whether there are particular patient subgroups more likely to experience benefit and further clarify optimal timing and administration. Nevertheless, it is expected that broad implementation of the published guideline will reduce the burden of this devastating illness on those babies delivered preterm, their families and the community.

Table 2. Summary of clinical recommendations.

Clini	ical recommendations	Grade of recommendation
In wo birth, neure	omen at risk of early preterm* imminent# use magnesium sulphate for oprotection of the fetus, infant and child:	A
*Whe	en gestational age is less than 30 weeks.	В
#Whe defin is plc close	en early preterm birth is planned or itely expected within 24 hours. (When birth inned, commence magnesium sulphate as to four hours before birth as possible.)	A
•	Intravenously with a 4g loading dose (slowly over 20-30 minutes) and 1g per hour maintenance dose via IV route, with no immediate repeat doses. Continue regimen until birth or for 24 hours, whichever comes first.	С
•	Regardless of plurality (number of babies in utero).	В
•	Regardless of the reason women (at less than 30 weeks gestation) are considered to be at risk of preterm birth.	В
•	Regardless of parity (number of previous births for the woman).	В
•	Regardless of anticipated mode of birth	В
•	Whether or not corticosteroids have been given.	В

- 1. Stanley FJ. Trends in perinatal mortality and cerebral palsy in Western Australia, 1967 to 1985. *BMJ* 1992: 304: 1658-63.
- Himpens E, Van der Broeck C, Oostra A, Calders P, Vanhaesebrouck P. Prevalance, type, distribution and severity of cerebral palsy in relation to gestational age: a meta-analytic review. *Dev Med Child Neurol.* 2008 May; 50(5):334-40. Epub 2008 Mar 18.
- The Anteratal Magnesium Sulphate for Neuroprotection Guideline Development Panel. Antenatal Magnesium Sulphate Prior to Preterm Birth for Neuroprotection of the Fetus, Infant and Child: National Clinical Practice Guidelines 2010 Feb. Available at: www.adelaide. edu.au/arch/antenatalMagnesium SulphateGuidelines.pdf.
- Mittendorf R, Dambrosia J, Pryde PG, et al. Association between the use of magnesium sulfate in preterm labour and adverse health outcomes in infants. Am J Obstet Gynecol. 2002; 186: 1111-8.
- Crowther CA, Hiller JE, Doyle LW, Haslam RR (ActoMgSO4 Collaborative group). Effect of magnesium sulphate given for neuroprotection before preterm birth: a randomised controlled trial. JAMA 2003; 290:2669-2676.
- Marret S, Marpeau L, Zupan-Simunek V, *et al* on behalf of the PREMA Trial Group. Magnesium sulphate given before very preterm birth to protect infant brain: the randomised controlled PREMAG trial. *BJOG* 2007; 114:310-318.
- Rouse DJ, Hirtz DG, Thorn E, *et al* for the Eunice Kennedy Shriver NICHD Maternal-Fetal Medicine Units Network. A randomised, controlled trial of magnesium sulphate for the prevention of cerebral palsy. *NEJM* 2008; 359: 895-905.
- Magpie Trial Follow-up Study Collaborative Group. The Magpie Trial: a randomised trial comparing magnesium sulphate with placebo for preeclampsia. Outcome for children at 18 months. *BJOG* 2007; 114: 289-99.
- Conde-Agudelo A, Romero R. Antenatal magnesium sulfate for the prevention of cerebral palsy in preterm infants less than 34 weeks' gestation: a systematic review and metaanalysis. *Am J Obstet Gynecol.* 2009 Jun; 200(6):595-609.

- Costantine MM, Weiner SJ, Shriver EK (NICHD). Maternal-Fetal Medicine Units Network (MFMU). Effects of antenatal exposure to magnesium sulfate on neuroprotection and mortality in preterm infants:a meta-analysis. *Obstet Gynecol.* 2009;114:354-64.
- Doyle LW, Crowther CA, Middleton P, et al. Antenatal magnesium sulfate and neurologic outcome in preterm infants – a systematic review. Obstet Gynecol. 2009;113:1327-33.
- McDonald JW, Silverstein FS, Johnston MV. Magnesium reduces N-methy-D-aspartate (NMDA) – mediated brain injury in perinatal rats. *Neurosci Lett.* 1990 Feb 5;109(1-2):234-8.
- Burd I, Breen K, Friedman A, et al. Magnesium sulfate reduces inflammation-associated brain injury in fetal mice. Am J Obstet Gynecol. 2010;202:292.e1-9.
- Turkyilmaz C, Turkyilmaz Z, Atalay Y, Soylemezoglu F, Celasun B. Magnesium pre-treatment reduces neuronal apoptosis in newborn rats in hypoxia-ischemia. *Brain Res.* 2002 Nov 15;955(1-2):133-7.
- Kuban KC, Leviton A, Pagano M, Fenton T, Strassfeld R, Wolff M. Maternal toxemia is associated with reduced incidence of germinal matrix hemorrhage in premature babies. *J Child Neurol.* 1992 Jan;7(1):70-6.
- Nelson KB, Grether JK. Can magnesium sulphate reduce the risk of cerebral palsy in very low birth weight infants? *Pediatrics* 1995; 95: 263-9.
- Doyle LW, Crowther CA, Middleton P, Marret S, Rouse D. Magnesium sulphate for women at risk of preterm birth for neuroprotection of the fetus. *Cochrane Database of Systematic Reviews* 2009, Issue 1. Art. No.CD004661. DOI:10.1002/14651858. CD004661.pub3.
- Sibai BM. Diagnosis, prevention and management of eclampsia. Obstet Gynecol. 2005 Feb;105(2):402-10.
- Hallak M, Cotton DB. Transfer of maternally administered magnesium sulphate into the fetal compartment of the rat: assessment of amniotic fluid, blood and brain concentrations. *Am J Obstet Gynecol.* 1993;169: 427-31.
- Rouse DJ. Magnesium sulphate for the prevention of cerebral palsy. *Am J Obstet Gynecol.* 2009; 200: 610-2.
- ACOG, Society for Maternal-Fetal Medicine. Committee Opinion No. 455: Magnesium sulphate before anticipated preterm birth for neuroprotection. *Obstet Gynecol.* 2010 Mar;115(3):669-71.

Benign vulval dermatoses



Dr Catherine Drummond FACD Dermatologist

The vulva is part of the skin and is affected by conditions common to all skin, both inflammatory and neoplastic.

The vulva is of course subject to the unique influences of anatomy, reproductive hormones and microbial flora, in a physical environment which causes friction, heat and sweating, and is subject to a variety of irritants. As a result, the appearance of a vulval dermatosis may not be typical of that dermatosis elsewhere on the body.

Because the vulva is skin, it is useful to enquire about history of dermatological conditions and to examine the rest of the skin, including scalp, mouth and nails. This article will discuss benign

inflammatory dermatological conditions of the vulva: dermatitis, chronic vulvovaginal candidiasis, psoriasis, lichen sclerosus, lichen planus and desquamative inflammatory vaginitis.

Dermatitis

Dermatitis (synonymous with eczema) is either endogenous or exogenous. Endogenous dermatitis is usually atopic dermatitis. These women have a personal and/or family history of eczema, asthma or hayfever. This means a genetic predisposition to dry, sensitive, itchy and easily irritated skin. Exogenous, or contact dermatitis, is either due to irritants or allergens. Irritant dermatitis is caused by chemical or physical injury to the skin barrier. The vulva is an area particularly prone to irritation from multiple sources and irritation is most commonly low-grade and cumulative. Allergic contact dermatitis is an immune-mediated delayed type hypersensitivity. It is less common and diagnosed by patch testing when dermatitis is recalcitrant. Common contact allergens include local anaesthetics, antibiotics, fragrances and preservatives.

Most vulval dermatitis is atopic dermatitis with superimposed irritant contact dermatitis. Dermatitis usually presents with pruritus on the vulval skin. Mucosal involvement manifests as burning and dyspareunia. Clinical features may be very subtle, but include erythema with poorly defined margins; hyperpigmentation and lichenification (thickening of the skin with increased skin markings – the skin's response to scratching and rubbing); hyperkeratosis; fissures; and excoriation. Chronic eczema is also known as lichen simplex (chronicus). Eczema is usually diffuse and bilateral, so if localised hyperkeratotic plaque is present, a biopsy should be performed to exclude lichen sclerosus and vulval intraepithelial neoplasia. If the plaque is erythematous or eroded, consider extramammary Paget's disease. Patch tests should be considered if there is a dermatitis which does not respond to conventional treatment.

General management includes environmental modification to minimise or modify exposure to irritants. A potent topical corticosteroid such as methylprednisolone aceponate 0.1 per cent fatty ointment is used until symptoms resolve, then as required for flares, which is to be expected as dermatitis is a recurrent condition. If recurrences are frequent, maintenance treatment with a weak corticosteroid ointment such as hydrocortisone one per cent daily may be required. An ultrapotent corticosteroid preparation such as betamethasone diproprionate ointment 0.05 per cent (optimised vehicle) may be required for lichenified dermatitis. Reduce the potency as the lichenification improves. Allergic contact dermatitis improves when the causative allergen is avoided.

Psoriasis

Vulval psoriasis is a manifestation of flexural psoriasis and localises in this area because of friction, sweating, heat and scratching. Psoriasis is usually intensely itchy in the vulva. There may be a family history of this disorder. Examination of the nails (pitting, onycholysis), scalp (scaly plaques) and skin (especially over extensor surfaces of elbows and knees) may provide clues. Often, the characteristic features of psoriasis in the vulva are not apparent – scale may be lacking and margins less well-defined. An important diagnostic clue is erythema involving the inguinal folds and extending into the gluteal crease. Fissures are common. Psoriasis never involves mucosa. Diagnosis is usually clinical and biopsy, if performed, may be non-specific. Psoriasis is less responsive to therapy than eczema and often requires maintenance treatment to prevent recurrence. Initial treatment to induce remission consists of a potent topical corticosteroid ointment such as methylprednisolone 0.1 per cent fatty ointment for several weeks. Once improvement occurs, a weak tar preparation such as LPC (liquor picis carbonis) two per cent in aqueous cream is usually effective. Calcipotriol 0.5 per cent ointment may be tolerated once initial improvement is achieved with corticosteroid.

Chronic vulvovaginal candidiasis

This is a disorder affecting a small percentage of women and is distinct from acute candidiasis. Pathogenesis is unknown but probably represents a hypersensitive reaction to the presence of candida organisms, rather than an infection. No defects in cell mediated or antibody immunity, either localised or generalised, have been discovered. Estrogen is a factor, as candidiasis does not occur in premenarchal girls or post-menopausal women unless other risk factors are present, which include hormone replacement therapy, diabetes and antibiotics. Many women are atopic. Symptoms are usually long-standing, predominantly pruritus and burning, with resultant dysuria and dyspareunia. Vaginal discharge is not characteristic. There is often a history of acute thrush, with exacerbation of symptoms premenstrually and after courses of antibiotics. There may have been positive swabs in the past, but not invariably. Appearance ranges from almost normal to significant vaginal erythema and oedema. There is a dermatitis on the surrounding vulval skin, often around the introitus, and on the medial buttocks. Always perform a low vaginal swab on the first presentation of a woman with vulval pruritus. Chronic candidiasis may coexist with other vulval dermatoses. A positive swab is not a diagnostic test – candida species are commensals and may not cause symptoms, while a negative swab does not eliminate the diagnosis, often because many women self-medicate with antifungal creams and tablets. The presence of non-albicans candida may not indicate a pathogen.

A trial of treatment may be the best diagnostic test. Treatment of chronic candidiasis involves an oral antifungal agent, most commonly fluconazole and itraconazole, for long enough to suppress the symptoms and then weaned slowly to avoid recurrence.



Images published with permission from DermNet NZ. (http://dermnetnz.org/)

This usually involves treatment over many weeks to months. Topical antifungal creams are invariably irritating with long-term use. Consideration should be given to contraception, drug interactions and monitoring liver function tests if therapy is prolonged. Fluconazole is given as an induction course: 150mg every three days for two weeks, then 150mg weekly. Itraconazole is prescribed as 100mg daily. Treatment is continued until symptoms are suppressed, then gradually withdrawn until the lowest dose which controls symptoms is reached and continued for six months. Many women relapse after cessation of this regime, requiring ongoing maintenance treatment. Hydrocortisone ointment one per cent is prescribed for the dermatitis on vulval skin for as long as needed. If non-albicans candida are thought to be the cause of symptoms, boric acid pessaries 600mg daily are prescribed until symptoms resolve, then once or twice weekly as maintenance treatment.

Lichen sclerosus

Lichen sclerosus is a chronic inflammatory dermatosis, thought to have an auto-immune pathogenesis, as it may be associated with thyroiditis and vitiligo. It can occur on non-genital skin. It can occur at any age but especially in peri-menopausal and postmenopausal women. It also occurs in premenarchal girls. It usually presents with pruritus but may be asymptomatic. It may also cause dysuria, dyspareunia, constipation and sleep disturbance. It has a characteristic clinical appearance: early with atrophic, pale papules and plaques, later with sclerotic plaques involving the vulva, perineum and perianal skin in a 'figure of eight' distribution. It does not involve the mucosa. It may result in loss of architecture with obliteration of the clitoral hood, resorption and adhesion of the labia minora and introital stenosis. Vitiligo may coexist and be difficult to differentiate. Vitiligo is characterised by complete depigmentation rather than pallor and there is no alteration in skin texture.

Biopsy is often indicated to confirm the diagnosis of lichen sclerosus, as this is a long-term condition which requires suppressive therapy, with a risk of scarring and malignancy. Biopsy is mandatory if there is a unilateral pale hyperkeratotic plaque to exclude vulvar intraepithelial neoplasia (VIN). Risk of development of squamous cell carcinoma is in the order of five to six per cent, but may be reduced by suppressive therapy.

Management consists of induction with ultrapotent topical corticosteroids to relieve symptoms. The potency of the corticosteroid is then reduced. The aim of treatment is to relieve symptoms, normalise appearance, prevent scarring, and, it is hoped, reduce the risk of squamous cell carcinoma. One suggested regime is betamethasone diproprionate 0.05 per cent ointment (optimised vehicle) twice daily for four weeks, then daily for four weeks. If clinical appearance has normalised, slow reduction of topical corticosteroid potency initially to methylprednisolone aceponate 0.1 per cent fatty ointment, then long-term maintenance treatment with hydrocortisone ointment one per cent, is achievable in many women. Ultrapotent corticosteroids can be reintroduced for flares in disease. Maintenance treatment with weak topical corticosteroids may be more appropriate than episodic ultrapotent preparations alone, as disease can recur asymptomatically and progress silently.

Surgery is indicated for division of adhesions. After this, treatment with ultrapotent topical corticosteroids needs to be recommenced immediately post-operatively to prevent adhesions reforming.

Women need to be reviewed at six-monthly intervals to check for disease recurrence, scarring and signs of malignancy – persistent hyperkeratosis, erythematous patches. A biopsy should be performed if there is any suspicion of malignancy.

Lichen planus

Lichen planus is a chronic inflammatory mucocutaneous condition. It is thought to have an immune basis as the histological hallmark is a lymphocytic infiltrate in the upper dermis directed against the basement membrane zone. It is usually idiopathic, although sometimes may be induced by contact allergens or infections such as hepatitis B and C. On keratinized skin of the vulva, it presents as pruritic violaceous erythematous papules and plaques, often with characteristic lacy scale. When it involves vaginal mucosa it causes pain and discharge. Oral mucosal involvement often coexists. This manifests as erythema and erosions on the vaginal mucosa. While speculum examination is ideal to assess the extent of involvement, it is often too painful to perform. Scarring can occur. Histopathology of mucosal lichen planus may not be diagnostic. Lichen planus may be difficult to control and requires oral or ultrapotent topical corticosteroids for suppression, with introduction of steroid-sparing agents once control has been achieved. Topical treatment may be possible with calcineurin inhibitors such as tacrolimus ointment 0.1 per cent (from a compounding pharmacy). Pimecrolimus cream (Elidel cream) often causes intolerable stinging. Systemic therapy may be required and agents used include hydroxychloroquine, actiretin and methotrexate.

Surgical treatment may be required for correction of scarring. If this is performed, it is vital to prevent reformation of scarring and adhesions with oral or ultrapotent corticosteroid ointments in the healing phase.

Desquamative inflammatory vaginitis

This condition may be confused with vaginal lichen planus, as it presents with pain and discharge with erythematous patches on the vaginal mucosa. However, there is no erosion (and therefore no potential for scarring) and erythema may appear glazed and petechial. It does not involve vulval skin. Histopathology is nonspecific, showing mixed inflammatory infiltrate. Most women are perimenopausal. It may be the same condition as Zoon's vulvitis and plasma cell vulvitis. The pathogenesis is unknown but thought to be inflammation due to alteration in vaginal microbial flora. Low vaginal swabs are non-diagnostic and may report polymorphs and gram positive cocci.

This condition responds to anti-inflammatory antibiotics, used intravaginally, such as clindamycin two per cent, mupirocin two per cent, or metronidazole 0.75 per cent creams, used daily for several weeks until symptoms improve and erythema resolves. This condition may be recurrent and maintenance treatment once or twice weekly is often required. Hydrocortisone one per cent ointment may be helpful for symptom relief.

Benign vulval dermatoses are chronic, recurrent disorders, which, like dermatological conditions in general, are not cured but controlled. Patient education to understand this concept and the need for ongoing maintenance treatment is important. A useful website for patients is **www.caredownthere.com.au**, written by a collaboration of practitioners from the fields of dermatology, gynaecology, sexual health, physiotherapy and psychotherapy.

Further information

- 1. Australian and New Zealand Vulvovaginal Society: www.anzvs.org.au .
- Fischer G, Bradford J. The Vulva: A Clinician's Practical Handbook. Family Planning New South Wales 2010.


Journal Club

Had time to read the latest journals? Catch up on some recent O and G research by reading these mini-reviews by Dr Brett Daniels.

Sling procedures for stress incontinence

These two articles focus on Australian experience with stress incontinence procedures, in particular on midurethral slings. Lee and Dwyer use Medicare data

and report that there has been a 75 per cent increase in the number of surgical procedures for stress incontinence performed in Australia between 1994 and 2008, rising to a total of 7000 procedures in 2008. It is noted that the use of Burch colposuspension has decreased greatly in this time, with midurethral slings showing a more than five-fold increase in use since 2000. The authors acknowledge that Medicare data presents only private sector procedures and that there is no adjustment made for changes in patient age or other demographic features. Stav and colleagues sought to identify risk factors which could lead to failure of midurethral slings, with five to 20 per cent of women reported to have persistent or recurrent urinary stress incontinence following a sling procedure. They retrospectively studied 1225 women receiving sling procedures in Melbourne between 1999 and 2007. Seventy-eight per cent were retropubic and 22 per cent were transobturator slings. They reported a subjective cure rate of 85 per cent. Multivariate analysis found that BMI over 25, a diagnosis of urodynamic mixed urinary incontinence, a history of previous incontinence surgery, intrinsic sphincter deficiency, or diabetes mellitus, all significantly increased the risk of sling failure. Women having concomitant prolapse surgery at the time of sling placement had a reduced rate of sling failure. Surgeons may find this study useful in planning surgical management of urinary stress incontinence for their patients.

Lee JK, Dwyer PL. Surgery for stress urinary incontinence in Australia: current trends from Medicare data. *MJA* 2010; 192: 422.

Stav K, Dwyer PL, Rosamilia A, Schierlitz L, Lim YN, Lee J. Risk factors of treatment failure with midurethral sling procedures for women with urinary stress incontinence. *Int Urogynecol J.* 2010; 21: 149-155.

Pregnancy outcome after threatened miscarriage

First trimester bleeding affects about 20 per cent of pregnancies and is a common cause of maternal anxiety and presentation to medical professionals. The authors performed a systematic review of studies of pregnancy outcome following confirmed viability after first trimester bleeding. That is, they included studies in which bleeding had occurred in the first trimester, but the pregnancy continued at least until viability was confirmed. They were able to identify 14 such studies and examined maternal and perinatal outcomes. They found that first trimester bleeding was associated with significantly higher risks of antepartum haemorrhage (both from placenta praevia and of unknown origin), preterm prelabour rupture of membranes, preterm delivery and intrauterine growth restriction. First

Timing of elective caesarean section

The timing of elective caesarean section is a topic on which there is considerable expert agreement that neonatal outcome is better if the delivery is performed after 39 weeks gestation. Wilmink and colleagues add to this literature with a retrospective cohort study of over 20,000 caesarean births. They compared a composite neonatal outcome measure (including intubation, NICU admission, need for respiratory support, sepsis, jaundice, NEC and Apgar scores under 3). They found a rate of 20.6 per cent for babies born before 38 weeks, 12.5 per cent if born between 38+0 to 38+6 weeks and 9.5 per cent for babies delivered after 39 weeks gestation. The corresponding adjusted odds ratios (95 per cent confidence interval) were 2.4 (2.1 to 2.8) and 1.4 (1.2 to 1.5), respectively for babies delivered before 38 and 39 weeks gestation.

It is, however, clear that many elective caesarean sections are performed before 39 weeks gestation. Nicholl and Cattell describe this as an 'evidence-practice gap'. They report a six-month case study in their unit in which changes, especially to the way in which caesarean sections were booked, resulted in the rate of elective caesareans booked before 39 weeks falling from 30 per cent to ten per cent, with there being no admissions to the neonatal nursery of babies born by elective caesarean before 39 weeks during this period. Their study, while small, highlights some of the barriers and possible solutions for implementing evidence-based practice in a clinical setting.

RANZCOG. Timing of Elective Caesarean Section (C-Obs 23). 2009.

Wilmink FA, Hukkelhoven C, Lunshof S, Mol B, van der Post J, Papatsonis D. Neonatal outcome following elective cesarean section beyond 37 weeks of gestation: a 7-year retrospective analysis of a national registry. *Am J Obstet Gynecol.* 2010; 202: 250.1-8.

Nicholl M, Cattell M. Getting evidence into obstetric practice: appropriate timing of elective caesarean section. *Australian Health Review* 2010; 34: 90-92.

trimester bleeding was also associated with a significantly higher risk of perinatal mortality and low birthweight babies. There was no significant relationship between first trimester bleeding and hypertensive disorders of pregnancy, mode of delivery or congenital malformations. Clinicians may find this study useful in counselling women regarding the effects of first trimester bleeding on an ongoing pregnancy.

Saraswat L, Bhattacharya S, Maheshwari A, Bhattacharya S. Maternal and perinatal outcome in women with treatened miscarriage in the first trimester: a systematic review. *BJOG* 2010; 117: 245-257.

Surgical Safety Checklist

Looking for ways to reduce surgical complications? Have you tried the Surgical Safety Checklist?

The checklist (see opposite page) is quick and simple to use and encourages teamwork to make the operating theatre safer.

The checklist is used at three crucial points of a surgical procedure:

- 1. Immediately before the administration of anaesthesia;
- 2. Before the first incision; and
- 3. Before the patient is taken from the operating theatre.

Much like a pilot and the team in the cockpit of an aircraft, the surgeons and theatre staff work through the checklist together, ensuring no preventable errors are about to be made.

By using the Surgical Safety Checklist in the audit process in your hospital, you can reduce surgical complication and accrue PR&CRM points.

Download a copy of the Surgical Safety Checklist from the RANZCOG website: www.ranzcog.edu.au/fellows/prcrmactivities. shtml#SurgicalSafetyChecklist .

Background to the Surgical Safety Checklist

The Surgical Safety Checklist is a World Health Organization (WHO) initiative aimed at reducing mortality rates and the incidence of surgical complications. This checklist was trialed at eight hospitals around the world and was associated with marked improvements in surgical outcomes. Results of the trial were published in *The New England Journal of Medicine*.¹ The authors reported that using the Surgical Safety Checklist led to a fall in post-operative complications rates by 36 per cent on average and death rates fell by a similar amount.

The Australian Federal Health Minister, the Hon Nicola Roxon MP, launched an Australian and New Zealand version designed to meet local clinical conditions. The checklist was developed by the Australian Commission on Safety and Quality in Health Care, in consultation with the Royal Australasian College of Surgeons, working closely with other specialist medical colleges, including the Australian College of Operating Room Nurses and experts in hospital care from the Federal and State Health Departments.

Reference

 Haynes A, et al. A Surgical Safety Checklist to Reduce Morbidity and Mortality in a Global Population. NEJM; Volume 360:491-499 Number 5.

SURGICAL SAFETY CHECKLIST (AUSTRALIA AND NEW ZEALAND EDITION)

Before induction of anaesthesia > > > > Before skin incision > > > > > > > > > Before patient leaves operating room					
SIGN IN	TIME OUT	SIGN OUT			
 PATIENT HAS CONFIRMED IDENTITY SITE PROCEDURE CONSENT SITE MARKED/NOT APPLICABLE ANAESTHESIA SAFETY CHECK COMPLETED 	CONFIRM ALL TEAM MEMBERS HAVE INTRODUCED THEMSELVES BY NAME AND ROLE SURGEON, ANAESTHESIA PROFESSIONAL AND NURSE VERBALLY CONFIRM PATIENT SITE PROCEDURE 	NURSE VERBALLY CONFIRMS WITH THE TEAM: THE NAME OF THE PROCEDURE RECORDED THAT INSTRUMENT, SPONGE, NEEDLE AND OTHER COUNTS ARE CORRECT HOW THE SPECIMEN IS LABELLED (INCLUDING PATIENT NAME)			
PULSE OXIMETER ON PATIENT AND FUNCTIONING DOES PATIENT HAVE A : KNOWN ALLERGY? NO YES DIFFICULT AIRWAY/ASPIRATION RISK? NO YES, AND EQUIPMENT/ASSISTANCE AVAILABLE RISK OF >500ML BLOOD LOSS (7ML/KG IN CHILDREN)? NO YES, AND ADEQUATE INTRAVENOUS ACCESS AND FLUIDS PLANNED PROSTHESIS/SPECIAL EQUIPMENT: IF PROSTHESIS (OR SPECIAL EQUIPMENT) IS TO BE USED IN THEATRE, HAS IT BEEN CHECKED AND CONFIRMED? YES	ANTICIPATED CRITICAL EVENTS ANTICIPATED CRITICAL EVENTS SURGEON REVIEW: WHAT ARE THE CRITICAL OR UNEXPECTED STEPS, OPERATIVE DURATION, ANTICIPATED BLOOD LOSS? ANAESTHESIA TEAM REVIEWS: ARE THERE ANY PATIENT-SPECIFIC CONCERNS? NURSING TEAM REVIEWS: HAS STERILITY (INCLUDING INDICATOR RESULTS) BEEN CONFIRMED? ARE THERE EQUIPMENT ISSUES OR ANY CONCERNS? HAS ANTIBIOTIC PROPHYLAXIS BEEN GIVEN WITHIN THE LAST 60 MINUTES? YES NOT APPLICABLE HAS THROMBOPROPHYLAXIS BEEN ORDERED? YES NOT REQUIRED SESSENTIAL IMAGING DISPLAYED? YES	 (INCLUDING PATIENT NAME) WHETHER THERE ARE ANY EQUIPMENT PROBLEMS TO BE ADDRESSED SURGEON, ANAESTHESIA PROFESSIONAL AND NURSE REVIEW THE KEY CONCERNS FOR RECOVERY AND MANAGEMENT OF THIS PATIENT 			

This checklist is not intended to be comprehensive, additions and modifications to fit local practice are encouraged.

Based on the WHO Surgical Safety Checklist, URL http://www.who.int/patientsafety/safesurgery/en, © World Health Organization 2008 All rights reserved.



Q & a attempts to provide balanced answers to those curly-yet-common questions in obstetrics and gynaecology for the broader O & G Magazine readership including Diplomates, Trainees, medical students and other health professionals.

An 80-year-old woman with a utero-vaginal prolapse has her symptoms well controlled with a ring pessary and regular topical estrogen cream. How often should her pessary be changed? Can you re-use them by simply washing and re-inserting at the same visit?

Dr Lynsey Hayward

FRANZCOG

а

Ring pessaries can be a very effective form of management for women with utero-vaginal prolapse, who are unfit for or do not wish to undergo surgical treatment. Ring pessaries can also be used as an interim measure in younger women completing their families prior to definitive surgery.

How often should a woman's pessary be changed or checked?

At the time of initial insertion, the woman should be assessed to ensure she cannot feel discomfort from the pessary, that it does not descend or displace on straining and that she can void adequately. A second check two to three weeks later is sensible to ensure there are no side effects such as discomfort, de novo stress or urge urinary incontinence, persistent prolapse symptoms or difficulty with defaecation.

Many women are able to be taught to remove, wash and replace ring pessaries. For those women who are unable to do so, they should be reviewed every four to six months, have the ring removed and the vagina checked for ulceration. Cube pessaries used for women with third or fourth degree prolapse apply suction to the vaginal walls and should be removed nightly. Vaginal ulcerations are reported in three to 24 per cent of long-term pessary users^{1,2,3} and there are case reports of ring incarceration and impaction with neglected pessaries^{4,5,6}. Ulceration usually occurs on a background of vaginal atrophy and the use of vaginal estrogen two to three nights a week can be helpful. In the case of ulceration, this regime should be increased to nightly application, with one to two weekly checks to ensure healing is occurring. You may need to consider a temporary removal of the pessary during this time. As patients age, a change (usually reduction) in the size or type of pessary may be necessary. There should be enough space to sweep one finger between the pessary and vaginal walls but not so much that it rotates on straining. A pessary that is too large can increase the risk of ulceration.

Bacterial vaginosis occurs in up to 32 per cent of pessary users. An acidifying vaginal gel used twice weekly can be used to decrease

this risk. Urinary tract infections are reported in up to 13 per cent of pessary users^{1,3,7,8} and women should be warned to look for signs of infection.

Reusing pessaries

The majority of pessaries are made from silicon which is nonporous and so does not absorb bacteria or odours. Washing with soap and water at clinic visits is sufficient but silicon pessaries are autoclavable and can also be sterilised with cidex or alcohol solutions, for example, Silicon pessaries naturally discolour but they only need to be replaced when there are signs of cracking, loss of shape or flexibility. A silicon pessary can last several years. The acrylic pessaries, such as the rigid gellhorn, can be washed and replaced, but should not be autoclaved or soaked in alcohol. They can be sterilised by soaking in cidex. Again, replacement is only necessary when there are visible signs of surface deterioration.

References

- Hanson LA, Schulz JA, Flood CG, et al. Vaginal pessaries in managing women with pelvic organ prolpase and urinary incontinence: patient characteristics and factors contributing to success. Int Urogecol J Pelvic Floor Dysfunct. 2006;17:55.
- Wu V, Farrell SA, Baskett TF, Flowerdew G. A simplified protocol for pessary management. Obstet Gynecol. 1997; 90:990.
- Powers K, lazarou G, Wang A, et al. Pessary use in advanced pelvic organ prolpase. Int Urogyn J Pelvic Floor Dysfunct. 2206; 17:160.
 Mohammed M, Sidra L, Haldipur H, Unuigbe A, Incarcerated
- Mohammed M, Sidra L, Haldipur H, Unuigbe A. Incarcerated appendices epiploicae through the posterior vaginal defect secondary to a ring pessary. J Obstet Gynaecol. 2008; 28(2): 252.
- Sankar A, Aziz A, Trottr P, Fox R. Outpatient management of incarcerated ring pessary: use of orthopaedic bone cutters. J Obstet Gynaecol. 2008; 28(2): 245.
- Frenando RJ, Sultan AH, Thakar R, Jeyanthan K. Management of neglected vaginal ring pessary. Int Urogyn J. 2007; 18 (1): 117.
- Moore KH, Foote A, Burton G, King J. An open study of the bladder neck support prosthesis in genuine stress incontinence. Br J Obstet Gynaecol. 1999;106: 42.
- 8. Alnaif B, Drutz HP. Bacterial vaginosis increases in pessary users. Int Urogynecol J Pelvic Floor Dysfunct. 2000;11: 219.

RANZCOG members are invited to submit questions, tips or interesting cases to Qජ්ය. Please send entries to Qජ්ය @ 0ප්රි Magazine via: (email) ranzcog@ranzcog.edu.au (fax) +61 3 9419 0672 (mail) 254-260 Albert Street, East Melbourne, VIC, Australia 3002

Emergency contraception *We've come a long way...or have we?*



Dr Caroline Harvey DRANZCOG Medical Director Family Planning Queensland

Emergency contraception (EC) has come a long way since the days when Family Planning clinics and other services provided cut-up sections of pink oral contraceptive pill strips and a dose or two of an antiemetic in tiny sealed plastic bags to women who were lucky enough to know about this option.

When evidence demonstrated the superiority of the progestogen-only regimen in 1998¹, these plastic bags were then loaded with 50 minipills – many more pills to swallow but less nausea and vomiting for these women 'in the know'. However, despite current 'over the counter' (OTC) availability of a dedicated product and clear evidence on its safety and efficacy, it is probably underutilised and poorly understood by many in the community and by some health professionals.

EC is defined as a medication or device used to prevent pregnancy after unprotected intercourse (including sexual assault) or after a recognised contraceptive failure. It has alternatively been called postcoital contraception or 'the morning after pill'. These terms are confusing and imply that EC pills can only be taken immediately, which is incorrect. They can be used, while with decreasing efficacy, for up to five days post intercourse.

There are no evidence-based absolute contraindications to hormonal EC except established pregnancy (due to a lack of efficacy rather than specific adverse outcomes) and allergy. Side effects are uncommon with the progestogen-only regimen and hormonal EC can be used more than once in a cycle if required. It will not provide protection for the rest of the cycle, so ongoing contraception should be addressed from the time of administration.²

What is used in Australia?

The oldest method of hormonal EC, the 'Yuzpe' method (named after the Canadian who described it), was introduced in 1974 and consisted of two doses of 100mcg of ethinyl estradiol and 500mcg of levonorgestrel, given 12 hours apart. Only a few countries ever licensed this method, but it was widely used off-label, including in Australia. It is associated with side effects, particularly nausea and vomiting, due to the high estrogen dose and was therefore usually administered with prophylactic antiemetics.

The progestogen method, using levonorgestrel (LNG), was found to be both more effective and associated with less side effects in a WHO study.¹ LNG is administered in two doses of 0.75g 12 hours apart, or a single dose of 1.5mg is equally effective for EC.³ Until 2002, there was no prescribable EC brand in Australia, so LNG EC was given off-licence as two doses of 25 minipills (this was understandably sometimes viewed by women with great trepidation). Postinor-2® became available on prescription in mid 2002 and then was rescheduled in January 2004 as a pharmacy supplied product. Since then, three other brands have been marketed – Levonelle-2®, Norlevo® (both containing two 0.75mg tablets) and more recently Postinor-1® which delivers the 1.5mg as a single tablet. The other available method of EC is insertion of a copper bearing intrauterine device (IUD) within five days of unprotected intercourse (UPSI). While an IUD is highly effective^{4,5} and has the advantage of providing immediate ongoing contraception, insertion needs to be done by a skilled medical practitioner. Historically, services able to provide IUD insertion within this timeframe are very limited in Australia, so this in practice is a rarely used option. It should be noted that insertion of a LNG IUD (Mirena ®) cannot be used as EC as it is not effective for this indication.

Other regimens available elsewhere

The antiprogestin, mifepristone, has been studied as an EC and is used in some countries for this indication. A *Cochrane* review⁵ found mid-dose (25 to 50mg) mifepristone to be superior in efficacy to other hormonal regimes and low-dose (less than 25mg) to be at least as effective as the commonly used LNG 1.5mg regime. Ulipristal, a selective progesterone-receptor modulator, has recently been marketed as EC in Europe. A randomised study comparing ulipristal with LNG as EC, found it to be more effective overall and to have higher effectiveness between 73 and 120 hours after UPSI.⁶

Efficacy

Currently used hormonal methods of EC prevent about 50 to 80 per cent of pregnancies.⁷ Efficacy rates for EC are estimated by comparing the number of pregnancies observed among a large number of women using the EC method to the number of pregnancies that would be expected in an equivalent number of women with the same coital history, but using no contraception, and is expressed as a percentage. The number of 'expected' pregnancies is based on a series of calculations based on numerous assumptions and suffers from the imprecision with which the day of ovulation can be known in any woman.⁸ The generally quoted efficacy rates (see Table 1) have been criticised as an overestimate and several recent investigators have attempted to recalculate the efficacy, suggesting that EC prevents, as a minimum, 50 per cent of pregnancies.⁸ It is known that efficacy for hormonal methods decreases with lengthening interval of administration after intercourse (see Table 2). This is not the case for a copper IUD, which is equally effective any time up to five days post intercourse.

Table 1.

Efficacy rates for emergency contraception methods.

Method	Time between dose and UPSI (hours)	Pregnancy rate %	Prevented pregnancies %
Yuzpe ²⁰	<72	2.0	74
LNG ³	<120	1.6	80
Mifepristone 10mg³	<120	1.3	83
Copper IUD⁴	<120	0.1	99

Table 2. Pregnancy rates relative to timing.°

Time interval between UPSI and EC administration (hours)	Pregnancy rate %
0-12	0.5
13-24	1.5
25-36	1.8
37-48	2.6
49-60	3.1
61-72	4.1

Mechanism of action

Possible reproductive targets for EC include follicular development, ovulation, sperm transport, fertilisation, implantation and corpus luteum function. As sperm are viable in the female reproductive tract for up to five (or sometimes seven) days, while ovum can only be fertilised within 24 hours of ovulation, the mechanism of action most likely differs depending on when hormonal EC is given in relation to the time of intercourse and the time of ovulation.¹⁰ Research has shown that the primary mechanism of action is by the prevention or postponement of ovulation through its effect on the LH surge¹⁰, but that this will work only if given at least two days before ovulation.⁷ The overall biological data overall strongly suggest that the most likely mode of action is thus prefertilisation. This is supported by (and explains) the reducing efficacy rates with greater time interval between coitus and administration described above. That is, the later hormonal EC is given, the more likely it is that the LH surge has already occurred and ovulation will not be prevented. There is no data to support the view that LNG can impair the development of the fertilised embryo or prevent implantation, but any post-fertilisation action cannot be completely excluded. However, it is clear that LNG does not disrupt an established pregnancy, defined as beginning with implantation, and is not considered an abortifacient.¹⁰

Who uses emergency contraception?

Information about the users of EC is conflicting, with some studies showing more users to be young and unmarried, while other studies have found more users to be older and in stable relationships. Similarly, findings as to whether users are at high risk of sexually transmitted infections (STIs) and unwanted pregnancy more generally, have differing findings.¹¹ An Australian study of sexual health clinic clients requesting EC found users were more likely to be a student, to have a regular sexual partner and less likely to have had an STI or previous unplanned pregnancy than controls.¹¹ It is clear that it is not accurate to stereotype the EC user as young, irresponsible and at risk of STIs, which seems to be a common perception.

Wide access to emergency contraception – what happens?

There has been considerable debate internationally over widening access to EC, including the concept of advance provision. From a public health perspective, wider availability has been supported by numerous reproductive and other professional health organisations, as it seems logical that ready access to EC should reduce the number of unplanned pregnancies, along with the rate of abortions. Detractors have voiced concerns that wide access might result in reduced use of regular contraception, encourage irresponsible behaviours and increase STIs. Evidence, however, does not support either of these suggested outcomes. While increased access to EC pills improves use, disappointingly, it has not shown in a systematic review to have a population effect¹², although there are no published population studies in the Australian context.

A *Cochrane* review found advance provision of EC did not reduce pregnancy rates when compared to conventional provision and this ready access did not change the use of regular contraception or sexual behaviours.¹³ Random controlled trials (RCTs) have been consistent with this encouraging finding (that ready access to EC does not negatively impact on sexual and reproductive health behaviours and outcomes), including studies specifically with teenagers.^{14,15} Follow-up at three years from a large trial, where 17,800 women had access to home supplies of EC in Scotland, found that routine use of more effective contraception actually increased amongst these women.

It seems that even when women have ready access to EC, including advance supply, they often don't use it after UPSI, most commonly due to a lack of recognition of the risk of pregnancy or a neglect of the perceived risk.¹³ Of 518 women seeking abortions in a Swedish study, 83 per cent knew of the ready availability of EC, but only 15 had used it to attempt to prevent the current pregnancy.¹⁶ The available data suggest abortion rates have remained unchanged for complex reasons, where women at risk for unintended pregnancy fail to use it when it is indicated.⁸

Barriers to emergency contraception use

Knowledge

Numerous studies have explored the levels of community knowledge about EC, but less is known about the situation in Australia. Two Australian studies found significant EC knowledge gaps amongst tertiary students in Adelaide¹⁷ and Cairns¹⁸, including poor understanding of the recommended timeframe, low levels of knowledge of the current 'over the counter' (OTC) status and misunderstandings about the mechanism of action. Many women also had poor knowledge of fertile times seeking in their cycles meaning they are not well able to assess their pregnancy risk after UPSI.¹⁷ There has been little research in Australia on clinician knowledge, but studies from other countries suggest that clinicians have poorer than expected knowledge about EC. Poorly informed clinicians are unlikely to provide opportunistic education and advance provision to the women who could benefit from this information and opportunity for future access.¹⁹

Provider and cost issues

While OTC supply has the potential to increase access generally at a population level, for individuals, pharmacy EC supply may add some specific barriers. Little has been published in the Australian situation, although it is of concern that more than 20 per cent of tertiary students in the Cairns study felt unable to purchase EC in a pharmacy where they may be recognised.¹⁸ While pharmacies are required to provide a designated private counselling area, in reality, this space may feel less than private to many women seeking EC. While most pharmacists have embraced the Schedule III listing and see EC supply as an extension of their role in the healthcare team, some women report seeking pharmacy supply as being confronting and difficult experience. These perceived barriers are likely to be even greater in secondary age students and marginalised groups. While a prescription is not required for EC, an advance prescription by a medical practitioner (which will then be dispensed by the pharmacist without need for a pharmacy 'supply consultation') is one strategy which could help overcome some perceived barriers for women.

EC is not Pharmaceutical Benefits Scheme (PBS) listed and may sell for upwards of A\$40 in some pharmacies, which may be a disincentive to its use for some women. Doctors can prescribe a PBS listed 30mg progestogen 'minipill' (for example, Microlut®) and advise on the '25 pills and repeat in 12 hours' regimen as used off label pre 2002. For a healthcare or pension card holder, a single script will give two EC treatments for approximately A\$5.

Conclusions

While we have come a long way with EC in Australia in terms of an available OTC dedicated product, there are still significant barriers to its use. While at a population level, international studies have not found that wide availability decreases unplanned pregnancy, it does not result in decreased use of regular methods or behaviour changes which would adversely affect other reproductive or sexual health outcomes. While complex factors unfortunately seem to prevent women taking EC, even when there is ready access, they won't have the chance to even consider its use if they misunderstand it or don't know about it at all. EC is a woman's last opportunity to prevent an unwanted pregnancy. Clinicians in Australia have a responsibility to inform women about emergency contraception and consider the benefits of offering an advance prescription to sexually active women not using a long-acting contraceptive method.

References

- Task Force on Post-ovulatory Methods of Fertility Control. Randomised controlled trial of levonorgestrel versus the Yuzpe regime of combined oral contraceptives for emergency contraception. *Lancet* 1998; 352: 428-433.
- Sexual Health and Family Planning Australia. Contraception: An Australian Clinical Practice Handbook. 2008. SHFPA, Canberra.
- von Hertzen H, Piaggio G, et al. Low dose mifepristone and two regimes of levonorgestrel for emergency contraception: a WHO multicentre randomised trial. *Lancet* 2002; 360:1803-1810.
- 4. Trussell J, Ellertson C. Efficacy of emergency contraception. Topical reviews. *Fertility Control Reviews* 1995; 4:8-11.
- Cheng L, Gulmezoglu A, Piaggio G, Ercurra E, Van Look P. Interventions for emergency contraception. *Cochrane Database of Systematic Reviews*. 2008, Issue 2.
- Glasier A, Cameron S, et al. Ulipristal acetate versus levonorgestrel for emergency contraception: a randomised non inferiority trial and meta analysis. *Lancet* 2010. Online 29 January.
- 7. Baird D. Emergency contraception:how does it work? *Ethics, Biosciences and Life* 2009; Vol 4,No.1.

- Greene M. Emergency Contraception: A Reasonable Personal Choice or a Destructive Societal Influence? *Clinical Pharmacology & Therapeutics* 2008; Vol 38, No.1.
- Piaggio G, von Herzen H, Grimes DA, et al. Timing of emergency contraception with LNG or Yuzpe regimen. Task force on post ovulatory methods of fertility control. *Lancet* 1999; 353:721.
- Allen R and Goldberg A. Emergency Contraception: A Clinical Review. Clinical Obstetrics and Gynaecology 2007; Vol 50, No.4, 927-936.
- Fox J, Weerasinghe D, et al. Emergency contraception: who are the users? International Journal of STD & AIDS 2004; 15,5:309-313.
- Raymond E, Trussel J, Polis CB. Population effect of increased access to emergency contraception pills: a systematic review. *Obstet Gynecol.* 2007; 109:181-188.
- Polis C, Grimes D, et al. Advance provision of emergency contraception for pregnancy prevention. Cochrane Database of Systematic Reviews 2007; Issue 2.
- Ekstrand M, Larsson M, et al. Advance provision of emergency contraceptive pills reduces treatment delay: a randomised controlled trial among Swedish teenage girls. Acta Obstetricia and Gynecologica 2008; 87:354-359.
- Gold M, Wolford J, et al. The effects of advance provision or emergency contraception on adolescent womens sexual and contraceptive behaviours. J Pediatric Adolesc Gynecol. 2004; 17:87-96.
- Aneblom G, Larsson M, et al. Knowledge, use and attitudes towards emergency contraceptive pills among Swedish women presenting for induced abortion. Br J Obstet Gynecol. 2009; 109,155-160.
- Calabretto H. Emergency Contraception knowledge and attitudes in a group of Australian university students. ANZJPH 2009; 33,3:234-238.
- Mohoric-Stare D, de Costa C. Knowledge of emergency contraception amongst tertiary students in far North Queensland. Aust and New Zealand Journal Obstetrics Gynaecology 2009; 49:307-311.
- Broekhuizen F. Emergency contraception, efficacy and public health impact. Current Opinion in Obstetrics and Gynecology 2009; 21:309-312.
- Trussell J, Rodriguez G, Ellertson C. Updated estimates of the effectiveness of the Yuzpe regimen of emergency contraception. *Contraception* 1999; 59, 147-151.

Obligations when dealing with patients under 18 years of age



Dr Sonia Grover FRANZCOG

Being involved in the care of younger, teenage women provokes a mixed response from clinicians, but one that is often mingled with some anxiety.

I suspect this partly reflects their own (embarrassing) recollections of being a teenager; thinking of the hassles of communicating with their own teenage children; knowing that teenagers don't always give you a straight answer; wondering whether you will need to ask the parent to leave the consultation; wondering whether you have the time for what will almost invariably be a longer than normal consult; and finally, that there may be medico-legal issues around decision-making, consent,

confidentiality and mandatory reporting that may impact on the consultation. Medico-legal issues always seem to have the effect of raising ones anxiety – not a good place to start a consultation.

As teenagers are often accompanied by an adult, it is worth ensuring that you immediately establish that the consultation is primarily with the young person – get the young person to introduce you to their accompanying adult or parent. If you actually know the mother already (possibly as she is a patient of yours), try to ensure that you are making the adolescent or young girl the focus of the consultation. In the first few minutes, it is also worth mentioning to the adult that it is usual practice to do some of the consultation with an adolescent without the adult or parent in the room. I often say to the accompanying adult that it is good practice for the young person to do some of this on their own, as having a medical consultation is a new experience. A young person will often talk to me more easily without another adult in the room.

'We know that teenagers actually don't mind about the gender or age of their doctor. What matters is the confidentiality and that the doctor is nonjudgemental."⁴

So why is this important medico-legally? I am not aware of anyone being sued because they didn't ask a parent to leave the room and hence failed to uncover a critical piece of information, but on the other hand, it is our obligation to provide care to the highest possible standards. Failing to ask a question regarding sexual activity (because we didn't want to ask in front of the mother, or asked and got the wrong answer because the mother was there), hence missing the chance to do a screening test for chlamydia in this higher risk population (under 25 years old and sexually active)¹ could in theory leave us responsible for the subsequent pelvic inflammatory disease and tubal obstruction.

Teenagers do need to know that the consultation is confidential $^{\!\!\!2,3}$ with the following exceptions – the young person is at risk from self-

harm, is about to harm someone else, or is being harmed (therefore mandatory reporting). In these circumstances, adolescents will usually give consent for breaking confidentiality, if you take the time to explain why it is necessary, although there can be rare occasions where you need to break confidentiality without their consent. There is very clear evidence that teenagers who have been informed that the consultation is confidential provide a more complete and accurate history.² We know that teenagers actually don't mind about the gender or age of their doctor. What matters is the confidentiality and that the doctor is nonjudgemental.⁴

As an example, a 15-year-old is brought to see you with irregular and painful periods, accompanied by her mother. You have a preliminary discussion, with your questions and body language directing all your attention to the young woman – about home, school and other activities. You then discuss her period problems. (It is often helpful having the mother present, as she will have recorded the dates of the menses and may tell you the number of days of missed school). Then ask the mother to leave. Explain the confidentiality rules. Go back over some of the home, school, activity and friendship-related activities, working from less threatening topics, to the more sensitive ones around sexual activity.⁵ Is she missing school because of period-related problems or are there other issues behind this? Do her friends smoke? Do her friends have partners? Ask these questions before you ask if she smokes or has a partner. Having uncovered that she has been sexually active for the last eight months, that her mother does not know and that she does not want her mother to know, you are faced with wanting to do some further tests and wanting to ensure that she has adequate contraception (or your next consult may well be for the unplanned pregnancy, which will be more difficult than sorting out contraception).

You now have to determine whether the young woman is able to consent for contraception use (therefore, is she 'Gillick competent')⁶ and what the mother will be told. You also need to organise to test for chlamydia. You may well take the opportunity to say that you suspect that her mother may not be so upset and that most parents would prefer to know that their daughters were being sensible and using contraception rather than risk getting pregnant, but if she feels that at this point, she does not want her mother to know, then the confidentiality requirements and your medico-legal obligation is to not break that.

To decide if the teenager is Gillick competent is to decide that she fully understands the treatment proposed. In fact, at the time of the ruling in 1985, one of the judges established guidelines on prescribing contraception to an under 16-year-old (*Fraser Guidelines*). ^{6,7} The UK ruling has been confirmed in Australia. In New Zealand, this has not occurred, but legal opinion suggests that the same principles would also apply given the widespread clinical usage of them.⁸

- To fulfill the *Fraser Guidelines* you will need to:
- Discuss with the young person the benefits of talking the issue over with her parents.

- Establish that the young person can understand the relevant information which means understanding the causal relationships (in this case, between sex, pregnancy and STIs, and the role of contraception and safe sex). To assess this you will need to ask her to explain back to you the information you have discussed.
- Believe that the young person will be having sex with or without contraception.
- Believe that the physical or mental health of the young person will suffer if the request is denied.
- Believe that it is in the young person's best interests to prescribe contraception and to respect her confidentiality.

With this 15-year-old, your assessment is that she does understand the need for contraception, the risks of using the pill and understands how to use it. She does not feel able to discuss this with her mother at the moment, but will think about it. You carefully document your discussion. You organise a urine PCR test for chlamydia and sort out how you will communicate these results without breaking her confidentiality. You agree that you will not tell her mother and that when her mother returns into the room you will say that you have had a discussion regarding options to control the irregular and painful menses and have opted to use the oral contraceptive pill, as this will tackle both the irregular and the painful period problems.

Assessing Gillick competence⁷ is about trying to ensure that the young person understands the risks and complications of a proposed medical treatment. The detail and extent you go to is directly proportionate to the riskiness and complexity of the issue. So Gillick competence for contraception would require less time, information and documentation than consent for a pregnancy termination.

If you undertake a clinical examination, then consenting for this also requires you to explain to her what you would like to do. As a 15-year-old, she has probably never had a genital examination before. She may feel more comfortable if you explain her anatomy to her whilst she holds a hand mirror. If you think an ultrasound is required, make sure that she is aware that this can be a transvaginal procedure, but that this is likely to be appropriate only if she is sexually active and is comfortable with it.⁹ Examinations without consent can be considered assault.

Mandatory reporting is another issue in the care of younger patients. This is relatively straightforward if you uncover a story of abuse or discover examination findings that are suggestive of abuse. Referral to a centre for assessment for child abuse should be made and clarify with that team whether you should report to the relevant authority or whether they will. Although the legislation does vary¹⁰, in all cases, doctors are obliged to report where abuse is suspected. Some areas of potential difficulty exist. If you are seeing a 14-yearold who is sexually active or pregnant with an 18-year-old boyfriend, are you obliged to report? It may be technically illegal, but you are not mandated to report if this is a consenting and equal relationship. If she is a 14 or 15-year-old with a 30-year-old partner, there would have to be serious concerns about whether she is being taken advantage of and thus you would want to ring the relevant department to seek advice. Likewise, a 16-year-old young woman with a mild to moderate intellectual disability, who is in a consenting relationship with a 16-year-old boy, with an equal level of intellectual disability is probably fine. However, the same girl in a relationship with an older male who is not intellectually disabled is likely to represent abuse and hence require reporting.

Yes, your consultation did take longer, but having done a careful consultation, you have avoided potential medico-legal problems and you have hopefully given the teenager a positive experience in terms of managing her reproductive health, contributing to her sense of self-respect and autonomy.

References

- Department of Health and Ageing, Australian Government. National Notifiable Diseases Surveillance System. (Accessed at: www9.health. gov.au/cda/Source/Rpt_5.cfm accessed 30/3/2010).
- Ford CA, Millstein SG, Halpern-Felsher BL, Irwin CEJr. Influence of physician confidentiality assurances on adolescents' willingness to disclose information and seek future health care. A randomized controlled trial. JAMA. 1997;278(12):1029-34.
- Veit FC, Sanci LA, Coffey CM, Young DY, Bowes G. Barriers to effective primary health care for adolescents. *Med J Aust.* 1996;165(3):131-3.
- Farrant B, Watson PD. Health care delivery: perspectives of young people with chronic illness and their parents. J Paediatr Child Health. 2004 Apr;40(4):175-9.
- Cohen E, Mackenzie RG, Yates GL. HEADSS, a psychosocial risk assessment instrument: implications for designing effective intervention programs for runaway youth. J Adolesc Health. 1991;12(7):539-44.
- 6 Gillick case. Gillick vs West Norfolk and Wisbech Area Health Authority (1985) AC 112.
- Wheeler R. Gillick or Fraser? A plea for consistency over competence in children. BMJ. 2006;332:807
- McLean K. Children and competence to consent: Gillick guiding medical treatment in New Zealand. (Accessed at: www.austlii.edu.au/ nz/journals/VUWLRev/2000/31.html#fn1).
- Royal Australasian College of Physicians. Genital examinations in girls and young women: A clinical practice guideline. (Accessed at: www. ranzcog.edu.au/publications/endorsedstatements/RACP_Paediatric_ Policy Vaginal examinations.pdf).
- Australian Institute of Family Studies. Mandatory reporting of child abuse. ISSN 1448-9112 (Access at: www.aifs.gov.au/nch/pubs/ sheets/rs3/rs3.html).



Improving outcomes for women with gynaecological cancer

In October 2009, The Royal Australian and New Zealand College of Obstetricians and Gynaecologists joined forces with Cancer Australia's National Centre for Gynaecological Cancers (NCGC) to develop a National Gynaecological Cancers Service Delivery and Resource Framework (the National Framework).

This National Framework will be used to guide improvements in the care and support of women with gynaecological cancers over the next ten years to ensure that all Australian women with, or at risk of, gynaecological cancer gain access to high quality care wherever they live in Australia.

The National Framework is part of NCGC's broader Gynaecological Cancers Workforce Initiative. This initiative includes the funding of State and Territory projects that will implement elements of the framework locally. It is guided by a Project Working Group, chaired by Dr Gerry Wain, Director of Gynaecological Cancer Services at Westmead Hospital, New South Wales, and a consumer reference group.

The National Framework includes all stages of the patient journey and has drawn on research findings; evidence-based guidelines and national and State/Territory policy directions; consumer and stakeholder consultation with key service providers; and policymakers around Australia. The work culminated in a national workshop held in Melbourne in late April 2010 attended by over 60 participants.

Based on women's needs, the National Framework addresses the issues of specialist and generalist service providers involved in caring for women with gynaecological cancer within and across services and sectors.

Challenges facing the whole gynaecological cancer workforce include:

- The broader healthcare issues, including access and the burdening costs of healthcare
- Increasing gynaecological cancer service demand
- Variation in practices in gynaecological cancer care
- A 'stretched and changing' workforce
- Balancing access, equity and efficiency to ensure the best use of valuable resources.

To address these and other challenges, the National Framework is underpinned by a patient (and family) centred approach to care. The following service delivery elements are identified that make up good practice gynaecological cancer care:

Pathway points

- Reducing risk and finding gynaecological cancer early
- Multidisciplinary management and support during treatment
- Follow-up and survivorship
- Management of recurrence.

Transecting elements

- Women-centred environment
- Supportive care
- Coordinated care
- Continuous quality improvement, clinical trials and research.

Each element identifies a broad objective to guide practice and the required service delivery components of care and supporting systems and processes.

The National Framework then addresses the workforce resources and skills needed to respond to these elements, including the increasing number of gynaecological oncology resources required to meet future service demand.

In order to make the best use of current and future resources, the National Framework challenges the gynaecological oncology workforce to consider its core functions and the potential role of other providers, including gynaecologists and general practitioners, to support women with gynaecological cancer at different points in the pathway.

The National Framework is due to be completed by June 2010. Once finalised, the National Framework will be forwarded to RANZCOG Executive, other professional colleges and State and Territory government health representatives to gain their support.



Supporting women living with gynaecological cancers

Susan Hanson

National Manager National Centre for Gynaecological Cancers Cancer Australia

For the first time, Australian women with a gynaecological cancer will be able to access reliable and current information about their condition from a single point.

The National Centre for Gynaecological Cancers (www. gynaecancercentre.gov.au) now offers easily understandable, accurate information on gynaecological cancers. The website covers cervical, endometrial, fallopian tube, and other uterine cancers, ovarian placental and gestational trophoblastic disease, vaginal and vulval cancers.

The introduction of this online resource is part of the Australian Government's \$5.1 million commitment to continue improving outcomes for women affected by gynaecological cancers, their families and carers. It further aims to support health professionals to provide best practice care through information regarding diagnosis, treatment and care.

Each year, over 4000 Australian women are diagnosed with a gynaecological cancer and 1500 will die of their disease. These women often face an uncertain future, with the added burdens of complex physical and emotional side effects from cancer treatment.

Having access to a consolidated range of quality and reliable information can reduce the burden of cancer and enable all affected to play an active role in decision-making with regards to their treatment, care and support options.

In order to reduce duplication of existing high quality information materials and improve the availability of information from a central source, the National Centre for Gynaecological Cancers developed the resources in consultation with national and international cancer organisations, including Cancer Council Australia, Cancer Council New South Wales and Victoria, the National Cancer Institute, the Gynaecological Cancer Society, National Breast and Ovarian Cancer Centres, Ovarian Cancer Australia and the University of Pennsylvania's online cancer resource OncoLink.

Access easily understandable, accurate patient information on gynaecological cancers at: www.gynaecancercentre.gov.au .

Letter to the Editor

Kjelland's rotational forceps

Of recent times, I have read with interest and concern, O & GMagazine articles related to the obstetric Kjelland's rotational forceps. It is sad to read some of the present opinions of this beautifully designed instrument.

I commenced my training when the caesarean section rate was around five per cent and carried a recognisable maternal mortality. The trilene bottle still hung on the bedhead, rectal avertin was widely used for eclampsia, pudendal block was the only regional anaesthesia available and general anaesthesia was listed as a cause of obstetric maternal death. Prolonged labour, between 12 to 20 plus hours, was not uncommon and as there was no restriction on the hours of employment, one could observe such a prolonged labour throughout its entirety. We were taught: 'Don't let the sun set twice on the progress of labour'. These confines forced us to attempt a successful vaginal delivery. As observation of a prolonged labour extends well beyond the hours of today's employment, a prolonged labour may now never be observed throughout its entirety by one individual. This results in earlier intervention of labour.

The management of the abnormal cephalic presentation, in the main occipito-posterior position, challenged the obstetrician's 'art of obstetrics'. A prolonged labour, cephalic manual rotation and the use of pelvic curve forceps, for example Neville-Barnes axis-traction forceps, often resulted in maternal and neonatal morbidity and sometimes mortality. Kjelland's forceps were rarely used at that time.

With the introduction during labour of intravenous hydration, regional spinal and epidural anaesthesia, and close monitoring of fetal wellbeing, prolonged labour was greatly assisted. This permitted uneventful progress to full dilatation and often resulted in spontaneous rotation to the occipito-anterior position. If rotation had not occurred, the *accoucheur* was faced with a persistent occipito-posterior position or a deep transverse arrest position. The straight Kjelland's rotational forceps were magnificent in this situation, permitting cephalic rotation at the level of pelvic arrest, with then subsequent descent to yield a safe vaginal delivery.

The Kjelland's forceps were one of the best designed surgical instruments we used. The successful use of the Kjelland's forceps was no doubt due to 'adequate supervised teaching' and 'gentle hands'. To correctly rotate the head required no more pressure than required to 'turn a key in a door'. At times, careful slow rotation was assisted when alternated with slow traction descent. If these principles could not be adhered to, then delivery by caesarean section was required.

In my experience, overzealous attempts at vaginal delivery using the Kjelland's forceps were prevented when the procedure was undertaken in the operating theatre, with adequate theatre preparation and staff to proceed immediately to caesarean section.

In summary, an obstetrician who was well-trained in the use of the Kjelland's rotational forceps and operated with gentle, dexterous hands obtained excellent results with this instrument. During my practising obstetric career, I often used the Kjelland's forceps with satisfaction and dexterity to obtain excellent results.

I do not accept oversimplified criticism of this beautifully designed instrument. The demise of the use of the Kjelland's forceps is a loss to 'the art of obstetrics' and must surely lead to an increasing incidence of caesarean section.

Dr Peter Monks FRANZCOG

A life-changing experience in Ethiopia

Dr Maurice Lichter FRANZCOG

After the luxury of 30 years of city practice, I undertook six weeks training in fistula surgery in Ethiopia, in preparation for volunteering in Africa. My wife, Robyn, joined me during the first two weeks at the Addis Ababa Fistula Hospital.

Addis Ababa is a city of vast contrast encompassing shanty housing, streetfront stores and an exciting, bustling market among opulent Western style hotels and restaurants, shadowed by developmentally arrested high rises. Sheep and cattle meander through the crowded streets adding to the hubbub of rusting blue and white taxis and what seems to be most of the 78 million inhabitants of Ethiopia.

The Addis Ababa Fistula Hospital (AAFH) is a haven of splendour with its beautiful trees and shrubs. The Medical Director, Professor Gordon Williams, is originally a urologist from England. His hospital statistics indicate 97 per cent of fistulae are cured, but nearly 50 per cent will have residual urinary incontinence. Since anticholinergic medication has been allowed into the country, control of urge incontinence has improved. Interestingly, male babies seem to cause 84 per cent of fistulae in the first pregnancy and 64 per cent in subsequent pregnancies.

Patients stay for an average of three weeks in the 120-bed hospital. The main ward houses four rows of 12 post-operative beds with no curtains or privacy. Recovering patients, wearing hospital issue nightgowns and distinctive multicolour patchwork blankets, sit in small groups in the sunshine. There are about 97 dialects in Ethiopia making communication a challenge. Despite this, patients nurture each other with grooming and recovery.

'Although few facilities are available for diagnosis and treatment, volunteering in Ethiopia gave me more than I could ever give back. I can thoroughly recommend the experience to reset values.'

With so many post-operative patients, I expected a significant, well-equipped operating facility. However, the one operating theatre contains four operating tables with dubious lighting and sterility. Everything is reused except syringes. Surprisingly, infection almost never occurs and there is a very low complication rate. Spinal anaesthesia is the rule and is overseen by two anaesthetists. I learned the technique as there was no dedicated anaesthetist in outreach centres. I was instructed in fistula repair and fortunately had the opportunity to do ureteric reimplantations.

Two nurses assist each surgeon and another handles the instruments. Nurse aides fetch sutures and instruments, remove bloody swabs from the buckets and manipulate the operating table in order to see the operating site. The limited available equipment is shared between operating sites. The theatre staff work tirelessly and without complaint. I was impressed by their multi-tasking. Since AAFH commenced operating, many of the old myths have been dispelled – single layer closures and closure of the fistula and the vagina in the same direction are often the most effective.

Two teams share post-operative care and management was not necessarily by the operating surgeons. Each team consists of doctors, nurses and nurse aides. One nurse aide is dedicated to the fly swat and the hand cleanser. With nine Ethiopians on the round and with help from patients, we were able to communicate between the many dialects quite effectively.

It is sobering to recognise that these patients are the injured survivors of the ordeal of prolonged, obstructed labour and delivery of a macerated fetus. The vast majority of patients in this predicament die in the peripartum period due to haemorrhage and infection. The Ethiopian maternal mortality rate is 1:27!

A physiotherapist runs group sessions every morning, followed by specific individual instruction. In the afternoon, another group session deals with pelvic floor rehabilitation.

Urodynamics studies are done within days of being ordered using a modern donated machine. A nurse does all the tests, interprets the results and plans appropriate treatment.

The myriad of cases I experienced at AAFH were confronting and often overwhelming. A ten-year-old was accidentally shot, causing a recto-vaginal fistula. A 50-year-old presented with a vesico-vaginal fistula (VVF) she'd had for 20 years because she never knew she could have it fixed. A woman in her mid twenties presented with VVF, having spent seven years in the fetal position hoping to cure it. She had contractures of all her limbs and weighed only 27kg on arrival. She had bilateral foot drop due to ischemia of the peroneal nerves after prolonged squatting.

After almost 12 months of physiotherapy, she was able to straighten her arms and legs, but shortening of her Achilles tendons will probably have to be corrected surgically.

The majority of cases were VVF or recto-vaginal fistula (RVF) and in many cases, both. Most patients were only 18 to 20 years old, some with vaginas so scarred that they have retrograde menstruation and described endometriosis.

Many unusual problems presented. A patient presented with sloughing of the anus and rectum after using a caustic poultice prescribed by a traditional healer to treat haemorrhoids. Another patient required re-catheterisation for urinary obstruction, after bladder augmentation with a loop of bowel revealed urine which looked like thick pea soup. Infection and mucous production blocked the catheter. A 23-year-old presented who had an RVF and VVF after a stillbirth. Most of her anterior vaginal wall had sloughed off leaving a dimple where her vagina was previously. Her negligible

Continued on page 64.



Entrance to the main hospital in Bahir Dar.



View of entrance to the Fistula Centre in Bahir Dar.



Recovering patients sit in the sunshine outside the Addis Ababa Fistula Hospital.

bladder remnant resulted in incontinence, but this was not her main complaint. In Ethiopian society, she is of no use if she is unable to have intercourse or attain pregnancy. Unfortunately, the degree of scarring precluded plastic surgery or the use of prosthetics to stretch the skin.

After my two weeks training at AAFH, I spent the next four weeks with Dr Andrew Browning in Bahir Dar, north-west Ethiopia. Arriving in the dark, I found rolling anything but my ankles on the broken rock was impossible. So, carrying my bag, I negotiated the car park with some difficulty before the short trip to the hospital.

The hospital here is a government institution that services two million local inhabitants. It is spread over a vast area joined by dirt tracks. The paediatric ward comprises several small rooms with three to four beds where children are nursed by their mother. The HIV centre is a new, clean, painted building paid for by donations. Big signs advertise the clinic with no concerns about patient confidentiality or privacy.

The outpatients area consists of a large central area where patients wait on broken benches for one to three days to be seen. The perimeter is punctuated by wooden doors, each housing a different specialty. The gynaecology area, like all these clinics, is one room with a desk, a lamp and a vinyl-covered bed. There are no sheets or curtains. There are paediatric, surgical and medical clinics and a resuscitation room with a smaller desk and a bigger bed – nothing else to be seen. I hope they had everything else hidden away.

The maternity building is a concrete structure with gaps for windows. The walls are raw plaster and the tiled floors are covered in dust from the outside paths. Bleeding patients lie in the corridor on plastic covered stretchers at ground level with relatives attending to their needs.

Labour ward beds are shared by patients who move to the delivery room in the second stage of labour. Four delivery options are available – a half-bed with foot stirrups, a narrow bed, an oldfashioned birthing chair and an old arm chair. A single bar radiator is available to warm cold babies. The Bahir Dar fistula centre is separately funded and in contrast to AAFH, there is no hand sterilising or fly swatter. Prophylactic antibiotics are used routinely and infection is rare. Here incisions are different and the dissection is not as wide. Patients are operated on early and urged to drink immediately, as intravenous fluid is expensive and limited. Catheters are removed earlier here with no ill effects. Dr Browning has developed an operation for stress incontinence which involves urethral plication and suburethral support from surrounding tissue. So far, this easily reproducible operation has a 75 per cent success rate and does not need expensive equipment, which is not available in Ethiopia anyway.

Despite my initiation at the AAFH, I was continually taken aback by confronting cases such as marriage at the age of eight, obstructed labour, a dead baby and a fistula by the age of 18, then, secondary amenorrhoea due to the trauma.

Patients in Bahir Dar receive numeracy and literacy lessons from the staff and learn enough to get by before they leave hospital. Tubal ligation is frequently requested at the time of fistula repair and it is done abdominally in Bahir Dar. After demonstrating vaginal tubal ligation, I was delighted that this became the accepted approach.

I had exposure to many different types of surgery and the opportunity to haggle with taxi drivers and explore the sights of Ethiopia. Although electricity and water are occasionally unavailable and the dial-up internet fails frequently, the overall experience is not to be missed.

The smiling faces of the patients and their appreciation for the help they are given cannot be adequately described. The interaction between the patients is wonderful to see and the effort made by the fistula centre to promote obstetric care in outlying areas will hopefully diminish the need for fistula surgery in the future.

The needs in Ethiopia are many and exposure to western advances is essential. Although few facilities are available for diagnosis and treatment, volunteering in Ethiopia gave me more than I could ever give back. I have learnt to do without many things I believed were essential and I will hopefully return on many occasions.

Fostering links with Fiji

Dr Kirsten Connan

Dr Daniel Jolley

Anaesthetist Royal Women's Hospital Melbourne While that may be true for holidaying among the beautiful beaches of the Mamanuca and Yasawa Islands or on Viti Levu's Coral Coast, life as a medical volunteer in Suva, Fiji, is an equally rewarding but altogether different experience.

From August 2009 to January 2010, we spent six months working and teaching at Fiji's main hospital in Suva and the affiliated Fiji School of Medicine.

Fiji is one of the most populous South Pacific countries, home to almost 900,000 people among the 110 inhabited islands. Despite being a popular tourist destination, poverty is a major challenge for many Fijians. Fiji is placed ninety-second on the Human Development Index and has a GDP per capita of US\$4121. The nation has an infant mortality rate of 16 per 1000 births, four times greater than Australia, and a maternal mortality of 75 per 100,000 births.

The nation's capital, Suva, serves as the centre for medical training and receives tertiary referrals for the entire Fiji Islands. The Fiji School of Medicine (www.fsm.ac.fj) has now been training doctors for Fiji and many other South Pacific nations for the last 150 years. Suva also serves as the largest provider of healthcare in the South Pacific through the Colonial War Memorial Hospital (CWMH). Our role was with both the Fiji School of Medicine as lecturers and as clinicians at CWMH.



Unexpected triplets at 35 weeks gestation. No antenatal care, straight from the boat after a two-day trip to the hospital.

Living in the Fiji Islands normally conjures up idyllic tropical island images of sunshine, endless beaches, crystal clear water, great seafood and a lazy lifestyle.

The CWMH department of obstetrics and gynaecology manages 6500 deliveries each year, all under the supervision of only three very busy fulltime specialists. The labour ward consists of a six-bed 'first stage' open room, with four individual rooms for 'second stage' management. Most days, the labour ward corridor served as an overflow postnatal ward. The antenatal waiting area was equally often overwhelmed, with many women having travelled more than a couple of days to be seen. Due to poor resources, access and limited education, antenatal bookings commonly occur in the third trimester, with many at first presentation when in labour. Maternal medical diseases such as diabetes, rheumatic heart disease, syphilis, tuberculosis and SLE (systemic lupus erythematosus) were a few of the many medical complications that present among the antenatal population. Preterm labour and PPROM were commonplace, with perinatal outcomes universally very poor at gestational ages under 32 weeks. Despite only a ten per cent caesarean section rate, we still saw more cases of ruptured uterus during our sixmonth term than during our postgraduate training. Gynaecological presentations commonly included ruptured ectopics (less than two per cent having ultrasounds in the first trimester); pelvic inflammatory disease; tubo-ovarian abscesses; extremely large fibroid uteruses and ovarian cysts; and most gynaecological cancer presentations at stage 3 or 4. All registrars learned Wertheim's hysterectomies.

Daily work in the O and G department was very busy, with significant service commitments competing with training of medical students, Diploma and Masters of O and G registrars. Nonetheless, the enthusiasm of the postgraduate trainees and the desperate need of many of the patients remained the motivator for the occasional 72-hour on-call consultant shifts. Ruptured uteruses, ruptured ectopics, postpartum haemorrhage, antenatal Eisenmenger's syndrome and complex laparotomies were among the many challenges occurring during on-call shifts. Excellent clinical supervision and collegiate support was always on hand within the O and G department. Sadly, there were six maternal deaths in just our short six-month term.

Anaesthesia offered similar contrasts to practice in Australia. In addition to the O and G theatre workload, CWMH maintains paediatric, general, orthopaedic, plastics, thoracic and urological



The CWMH labour ward desk for handover.

surgical services, as well as emergency neurosurgical management. These demands were covered by only four operating theatres. The anaesthesia department also managed the six-bed ICU, frequently needing to close elective theatres in order to use anaesthesia monitors for ICU patients.

Many theatre patients suffered complex medical problems impacting both their surgery and anaesthesia. Diabetes and coronary artery disease are growing problems in Fiji and among the younger obstetric population, rheumatic heart disease is not uncommon.

Anaesthesia equipment, monitoring and drug shortages were a common problem, sometimes requiring cancellation of elective theatres. Many of the drugs available were those no longer used for anaesthesia in Australia, nonetheless, it was quite satisfying to be able to deliver a safe and effective anaesthetic using only these older agents. The resourcefulness and skill of the local anaesthesia trainees was impressive, working in an environment far outside the comfort zone of the typical Australian anaesthetist.

Anaesthesia involvement in obstetrics was restricted to theatres and ICU. Labour analgesia was essentially unavailable. Although once used in the past, nitrous oxide had now fallen into disuse on the labour ward due to a lack of training and motivation. Additionally, a lack of staffing and facilities made it impossible to offer a spinal or epidural analgesia service.

'The Damocles sword often hanging over Pacific trainees was the looming responsibility of returning once qualified to their native country as the sole obstetriciangynaecologist or anaesthetist for an entire island nation.'

Teaching was both the primary focus and the most satisfying aspect of our time while in Fiji. The Fiji School of Medicine trains both undergraduate and postgraduate doctors for the entire South Pacific. Standards are comparable to Australia and New Zealand for undergraduate teaching, while postgraduate teaching relies heavily on expat doctors from Australia, New Zealand and other countries.

In addition to formal tutorials, assignments and exams, a large component of our teaching was clinical; on the wards, in ICU or in theatre. The Fijian and Pacific trainees had a strong thirst for knowledge. The Damocles sword often hanging over Pacific trainees was the looming responsibility of returning once qualified to their native country as the sole obstetrician-gynaecologist or anaesthetist for an entire island nation.

While resource scarcity is an ongoing challenge for Fiji, much has and can be achieved through further education and training. It is easy to forget that many of the advances in modern medical care are the result of changes in practice, knowledge and skills rather than the result of expensive new drugs or equipment. We plan to regularly return to Fiji to continue this journey, as well as build upon the valuable relationships forged with local doctors and further foster links between medicine in the South Pacific and Australia.

Kirsten and Daniel are returning to Fiji in August 2010 to run Advanced Life Support in Obstetrics (ALSO) and Obstetric Anaesthesia courses respectively.

The delivery rate at CWMH for the first three months of 2010 have seen increasing birth rates, with a rate of 924 in March (projecting to over 10,000 for the year)!

A Launceston odyssey Reflections on a SOLS experience

Dr Wah Hin LeeMy spirit of travel and adventure was not dampened by the bitterly cold wind and
blizzardly rain as I descended on the shores of Launceston, Tasmania.

I was to commence a 30-day position working in the maternity unit at Launceston General Hospital (LGH) as a locum obstetrician. As I have never practised in this part of Australia, I must admit it was with some trepidation that I ventured into this unknown territory, wondering what pitfalls and misadventures could possibly lie ahead of me.

The staff of the Specialist Obstetrician Locum Scheme (SOLS) were excellent in preparing the ground work. Documentation and registration with the Tasmanian Medical Council and liaison with the administration of LGH were very efficiently carried out. The crossing of the T's and dotting of the I's were completed long before my arrival at the hospital.

A late model SUV was made available for me on my first day at LGH. With GPS navigation guiding me, I had no problems finding my way to a fully-serviced townhouse complete with modern conveniences and a fridge well stocked with the basic necessities. With a pleasantly warm heating system during the bitter Tasmanian winter, it did make my settling into Launceston a warm and welcoming one.

My first day in Launceston was nothing but remarkable and exciting. I must admit that I have never met friendlier people. From the moment I stepped into the hospital, there was a feeling of warmth and hospitality by the staff and the department. The labour ward was pleasant, the outpatient clinics friendly and courteous.

The Director, Dr Amanda Dennis, was clearly very much in control and has been running the unit like a good admiral in command. Orientation with a morning cup of tea at the local café started off a very pleasant 30-day tour of duty in Launceston and a thoroughly exciting and professionally rewarding experience. Dr Dennis was most impressive and helpful. The mission of the hospital was made very clear to me and I soon got to understand quite succinctly the hopes, aspirations and visions of the maternity department.

The work was indeed enjoyable. There was a true spirit of camaraderie and while all staff from the director downwards were assigned duties appropriately, everyone chipped in when the need arose, or when there was a situation to render help or professional assistance. Nothing was too hard or too demanding. For me, being rostered only one weekend in four and one day a week, life could only be said to be wonderful. I felt I was somewhat underworked and indeed I was beginning to be concerned if I was earning my keep.

The work schedule left me with plenty of time to wander around and explore some of the beautiful sights and sounds of the Tasmanian wilderness.

Dr Dennis' Chief of Staff was an experienced senior registrar who had been in the United Kingdom for a number of years. He helped manage the unit as second in command and arranged the handover of work and duties on the first day. When I departed on the last day, he was there to pass the baton onto the obstetrician whom I was providing the locum cover for. As the senior registrar who should be well informed of the problem cases in the department, his duty was to identify issues and resolve them either in the clinics, in the labour ward, or in the operating theatres. It is obvious that a competent senior registrar enables the unit to work a lot better and makes the life of a director easier. Certainly from a locum's point of view, it is a godsend.

As I was the charter President of the Rotary Club of Shanghai, I was socially preoccupied with invites from the Rotarian fraternity in Launceston, but perhaps equally important was the fact that I had been well looked after by my medical colleagues. Dr John Grove, no doubt a luminary of the RANZCOG fraternity, had given me an excellent insight into the workings of the College, the who's who and who had done what in the College political hierarchy. It was indeed illuminating. I certainly had a much better insight into the functions of RANZCOG.

Some of the junior medical staff had also been most accommodating and invited me out for meals at the local restaurants and cafes of Launceston.

I was also very impressed by the cordial atmosphere of the mini 'United Nations' at the unit. I have worked in many parts of Australia and I know that most of the remote and rural hospitals have a high percentage of overseas doctors. Launceston is probably not much of an exception. I conducted a ward round one day and in the entourage was the fair dinkum Australian from Tasmania, the Nigerian registrar, a Filipino doctor and a very diminutive junior registrar from Myanmar.

Like all good things, they eventually need to come to an end and I bade farewell to Launceston and Tasmania. I have become wiser in many ways, socially, politically and professionally. I have made new friends, renewed acquaintances and seen a new aspect of professional practice that I can adopt to enrich my professional life. I congratulate Dr Dennis and her staff for running such a fine unit.

For those who are interested in embarking on a career in locum work, SOLS could be your primary contact. The service will assist with the necessary documentation, including medical registration with the respective medical boards and councils. Subsidies for registration fees will be provided by SOLS.

Contact the SOLS Coordinator at RANZCOG for further information about the opportunities that may currently be available in Australia:

(t) +61 3 9412 2912

(e) sols@ranzcog.edu.au (w) www.ranzcog.edu.au/SOLS/index.shtml

College Statements Update March 2010



Michael Permezel FRANZCOG Chair, Women's Health Committee

The Women's Health Committee (WHC) approved four new statements in March 2010, which were subsequently endorsed by Council. New and revised College statements can be viewed on the College website at: www.ranzcog.edu.au/womenshealth/statementsupdate.shtml .

New College Statements C-Obs 35: Prenatal Screening for Fetal Abnormalities

All women need to be counselled with respect to available prenatal screening. In this often complex area of clinical practice, it is essential that all health professionals providing antenatal care have a thorough understanding of the more common problems and the available screening. Many clinicians are concerned that this is increasingly resource intensive and those funding

healthcare have a responsibility to ensure that both the counselling time and screening tests are adequately funded.

C-Obs 31: Routine Intrapartum Care in the Absence of Pregnancy Complications

A number of separate College statements deal with aspects of intrapartum care, including the provision of adequate services (for example, access to theatre), fetal surveillance, intrapartum analgesia and management of the third stage of labour. This statement synthesises key aspects of those statements and considers other important areas of intrapartum management, including assessing the progress of labour and the management of failure to progress. While not prescriptive, the statement provides a broad framework that the College hopes will be useful in formulating institutional guidelines for intrapartum care.

C-Gyn 26: Long-term Health Consequences of PCOS

Dr Digby Nan Kee developed this statement in response to a number of requests to highlight the fact that the long-term consequences of polycystic ovary syndrome (PCOS) extend beyond the obvious immediate reproductive sequelae.

C-Obs 29b: Progesterone: Use in the Second and Third Trimester of Pregnancy for the Prevention of Preterm Birth

The Women's Health Committee is very grateful to Dr Jon Hyett for his assistance in developing a statement on progesterone use for the prevention of preterm birth. The key research studies are summarised with a view to assisting Fellows with management decisions in this contentious and difficult area of clinical practice. Whilst studies will likely inform further (for example, the Progress study), the Women's Health Committee recognises that Fellows still have to make clinical decisions with the knowledge available at this point in time.

Revised College Statements Endorsed Without Significant Changes

• C-Gen 2: Guidelines for Consent and the Provision of Information Regarding Proposed Treatment

- C-Gen 7: Guidelines for Gynaecological Examinations and Procedures
- C-Gen 8: Diethylstilboestrol (DES) Exposure in Utero
- C-Gyn 10: Sterilisation Procedures for Women with an Intellectual Disability
- WPI 11: Obstetrician's Competence and Performance
- WPI 16: Clinical Training Whilst Pregnant

Statements of Other Bodies Endorsed by Council

Home Fetal Heart Monitoring – ANZSA

This statement was developed by the Australian and New Zealand Stillbirth Alliance (ANZSA) in response to a marketing initiative whereby women have been encouraged to undertake their own home fetal heart rate monitoring. This obviously has the potential to lead to misinterpretation and false reassurance and all fetal heart rate monitoring requires clinical oversight.

New Statements Under Development

- Term premature rupture of the membranes (PROM)
- Delivery of the fetus at caesarean section
- Caesarean section performed by non-obstetricians.

Prescriber Status for Mifepristone

RANZCOG has developed an aide package to assist Fellows with their application to the Therapeutic Goods Administration (TGA) to become an authorised prescriber for mifepristone. If you would like a copy of the aide to be emailed to you, please contact RANZCOG on +61384150408.

Want to locum in rural Australia?

Register as a SOLS Locum!

Do you want to: Help your rural colleagues?



For more information: www.ranzcog.edu.au/sols/index.shtml (03) 9412 2912 | sols@ranzcog.edu.au

The Specialist Obstetrician Locum Scheme is funded by the Australian Government

Specialist Obstetrician Locum Scheme

C-Gyn 26: Long Term Health Consequences of PCOS

Date of this document: March 2010 First endorsed by Council: March 2010 Next review due: March 2013

Introduction

Polycystic ovary syndrome (PCOS) is the most common endocrine abnormality in women of reproductive age and may be associated with anovulation, infertility, hyperandrogenism and long-term metabolic sequelae. Insulin resistance is central to this syndrome, driving increased androgen levels and resulting in impaired glucose tolerance and potentially an increase in cardiovascular disease. The prevalence of this condition is estimated at six to seven per cent of the population, or 400,000 Australian women.¹ Obesity aggravates insulin resistance and as obesity in the community increases, the complications from PCOS are expected to rise. Women with PCOS should be fully informed about the long-term health consequences of this condition and advised about how they may reduce their risk.

Diagnosis

The diagnostic criteria for PCOS remain controversial. However, all diagnostic approaches require that secondary causes (adult onset congenital adrenal hyperplasia, hyperprolactinaemia and androgen secreting neoplasms) should first be excluded.^{2,3} Currently, the most commonly accepted consensus criteria for diagnosis of PCOS are the Rotterdam criteria⁴, agreed by the European Society of Human Reproduction and Embryology (ESHRE) and the American Society for Reproductive Medicine (ASRM).

The Rotterdam criteria suggest that two of the three following symptoms are required for the diagnosis of PCOS:

- Polycystic ovaries (either 12 or more peripheral follicles or increased ovarian volume)
- Oligomenorrhea or anovulation
- Clinical and/or biochemical signs of hyperandrogenism.

The Rotterdam criteria will include a number of phenotypes of PCOS, including mild phenotypes that would be excluded by previous criteria. PCOS should not be over-diagnosed and diagnosis should be in strict accordance with current international criteria.

Metabolic consequences

Women with PCOS, particularly if they are obese (BMI greater than 30kg/m²), are at increased risk of impaired glucose tolerance (IGT), type 2 diabetes (T2D) and metabolic syndrome (see Table 1). The investigation and management of metabolic dysfunction in PCOS is therefore of prime importance. A study of a large cohort of Australian women with PCOS¹ revealed an incidence of IGT of 15.6 per cent and T2D of four per cent. Most women in this study were also obese with a mean BMI of 35kg/m². Although obesity exacerbates insulin resistance, women with PCOS are insulin resistant independent of obesity. Lean women with PCOS have a two-fold increase in incidence of T2D compared to controls.⁵ Women with PCOS are also at increased risk (OR 2.94) of gestational diabetes.⁶

Fasting glucose levels are poor predictors of glucose intolerance risk in women with PCOS and therefore screening for IGT should be by a two-hour oral glucose tolerance test (OGTT).⁷ However, there does not appear to be a consensus on who should be screened.

Some authorities recommend that all women diagnosed with PCOS should be screened with a two-hour OGTT.³ Other authorities recommend that an OGTT should be given to women with a fasting blood sugar of 5.6mmol/l or greater, BMI greater than 30kg/m², or a strong family history of gestational diabetes.² Currently, both strategies appear to be acceptable.

Longitudinal studies have also confirmed an increased incidence of T2D over time and conversion from IGT to T2D.⁸ Consideration should therefore be given to repeat screening³ based on the key predictors for the development of diabetes – age, BMI, family history. Measurement of insulin levels has little practical utility in PCOS and is not recommended.^{2,3}

There is increasing recognition of the metabolic syndrome as a risk for the development of cardiovascular disease. This syndrome is increasingly common in Western societies and the prevalence in women with PCOS has been found to be up to 45 per cent.⁹ Women with PCOS should be screened for cardiovascular risk by determination of BMI, fasting lipid and lipoprotein levels and metabolic syndrome risk factors.³

Table 1.

Metabolic syndrome

- Elevated blood pressure (greater than or equal to 130/85)
- Increased waist circumference (greater than or equal to 88cm)
- Elevated fasting blood glucose levels
- Reduced high density lipoprotein cholesterol levels
- Elevated trigyceride levels

Cardiovascular risk

Prospective studies have not as yet identified an increase in cardiac events in women with PCOS.¹⁰ However, there is indirect evidence of increased cardiovascular risk. The Nurses Health Study has revealed an increasing risk of cardiovascular disease with increasing oligomenorrhea.¹¹ Studies in premenopausal women with PCOS have also revealed an increase in sub-clinical atherosclerosis compared to controls.^{12,13} Hypertension should be actively treated in women with PCOS, but as yet, there is insufficient evidence to recommend routine treatment with lipid-lowering drugs such as statins.² Women diagnosed with PCOS should be strongly advised to refrain from smoking.

Obstructive sleep apnoea

Sleep apnoea is an independent risk factor for cardiovascular disease and is more common in PCOS. The difference in prevalence of sleep apnoea between PCOS and controls remains significant even when controlling for BMI.^{14,15} Women diagnosed with PCOS should be asked about the symptoms of sleep apnoea (snoring, daytime fatigue/somnolence) and offered investigation and treatment if indicated.

Cancer

Obesity, which is common in PCOS, is a recognised risk factor for several different cancers. It is also well recognised that oligomenorrhea or amenorrhea in women with PCOS may predispose to endometrial hyperplasia and carcinoma.¹⁶ Women with oligomenorrhea or irregular bleeding should be investigated according to local protocols. This may involve trans-vaginal assessment of endometrial thickness, endometrial sampling or hysteroscopy. In women with PCOS and oligomenorrhea or amenorrhea, the induction of regular withdrawal bleeds (at least every three to four months) is advisable using cyclic progestagens for at least 12 days or the oral contraceptive pill.¹⁷ A levonorgestrel-releasing intrauterine contraceptive device (for example, Mirena) is also a valid option.

Risk reduction

Lifestyle intervention through diet and exercise are the key treatments for reducing risk in PCOS. Women with PCOS who are not obese should be strongly advised to maintain their BMI in the normal range. Modest weight reduction (five to ten per cent) is associated with a significant improvement in metabolic indices.¹⁸ Dietary advice should focus on total calorific intake and on the balance of evidence. Low glycaemic index diets are preferred.¹⁹ Regular exercise (30 minutes of aerobic exercise a day) has been shown to decrease central obesity and increase insulin sensitivity and is therefore strongly recommended.²⁰ Women diagnosed with PCOS should be advised of the long-term health benefits of maintaining a healthy lifestyle.

Drug therapy

Insulin-sensitising agents such as metformin have a role when IGT or T2D has been diagnosed. However, there is no current evidence indicating that these drugs lower cardiovascular risk and their routine use in PCOS is not recommended.^{2,3,7} Trials suggest that metformin is not superior to lifestyle intervention in improving cardio-metabolic risk or progression to T2D.^{21,22}

Ovulation induction

Ovulation induction in PCOS is not specifically addressed in this statement, although randomised trials suggest that clomiphene is the first-line treatment and more effective than metformin. However, pregnancy when obese is associated with many increased risks. Another College statement (C-Gyn 2) has recommended that ovulation induction agents should not be given if the BMI is greater than 35kg/m².

Other related College statements

C-Gyn 2: Ovarian Stimulation in Infertility

References

- Dabadghao P, Roberts BJ, Wang J, et al. Glucose intolerance abnormalities in Australian women with polycystic ovary syndrome. *Med J Aust.* 2007; 187:328-331.
- 2. Long-term consequences of polycystic ovary syndrome RCOG Greentop Guideline No. 33 December 2007.
- Polycystic Ovary Syndrome: ACOG Practice Bulletin. Obstetrics and Gynaecology 2009; 108:936-949.
- ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril*. 2004; 81:19-25.
- Dunaif A, Segal KR, Futterweit W, Dobryjansky A. Profound peripheral insulin resistance, independent of obesity, in polycystic ovary syndrome. *Diabetes* 1989; 38:1163-1174.
- Boomsma CM, Eijkemans MJ, Hughes EG, Visser GH, Fauser BC, Macklon NS. A meta-analysis of pregnancy outcomes in women with polycystic ovary syndrome. *Hum Reprod Update* 2006; 12: 637-83.
- Kelsey E, et al. Glucose intolerance in polycystic ovary syndrome a position statement of the Androgen Excess Society. *Journal of Clin* Endocrinol Metab. 2007; 92(12): 4546-4556.
- Legro RS, Kunselman AR, Dobson WC, Dunlaif A. Prevalence and predictors of risk for type 2 diabetes mellitus and impaired glucose tolerance in polycystic ovary syndrome: a prospective controlled study in 254 affected women. J Clin Endocrinol Metab. 1999; 84:165-169.

- Glueck CJ, Papanna R, Wang P, Goldenberg N, Sieve-Smith L. Incidence and treatment of metabolic syndrome in newly referred women with polycystic ovary syndrome. *Metabolism* 2003; 52:908-915.
- Wild S, Pierpoint T, McKeigue P, Jacobs H. Cardiovascular disease inwomen with polycystic ovary syndrome: results of a 31 year followup study. *Human Fert. (Camb)* 2000; 3:101-105.
- Soloman CJ, Hu FB, Dunaif A, Rich-Edwards JE, Stampler MJ, Willett WC, et al. Menstrual cycle irregularity and the risk for future cardiovascular disease. J Clin Endocrinol Metab. 2002; 87:2013-7.
- Christian RC, Dumesic DA, Behrenbeck T, Oberg AL, Sheedy PF, Fitzpatrick LA. Prevalence and predictors of coronary artery calcification in women with polycystic ovary syndrome. J Clin Endocrinol Metab. 2003; 88:2562-8.
- Talbot EO, Zborowski JV, Rager JR, Boudreaux MY, Edmundowicz DA, Guzick DS. Evidence for an association between metabolic cardiovascular syndrome and coronary and aortic calcification among women with polycystic ovary syndrome. J Clin Endocrinol Metab. 2004; 89:5454-61.
- Fogel RB, Malhotra A, Pittman SD, Dunaif A, White DP. Increased prevalence of obstructive sleep apnoea in obese women with polycystic ovary syndrome. J Clin Endocrinol Metab. 2001; 86:1175-80.
- 15. Gopal M, Duntley S, Uhles M, Attarian H. The role of obesity in the increased prevalence of obstructive sleep apnoea syndrome in patients with polycystic ovary syndrome. *Sleep Med.* 2002; 3:401-4.
- 16. Chamlian DL, Taylor HB. Endometrial hyperplasia in young women. *Obstet Gynaecol.* 1970; 36:659-66.
- Writing group for the PEPI trial: effects of hormone replacement therapy on endometrial histology in postmenopausal women. The Postmenopausal Estrogen/Progestin Interventions (PEPI). JAMA 1996; 275:370-5.
- Norman RJ, Daview MJ, Lord J, Moran LJ. The role of lifestyle modifications in polycystic ovary syndrome. *Trends Endocrinol Metab.* 2002; 13:251-257.
- 19. Marsh K, Brand-Miller J. The optimal diet for women with polycystic ovary syndrome? *Br J Nut.* 2005; 94(2):154-65.
- Tuomelehto L, Lindstrom J, Eriksson JG, Valle T, Hamalainen H, Ilanne-Parikka P, et al. Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Eng J Med. 2001; 344:1343-50.
- Tang T, Glanville J, Hayden CJ, White D, Barth JH, Balen AH. Combined lifestyle modification and metformin in obese patients with polycystic ovary syndrome. A randomized, placebo-controlled, doubleblind multicentre study. *Hum Reprod.* 2006; 21:80-9.
- Hoeger K , Davidson K, Kochman L, Cherry T, Kopin K Guzick DS. The impact of metformin, oral contraceptives, and lifestyle modifications on polycystic ovary syndrome in obese adolescent women in two randomized, placebo-control trials. *J Clin Endocrinol Metab.* 2008; 93: 4299-306.

RANZCOG Application Aide -TGA Prescriber Status for Mifepristone and Misoprostol

For those seeking to become an authorised prescriber for Mifepristone and Misoprostol, contact RANZCOG for a free application aide:

Nola Jackson Women's Health Officer (t) +61 3 8415 0408 (e) njackson@ranzcog.edu.au

C-Gyn 29(b): Progesterone: Use in the Second and Third Trimester of Pregnancy for the Prevention of Preterm Birth

Date of this document: March 2010 First endorsed by Council: March 2010 Next review due: March 2013

Three recent randomised controlled trials have stimulated interest in the use of progesterone for the prevention of preterm birth. These trials demonstrated that progesterone reduces the risk of preterm birth in women with a previous history of spontaneous preterm birth and in women found to have a short cervix using a standardised transvaginal technique at the time of the routine anomaly scan (median 22 weeks gestation).^{1,2,3} Whilst systematic review and meta-analysis shows a clear reduction in the rate of preterm birth in these groups, data demonstrating an improvement in neonatal outcome is more limited.⁴

Women with a previous history of spontaneous preterm birth

Systematic review and meta-analysis of randomised trials suggests that progesterone reduces the prevalence of preterm birth in women with a previous spontaneous preterm delivery (Relative Risk [RR] for birth <34 weeks 0.15 [95% CI: 0.04-0.64]).^{4,5,6} Data demonstrating an improvement in neonatal outcome is incomplete, although there is evidence that the prevalence of low birthweight (less than 2500g) infants is reduced.⁴ Secondary analysis of one randomised controlled trial suggests that the use of progesterone is more effective in prolonging pregnancy of women with a history of spontaneous preterm birth before 34 weeks gestation.⁷ Two recent randomised controlled trials also suggested that intervention was more effective at preventing preterm birth at less than 34 weeks than under 37 weeks gestation.^{1,2}

Asymptomatic women with a short cervix at 18 to 23 weeks

A short cervix is associated with an increased risk of preterm birth (see RANZCOG College Statement *C-Obs 27: Measurement of Cervical Length in Pregnancy for Prediction of Preterm Birth*). There is only one randomised controlled study that has examined the effectiveness of progesterone in this population.³ This trial recruited women found to have a short cervix (under 15mm) at the time of the routine anomaly scan (median 22 weeks gestation). The trial showed a significant reduction in the rate of preterm birth before 34 weeks (RR 0.58 95% CI: 0.38-0.87) as well as a reduction in neonatal morbidity, although only the reduction in the prevalence of neonatal sepsis was significant.^{3,4} The reduction in the rate of preterm birth is as significant as that described using cervical cerclage in this situation.⁸

This data is supported by secondary analysis of a randomised controlled trial that was primarily designed to examine the effectiveness of progesterone for the prevention of preterm birth in women with a previous history of spontaneous preterm birth.^{9,10} Whilst this trial had shown no benefit for progesterone in the whole cohort, the secondary analysis did show that progesterone given to women with a short cervix (less than 28mm) was effective at reducing the rate of early (less than 32 weeks) preterm birth and at preventing neonatal admission.

These two cohorts have used different cervical lengths to define the high-risk population. Whilst progesterone is effective in preventing preterm birth for women with a short cervix, the most effective cutoff for defining a high-risk group that should be treated has yet to be determined.

Other indications

A small number of patients with a history of uterine malformation or of 'cervical incompetence' were deemed to be at high risk of preterm delivery and included in one of the recent randomised controlled trials, but the numbers do not give sufficient power to determine whether treatment for these indications is effective.¹ Progesterone has been shown to be of no value in reducing the prevalence of preterm labour in twin or higher order multiple pregnancies.^{11,12,13} There is limited data supporting its use as a long-term tocolytic for women who present with threatened preterm labour at less than 34 weeks gestation and further research is needed to examine the role of progesterone in this context.¹⁴

Route of administration, dosage and safety

A variety of progestins have been used in these trials. The US datasets predominantly use 17-alpha-hydroxyprogesterone caproate, given as a weekly intramuscular injection, but this preparation is not currently available in Australia.

Vaginal pessaries of progesterone are available and have the advantage of using a natural rather than synthetic preparation with high uterine bioavailability and few systemic side effects, although vaginal irritation can be problematic. This route of administration has been studied using doses of 90mg to 400mg and the optimal dosage is not clearly established. In women with a previous history of spontaneous preterm birth, the trial of Fonseca *et al.*¹showed that a dose of 200mg/day was effective, whilst that of O'Brien *et al.*⁹ found a dose of 90mg/day caused no reduction in the rate of preterm birth. In women with a short cervix, a dose of 100mg/day appeared to be effective and this is supported by the secondary analysis of the O'Brien study, where this subgroup showed a therapeutic benefit using 90mg/day.^{3,10}

Timing of therapy has also varied between studies, starting as early as 16 weeks of gestation in women with a previous history of spontaneous preterm delivery and continuing to 37 weeks in some trials. Early cessation of 17 alpha-hydroxyprogesterone caproate has been associated with an increased risk for recurrent preterm delivery.¹⁵

Commencing progesterone therapy in the second trimester of pregnancy appears to be safe for both the mother and the fetus and no teratogenic effects have been observed. Infants recruited to the National Institute of Child Health and Human Development (NICHD) trial, whose mothers received 17 alphahydroxyprogesterone caproate, were followed to four years of age and no detrimental effects were observed.¹⁶

Continued on page 74.

Recommendation

Progesterone therapy may be considered for all women who have a history of previous spontaneous preterm birth or who are found to have a short cervix at the time of the routine morphology scan. Further research is needed to improve our understanding of the effectiveness of this treatment in reducing neonatal mortality and morbidity. Participation in relevant clinical trials should be encouraged.

References

- Fonseca EB, Bittar RE, Carvalho MHB, Zugaib M. Prophylactic administration of progesterone by vaginal suppository to reduce the incidence of spontaneous preterm birth in women at increased risk: A randomised placebo-controlled double-blind study. *Am J Obstet Gynecol.* 2003; 188: 419-424.
- Meis PJ, Klebanoff M, Thom E, Dombrowski MP, Sibai B, Moawad AH, Spong CY, et al. Prevention of recurrent preterm delivery by 17 alphahydroxyprogesterone caproate. N Engl J Med. 2003; 348: 2379-2385.
- Fonseca EB, Celik E, Parra M, Singh M, Nicolaides KH. Progesterone and the risk of preterm birth among women with a short cervix. N Engl J Med. 2007; 462-469.
- Dodd JM, Flenady VJ, Cincotta R, Crowther CA. Progesterone for the prevention of preterm birth. *Obstet Gynecol.* 2008; 112: 127-134.
- Sanchez-Ramos L, Kaunitz AM, Delke I. Progestational Agents to Prevent Preterm Birth: A Meta-Analysis of Randomized Controlled Trials. *Obstet Gynecol.* 2005; 105: 273-279.
- Mackenzie R, Walker M, Armson A, Hannah ME. Progesterone for the prevention of preterm birth among women at increased risk: A systematic review and meta-analysis of randomized controlled trials. *Am J Obstet Gynecol.* 2006; 194: 1234-1242.
- Spong CY, Meis PJ, Thom EA, Sibai B, Dombrowski MP, Moawad AH, Hauth JC, *et al.* Progesterone for prevention of recurrent preterm birth: Impact of gestational age at previous delivery. *Am J Obstet Gynecol.* 2005; 193: 1127-1131.
- Berghella V, Odibo AO, To MS, Rust OA, Althuisius SM. Cerclage for Short Cervix on Ultrasonography. Meta-Analysis of Trials Using Individual Patient-Level Data. *Obstet Gynecol.* 2005; 106: 181-189.

- O'Brien JM, Adair CM, Lewis DF, Hall DR, DeFranco EA, Fusey S, Soma-Pillay P, et al. Progesterone vaginal gel for the reduction of recurrent preterm birth: primary results from a randomized, doubleblind, placebo-controlled trial. Ultrasound Obstet Gynecol. 2007; 30: 687-696.
- Defranco EA, O'Brien JM, Adair CD, Lewis DF, Hall DR, Fusey S, Soma-Pillay P. Vaginal progesterone is associated with a decrease in risk for early preterm birth and improved neonatal outcome in women with a short cervix: a secondary analysis from a randomized, doubleblind, placebo-controlled trial. Ultrasound Obstet Gynecol. 2007; 30: 697-705.
- Rouse DJ, Caritis SN, Peaceman AM, Sciscione A, Thom EA, Spong CY, Varner M, et al. A Trial of 17 alpha-hydroxyprogesterone caproate to prevent prematurity in twins. N Engl J Med. 2007; 357: 454-461.
- Norman JE, Mackenzie F, Owen P, Mactier H, Hanretty K, Cooper S, Calder A, et al. Progesterone for the prevention of preterm birth in twin pregnancy (STOPPIT): A randomised, double-blind, placebocontrolled study and meta-analysis. *Lancet* 2009; 373: 2034-40.
- Caritis SN, Rouse DJ, Peaceman AM, Sciscione A, Momirova V, Spong CY, lams JD, *et al.* Prevention of preterm birth in triplets using 17 alpha-hydroxyprogesterone caproate: a randomized controlled trial. *Obstet Gynecol.* 2009; 113: 285-292.
- Borna S, Sahabi N. Progesterone for maintenance tocolytic therapy after threatened preterm labour: A randomised controlled trial. *Aust* NZ J Obstet Gynaecol. 2008; 48: 58-63.
- Rebarber A, Ferrara LA, Hanley ML, Istwan NB, Rhea DJ, Stanziano GJ, Saltzman DH. Increased recurrence of preterm delivery with early cessation of 17-alpha- hydroxyprogesterone caproate. *Am J Obstet Gynecol.* 2007; 196: 224.e1-224.e4
- Northen AT, Norman GS, Anderson K, Moseley L, Divito M, Cotroneo M, Swain M, et al. Follow-up of children exposed in utero to 17 alpha-hydroxyprogesterone caproate compared with placebo. Obstet Gynecol. 2007; 110: 865-872.

Useful Links

Dodd JM, Flenady V, Cincotta R, Crowther CA. Prenatal administration of progesterone for preventing preterm birth in women considered to be at risk of preterm birth. *Cochrane Database of Systematic Reviews* 2006, Issue 1. Perinatal Trials: PROGRESS.

C-Obs 31: Routine Intrapartum Care in the Absence of Pregnancy Complications

Date of this document: March 2010 First endorsed by Council: March 2010 Next review due: March 2013

Service requirements for the provision of intrapartum care

Women in labour should have timely access to obstetric, midwifery, neonatal paediatric, anaesthetic, operating theatre and resuscitation services in labour and for at least several hours after birth. Further requirements include: access to intensive care specialist consultation; haematology and blood bank services (including specialist haematological consultation); and policy documents detailing methods of accessing emergency assistance. Even among women without pregnancy complications, women in labour and their babies can rapidly develop complications where timely access to these services may be life-saving. For this reason, birth centres are ideally placed within (or immediately adjacent to) an appropriately resourced 24-hour obstetric facility. Where, by virtue of remote location, such on-site services cannot be provided, patients should be informed of the limitations of services available and the implications for intrapartum and postpartum care. Antenatal transfer to a centre with more comprehensive services should be considered.

In circumstances where transfer may be necessary, formal systems must be in place to ensure the safe and timely transfer of women and/or their babies who require specialist treatment. These arrangements should be collaborative and hold the safety of mother and baby as paramount.

All transfers should be documented for future review. Such information is valuable for planning and resourcing improvements of those units requiring transfer capability. Amongst women selected for low obstetric risk, approximately 25 per cent will develop peripartum complications necessitating transfer to an obstetrician-led service.²

Admission to the delivery suite

Clinical assessment

All members of the clinical team (midwives and medical practitioners) should be aware of the admission of a patient in labour.

The woman should be assessed with a careful history and examination taking particular care to note:

- History: Gestational age (check ultrasound dates); past history (obstetric, gynaecological, medical and surgical); medications; pregnancy complications; investigation results (ultrasounds, group B streptococcus status, blood group and ab screen, full blood exam and haemoglobin, glucose tolerance test, infectious disease screen).
- 2. **Clinical Examination:** General and abdominal examination. A vaginal examination is indicated for women admitted in apparent labour unless contraindicated by an antepartum haemorrhage or ruptured membranes not in labour.
- 3. **Investigations:** 'Admission Cardiotocograph' (CTG). See Intrapartum Fetal Surveillance Guidelines.

Routine care in labour

Care for women is optimised where there is one-to-one midwifery support in labour in an obstetrician-led collaborative service.

Communication and support

The team responsible for the care of the woman in labour should be given an opportunity to introduce themselves, explain procedures as clearly as possible and actively involve the woman and her support person in management decisions in labour.

Maternal observations

Each unit should have a prescribed regimen for taking, recording and notifying observations in labour such as pulse rate, blood pressure, respiratory rate, temperature, contraction duration/ frequency/intensity, abdominal palpation findings, vaginal examination findings and presence/colour of the amniotic fluid. The World Health Organisation (WHO) recommends that such information be graphically displayed on a partograph to facilitate review of a woman's progress in labour. Standards exist for such a document and are available from WHO's *Pregnancy, childbirth, postpartum and newborn care - A guide for essential practice.*

Fetal surveillance

See RANZCOG Clinical Guidelines: Intrapartum Fetal Surveillance.

Activity

Although walking during the first stage of labour is often recommended, it does not reduce the need for oxytocin or caesarean section.

If walking and showering take place for maternal comfort, maternal observations and fetal surveillance must not be compromised.

Ambulation during labour has no effect on use of oxytocic, operative delivery or neonatal outcome. Women should be encouraged to ambulate freely according to comfort, where it does not compromise maternal and fetal observations in labour.

Analgesia in labour

See RANZCOG/ANZCA/RACGP/ACRRM Position Statement on the Provision of Obstetric Anaesthesia and Analgesia Services.

Patients should be informed prospectively of the obstetric anaesthesia and analgesia services offered by an institution. Where such facilities are limited, patients should be informed and offered transfer antenatally to a centre with more comprehensive services. Both pharmacological and non-pharmacological methods of pain relief should be both available and offered to women in labour.

Where epidural analgesia is being employed:

- Consideration should be given to fluid loading, particularly where the patient may be relatively dehydrated after some hours in labour.
- Particular attention should be made to bladder care with anticipation of difficulties with voiding.
- Continuous electronic fetal monitoring should be instituted as per the RANZCOG Intrapartum Fetal Surveillance Guidelines.

Fluids and oral intake

There is some data to suggest that inadequate hydration may increase the length of labour and the need for oxytocin augmentation in labour. However, women should be encouraged to only have clear fluids and light diet in the active phase of labour to minimise the small risk of aspiration pneumonitis. Intravenous fluid replacement may be considered in women unable to tolerate oral intake.

Intravenous fluid loading prior to epidural catheter placement is mandatory and it may be recommended in other situations where hydration is considered to be inadequate.

Antibiotics

Indications for antibiotic administration in labour:

- For prevention of early onset group B streptococcus (GBS) infection is indicated according to institutional guidelines. See C-Obs 19: Screening and Treatment for Group B Streptococcus.
- 2. For women at risk of chorioamnionitis or if other bacterial infection is suspected, for example, fever over 38 on one occasion or above 37.5 on two occasions.
- 3. Rupture of membranes after 18 hours.
- 4. For women with cardiac lesions susceptible to infective endocarditis.

Amniotomy (ARM)

Indication

1. Induction of labour

Women not in labour or in 'early/spurious labour' may elect to have an ARM performed. This should be done in the knowledge that this may be inducing rather than augmenting labour.

2. Augmentation of labour

Routine amniotomy in labour has both benefits and adverse consequences. In considering amniotomy for the augmentation of labour, the following should be noted:

- i. Evidence that routine ARM shortens labour is largely lacking (Cochrane review 2009).
- ii. The risk of infection increases following rupture of the membranes, whether by amniotomy or following spontaneous rupture of the membranes.
- iii. ARM provides useful information on fetal wellbeing (liquor volume and colour). The presence of scant amniotic fluid or meconium staining of liquor at ARM enables identification of a fetus that would benefit from continuous electronic fetal monitoring (CEFM) when this may not otherwise be known.

Relative contraindications to amniotomy

- Hepatitis B, hepatitis C, herpes simplex virus (HSV) and HIV infection in order to minimise the hazards to the fetus of ascending infection.
- 2. Presenting part high and mobile.
- 3. Any antenatal suspicion of vasa praevia.

Failure to progress in labour

Principles

The purpose of oxytocin augmentation is to shorten the duration of labour when it is abnormally prolonged, correct inefficient uterine action and lessen the need for operative intervention. Never is the influence of parity as great as when considering augmentation of a labour that is progressing slowly.

Frequency of cervical examinations in labour

Most trials examining frequency of cervical assessments, including those involving assessment of the active management of labour, have included two-hourly cervical assessments. This enables dystocia to be diagnosed and corrected early, but needs to be weighed against the added maternal discomfort of more frequent examinations and the potential for introducing infection. A compromise position is the common practice of four-hourly examinations in the first stage of labour.

Where full dilatation is not apparent clinically two hours after a patient is 9cm dilated, a further vaginal examination is beneficial to confirm full dilatation or allow the diagnosis of 'failure to progress' if full dilatation has not occurred.

Reassessment should occur after two hours in the second stage of labour in a primigravida and one hour in a multigravida.

Definition of failure to progress

1. First stage – before 'established labour'

No upper limit to the length of the 'latent phase' of labour can be defined. It is not uncommon for labour to 'stop and start' with multiple episodes of early/spurious labour before labour is finally established. Nevertheless, recurrent or prolonged episodes of spurious labour may contribute to a legitimate decision to induce labour in some women.

2. First stage – in 'established labour' (defined above)

Primigravida

The tenth centile for progress of cervical dilatation in labour is 1.2 cm/hour in a primigravida. The decision as to when to initiate augmentation of labour in the presence of slow progress requires individual consideration including the following:

	Threshold for oxytocin use	
	lower (e.g.Dublin)	higher (e.g.NICE)
labour	shorter	longer
frequency, duration and intensity of contractions	1	\downarrow
continuous electronic fetal monitoring (CEFM)	1	\downarrow
pelvic floor damage	\downarrow	1
postpartum haemorrhage	\downarrow	1
instrumental delivery	\downarrow	1

The threshold at which slow cervical dilatation merits a recommendation for oxytocin infusion is therefore:

- i. Appropriately individualised with an informed discussion between the patient and her carer.
- ii. Commonly at 1 cm/hour for most women in spontaneous labour but may be as high as 1 cm/two hours in women prioritising low intervention.

Multigravida

The tenth centile for progress of cervical dilatation in labour is 0.9cm/hour in a multigravida. Augmentation of labour in the multigravid labour should proceed only after careful assessment and then with caution, as it carries the particular risk of uterine rupture which is exceedingly uncommon in the primigravida.

3. Second stage

Progress is judged solely in terms of cervical dilatation and head descent in the first stage of labour. Progress in second stage includes flexion, rotation and descent of the head. Normal second stage for a primigravida is up to two hours and up to one hour for a multigravida. When these times are exceeded, assessment should occur by a medical practitioner with the view to correcting dystocia or effecting delivery.

Delivery

Positioning

Preparation for spontaneous delivery should take into account the patient's parity, preference for positioning during delivery, the progress of labour, presentation of the fetus and any complications of the labour. If it is anticipated that significant fetal manipulation may be required (twins, breech, anticipated shoulder dystocia), the patient should be delivered in the lithotomy position.

There is good evidence that flexion and abduction of the hip joint increases the size of the pelvic outlet (McRoberts' position). This knowledge may assist with normal birth as well as shoulder dystocia.

All women should birth in a position where they can rapidly access treatment in the event of sudden unexpected complications such as maternal collapse or shoulder dystocia. Management of the obese patient in labour is particularly important in this respect. Birth in water is not recommended (see College Statement *C-Obs 24: Warm Water Immersion in Labour*).

The accoucheur

The role of the accoucheur at delivery is to minimise maternal perineal trauma, prevent fetal injury and provide initial support of the newborn. The accoucheur should control the fetal head at crowning (to avoid precipitous expulsion) and support the perineum to reduce perineal tears.

Episiotomy

There is no benefit to 'routine' episiotomy. Episiotomy should be considered where there is:

- 1. A high likelihood of severe laceration.
- 2. Soft tissue dystocia.
- 3. A requirement to accelerate the birth delivery of a compromised fetus.
- 4. A need to facilitate operative vaginal delivery.
- 5. A history of gemale genital mutilation (FGM).

The third stage of labour

'Active' management of the third stage of labour includes oxytocic administration followed by assisted delivery of the placenta and is recommended for all women. 'Expectant' management cannot be recommended on the basis of evidence and is associated with approximately a two-fold increase in the incidence of postpartum haemorrhage and an increased risk of blood transfusion when compared with active management. A poorly contracted uterus poses an increased risk of the potentially fatal complication of uterine inversion. Women should be informed of the reasons for this recommendation prior to labour.

Oxytocic administration

Administration of an oxytocic with delivery of the anterior shoulder may maximise the benefit in terms of preventing postpartum haemorrhage. Caution must be exercised if there is the possibility of an undiagnosed second twin (therefore, no ultrasound in pregnancy).

A number of oxytocic regimens have been used and each has its passionate advocates. The most popular regimens are oxytocin 5 or 10 units (intramuscularly or intravenously) OR Syntometrine 1ml intramuscularly (ergometrine 0.5 mg + oxytocin 5 units).

Ergometrine commonly produces nausea and vomiting and may lead to raised blood pressure. Care should be taken in patients with hypertension or heart disease. When intramuscular ergometrine is used, side effects have, in general, been found to be mild. Most studies have shown a slight increase in the incidence of manual removal of the placenta with active management of the third stage of labour.

Assisted delivery of the placenta

It is absolutely essential to ensure the uterus is well contracted and the placenta separated before controlled cord traction is applied.

Newborn care

At birth, the neonate should be immediately assessed as to the need for resuscitation. Where neonatal resuscitation is not required, the neonate should be placed in close proximity to the mother such that the baby can be kept warm and continue to be observed by the responsible carer. Apgar scores should be completed at one and five minutes.

Regular observations of the neonate should continue during the early neonatal period including:

- Neonatal respiration, pulse rate, colour, tone and reflex irritability
- Observing for any evidence of respiratory distress (grunting, nasal flaring, intercostal retraction, tachypnoea, cyanosis).

Following a vacuum-assisted delivery, additional neonatal observations may be recommended (See *C-Obs 13a: Vacuum-assisted Birth*).

Intramuscular vitamin K administration is recommended.

Early postpartum care

All women should be cared for in the early postpartum period by caregivers experienced in the management of the early puerperium and its complications. This applies also to women being cared for in a 'recovery area' after an operative procedure and to women who have been admitted to an area of high dependency or intensive care. Regular maternal observations should include:

- Pulse rate, blood pressure and temperature
- Palpation of the uterine fundus to exclude atony
- Inspection of the perineum to exclude excessive postpartum blood loss or development of vulval haematoma.

Complications such as haemodynamic instability, excessive PV blood loss or evidence of expanding haematoma necessitate immediate notification of the responsible obstetrician.

Clinical audit

Perinatal outcomes and obstetric intervention must be subject to regular multi-disciplinary clinical practice audit, supported by robust, systematic data collection systems. Where there has been transfer between birthing units or models of care, this must be flagged in the data collection system and subject to clinical audit by both the referring and receiving clinical teams.

References

- World Health Organisation (WHO). Care in normal birth: A practical guide. Report of a technical working party. 1996 Geneva: Publication no WHO/FRH/MSN/96.24. King Edward Memorial Hospital (KEMH) Obstetric Clinical Care Unit: Management of normal labour. 2002.
- Stern C, Permezel M, Petterson C, Lawson J, Eggers T, Kloss M. The Royal Women's Hospital Family Birth Centre: The first ten years reviewed. *Aust NZ J Obstet Gynaecol.* 1992; 32: 291-5.
- Institute for Clinical Systems Improvement (ISCI). Health Care Guideline: Prevention, diagnosis and treatment of failure to progress in obstetrical labor. 2003. Society of Obstetricians and Gynaecologists (SOGC) Policy Statement Number 89: Attendance at Labour and delivery guidelines for obstetrical care. 2000.
- Enkin M, Keirse M, Neilsen J, et al. A guide to effective care in pregnancy and childbirth. Melbourne: Oxford University Press.
- Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG). *Clinical Guidelines: Intrapartum Fetal Surveillance*. 2002.
- Hodnett ED. Continuity of caregivers for care during pregnancy and childbirth (Cochrane Review). In: *The Cochrane Library*, Issue 3, 2004 Chichester, UK: John Wiley & Sons, Ltd. (Amended 1999)
- Hodnett ED, Gates S, *et al.* Continuous support for women during childbirth (Cochrane Review). In: *The Cochrane Library*, Issue 3, 2004 Chichester, UK: John Wiley & Sons, Ltd. (Amended 2003)
- Pregnancy, childbirth, postpartum and newborn care a guide for essential practice. World Health Organization, Department of Making Pregnancy Safer. 2006. Accessed at: www.who.int/reproductivehealth/ publications/maternal_perinatal_health/924159084X/en/index.html

Other related College statements

- C-Obs 21: Management of the Third Stage of Labour
- C-Gen 2: Guidelines for Consent and the Provision of Information Regarding Proposed Treatment

Have you changed your address or email account recently?

Have you notified the College of these changes?

If not, please update your contact details via the RANZCOG website (www.ranzcog. edu.au) and follow the link to 'Update contact details' or call 03 9417 1699 to notify the College of your changed contact details.

C-Obs 35: Prenatal Screening for Fetal Abnormalities

Date of this document: March 2010 First endorsed by Council: March 2010 Next review due: March 2013

1. General Principles

All women, irrespective of their geographical location, resources or chosen model of antenatal care, are entitled to informed prenatal screening and diagnostic testing for fetal abnormalities or genetic conditions that may impact on the future life and health of their baby.

Pre-pregnancy counselling for inherited genetic conditions is of particular importance in patients where the possibility of pre-implantation diagnosis is available.

1.1 Screening

At pre-pregnancy or early in the antenatal period counselling should address:

- A detailed clinical assessment of any particular risk factors for fetal abnormalities or familial genetic conditions.
- b. Maternal serum screening and nuchal translucency assessment to evaluate the risk of chromosomal abnormalities, irrespective of maternal age.
- c. Screening for thalassaemia syndromes.
- d. Availability of carrier status screening for genetic conditions of perceived high prevalence or consequence.
- e. A midtrimester ultrasound for fetal structural abnormalities.

1.2 Diagnostic testing

If an increased risk for fetal genetic or structural abnormality is identified from the above screening procedures, all women should have access to timely diagnostic testing. This may involve either:

- a. Invasive testing for fetal genetic/chromosomal abnormality, or infection by amniocentesis, or chorionic villus sampling; or
- b. Specialised imaging (ultrasound/MRI) for further diagnosis or evaluation of suspected structural abnormality to obtain maximal prognostic information.

2. Prenatal screening tests for fetal abnormalities

In Australia and New Zealand, maternal serum screening and obstetric ultrasound are widely used to identify pregnancies at increased risk of chromosome and structural anomalies. Initial screening tests may lead to an offer of further testing (tertiary ultrasound, chorionic villus sampling or amniocentesis) to definitively identify the presence of a chromosomal or structural fetal anomaly. In the event of the diagnosis of an anomaly, the woman and her partner may choose to terminate or continue with the pregnancy. Prenatal screening is best implemented in the context of a comprehensive program that coordinates pre-test counselling and information; biochemical and ultrasound measurements; post-test interpretation; counselling and support during decision-making; and, where indicated, follow-up consultations and diagnostic testing.

3. Clinical assessment

A detailed medical history and clinical examination should be obtained in order that any predisposition to fetal abnormality can be ascertained and particular needs of the couple can be addressed.

Where possible, this should take place at a pre-pregnancy visit or alternatively at the earliest opportunity in pregnancy.

4. Counselling and information

All pregnant women should be advised of the availability of prenatal screening at pre-pregnancy counselling or as early as possible in pregnancy to allow time to discuss the options available and facilitate an informed choice.

The clinician providing the counselling should do so in the appropriate context including:

- The resources available including, where necessary, recognition of the patient's geographical location.
- The patient's clinical circumstances (for example, gestational age).
- The financial costs involved, where this is relevant.

Information should be given in a way that is easily understood and culturally appropriate. Written information is particularly valuable for many patients.

Information provided should include:

- The difference between screening and invasive diagnostic testing.
- The relative advantages and disadvantages of the available screening tests
- Details of the nature, purpose, limitations and consequences of screening
- That screening is entirely voluntary and that there will be no change to pregnancy management if a woman and her partner choose not to have any screening tests.
- Practical aspects of screening, including the conditions that are being screened for, the type of tests, the timing of tests and the approximate costs involved.
- The possibility of diagnosing fetal anomalies other than those for which the screening programs are designed.
- The nature of results (often expressed as a numerical risk estimate) and the offer of a follow-up diagnostic test if an 'increased risk' (positive) result is obtained.
- That termination of pregnancy is an option in the event that an abnormality was diagnosed.
- An assurance that continuation of the pregnancy is a valid option should an abnormality be diagnosed and that couples would receive appropriate counselling and care in preparation for birth.

5. Referral for further counselling

In some circumstances, access to further counselling may be sought from one or more of the following: an obstetrician (general practitioner or specialist), a paediatrician, a paediatric surgeon, a geneticist or a genetics counsellor.

6. Education, training and continuing professional development

Health professionals caring for pregnant women should:

- Have had education and training with respect to the clinical assessment and testing that is available for prenatal screening; and
- Participate in continuing professional development (CPD) and through seminars, courses, journals and other printed material, maintain an awareness of the most up-to-date evidence-based practice.

Useful links

Nuchal Translucency – Ultrasound, Education and Monitoring Program. Access at: www.nuchaltrans.edu.au .

The Fetal Medicine Foundation (FMF) credentials ultrasound operators and provides ongoing quality assurance for operators working outside Australia. Access at: www.fetalmedicine.com .

CPD Points for Past Meetings

Have you attended a conference and don't know how many CPD points to claim?

Download the 'point for past meetings' list from the website and check if your meeting is listed.

www.ranzcog.edu.au/meetingsconferences/ pastmeetings.shtml

Points for attendance at all RANZCOG accredited meetings are detailed on this list as well as some of the larger overseas meetings.

If you are attending an overseas meeting that is not included on this list please send a copy of the scientific program to:

Val Spark

CPD Senior Coordinator (t) +61 3 9412 2921 (f) +61 3 9419 7817 email: vspark@ranzcog.edu.au



Nuchal Translucency Online Learning Program



Purpose

The Nuchal Translucency Online Learning Program (NTOLP) is designed to replace the theoretical course that is conducted for operators who wish to become credentialed to perform Nuchal Translucency scans.

Content

The NTOLP covers eight topics:

- 1. Principles of screening
- 2. Practicalities of NT measurement
- 3. NT and chromosome abnormality
- 4. Biochemical screening
- 5. 12-week anomaly scan
- 6. Screening test results and informed choice
- 7. Screening and multiple pregnancy
- 8. Increased NT and normal chromosomes

Features

This site uses many elements to engage and interest the learner. Some examples are:

- Interactivity mouse over, prediction tasks and multiple choice questions
- Customised images graphs, detailed diagrams, flash animations and ultrasound scans
- Illustrations and text
- Discussion Forums

The course is now live and costs A\$165.00 incl. GST per individual. Please visit www.nuchaltrans.edu.au/ for further details or to enrol. This program is co-located with The Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG) and development has been funded by the Australian Department of Health and Ageing.

Council Meeting Report 5 March 2010

Penelope Griffiths

Director of Corporate Services

Report from the President

The President presented his report including the following major items for information of Council:

- Maternity reforms. Consultative groups working in this area include the NHMRC guidance group to develop a guidance document for use of practitioners engaged in collaborative models of maternity care and the multidisciplinary Maternity Services Advisory Group, set up to advise Government on maternity care reform.
- Governance review of the College has been conducted with a new governance model developed by the governance working party. This model has subsequently been approved by plebiscite by a majority of votes and will be instituted from November 2010.
- A RANZCOG training program working party is to be established. The proposed terms of reference are included in the report from the Executive Committee.
- National E-Health.
- Meeting with Medical Benefits Task Review Group re development and implementation of a new Medicare Benefits Schedule (MBS) Quality Framework and new listing process for item numbers in the MBS.
- AusAID meeting, Canberra, 11 December 2009.
- RANZCOG 2010 ASM, Adelaide, 21-24 March 2010.
- Meeting with retired/senior RANZCOG Fellows, College House, Melbourne, 12 February 2010.
- The Executive Committee meeting is moving to electronic documentation.

Report from the CEO

The CEO presented his report including the following major items for information of Council:

- Implementation of the National Registration and Accreditation Scheme (NRAS) and issues raised by the Medical Board of Australia (MBA).
- College Appeals processes.
- Health workforce planning.
- The new Rural Health Continuing Education Program (RHCE) will replace the Support Scheme for Rural Specialists (SSRS).
- Recommendations from the MedEd09 Conference, October 2009, have been endorsed by the Committee of Presidents of Medical Colleges (CPMC) and the CEO has been nominated by the CPMC to a cross-stakeholder group to progress implementation of these recommendations.
- Report from the Director of Education and Training.
- College tender to the Commonwealth Government to administer a component of the GP Procedural Training Support Program.
- College application under the Australian Government's Innovative Clinical Teaching and Training Grants program.
- College Budget for 2010-2011 is underway.

Resignation of Editor, ANZJOG

Advertisement of the Editor's position for the Australian and New Zealand Journal of Obstetrics and Gynaecology (ANZJOG) is underway following notice of the resignation of Professor David Ellwood, as from the end of September 2010.

The President minuted a note of commendation to Professor Ellwood on his tenure in the position of Editor of *ANZJOG*.

Meeting with Medical Council of New Zealand

On 29 January 2010, the College President, Dr Ted Weaver, the New Zealand Vice President, Dr Digby Ngan Kee, and Chief Executive Officer, Dr Peter White, met with representatives from the Medical Council of New Zealand. Discussions were held regarding the continuing improvements to the common features of both Australian and New Zealand international medical graduate (IMG) and overseas-trained specialist (OTS) assessment processes.

Research Project

Measures are being reviewed by the Research Assessment Subcommittee to ensure timely turnaround of research project proposals. The bank of assessors and volunteer mentors has expanded and guidelines are to be developed to make best use of these resources.

Deferment of Training Policy

Background information on the reasons for introduction of a College policy on deferment of training had been circulated to Council.

Following discussion, Council endorsed the new policy on deferment of training.

XXIst AOFOG Congress (AOCOG)/ RANZCOG 2009 ASM

Auckland, New Zealand, 26-30 March 2009.

The Auditor's Report from the XXIst AOFOG Congress (AOCOG)/ RANZCOG 2009 ASM has been received.

Rotation Schedule for RANZCOG Annual Scientific Meetings

The following motion was put and carried.

THAT the rotation of forthcoming RANZCOG Annual Scientific Meetings be scheduled as follows: RANZCOG 2012 ASM – Australian Capital Territory RANZCOG 2013 ASM – New South Wales RANZCOG 2014 ASM – Western Australia RANZCOG 2015 ASM – Queensland RANZCOG 2016 ASM – Tasmania

RANZCOG Expert Witness Register

A working party has been established to review the College statement guidelines for the Expert Witness Register.

COGU Ultrasound In-Hospital Clinical Assessment (IHCA)

Background information had been circulated to Council about significant revisions and changes to the format of the current In-Hospital Clinical Ultrasound Examination (ICUE).

Council approved the following recommendations:

THAT the name of the revised assessment be changed to COGU Ultrasound In-Hospital Clinical Assessment (IHCA).

THAT the IHCA apply from 1 January 2011 for all COGU trainees, subject to satisfactory review of the IHCA by each committee at the end of 2010, with the exception of those trainees eligible to do the IHCE in 2010, having the option of completing either the current examination or the revised assessment.

THAT all existing regulations pertaining to the old ICUE remain in place for the IHCA (except for Regulation 15.15.4 which will be amended to allow COGU trainees to complete the assessment after 12 months of subspecialty training).

THAT the fee for the IHCA be reduced from the current ICUE fee of A\$830 to that charged for the ITP IHCA (\$315).

Accreditation of GO Training Unit

A report was noted that applications for the re-accreditation of the following gynaecological oncology (GO) training units have been approved by the GO Subcommittee:

- Christchurch Women's Hospital, Christchurch, New Zealand
- KK Women's and Children's Hospital, Singapore

Election of President for the period 2010-2012

Council approved the following recommendation:

THAT Dr Rupert Sherwood be declared duly elected President of the Seventh RANZCOG Council for the period November 2010 to November 2012.

Vice President Sherwood was congratulated by the President and Council on his election as incoming President. Dr Sherwood advised that he looks forward to taking up the College Presidency and thanked the Council for their support of his nomination.

MRANZCOG Research Assessors and Mentors Required

Do you have a strong research background?

Would you like to support the development of research skills amongst our ITP trainees?

College House is looking for research assessors and mentors to provide appropriate feedback and guidance to trainees undertaking their research proposals and projects. The research project is a compulsory requirement within the ITP/Elective training program.

What is required?

You would be required to read the trainee's research proposals and provide feedback relating to the stated aims, hypothesis, project background, literature review, method, study design, statistics collection and analysis. This feedback is completed on a prepared template. As the College will also be conducting random audits on completed projects, you may be asked to assess a completed project as well.

RANZCOG is also preparing a mentor list so that trainees can be referred to a suitable research mentor if required. This position does NOT mean you would be mentoring a trainee for the duration of their research. Rather, you would be asked to provide timely advice and/or support on a needs only basis. Effective mentoring is not location specific and can utilise a range of technologies such as online, email and telephone communication.

If you are interested in being an assessor or mentor please send an email detailing your research interests and expertise to: Frances Gilleard (e) fgilleard@ranzcog.edu.au

If you have any questions please contact: Bronwyn Robinson (t) +61 3 9412 2979 (e) brobinson@ranzcog.edu.au

REQUESTS FOR EXTENSION TO CONTINUING PROFESSIONAL DEVELOPMENT (CPD) PERIOD

Extension requests - six months and greater

Have you been absent from medical practice for a period greater than six months due to maternity leave, ill health or other exceptional circumstances?

If so, why not apply for an extension to your current Continuing Professional Development (CPD) period?

APPLICATION

Requests for extensions can be made in writing to the Chairman of the Continuing Professional Development Committee (CPDC). Proof of maternity leave, ill health or exceptional circumstances must be supplied.

PROCESS

The Chairman of the CPDC will consider requests for extension of six to 12 months. Requests greater than 12 months will be considered by the full CPDC, which meets three times a year (March, July and November).

If you are absent from practice for a period greater that two years, please see the re-entry policy following a prolonged absence from practice at: www.ranzcog.edu.au/publications/statements/wpi13.pdf.

For further queries contact:

Val Spark CPD Senior Coordinator Ph: +61 3 9412 2921 Fax: +61 3 9419 7817 E-mail: vspark@ranzcog.edu.au



The Royal Australian and New Zealand College of Obstetricians and Gynaecologists

WANTED: VOLUNTEER FACILITATORS FOR RANZCOG BASIC SURGICAL SKILLS WORKSHOPS

Fellows and Year 5 and 6 Trainees are needed to act as facilitators at the RANZCOG Basic Surgical Skills (BSS) workshops conducted annually in each State in Australia and in New Zealand. Attendance at a BSS workshop is compulsory for all Year 1 RANZCOG Trainees.

These practical, interactive two-day workshops are run on weekends and cover theatre etiquette, handling instruments, knot tying, incision/closure, episiotomy repair, haemostasis, electrocautery and stacks, hysteroscopy and laparoscopy.

Facilitators provide hands-on teaching and advice during the workshop and help with setting up on the day. Time commitment: ONE weekend per year.

Applications and enquiries: Shaun McCarthy, Training Services Manager tel +61 3 9412 2917, *fax* +61 3 9419 7817, *email: smccarthy@ranzcog.edu.au*

RANZCOG Research Foundation Scholarship Recipients

Professor John Newnham

Chair, Scholarship Selection Committee

Professor David Healy Chair, Board of Directors

The RANZCOG Research Foundation supports scientific and clinical research in the fields of obstetrics, gynaecology, women's health and the reproductive sciences through the awarding of various scholarships, fellowships, travel awards and other grants. Each year, approximately \$120,000 is disbursed, helping to support early career researchers in their work. Our recipients have a strong record of subsequent achievement in research and in academic careers in Australia and overseas. We are pleased to provide you with the following summaries of research recently completed with funding from the RANZCOG Research Foundation.

Arthur Wilson Memorial Scholarship, 2007-2008

Dr Yasmin Jayasinghe

The Oncogenic Risk of HPV in Children and Adolescents: Risk Factors for Cervical Carcinoma and High Grade Dysplasia Under 25 Years of Age

Young women have high rates of human papilloma virus (HPV) infection and cervical dysplasias, treatment of which may be associated with risk of future preterm birth. The World Health Organisation now recommends that Pap screening not be performed prior to 25 years of age. However, most cervical carcinomas in young women are small screen-detected lesions that have an excellent prognosis and are amenable to fertility-sparing surgery. Worldwide uptake of the HPV vaccine is 30 per cent, therefore lack of cervical screening in young women may result in increased cancers diagnosed in slightly older women. Therefore, evaluation of risk factors for invasive or high-grade disease in women under 25 years of age is a focus of our investigation. Some have suggested targeted screening of young women who have been sexually abused. Preliminary analysis shows that early disease (under 25 years of age) is associated with unwanted adolescent sexual experiences (suggesting that these women may benefit from revised screening and vaccination guidelines), whilst childhood exposure (under 16 years of age) does not increase the risk of disease at a younger age over and above the risk for disease onset after 25 years of age. Biological factors related to the cervix are likely to play an important role in triggering disease in young women.

Arthur Wilson Memorial Scholarship, 2008-2009

Dr Stephen Tong

Development of a Bifungal Drug to Treat Diseases such as Ectopic Pregnancies and Cervical Cancer

Over the past decade, there has been intensive research in developing an exciting new class of drug – RNA-based medicines that can silence key genes to disrupt diseases. Using the generous support of the RANZCOG Research Foundation's Arthur Wilson Scholarship, we have successfully engineered a way to also make these RNA drugs – termed short interfering RNAs – immune stimulatory. It means that the one drug can both disrupt disease by silencing key genes and activate the immune system to invade, and contribute to disease clearance. Such a therapy would be particularly suitable for ectopic pregnancies, where genes important to trophoblast survival can be switched off and macrophages recruited to mop up cellular debris; and cervical cancer, where foreign cancer-causing genes inserted by human papilloma virus (E6/E7) can be silenced and the immune system called in to help and effect a cure.

Fotheringham Research Scholarship, 2008-2009

Dr Rosalie Grivell

Skin and Subcutaneous Fascia Closure at Caesarean Section: A Randomised Controlled Trial

Caesarean section is a common operation and for different parts of the operation, techniques vary widely between surgeons. Wound infection, other wound complications and post-operative pain are common after caesarean section and have the potential to significantly impact on maternal health and wellbeing, the woman's ability to care for her infant, and her overall experience of the postpartum period. It is therefore important to establish which methods of skin closure and subcutaneous fascia closure are associated with the lowest rates of wound infection and other wound complications.

The Fotheringham Research Scholarship has allowed me to spend valuable time on a large scale clinical trial from its initiation and protocol development stage through to implementation and follow-up. I have been able to take primary responsibility for all aspects of trial management with support as needed from my supervisors. The knowledge and education I have obtained throughout this time has been extremely valuable and will continue to be useful throughout my research career. Without this opportunity from the RANZCOG Research Foundation, I would not have been able to have this experience at a crucial time of my career.

Luke Proposch Perinatal Research Scholarship, 2008

Dr Michele Kwik

Prediction of Intrauterine Growth Restriction

This study seeks to predict intrauterine growth restriction (IUGR) by measuring a combination of physiological and angiogenic parameters in populations at low and high-risk of IUGR.

First trimester serum was analysed for pregnancy associated plasma protein-A (Papp-A) and angiopoietin-2 (Ang-2). Uterine artery Doppler studies were performed at the time of the 18 to 20 week morphology scan. Investigators were blinded to the results. This is the first study to date that investigates the combination of Ang-2 and uterine artery Doppler studies in screening for IUGR. In the low-risk cohort (n=406), there was no statistically significant association between the combination of tests and IUGR, suggesting no benefit for use in screening for IUGR in this population. However, there was a statistically significant association between the combination of tests and subsequent development of preeclampsia (low Ang-2 p=0.04 and low Papp-A p=0.001). In the high-risk cohort (n=54), there was an association with preeclampsia (p=0.022), but not with IUGR. However, the tests were associated with a high negative predictive value (96 to 100 per cent), suggesting that these results would be useful for re-assigning high-risk women into low-risk models of care. This would allow for a more efficient use of resources and should be investigated further with a larger study.

Luke Proposch Perinatal Research Scholarship, 2009

Dr Wan Tinn Teh

Hypoxic Regulated mRNA Measured in Maternal Blood as a Novel Biomarker for Intrapartum Fetal Distress

Timely delivery to prevent catastrophic injury from hypoxic stress (oxygen starvation) to the fetus during labour, while avoiding overly aggressive interventions to effect delivery, is a challenge for obstetricians. The recent find that RNA (ribonucleic acid) from the fetus circulates in maternal blood presents a novel avenue for biomarker discovery. It might be possible to take a blood sample from the mother and analyse this to determine how hypoxic the baby may be. We aim to examine whether RNA coding hypoxic regulated genes in maternal blood are increased in association with fetal hypoxia (high umbilical cord lactates at delivery). We recruited 48 women undergoing induction of labour where blood samples were collected across labour: before induction started (no/little hypoxic genes should be expressed); during active labour; and crucially, a final sample at the moment of delivery (a proportion of babies will be hypoxic). To analyse the samples, we are using laboratory approaches that measure a suite of genes belonging to the hypoxia response pathway in each sample. Preliminary results have been promising, showing that multiple hypoxic genes are increased in expression in cases where fetal distress has occurred (high fetal lactate), but are not increased in the control group.

Taylor-Hammond Research Scholarship, 2008

Dr Bradley de Vries

An Investigation into the Mechanism of Intrapartum Vertical Transmission of HIV and Other Viruses

We investigated mechanisms of viral transmission, using the observation that elective caesarean section reduces the vertical transmission of HIV by more than 50 per cent. We hypothesised that neonates born vaginally swallow more blood than those born by elective caesarean. Gastric aspirates from 20 neonates were tested for red blood cells using flow cytometry. The average volume of maternal blood was 1.9uL in the vaginal delivery group and 1.5uL in the caesarean section group. These volumes are unlikely to explain HIV transmission and represent less than one per cent of the infectious inoculum from a single breast feed. Fetal blood was present in 40 per cent of samples suggesting neonatal microtrauma and that gastric suctioning should be avoided when the mother is infected. In a parallel study, we detected several instances of transplacental haemorrhage from mother to fetus. We are testing more gastric aspirates of infants born vaginally and so far none of 21 infants have swallowed sufficient blood to explain HIV transmission. We are also commencing a study to estimate background rates of transplacental transfusion.

I would like to thank my co-investigators Esther Aklilu, Ross Brown, Dr Priya Sivadas and Professor Heather Jeffery. I would especially like to thank the RANZCOG Research Foundation for the opportunity to perform this research.

Taylor-Hammond Research Scholarship, 2009

Dr Jane Hirst

Understanding and Prevention of Stillbirth in Vietnam: Application of a Classification System to Identify Antecedent Risk Factors

Stillbirth remains a major problem globally with an estimated 3.5 million babies dying before or during birth every year. Unless causes and circumstances surrounding these deaths are understood, progress in death reduction will be difficult to monitor and achieve. My project aimed to try and reduce the rate of unexplained stillbirth in a major Asian obstetric facility within local resource constraints. From December 2008 to January 2009, I collected data on stillbirths in the Tu Du Maternity Hospital, Ho Chi Minh City, Vietnam. Over 31 days, there were 4816 births and 122 stillbirths, including late terminations of pregnancy. Using the PSANZ-PDC (Perinatal Death Classification) guideline as a reference tool, parental consent was obtained to examine 107 stillborn babies, placentae and conduct maternal interviews. Using this information, the unexplained stillbirth rate was reduced from 53 to 22 per cent (P<0.0001). In addition, several useful epidemiological characteristics were identified, including socio-economic status, ability to access antenatal care, as well as nutritional and behavioural factors, which may have contributed to the death. This project was also the first time the placental histopathology had been performed in this population. This scholarship has given me the opportunity to conduct a detailed study in a very challenging and exciting setting.

Lifetime Achievement Award

Professor Malcolm Coppleson

Dr Louise Farrell FRANZCOG

Professor Malcolm Coppleson has been awarded a Lifetime Achievement Award by our American colleagues of the American Society for Colposcopy and Cervical Pathology (ASCCP).

The Lifetime Achievement Award was established in 2008 to recognise a 'lifetime corpus of work' by an individual in the field of colposcopy. Malcolm has been honoured as a pioneer in recognising the value of colposcopy as a diagnostic technique for the evaluation of the abnormal cervix; for the establishment of the first Australian colposcopy clinic; for co-authoring the first widelyused English language textbook on colposcopy; and for his teaching in many parts of the world, which established colposcopy as the triage procedure for the evaluation of abnormal cervical cytology. The other recipients of the Lifetime Achievement Award 2010 were Professor Harald zur Hausen, Professor Margaret Stanley OBE, Raymond H Kaufman MD and Eduardo Franco MD.

This award joins many others in Malcolm's distinguished career. He was honoured with an Order of Australia in 1994 for service to medicine in the field of gynaecology, particularly the prevention and treatment of gynaecologic cancer. It began in 1954 when he was awarded the Joseph Foreman Travelling Fellowship, which he used to go to Oxford, UK, where he trained with Professor Stallworthy, who encouraged him to go to Germany to train in colposcopy. When Malcolm returned to Australia, he set up a colposcopy clinic at the Royal Prince Alfred Hospital (RPA) in Sydney.

After Malcolm's return to Sydney in 1956, he concentrated his research interests with his co-investigator Bevan Reid on molecular biology studies that introduced many new concepts on the origin, nature and management of early cervical cancer and its precursors and that promoted an individualised and conservative approach to management. In 1959, he published an article titled 'The use of colposcopy in the early detection of cervical cancer' in the Medical Journal of Australia. He went on to have many more articles published on malignancy and re-malignant conditions of the female genital tract. In 1967, Malcolm and Bevan published a book, Preclinical Carcinoma of the Cervix Uteri: Its Nature, Origin and Management. Malcolm obtained his MD with his thesis, 'A colposcopic study of the human cervix uteri in health and disease'. He has contributed chapters to a number of books. Malcolm has served on the editorial boards of 11 medical journals and has spoken at innumerable conferences and courses.

In 1971, Malcolm published a book with Bevan Reid and Ellis Pixley, *Colposcopy: A Scientific and Practical Approach to the Cervix in Health and Disease*. This textbook would have three editions, the last in 1986. It was also translated into Spanish and Italian. In 1981, Malcolm was the editor and co-author of a two-volume text, *Gynaecologic Oncology: Fundamental Principles and Clinical Practice*.

From 1979, Malcolm founded the Department of Gynaecologic Oncology at RPA and was head of the unit until 1991. He was made a clinical professor of the University of Sydney in 1991, the first VMO to be awarded this title. He has served on many societies. He was a member of the Australian Council of the Royal College of Obstetricians and Gynaecologists (RCOG) from 1961 to 1968 and Honorary Treasurer from 1966 to 1968. He had a number of roles within the College, including Chairman of the Examinations Committee from 1973 to 1975. He was President of the Clinical Oncological Society of Australia from 1986 to 1987 among other previous roles in the Society.

Malcolm was founding President of the Australian Society of Colposcopy and Cervical Pathology (ASCCP) from 1972 to 1987. He was President of the International Federation of Cervical Pathology and Colposcopy (IFCPC) from 1984 to 1987 and served on their Executive from 1975 to 1984. He has been awarded honorary membership of the British, Argentinian and Singaporean colposcopy societies. Malcolm was also awarded a first Founder's Medal by the British Society for Colposcopy and Cervical Pathology (BSCCP).

Professor Coppleson has made an enormous contribution to our understanding and management of female genital tract malignancy and its precursors. We congratulate him on his award.



Professor Coppleson with his Lifetime Achievement Award, presented by the American Society for Colposcopy and Cervical Pathology.

Sydney IVFRANZdonatesWomultrasoundHealmachine to PNG2009

Prof Glen Mola

Head of O and G, School of Medicine and Health Sciences University of Papua New Guinea

Last year, Sydney IVF Newcastle donated a very modern ultrasound scan machine to Port Moresby General Hospital, Papua New Guinea. There were some hiccups in transporting the machine to Papua New Guinea – somehow the transducer heads became separated from the machine. However, these finally arrived in February and we have now set up the machine. It will be installed in our soon-to-be-opened, newly refurbished labour and delivery suites at Port Moresby General Hospital.

The Port Moresby General Hospital labour ward cares for more than 11,000 confinements every year (about 1000 per month, except when many women accompany their families to their rural villages for Christmas holidays when the numbers go down to 700 to 800). Amongst this very large obstetrical workload, there are at least five to six serious complications each day for which this ultrasound scanner will prove extremely valuable.

On behalf of the midwifery and obstetric teams at Port Moresby General Hospital, I would like to sincerely thank the team at Sydney IVF Newcastle for this generous donation.



Prof Mola using the donated ultrasound machine with a patient at Port Moresby General Hospital, PNG.

RANZCOG Women's Health Award 2009

Balveena Dhaliwal, a fourth year student in the MBBS program at Monash University's Sunway campus in Malaysia has been recognised for clinical excellence by the Royal Australian and New Zealand College of Obstetricians and Gynaecologists. The RANZCOG Women's Health Award 2009 recognised Balveena's achievement of obtaining the highest mark of any student in the Monash medical curriculum for clinical skills in women's health, as determined by the Observed Structured Clinical Examination (OSCE) and in-semester academic grading.



RANZCOG Fellow, Dr Danielle Wilkins, with Ms Balveena Dhaliwal, RANZCOG Women's Health Award 2009 winner.

Text and photo source: www.med.monash.edu.au/news/2010/mbbsmalaysia-high-achiever.html .



News from the Historical Collections

Di Horrigan, Librarian Grainne Murphy, Museum Curator Ros Winspear, Archivist

Gifts to the Archives, the Frank Forster Library, the Museum and the College Collection July 2009 to March 2010

We wish to thank the following Fellows and Friends who have generously donated items to the Collections and also contributed funds to support the ongoing expansion and maintenance of the Collections:

- Dr Geoffrey Bishop (Vic)—RACOG ephemera, personal papers
- Mrs Prue Forster (Vic)—Books, Gowllands otoscope, Tycos sphygmomanometer
- Dr David Knight (ACT)—MRCOG case record book
- Dr Richard Lewis (NSW)—MRCOG case record book
- Military Chapter, RANZCOG—RAN and RAAF ephemera (shields and hat); commemorative coins
- Prof Glen Mola (PNG)—Ventouse vacuum extractor
- Dr Jock Murray (NSW)—RCOG gown, RACOG gown
- O&G Society of Singapore—Crystal vase on stand
- Dr James Roche (NSW)—Brass figurine of doctor and baby
- Prof H Sathananthan (Vic)—IVF books, DVD
- Dr Michael Simcock (NSW)—Books
- Dr Margaret Smith (WA)—MRCOG case record book
- Mrs Jane Stops (NSW)—Books

Donations to the Friends of the College Collection

July 2009 to March 2010

Donations amounting to A\$2950 have been received from the following Fellows and Friends:

- Dr Mark Beale (NSW)
- Dr John Campbell (Qld)
- Dr Deryck Charters (Qld)
- Mrs Joan Cope (NSW)
- Mr Arthur Day (Vic)
- Prof Caroline de Costa (Qld)
- Dr Louise Farrell (WA)
- Prof Ian Fraser (NSW)
- Dr Euan Howell (Vic)

- Dr David Knight (ACT)
- Dr Mark Jalland (Vic)
- Dr Maxwell Michael (Vic)
- Dr David Morton (NSW)
- Mr Ian Ross (Vic)
- Dr Malcolm Stening (NSW)
- Prof William Walters (NSW)
- Dr Ted Weaver (Qld)

Do you have a RACOG Fellow's gown that you no longer need?

If so, the Image and Regalia Working Party would like to hear from you as they are keen to obtain RACOG Fellow's gowns that are no longer used by their owners. The aim is to build up the existing collection of gowns at the College. We plan to have the gowns available for the use of members of Council, new Fellows being presented with their Fellowship and for hire by Fellows for special occasions (a fee is charged for the hire of the gowns to cover postage and handling).

- The gowns can be upgraded to a RANZCOG gown with the addition of silver braid.
- The collection of gowns is kept in a special storage area and maintained in excellent condition.
- The gowns are used by the Council members at every College function including Council meetings.

Any enquiries please contact:

Ros Winspear

Coordinator, Image & Regalia Working Party ph: +61 3 9412 2934 fax: +61 3 9419 0672 email: rwinspear@ranzcog.edu.au
Obituaries

Dr Jean Murray-Jones

1921 - 2009

Jean Murray-Jones was born in South Australia on 30 October 1921. She moved to Perth as a small child in 1925 when her father was appointed Chief Veterinary Officer of Western Australia. She returned to South Australia to attend university, there being no medical school in Western Australia. After graduation in 1944, she came back to Perth to commence her residency at Royal Perth Hospital and then went to the Royal Children's Hospital, Perth (now Princess Margaret Hospital). In 1949, she went to the UK where she first worked at the West Middlesex Hospital in Islington for two years and then the Jessup Hospital for Women for a year. She went to Cumberland Infirmary as a gynaecology registrar for 12 months and gained her MRCOG in 1953. Jean returned to Melbourne in 1954 where she worked at the Queen Victoria Hospital.

In 1955, Jean returned to Perth and initially worked in Victoria Park as a general practitioner. Later, she set up private practice in Ord Street, West Perth. She had a beautiful Federation style house which served both as her home and her rooms. Jean worked as a clinical assistant at King Edward Memorial Hospital (KEMH)) from 1955 to 1974. Thereafter, she worked solely in private practice with a clientele that had a strong representation of troubled young women. In 1985, Jean was elevated to the Fellowship of the RCOG.

Jean was an only child and never married. Her father, a Lieutenant Colonel who served in both world wars, died in Perth in 1944. Jean was always extremely close to her mother, who acted as the receptionist in her practice.

Jean became a Foundation Fellow of the RACOG in 1979. She maintained a strong interest in College affairs and was a regular attendee at College meetings and educational events of the RACOG and KEMH. In the last decade, her health was poor and she was diminished greatly by dementia. Jean died on 27 December 2009. She appears to have no surviving relatives and has left a substantial bequest to the College in the form of her house in West Perth.

Dr Louise Farrell

FRANZCOG Perth, Western Australia

RANZCOG Application Aide -TGA Prescriber Status for Mifepristone and Misoprostol

For those seeking to become an authorised prescriber for Mifepristone and Misoprostol, contact RANZCOG for a free application aide:

Nola Jackson Women's Health Officer (t) +61 3 8415 0408 (e) njackson@ranzcog.edu.au

Are you interested in donating items to the Historical Collections?

We welcome enquires regarding donations.

If you have any items that you believe might be of value to the Historical Collections and you would be interested in donating them, please see the instructions below:

- Compile a list of items with a brief description. For books, include author, title, publisher, place and date.
 For archival and personal papers, include details.
 For museum items, include a brief description and the history of how you acquired it and attach a photograph.
- Email or post the list to one of the Historical Collections staff at the College.
- Contact the staff by telephone if you wish to discuss any items.

We look forward to hearing from you and would be delighted to consider any items you may wish to donate.

Librarian: Di Horrigan	Tuesday 9am-5pm
ph: +61 3 9412 2927	email: dhorrigan@ranzcog.edu.au
Museum Curator: Gráin	Monday 9am-5pm
ph: +61 3 9412 2927	email: gmurphy@ranzcog.edu.au
Archivist: Ros Winspear	Mon, Wed, Thu 9am-5pm

email: rwinspear@ranzcog.edu.au

ph: +61 3 9412 2934

College ConneXion

Is there an event you'd like to advertise? Want to know the latest College news or clinical information?

> Check out *College ConneXion*, RANZCOG's notice board.

www.ranzcog.edu.au/connexion/index.shtml