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Maternal death

The Royal Australian and New Zealand College of Obstetricians and Gynaecologists

O&G Magazine

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From the President

Prof Michael Permezel
President

Three months into the Eighth RANZCOG Council and an enormous amount of valuable work has already been accomplished by both College staff and those Fellows and Trainees who so generously give their time to the College. Thank you to all those that serve on the various College committees; assist with the development of a guideline or statement; give a talk at one of the many exam-preparation courses; join a board of examiners; are involved in hospital accreditation visits; or contribute in other ways to the work of the College. You are all great servants of the College and should be recognised as such.

Many of you have expressed your dismay at recent or projected cuts to some Australian public hospital budgets. While the blame for these cuts is in dispute between commonwealth and state politicians, the consequences are being borne by both patients and Trainees. Many elective gynaecological surgical lists have been cancelled – surgery that is not only critical to patient care, but also to training in gynaecological surgery. Safe working hours, reduced training opportunities overseas and the medicalisation of gynaecology have all meant some Trainees have found it increasing difficult to gain what they perceive to be the necessary surgical experience. Trainees are understandably disappointed if their surgical training opportunities are further reduced by budget cuts. It is important that health services become aware that their obligations extend beyond clinical service delivery and include the teaching and training of the future specialists. The current funding models do little (if anything) to reward the health service that provides excellence in training relative to a hospital where training struggles to achieve priority and administrators focus largely on the bottom line. In my last column, I alluded to the necessity of introducing financial incentives for health services to deliver the best-quality healthcare. It is equally important that the funding model also rewards quality in training.

> 'No longer can the number of training registrars at a given hospital be determined by the required number of service registrars. Trainees must be placed according to the quality of training provided by individual hospitals.'

The College can (and does) lobby the various agencies with respect to quality in women's healthcare and quality in training. For example, the College has recently had several meetings with Health Workforce Australia (HWA) and is progressively meeting Health Department representatives across all Australian states and New Zealand. All the medical colleges are working with HWA to determine optimal numbers of new Fellows, and thus the

predetermined number of Trainees. No longer can the number of training registrars at a given hospital be determined by the required number of service registrars. Trainees must be placed according to the quality of training provided by individual hospitals. The College has extensive quantitative data on where Trainees are receiving their procedural training in gynaecological surgery. Qualitative data is more interpretive, but has been greatly facilitated by the Hospital Accreditation Visit process. An integrated training program (ITP) comprising hospitals in which clinical training has slipped as a priority, might have correspondingly fewer training positions than an equivalent ITP hospital where registrar training is prioritised. A hospital having its 'Trainee Registrar' compliment reduced would need to explore other (possibly more expensive) approaches to clinical service delivery.

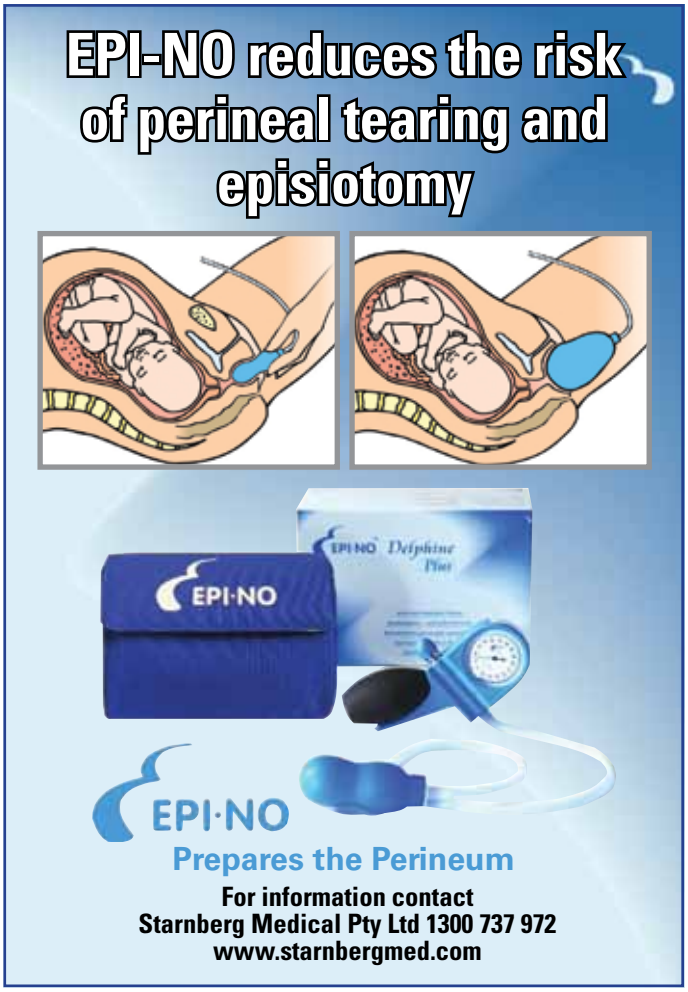
Along similar lines, all Fellows should now be aware of the significant changes to the training program to be introduced for Trainees commencing in 2014. Among some very significant changes is an acknowledgement that not all new O and G specialists will be experts across all areas of the specialty. With the exception of the subspecialties, there is an implied assumption in the current FRANZCOG training program that all new Fellows will be able to do everything. The revised training program further develops the concept of Core (~ITP) Training that is common to all Trainees, but also acknowledges that FRANZCOG Trainees may choose to develop 'special interests' during the two years of Advanced Training. In December, the College was fortunate to host Prof Wendy Reid, Vice-President Education, RCOG, and Deputy Chief Executive, Dr Michael Murphy. The two visitors provided important insights into many aspects of RCOG training – including the implementation of 'Advanced Training Skills Modules' as a means of defining training in areas of special interest for RCOG advanced trainees.

Few areas of practice better exemplify stratified training than laparoscopic surgery. All Trainees must achieve a basic level of competence in laparoscopic surgery during their four years of Core Training. Secondly, a substantive proportion (but not all) of Advanced Trainees will further develop those skills to a higher level following Core Training. Thirdly, a much smaller number of Advanced Trainees (or new Fellows) may undertake a two-year program of 'fellowship training' – as recently advertised by the Australasian Gynaecological Endoscopy Society (AGES). This latter group has created much interest and discussion. From the College's perspective, like all gynaecological surgery, the highest priority must be that new Fellows are able to provide the services needed by women in Australia and New Zealand. There is no debate about the need for basic laparoscopic competency at the completion of Core Training and also no debate that a substantial number of Trainees will build on those skills during

Advanced Training. The College must nevertheless ensure, if there is to be an expansion in the numbers of those undertaking AGES fellowship training posts, this does not compromise these Core and Advanced Training imperatives.

The introduction of Medicare rebates for 'eligible midwives' has not led to a rush of medical practitioners ready to sign collaborative agreements. The Government now plans to remove the obligation for an 'eligible midwife' to have a 'collaborative agreement with a medical practitioner' and, additionally, accept a 'collaborative agreement with a health service' as an alternative. The need for adoption of consensus consultation and referral guidelines has become urgent. RANZCOG, the Australian Medical Association and the National Association of Specialist Obstetricians and Gynaecologists are working together to ensure the current high standards of maternity care in Australia do not become compromised. Implicit in achieving this objective is the need for clear consultation and referral guidelines for midwives and obstetricians, with the term 'obstetrician' intended to include both specialist and GP obstetricians. As stated in the Summer issue of *O&G Magazine* column, senior representatives of both the Australian College of Midwives (ACM) and RANZCOG met at College House in December 2011, and produced a Joint ACM-RANZCOG Consensus Consultation-Referral Guideline. Unfortunately, the ACM Board chose not to endorse this document and the ACM continues to go down its own path, despite repeated requests from RANZCOG. It can only be hoped that the ACM will see that adopting a compromise position, as reached in December 2011, will be in the best interests of the women we all serve.

Homebirth is never long out of the news. Prominent in the media has been a drive by some in Victoria to expand the current homebirth programs at Western and Southern Health. The position of the College remains unaltered: the best possible outcomes for women and their babies are achieved in hospital. It is particularly concerning that active promotion of a home-birthing option by health services will be interpreted by many women as meaning this option is 'safe'. Almost no obstetrician would claim that there is an identifiable group of 'ultra-low-risk' pregnancies that are devoid of unexpected complications that may benefit from a close proximity of services only available in hospital. Similarly, no homebirth can be prospectively regarded as 'safe' in the context of a level of risk that parents would accept for their child in other areas of health practice. As suggested earlier, health services might be less keen to expand their homebirth numbers if they had to finance the indemnity risk (that is, the true actuarial cost) associated with that practice. Nevertheless, we all hope that those women who continue to choose to birth at home have the best possible health supports to minimise the occurrence of preventable adverse outcome for both mother and child.



From the CEO

Dr Peter White
CEO

As I sit down to write this column, the month of February has almost ended. With a Board meeting held recently in early February and various other activities already scheduled, it will not be long before March Council Week is here. Certainly, the festive season that was approaching when I wrote the previous column is now fast receding.

The activity of the College continues to increase, both in variety and complexity. While it is often difficult to single out particular activities as being of greater significance than others at any given time; there are three major activities in which the organisation will engage this year that I will note here: accreditation of the College through the process conducted by the Australian Medical Council (AMC) for the purposes of accreditation by the Medical Board of Australia (MBA) and the Medical Council of New Zealand (MCNZ); introduction of a revised continuous professional development (CPD) program for Fellows; and introduction of the revised FRANZCOG Training Program for Trainees commencing training from 1 December 2013.

The Standards relevant to the accreditation process that the College is subject to cover in detail all aspects of what I have referred to on previous occasions as the 'core activities' of the College; in other words, the various aspects of the training programs offered by the College (with particular emphasis on the FRANZCOG training program, but also in relation to the associated subspecialty training programs and the Certificate of Women's Health/Diploma programs), including, curriculum content and implementation, assessment processes, involvement of Trainees, evaluation processes, review/appeal processes and continuing professional development. Work on the College's accreditation submission, which is due for submission at the end of March, has already commenced and I thank those who are involved in reviewing the responses to each Standard for their work in this regard.

> '...the College is taking a proactive, strategic approach to the development and implementation of information and communication technology (ICT) initiatives, the College Board having approved an ICT Strategic Plan during 2012.'

The Accreditation Team will be chaired by Prof Kate Leslie, the Immediate Past-President of the Australian and New Zealand College of Anaesthetists (ANZCA) and current Chair of the Committee of Presidents of Medical Colleges (CPMC). The process typically involves visits by team members to a sample of training sites over one week, followed by meetings and discussions with key College committees and officers during a second week. The accreditation team visits are scheduled for late August and early September.

I have indicated previously that the College is taking a proactive, strategic approach to the development and implementation of information and communication technology (ICT) initiatives, with the College Board having approved an ICT Strategic Plan during 2012. Part of that strategy involves the development of an online facility to support the introduction of a revised CPD program for

RANZCOG Fellows. The program, based on the framework that underpins the FRANZCOG Curriculum, will enable Fellows to readily include a wide range of activities in their CPD that reflect their practice profile at various stages of their career. As previously discussed, the program has been successfully trialled prior to the implementation phase and final development of the ICT platform to support use of the program, with the College Board having considered the implementation of the program at its February meeting. This is an exciting development that, it is hoped, will pave the way for development of an online training e-portfolio for Trainees.

Preparations for introduction of the revised FRANZCOG Training Program for Trainees commencing training from 1 December 2013 continue, with consultation on draft regulations that will underpin the revised program currently taking place. It is envisaged that the regulations will be considered for adoption at the March meetings of the Board and Council, to allow for communication with groups such as Fellows, Trainees, jurisdictions and others identified through the original consultation process to occur in the following months. Part of the work flowing from the framing of regulations for the revised training program will be the identification of aspects of the new program that the College will look to incorporate into the requirements that apply to Trainees currently in the training program, as well as the development of enhanced electronic systems for recording and checking progress of Trainees through the program.

In the last issue I foreshadowed work on the College's Strategic Plan for the period November 2012 to November 2014, with a planning meeting that involved members of the Board as well as senior College staff having been held during the course of Council Week in November 2012. Since then, further work has been undertaken and the Strategic Plan was considered by the Board at its meeting in early February with a recommendation on its adoption to be put to Council at its meeting in March. The Plan is wide-ranging in scope, reflecting the priorities for the College during the relevant period, as well as providing an indication of the range of activities that currently constitute the work of the College.

Work continues to grow, in not only areas that may be considered 'core' College activity, but also in areas such as externally funded projects. In the latter part of 2012, the College was advised of the continuation of funding for the Specialist Obstetrician Locum Scheme (SOLS), this time under the banner of the Rural Obstetric and Anaesthetic Locum Scheme (ROALS) to reflect the inclusion of general practitioner anaesthetic locums in to the scheme as administered through RANZCOG, with the amount involved in the contract over the period to 30 June 2014 some $4 million. Combined with funding for extending the administration and associated educational project and specialist international medical graduate (SIMG) up-skilling work conducted under the Specialist Training Program, this represents a healthy state of targeted externally funded projects being conducted by and through RANZCOG.

Another area in which the College has been cognisant of a need to increase its activities has been the evaluation of its training programs. As part of this, an Evaluation Framework document for the College was approved by the Board at its meeting held in early February. This document provides an overarching framework by which all evaluations conducted by the College can be guided. While Year 2 and Year 4 Trainee surveys, confidential feedback from Trainees and some other evaluation mechanisms have been in place for some time, this framework brings together existing evaluation plans and tools, and incorporates them into a unified model of quality improvement. Initial work will be progressed in relation to evaluation of the FRANZCOG Training Program, and members will be informed of developments in this area in future *O&G Magazine* columns.

The College continues to rely on the members of regional committees, those who supervise Trainees, assist in examinations and otherwise contribute to College activities, whether formally or informally. All such contributions are greatly appreciated and the College is aware that they can never be taken for granted. As the work of the College grows, there is, however, a need for the involvement of an increasing number of members and I would urge any RANZCOG member who is interested in becoming involved to contact a member of staff or a member of their regional committee, including Councillors, for further information. The rewards, while perhaps not tangible, nor even readily apparent in advance, can be satisfying and long-lasting on both a professional and personal level.

Editorial

Dr Alexa Bendall
FRANZCOG Trainee

The death of a mother: tragic, horrific, incomprehensible and probably the most terrifying scenario that we face as medical professionals working in obstetrics.

The sad fact remains: women continue to die in childbirth and the puerperium. Birth, pregnancy and the postpartum period are still dangerous. These facts cannot be pushed to the back of our consciousness and, moreover, as responsible clinicians, we must confront them in order to stop women dying from preventable or treatable conditions.

The maternal mortality rate (MMR) in Australia currently sits at 8.4 per 100 000 live births. Thanks to improved recognition and treatment of high-risk presentations in the antenatal, intrapartum and postpartum periods, the MMR in Australia has dropped dramatically compared to even 50 years ago and is exponentially better than in areas with poor access to obstetric and midwifery care. For comparison one only needs to look to Sub-Saharan Africa, where the maternal mortality rate is just under 500 per 100 00 live births.

As outlined by Dr Jayaratnam, in his article on maternal 'near miss' events, the dramatic improvement in the MMR in the developed world does not mean we can rest on our laurels. There is always room for improvement. By analysing cases of women who suffered severe morbidity we are able to identify areas in which we can continue to improve as we strive to provide the best care possible for women.

In this issue of *O&G Magazine*, we examine a number of the more common causes of maternal mortality, ranging from the elusive and terror-inducing amniotic fluid embolus to the more clearly understood, but no less terrifying, postpartum haemorrhage. We discuss eclampsia/pre-eclampsia, cardiac disease, thromboembolic disease and sepsis. Importantly, this issue also covers what is sometimes the forgotten killer – psychiatric disease – as well as the crucial role of teamwork in managing life-threatening situations in obstetric care.

For all our readers in general practice there is an article that is of particular pertinence about the crucial role you can play, as primary care providers, in detecting women at risk and initiating treatment where suitable, including organising referral with appropriate urgency.

It is our hope that everyone can learn something from this issue and that we can work towards lowering the maternal mortality rate further. Even one death is one too many.

On a less solemn, but no less important note, the accuracy and relevance of the material published in this magazine is of the utmost importance to the *O&G Magazine* committee. In the interest of achieving these objectives, we are pleased to announce that a number of articles in each issue will now be peer reviewed. In the coming months we will be inviting select members of the College to become peer reviewers. We also welcome any interested members to contact us directly.

Reporting maternal death in Australia

The challenges and benefits of collecting national data.

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Maternal deaths are rare events in Australia, with an estimated 8.4 deaths per 100 000 women who give birth.[1] This statistic compares favourably with New Zealand (2006–10 maternal mortality rate 17.8 [95 per cent CI 13.5–23] maternal deaths per 100, 000 live births[2]) and is significantly lower than Australia's regional neighbours in Timor Este and the Oceania region (maternal mortality rate 300 maternal deaths per 100 000 live births and 200 maternal deaths per 100 000 live births, respectively[2]) and, internationally, with Sub-Saharan Africa, where there are almost 500 maternal deaths per 100 000 live births.[2]

Australia instigated the practice of reviewing and reporting maternal deaths for the triennium 1964–6; maternal deaths have decreased by nearly two-thirds since this time.[1] The next maternal death report, covering the years 2006–10, will be published in 2013. These national reports are informed by the UK's 'Saving Mothers' Lives' series, which internationally are considered the gold standard in obstetric mortality review. 'Observational methodology is used to generate hypotheses, show trend lines and make recommendations that may lead to improvements in maternal health.'[3] Key to this methodology is the adoption of a focus on learning, not simply the counting and reporting of numbers. 'Maternal Deaths in Australia 2006–10' report is currently being prepared as an output of the Maternal Mortality project. The Maternal Mortality project is part of the larger National Maternity Data Development Project funded by the Department of Health and Ageing that the National Perinatal Epidemiology and Statistics Unit (NPESU) is undertaking in collaboration with the Australian Institute of Health and Welfare.

Identifying maternal deaths

There is no national standard for notification of maternal deaths in Australia, with methods varying by jurisdictions. Maternal deaths are usually notified to the Department of Health, or equivalent, in each jurisdiction. The most common form of identification is direct notification by health professionals and hospitals involved in a death. In other cases, deaths may be reported by the coroner or the Registrar of Births, Deaths and Marriages. The latter follows identification by use of the 'pregnancy tick box' at death registration (which indicates if a woman has been pregnant within the year preceding her death). Some jurisdictions search for maternal deaths using hospital administrative data collections. Nationally, there is no mandatory reporting requirement, meaning health professionals are not obliged to report a maternal death or to provide information for use at confidential enquiry. An overall lack of clarity and consistency in reporting requirements means maternal deaths have been historically under-reported in Australia, with evidence showing up to 20 per cent of maternal deaths are never reported or investigated.[4]

Jurisdictional level reporting

Following notification of a maternal death to the relevant authority, all deaths undergo some form of a confidential enquiry. These enquiries are undertaken by specifically convened jurisdictional Maternal Mortality Committees (MMCs). Information is collected through audit of the medical notes, inclusion of the coroner or autopsy data and through interview with the relevant staff associated with the case. Each jurisdiction has varying policy, legislation and methods for collecting data on maternal deaths and in turn the quality and amount of data collected varies by jurisdiction, as does the consistency and timeliness of review. The confidential enquiry can be subject to significant delays, with some reviews occurring up to two years after the death has occurred. At the national level, these inconsistencies across jurisdictions mean the details of any particular woman and any conclusions or classifications made by jurisdictional committees can be difficult to compare.

National-level reporting

In contrast to the UK and New Zealand, national maternal death review is not the primary method of review in Australia and is undertaken on an ad hoc basis, subject to funding. A national reporting form, which collects a standard set of information on all deaths, is distributed to each jurisdiction and data commencing with the 1994–6 triennium has been provided to the NPESU on a voluntary basis. Limited resources impact on the timeliness of supply, the quality of information and the capacity to draw useful comparisons or conclusions. Legislative privacy restrictions that are intended to protect the privacy of individual women and promote open disclosure at confidential enquiry result in the limited transfer

of information from the jurisdictional committees to the national committees. For the 2006–10 maternal death report, the data provided to the NPESU varied by jurisdictions, which impacts the utility of national reporting.

Recent developments in maternal death review

In 2011, the National Maternal Mortality Advisory Committee was reconvened. This committee – mainly consisting of senior clinicians, many of whom are dedicated to the improvement of maternal health, with rich experience in maternal death review – has provided an excellent opportunity for discussion and exploration of options for the improvement of the current maternal death reporting system. Progress to date includes the development and piloting of a nationally standardised National Maternal Death Reporting Form, intended for implementation in 2013; the development of a nationally standardised system of classification for deaths related to psychosocial morbidity that is currently under review by the MMCs; and the undertaking of a national data linkage study on maternal deaths that, for the first time, will provide an accurate assessment of the number of maternal and late maternal deaths in Australia. It is intended that these developments will improve the consistency of maternal death ascertainment and data collection. Novel collaborations with comparable patient death enquiry bodies such as the Australia and New Zealand Audit of Surgical Mortality and the New South Wales Clinical Excellence Commission, aim to facilitate the development of effective national-level reporting in a state-based system.

The proposed introduction of a rapid maternal death surveillance system represents a significant change to the process of national maternal death review. The Australasian Maternity Outcomes Surveillance System proposes to collect monthly notifications on in-hospital maternal deaths Australia-wide. This system will allow rapid response to emerging trends in maternal mortality, such as the 2009 H1N1 epidemic. It may also serve to raise the culture of awareness of maternal death reporting at the hospital level with the long-term effect of improving jurisdictional maternal death notification.

Conclusion

Australia has one of the lowest maternal mortality rates in the world. Confidential enquiry is an internationally adopted method of review for maternal deaths. Work is underway to develop a systematic national approach for the reporting of maternal deaths in Australia. Mandatory notification requirements, improved consistency in the information collected and the responsible sharing of information between coroners, jurisdictional committees and national committees has the capacity to significantly enhance the current system. Recent developments in the process of national review indicate a climate of progress and change. Continued support and leadership from RANZCOG and the Australian College of Midwives is essential in the development of sustainable national reporting system of maternal deaths and in the adoption of a focus on learning and translation of findings into practice.

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Maternal mortality in New Zealand

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We have come a long way in the six years since maternal death investigation was reinstated in New Zealand.

The recent history of maternal mortality investigations in New Zealand has been outlined in previous issues of *O&G Magazine*.[1,2]

Under the New Zealand Public Health & Disability Act 2000, authority was given to set up mortality committees. These now are four in number: Child and Youth; Perinatal and Maternal; Family Violence; and Perioperative. Initially, the mortality committees reported to the Minister of Health, but since 2010 they have reported to the Health, Quality and Safety Commission (HQSC).

In 2006, the Perinatal and Maternal Mortality Review Committee (PMMRC) established the Maternal Mortality Review Working Group, a multidisciplinary group – including obstetrics, midwifery, anaesthetics, obstetric medicine, pathology, psychiatry and a health manager – to look at maternal deaths. When the working group started, there had been no in-depth review of maternal deaths in New Zealand since 1996, when the then Maternal Deaths Assessment Committee reported on the triennium 1989–91. The working group reports to the PMMRC, which in turn reports to the HQSC, which then advises the Minister of Health.

The PMMRC set up a network of local coordinators in all district health boards to identify and notify cases. Deaths are also drawn to the PMMRC's attention by clinicians, the Coronial Service, media reports, death notices and by cross-referencing notified deaths with the mortality collection of Births, Deaths and Marriages. Previous reports reported delay in identification, or non-identification, of cases. It is believed that currently the ascertainment of cases in New Zealand is as complete as it can be. Information is collected by review of notes, by a comprehensive maternal death data form and reports of individual practitioners. Where available coronial findings and any serious or sentinel event investigations, or internal enquiries are also reviewed. The cases are reviewed in detail by at least two members of the working group and all cases then come for discussion at the working group meetings, which are held three times a year. Following the review of cases, contributory factors are identified and potential avoidability is determined.

Table 1. Maternal mortality ratio (per 100 000 maternities) and cause of maternal death 2006–10.

Classification and cause of maternal death	2006	2007	2008	2009	2010	2006–2010		
	n	n	n	n	n	n	%	Ratio (95% CI)
Direct maternal death	**6**	**5**	**4**	**5**	**1**	**21**	**37**	**6.5 (4.1–10.0)**
Amniotic fluid embolism	3	–	1	4	1	9		
Postpartum haemorrhage	1	1	1	–	–	3		
Pulmonary embolism	–	1	1*	–	–	2		
Peripartum cardiomyopathy	–	1	–	–	–	1		
Pre-eclampsia/eclampsia		2	1	1		4		
Sepsis	2	–	–	–	–	2		
Indirect maternal death	**7**	**5**	**5**	**9**	**7**	**33**	**58**	**10.3 (7.1–14.4)**
Pre-existing medical condition	2	4	2	1	2	11		
Non-obstetric sepsis	–	1	–	5	1	7		
Intracranial haemorrhage	1	–	–	–	1	2		
Suicide	4	–	3	3	3	13		
Unclassifiable	**2**	**1**	**–**	**–**	**–**	**3**	**5**	**0.94 (0.19–2.7)**
Total	**15**	**11**	**9**	**14**	**8**	**57**		**17.8**
*Pulmonary embolism and sepsis								(13.5–23.0)

The PMMRC now has accumulated data for the five years 2006–10 and these data were reported in June 2012, with the release of the PMMRC Sixth Annual Report.[3] Work is well underway for reporting 2011 deaths and this report should be available for publication in the middle of 2013.

In the five-year period 2006–10, 57 deaths were recorded, giving a maternal mortality ratio of 17.8 (95% CI 13.5-23) per 100 000 maternities. The three leading causes of maternal death in New Zealand in this five-year period were suicide, pre-existing medical conditions and amniotic fluid embolism. Contributing factors were identified in 53 per cent of deaths and 32 per cent of deaths were thought to be potentially avoidable.[4] These included contributory factors relating to organisational and/or management (32 per cent of deaths), relating to personnel (30 per cent of deaths) and barriers to access to, or engagement with, care (37 per cent of deaths). The most frequent barriers recorded were no or infrequent antenatal care, late booking and lack of recognition of the complexity or seriousness of a condition by the woman or her family.

In 2006–10, Maori and Pacific mothers were more likely than New Zealand European mothers to die in pregnancy or in the first six weeks postpartum. The estimate for Indian mothers was also higher than for New Zealand mothers, but this difference was not statistically significant.

Some issues are common to all maternal death reports from similar countries. Suicide was an unexpected leading cause of maternal death. There were 13 cases and, in addition, a further four late postpartum suicide deaths (from 42 days to one year) were reviewed, (even though these cases do not contribute to the maternal mortality ratio). Of the 13 deaths within the New Zealand definition of maternal mortality, seven occurred during pregnancy and six postpartum or post-termination of pregnancy. Eleven of the 13 deaths from suicide were by violent means. Over half had a history of alcohol or drug usage that included, in four, use at the time of death. It was noted that there were difficulties in providing appropriate care to women when information was kept by different organisations or practices, and where that information was not available to other practitioners involved in the woman's care. Following these reviews, the PMMRC recommended the development of a comprehensive perinatal and infant mental health service aimed at reducing maternal mortality and morbidity. A comprehensive perinatal and infant mental health service includes:

- screening and assessment;
- timely interventions, including case management, transition planning and referrals;
- access to respite care and specialist inpatient care for mothers and babies; and
- consultation and liaison services within the health system and with other agencies, for example, primary care and termination of pregnancy services.

The PMMRC has also recommended that a mother and baby unit be provided in the North Island of New Zealand. One of the earliest workshops held by the PMMRC focused solely on maternal mental health issues, and it has been on the program of subsequent workshops. A recent Ministry of Health report titled 'Healthy Beginnings: developing perinatal and infant mental health services in New Zealand' (published January 2012) also highlighted the need for a mother and baby unit in the North Island and an analysis of current maternal health services in the Auckland Region is in the process of reporting on this recommendation.

There were a number of coincidental deaths in 2006–11, six of which were due to motor vehicle crashes. The committee felt that four of these six deaths were potentially avoidable had seat belts been worn and, as a result, a poster demonstrating wearing of seat belts in pregnancy has been developed and is available from the HQSC.

There are a number of parallel processes in investigation of maternal deaths. Local review of maternal deaths is important and valuable, particularly in looking for correctable systemic contributing causes where these are present, but is done variably depending on locality. The coronial process operates to its own recently revised legislation. (Deaths that must be reported to the Coroner include any death that occurred while the woman concerned was giving birth, or that appears to have been a result of that woman being pregnant or giving birth.) While accident compensation legislation has largely removed liability in tort, there still may be an investigation by the Accident Compensation Corporation to see if cover is available. The Health & Disability Commissioner has a parallel, but different, role in investigation of health complaints. Finally, there is the potential for referral to the Health Practitioners Disciplinary Tribunal and, ultimately, regulatory bodies.

The PMMRC holds a national workshop when the PMMRC Report is released. This year's workshop will be held at Te Papa in Wellington on 12 June.

Is the PMMRC reporting worthwhile?

In late 2012, the HQSC commissioned a survey of health professionals regarding readership and usage of the Sixth Annual Report of the PMMRC reporting 2010 perinatal data and 2006–10 maternal data. Although the sample was small, all rated the report 'very' or 'quite' helpful in raising awareness about perinatal and maternal mortality; as a tool to inform practice, 97 per cent rated the report 'very' or 'quite' helpful; and, as a tool to influence change, 56 per cent rated the report 'very helpful' and 31 per cent 'quite helpful'.

The full report and further information on the PMMRC can be found on the HQSC website: http://www.hqsc.govt.nz/our-programmes/mrc/pmmrc .

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Learning lessons in the UK

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The UK's Confidential Enquiries into Maternal Deaths has been in existence for more than 50 years. In that time much has changed, but the learning process remains the same.

Death in pregnancy is rare in developed countries such as Australia and the UK, but it still happens. When it does, our reaction, after the first shock, is to investigate and try to prevent the same thing happening again. Learning lessons from tragedy, however, is no easy task. There is a natural tendency to look for scapegoats. Organisations are reluctant to find fault with their own systems. Individuals directly involved feel guilty and either reproach themselves needlessly or become over-defensive when facing official enquiries.

Having seen these reactions many times in various countries, I am full of admiration for the people who set up the UK's Confidential Enquiries into Maternal Deaths (CEMD) in the early 1950s. They might easily have decided not to bother – maternal mortality had already fallen by three-quarters in 15 years, thanks to sulphonamide and penicillin eliminating puerperal sepsis – but instead of feeling complacent, that optimistic post-war generation wanted to tackle the remaining causes.

Their new initiative involved a national panel of obstetricians receiving detailed summaries of all maternal deaths across the country. The Ministry of Health published a report of their findings every three years, after which the papers on individual cases were destroyed. The published report contained no identifying details, so the staff involved could be completely frank about what had happened.

My generation of obstetricians grew up with this system, established in an era when government and doctors trusted each other. Confidentiality was never breached (though lawyers sometimes tried) and professionals knew their anonymous reports would be used to good effect. There was no extra pay for members of the national panel or for the Regional Assessors who organised local reporting: an invitation to do the job was a mark of respect from one's colleagues and was reward enough.

The triennial reports became best-sellers in the Royal College of Obstetricians and Gynaecologists (RCOG) bookshop, not just because they were required reading for the MRCOG, but because they told the truth. Death is not an arbitrary outcome measure dreamed up by managers. It makes unpleasant reading, though, and during my 20 years as a Central Assessor there were times when I was moved to tears by first-hand accounts from midwives and doctors, anonymised by marker pen or correction fluid even before their reports reached me.

During those 20 years there were many changes, though not to the basics such as confidentiality. The number of specialties involved in the panel increased, finally including a psychiatrist and a family doctor as well as a pathologist and members from the acute hospital services. The arrival of midwives was important and prompted a handwritten letter from the ex-Chief Medical Officer who had set up the enquiry (and was now approaching his 100th birthday). He had wanted midwives on the original panel in 1952, but had been overruled. The midwives are now the UK CEMD's strongest supporters.

The purpose has always been to learn lessons, as shown by the title of the most recent reports, 'Saving Mothers' Lives'. How do we do this? The process, now known as the 'audit loop', has five steps, the first being identification of cases. We have always insisted that every maternal death, from around the time of conception until well after delivery, must be reported. If you try to be selective you risk missing important messages, particularly about poor people. It can be hard work to find out about the deaths of socially excluded women outside hospital, and not all countries want to do so. Unfeasibly low maternal mortality rates look good in international comparisons, but you can be sure that they don't include the deaths of migrants or even the indigent poor.

Step two is to gather information. We encourage those involved to tell us not only what happened, but also what lessons they have learned. Their thoughts can be insightful and may be quoted in the report. By contrast, the paperwork from a hospital's own internal investigation, often sent to us in full nowadays, is rarely helpful. It is not that hospitals want to cover things up, but proper investigation of a maternal death needs experience of many similar events, which is now, thankfully, impossible for a single hospital to achieve. This is one reason why a national, or at least state-wide, enquiry is essential.

Step three of the audit loop is to analyse the results. Statistical help is needed, but the CEMD has always relied mainly on clinicians. Cases are grouped by cause – hypertensive disorders, sepsis, haemorrhage and so forth – and when each group is looked at patterns begin to emerge. You start to see who is at risk and what are the first signs of a life-threatening problem, and you can suggest what steps should be taken to avert disaster.

This pattern recognition is just like gaining clinical experience and teaches powerful lessons. Over the years we learned, for example, that the only symptom of ectopic pregnancy may be diarrhoea, fatally dismissed as a 'tummy bug'. We discovered how often a woman with postpartum psychosis seems to be managing well until she kills herself in a chillingly violent way. Recently, we found that some ethnic groups, even when well integrated in the UK, still have persistently high maternal mortality rates.[1]

Step four is to formulate recommendations. In the past, readers were left to decide for themselves what action to take, but in recent years we have become more prescriptive. To my surprise, clinicians like being told what to do. Recommendations are now targeted at

specific groups – clinicians, managers and politicians. Nobody can be forced to comply, but external pressure to do so comes when recommendations are incorporated into hospital protocols, RCOG guidelines and the National Health Service's indemnity scheme.

Nevertheless, the final step – implementation – remains the most difficult. It requires a readership that pays attention to the report. Some specialties, such as obstetric anaesthesia, have a tradition of doing so, but others, such as emergency medicine, do not. Even for obstetricians and midwives, the sheer number of recommendations can be daunting, which is why we recently developed a 'top ten' list of recommendations that must be prioritised.

In the National Health Service, clinicians cannot improve the service without the co-operation of managers and pressure from politicians. Engaging the attention of these groups requires some flair, and publicity helps. The launch of each report is a media event, which needs careful handling. A well-informed public is fundamental to improving women's health, but we don't want to scare women with a litany of fatal cases.

The CEMD has had its detractors, particularly in the early days of evidence-based medicine, when the process was criticised for being based on expert opinion rather than on randomised controlled trials. There is, however, a symbiotic relationship between the CEMD, epidemiology and laboratory science. The reference list of many a research paper shows it was inspired by CEMD findings, and the CEMD's recommendations are informed by the latest research. For example, in 1994, we drew attention to current research on hypertensive disorders[2], and our next report[3] endorsed treatment with magnesium sulphate, following a recent major RCT.[4] With some life-threatening conditions, however, adequately powered trials are not possible and there is no alternative to expert opinion.

Is there evidence that the CEMD has succeeded in saving mothers' lives? Without control groups blinded to the recommendations it is hard to be certain, but the timing of some improvements is highly suggestive. For example, from 1991 onwards thromboembolism was, by an increasing margin, the leading cause of direct maternal deaths in the UK. After the RCOG issued recommendations on thromboprophylaxis at caesarean section there was a sharp fall in deaths in this group, and the CEMD recommended extending the guideline to other women at risk. In 2004, the RCOG published a new thromboprophylaxis guideline that was followed by a statistically significant fall in deaths from thromboembolism.[1]

The fact remains, however, that despite its effectiveness and universal clinical support, the UK CEMD has been around for over half a century. Its longevity became an increasing irritant to a health service that likes to promote change, and in 2011 it sank into a bureaucratic quicksand and disappeared. Paradoxically, this happened just as many other countries, from South Africa to Kazakhstan[5,6], were adopting and adapting the UK model. Fortunately, the UK Enquiry is re-emerging under the auspices of the National Perinatal Epidemiology Unit and there is good reason to hope that under new management the process of saving mothers' lives will continue.[7]

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Maternal near miss

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Examination of cases of women who survived a severe complication of pregnancy, childbirth or the puerperium is increasingly being recognised as a potentially useful tool in assessing the quality of healthcare.

A maternal death is one of the most devastating complications in obstetrics, with wide-ranging implications for both the family and the staff involved. Thankfully, maternal mortality is now a rare event in Australia, which has one of the world's lowest maternal mortality rate (MMR).[1] Mortality rates in the developed world have plateaued over the last 40 years, while mortality rates in the developing world, though still high, have shown significant improvement over this time.[2] The epidemiology of maternal deaths has also changed, particularly in the developed world, where indirect obstetric causes such as venous thromboembolism (VTE), psychiatric disease and cardiac disease have overtaken direct obstetric causes such as postpartum haemorrhage (PPH) and pre-eclampsia as primary causes of mortality. One of the many primary reasons for improvement in the maternal mortality rates has been an understanding of the aetiology of maternal deaths and a subsequent improvement in clinical care. Confidential enquiries into maternal deaths (CEMD) have, in this regard, been a valuable tool, providing a thorough evaluation of the quality of healthcare and recommending improvements.[3]

However, as the MMR has improved, reviews of maternal deaths, though useful, do not necessarily reflect the scope of complications in obstetric practice. Resource-rich countries, such as Australia, have a low MMR and to attain a large enough sample to identify trends in quality of maternal healthcare takes from three to ten years.[3] This is reflected in the infrequent publications of the Australian Institute of Health and Welfare maternal mortality statistics. Furthermore, the circumstances related to a maternal death in a developed country are often very particular to the individual case and the value of this information for general use might be limited. This is certainly the case in many developed countries where maternal deaths tend to be the result of rare complications and an indication of the complexity and unpredictability of the condition, for example, amniotic fluid embolism or complex cardiac disease. In countries where maternal deaths are relatively frequent, CEMD give a better indication of problems in the health system, provided most deaths occur within health institutions. However, in resource-poor settings the vast majority of deliveries are still undertaken in the 'home' setting, thus creating difficulties with collection of data. Additionally, maternal deaths per institution in these countries are still uncommon, even in countries with a high MMR. Therefore, the relevance of such deaths to small district units may not highlight local issues and processes that require examination.

Review of cases of near miss has the potential to highlight both deficiencies and positive elements in the provision of obstetric services and to provide information about the epidemiology of

Table 1. The proposed WHO near-miss criteria

Clinical criteria	
Acute cyanosis	Loss of consciousness lasting >12 hours
Gasping	Loss of consciousness and absence of pulse/heart beat
Respiratory rate> 40 or <6/min	Stroke
Shock	Uncontrolled fit/total paralysis
Oliguria	Jaundice in the presence of pre-eclampsia
Clotting failure	
Laboratory-based criteria	
Oxygen sats < 50% for >60 mins	Ph <7.1
PaO_2/FiO_2 < 200mmHg	Lactate > 5
Creatinine > 300μmol/l or >3.5mg/dl	Acute thrombocytopenia (< 50000 platelets)
Bilirubin > 100μmol/l or > 6.0 mg/dl	Loss of consciousness and the presence of glucose and ketoacids in urine
Management-based criteria	
Use of continuous vasoactive drugs	Intubation and ventilation for > 60 mins not related to anesthesia
Hysterectomy following infection or haemorrhage	Dialysis for acute renal failure
Transfusion > 5 U of red cell transfusion	Requiring CPR

Say L, Souza, JP, Pattinson RC. Maternal near miss – towards a standard tool for monitoring quality of maternal health care. Best Practice and Research Clinical Obstetrics and Gynaecology. *23 (2009) 287-296.*

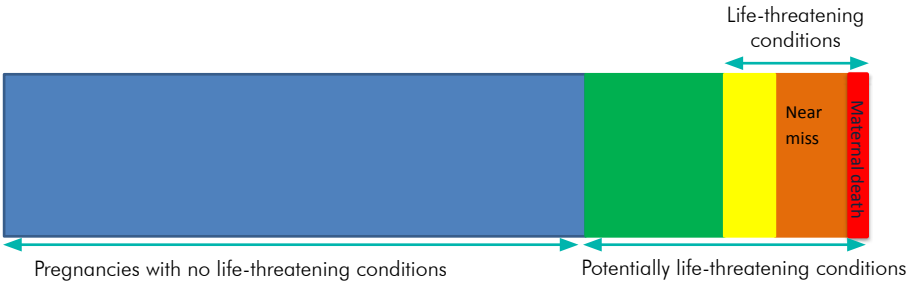

Figure 1. Classification of pregnancies at CBH.

severe obstetric complications.[4] This is analogous to the review of neonatal near misses that highlights the potential for improvements in intrapartum care.[5] Furthermore, as a result of their near-death experience and often complicated recoveries, near misses do have significant psychological and physical morbidity, as well as issues related to resource utilisation that need to be continually addressed to attain the best clinical outcomes. Auditing near misses ensures we do not forget those lessons from our past that have resulted in the dramatic reductions in maternal mortality.[3]

The concept of maternal near miss is not a new one with it known by various names, such as severe acute maternal morbidity (SAMM), over the last 20 years. In recent years, a considerable body of research has focused on the analysis of near miss with a view to understanding health system failures in relation to obstetric care.[6] Some clinicians argue that the auditing of cases of severe adverse maternal events already occurs within institutions, with root cause analysis (RCA), and at a national level, with the Australasian Maternity Outcomes Surveillance System (AMOSS). While mechanisms exist within most hospitals for the reporting of severe adverse events and for the performance of RCAs, these are designed to identify deficiencies within the hospital system or in the provision of care and thus do not incorporate suitable mechanisms for the detection of all individual cases of maternal near miss.[4] The AMOSS is now established in hospitals across Australia with the aims of improving knowledge of rare obstetric disorders and their management in Australia, and translating research findings into policy, clinical guidelines and educational resources for clinicians. However, AMOSS has been developed to deal with national trends and rare events and not audit data in individual units or area health services.[4]

Despite the collection of data, assessment of obstetric morbidity has been hampered by the various criteria used to define a near miss. What had been lacking is a standardised definition of a near miss, with criteria that can be applied in a systematic and consistent manner allowing a standardised comparison between units and within obstetric units over time. A WHO working party addressed this issue in 2009[6] and came up with a number of parameters for inclusion as a near miss (see Table 1).

The use of these broad categorisations in defining near miss has limitations and advantages, but what the categorisations encompass is a set of criteria that are consistent, validated and can be applied in both resource-rich and resource-poor settings.

A study at the Cairns Base Hospital (CBH), in Far North Queensland (FNQ), that began in 2009, was the first in Australia to incorporate the newly developed WHO criteria. At CBH we were concerned with adapting this tool to assess our own obstetric performance in the region. Collecting near miss data involved identifying those pregnancies that may be at risk, such as ruptured ectopic pregnancies or HELLP syndrome, and then further refining the process using the WHO criteria proposed to identify those cases that are true near misses (see Figure 1).

In identifying pregnancies that may be at risk, a combination of clinical and management-based criteria from the WHO working party were incorporated (see Table 2).

Table 2. CBH criteria for potential cases of obstetric near misses

Any APH requiring delivery for maternal reasons	Any admission to ICU
Any PPH requiring operative interventions	Severe pre-eclampsia complicated by HELLP, renal failure or other major morbidity
Any postnatal cases that returned to OT	Any pulmonary embolus
Any ruptured ectopic pregnancy	Any patient experiencing severe shock or collapse
Any condition that required immediate medical assessment	

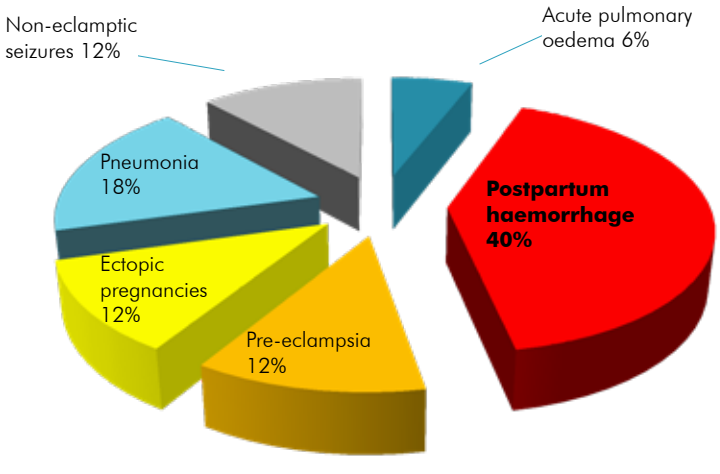

Figure 2. Obstetric near misses at CBH

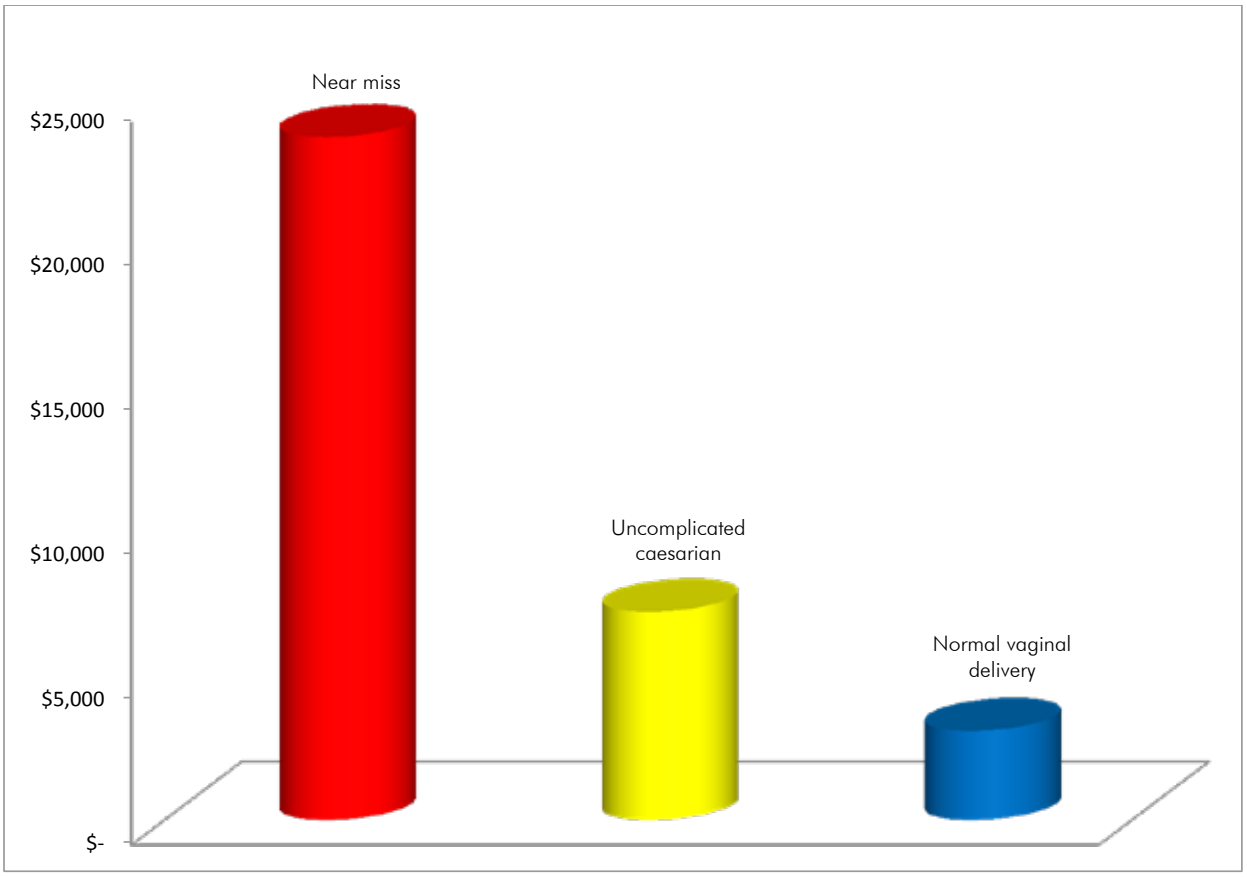

Figure 3. Cost of admission at CBH (comparison of near miss with uncomplicated delivery). Cost of admission was calculated by duration of stay in hospital, but did not incorporate other miscellaneous expenses such as use of blood products/factor 7 that would have significant increased the costs of cases of near miss.

Over the 12 months of the study, we found 58 cases considered by staff to fulfil the criteria outlined in Table 1. Of those cases, 18 were subsequently classified as not potentially life threatening and as responding promptly to care; 40 were considered to fall in the category of having potentially life-threatening conditions; of these 17 met one or more of the WHO criteria for having life-threatening conditions and were classified as true near misses (see Figure 2).

The most common cause of a near miss was PPH followed by pre-eclampsia and VTE. These conditions previously often resulted in death, but with improved management have become rare causes of maternal mortality in the developed world. Non-obstetric near misses accounted for a significant number of cases, reflecting the high level of comorbidity in FNQ. Additionally, Indigenous women were over represented in our data. The significant utilisation of resources by cases of near miss is also highlighted with the cost of admission being five- to ten-fold greater than an uncomplicated delivery (see Figure 3).

The results indicate the continued significant morbidity of these conditions as well as the need for ongoing training and simulation sessions for obstetric staff in the recognition and management of severe obstetric morbidity. It also highlights the significant resource allocation that is required in the management of near misses and the continued need to be alert to the presence of possible obstetric complications.

In summary, while review of maternal deaths and identification of avoidable contributing factors will undoubtedly continue to occur both nationally and in individual hospitals, incorporation of maternal near-miss analysis in assessing the process of obstetric care will be a valuable contribution in taking necessary action to improve the quality of care. The addition of near-miss audits will allow the care of critically ill women to be analysed, deficiencies in the provision of care to be identified and comparison within and between institutions and countries to be carried out over time. This will, ultimately, improve the quality of obstetric care and further reduce maternal morbidity and mortality.

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Indication For use in females of childbearing age for the medical termination of a developing intrauterine pregnancy in sequential combination with a prostaglandin analogue, up to 49 days of gestation. **Contraindications** Known hypersensitivity to mifepristone or any excipients; chronic adrenal failure; severe disease necessitating exogenous steroid administration; known or suspected hypocoagulation diseases or treatment with anticoagulants; uncertainty about pregnancy age; suspected ectopic pregnancy; contraindication to prostaglandin. **Precautions** Remove any intrauterine contraceptive device before prescribing mifepristone. Adjust long-term corticosteroid therapy because of the potential decrease in corticosteroid efficacy during the 3–4 days after mifepristone intake. Use with caution in patients with suspected acute adrenal failure or risk factors for, or established, cardiovascular disease. **Special warnings and precautions** Not recommended in patients with anaemia, renal failure, hepatic impairment or failure, or malnutrition. Exclude ectopic pregnancy and confirm gestation before prescribing mifepristone. Inform patients of the need to combine treatment with a prostaglandin analogue, the need for follow-up within 14–21 days after intake to confirm complete expulsion, and the non-negligible risk of failure, which may require termination by another method; provide patients with appropriate medications as necessary on discharge; fully counsel patients regarding the likely signs and symptoms of mifepristone, the prostaglandin analogue, and medical abortion; and ensure patients have direct access to the treatment centre. Expulsion may occur before intake of the prostaglandin analogue (about 3% of cases). This does not preclude need for follow-up. The following risks must be explained to all patients: ***Non-negligible risk of failure:*** Occurs in up to 7% of cases (with about 1% having continuing pregnancy), making follow-up mandatory. ***Bleeding:*** Prolonged vaginal bleeding, which may be heavy, can occur for 10–16 days after intake. Bleeding occurs in almost all cases and is not proof of complete expulsion. Excessive bleeding may require haemostatic curettage (up to 5% of cases) or a blood transfusion (0.1–0.2% of cases). Inform patients to remain near the treatment centre with precise instructions on who to contact and where to go if any problems emerge, particularly very heavy vaginal bleeding, until complete expulsion has been confirmed. Bleeding (even light) that persists beyond the 14–21-day follow-up should be checked within a few days until it has stopped. Persistent vaginal bleeding could signify incomplete abortion, ongoing pregnancy, or unnoticed extra-uterine pregnancy, and appropriate treatment should be considered. Patients with haemostatic disorders with hypocoagulability or anaemia should seek advice from specialised consultants if considering abortion. ***Infection:*** Serious bacterial infection, including fatal septic shock (very rare), has been reported following the use of mifepristone; however, no causal relationship has been established. Fever, severe abdominal pain, or pelvic tenderness in the days after a medical abortion may indicate infection. Exclude sepsis if abdominal pain, discomfort, or general malaise is reported more than 24 hours after intake. Very rarely, deaths have been reported in patients who presented without fever, with or without abdominal pain, but with leukocytosis with a marked left shift, tachycardia, haemoconcentration, and general malaise. Prevent Rhesus allo-immunisation if patient is Rhesus negative. **Interactions with other medicines** Ketoconazole, itraconazole, erythromycin, and grapefruit juice may inhibit mifepristone metabolism. Rifampicin, dexamethasone, St John's Wort, and certain anticonvulsants may induce mifepristone metabolism. Use with caution when administering with drugs that are CYP3A4 substrates and have a narrow therapeutic range. **Adverse effects** Very common: nausea, vomiting, diarrhoea, dizziness, gastric discomfort, abdominal pain, headache, vaginal bleeding, uterine spasm, fatigue, and chills and fever. Common: prolonged post-abortion bleeding, spotting, severe haemorrhage, endometritis, breast tenderness, heavy bleeding, and fainting. For uncommon, and rare and very rare adverse events, please refer to the full PI. Foetal malformations cannot be excluded if medical abortion fails and pregnancy persists. **Dosage and administration** One 200 mg tablet of mifepristone orally, then a prostaglandin analogue 36–48 hours later.

GyMiso® (Misoprostol) Minimum Product Information

Indications Use in females of childbearing age for the medical termination of a developing intrauterine pregnancy in sequential combination with a mifepristone 200 mg tablet, up to 49 days of gestation. **Contraindications** Known hypersensitivity to misoprostol (or any prostaglandin) or any excipients; known or suspected hypocoagulation diseases or treatment with anticoagulants; uncertain pregnancy age; suspected ectopic pregnancy; contraindications to mifepristone. **Precautions** Remove any intrauterine contraceptive device before intake. Do not administer if patient intends to carry a viable pregnancy to term. Use with caution in patients with risk factors for, or established, cardiovascular disease, or a history of epilepsy or asthma. **Special warnings and precautions** Refer to the Mifepristone Linepharma Minimum PI for special warnings and precautions for medical abortion including the non-negligible risk of failure, bleeding, and infection. **Interactions with other medicines** No drug interactions have been attributed to misoprostol. **Adverse effects** *Misoprostol*: Frequent: nausea, vomiting, diarrhoea, abdominal pain. Very frequent: uterine contractions in the hours after intake, vaginal bleeding, headache, dizziness, chills and fever. For adverse effects resulting from combined mifepristone and misoprostol, refer to adverse effects in the Mifepristone Linepharma Minimum PI. **Dosage and administration** To be taken 36–48 hours after mifepristone. Dosage is four 200 µg tablets taken orally or buccally (2 hours before or after a meal) as one 800 µg dose or two 400 µg doses, ie, two tablets followed 2 hours later by another two tablets. If abortion has not occurred after 1–7 days, a repeat dose of misoprostol may be offered.

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Lessons for general practice

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The Saving Mothers' Lives report makes valuable recommendations for general practitioners.

Chapter 14 of the Centre for Maternal and Child Enquiries (CMACE) report, titled Saving Mothers' Lives, is dedicated to general practice and it makes for interesting reading. The chapter identifies and highlights the key issues for GPs raised by the review of 64 maternal deaths. Although the scope of Australian GPs' involvement in maternity care is more diverse than in the UK, ranging from minimal through to providing advanced operative procedures, the recommendations in the report remain pertinent and our patients will benefit if we heed their advice. I would recommend all GPs read this chapter, along with the 'Top 10' recommendations and the back to basics sections. Consider printing these chapters out and sharing them with colleagues during your next clinical meeting or journal club.

Specific recommendations for general practice

Based on the findings of the review, it is recommended that all GPs should make themselves aware of the clinical features and practice guidelines for the following conditions listed below. Alongside each condition are suggested guidelines:

- Venous thromboembolism and pulmonary embolism:
  - Reducing the Risk of Thrombosis and Embolism during Pregnancy and the Puerperium, RCOG[1]; and
  - Clinical Practice Guideline for the Prevention of Venous Thromboembolism in Patients Admitted to Australian Hospitals, NHMRC.[2]
- Pre-eclampsia and eclampsia:
  - Guidelines for the Management of Hypertensive Disorders of Pregnancy, 2008, SOMANZ[3]; and
  - The Management of Hypertensive Disorders During Pregnancy, NICE.[4]
- Asthma:
  - Asthma Management Handbook, National Asthma Council of Australia[5] ; and
  - British Guideline on the Management of Asthma, British Thoracic Society.[6]
- Mental health:
  - Clinical Practice Guidelines for Depression and Related Disorders – Anxiety, Bipolar Disorder and Puerperal Psychosis – in the Perinatal Period. Beyond Blue[7]; and
  - Antenatal and Postnatal Mental Health, NICE.[8]
- Obesity:
  - Management of Women with Obesity in Pregnancy, RCOG/CMACE.[9]

Learning points arising from specific causes of death

This section of the chapter examines clinical conditions that resulted in maternal death, explores the role of GPs in each case and makes recommendations designed to improve the standard of care. Below, each condition with the formal recommendations is listed along with some additional general comments.

Venous thromboembolism

- Breathlessness is common in pregnancy, but can be the result of a pulmonary embolism, especially if a woman has risk factors.
- Heart rate, $Sa0_2$ and spirometry are useful clinical signs.
- Making, or excluding, a diagnosis of deep vein thrombosis requires hospital-based investigations i.e. CTPA or V/Q scan.
- Women are at risk of venous thromboembolism (VTE) across all trimesters, with the greatest risk during the postpartum period.
- Severe hyperemesis is a risk factor for VTE, owing to dehydration. Admission for rehydration and thromboprophylaxis may be required if it fails to respond adequately to antiemetics.
- Needle-phobic women can respond to psychological treatment to avoid compromising their obstetric or emergency care.

Eclampsia

- Routine observation of urine and blood pressure should be conducted every time a pregnant woman is seen by a GP, especially if she is feeling unwell.
- Jaundice in a pregnant woman requires immediate investigation and admission.
- Antihypertensives should not be commenced without the involvement of the clinician responsible for overseeing the care of the pregnancy.
- Reduced fetal movements, itching, oedema, haematemesis, thirst, nausea, vomiting, epigastric pain, belching and offensive faeces are all symptoms of severe disease.
- Epigastric pain is a common symptom of significant pre-eclampsia.
- HELLP syndrome may be present before other symptom of pre-eclampsia.

Bleeding or abdominal pain in pregnancy

Ectopic pregnancies commonly present with diarrhoea, vomiting and abdominal pain in the absence of vaginal bleeding. Remember: 'Every woman is pregnant until proven otherwise.' Ask about the possibility of pregnancy and, if in any doubt, perform a pregnancy test.

Sepsis

Deaths from genital tract sepsis have risen. Community-acquired group A streptococcal disease is a common cause. Disease can progress rapidly from first symptom to death over 12–24 hours.

- Parenteral penicillin is an appropriate community treatment in a case of puerperal sepsis.
- All pregnant women/recently delivered women should be informed about the risks and signs and symptoms of infection and how to take steps to prevent its transmission.
- All clinicians should be aware of the classic signs and

symptoms, and sepsis must be considered in all recently delivered women who feel unwell and/or have pyrexia.
- Sepsis is often insidious in onset. Vital signs should always be checked in women who have any signs or symptoms of possible infection.

Women with sepsis can deteriorate and die rapidly. Abdominal pain, fever and tachycardia are indications for emergency admission for intravenous antibiotics.

GPs should have a lower threshold for performing a throat swab in respiratory tract infections, along with a lower threshold for prescribing antibiotics in pregnancy. The Centor Criteria can be helpful in deciding which women are more likely to be suffering from a Group A streptococcal infection[10]:
- history of fever;
- tonsillar exudate;
- no cough; and
- tender anterior cervical lymphadenopathy.

Cardiac disease
- Not all chest pain in pregnancy is heartburn: it may be ischaemic, especially if a woman has risk factors.
- Breathlessness in pregnancy may be caused by cardiac conditions and needs to be carefully assessed.

Epilepsy
- Epileptic women of child-bearing age need to be informed of the risks of epilepsy in pregnancy before conception.
- If fits increase or recur in pregnancy, a woman should be referred to a neurologist and be seen within a week.
- Lamotrigine levels need monitoring during pregnancy.
- Pregnant women should be advised to shower rather than bathe and not to lock the bathroom door.
- The Australian Pregnancy Register for Women on Antiepileptic Medication[11] is a voluntary national registry of women who become pregnant while taking antiepileptic drugs (AEDs) with the aim to assess the risks to babies exposed to AEDs, to compare the efficacy of various drugs and to observe possible improvements in management practice over time.

Asthma
Assessment of severity of a woman's asthma is critical and must be communicated to the clinician responsible for pregnancy care. In general, the effect of pregnancy on asthma severity is: one-third improves, one-third is stable and one-third deteriorates. Short- and long-acting beta agonists and inhaled corticosteroids are safe in pregnancy.

Most pregnant women with asthma can be cared for in primary care. Only women with persistent poor control, a history of hospital admission as an adult, respiratory arrest or continuous or frequent courses of oral steroids would require a specialist referral to a respiratory physician during pregnancy.

Psychiatry and substance misuse
Women with a history of psychosis have at least a 50 per cent risk of perinatal recurrence. A family history of psychosis also increases the risk of puerperal psychosis. Care planning for all stages of the pregnancy and postnatal period is critical.
- GPs need a higher level of awareness for women who may be misusing drugs and/or other substances.
- GPs should screen all pregnant women for intimate partner violence.[12]
- GPs should screen all pregnant women for depression.[8]
- GPs should be proactive in maintaining contact with women with significant mental health problems, recognising those women may not initiate contact themselves. Book women in for more than the routine schedule of appointments and follow them up if they fail to attend.

A number of suicides or drug-related deaths occurred around the time of child-protection case conferences, suggesting that these are a period of increased risk. Additional support and surveillance through these difficult periods is required, especially if the child is removed.

Management of pre-existing disease
Serious pre-existing medical conditions require consultant involvement in pregnancy care. In the Australian context, this may vary from case discussion or consultation via telemedicine, through to shared care or referring a woman for tertiary care.

Communication
Referral is not treatment. If you are concerned or think an urgent response is required, telephone a senior clinician. However, always back up a fax, email or phone call with a written letter. Write high-quality referrals that specify the current problem and reason for referral, past history, any mental health issues (even if not directly relevant to the presenting problem), all medications she is on or has recently stopped and investigations to date.

GPs and practice staff should know how to access professional translation services rapidly. This is a major recommendation. Australian medical practitioners can register for free interpreting services through the Translating and Interpreting Service (TIS) National.[13] For urgent interpreting services call TIS on 131 450.

Conclusions
GPs are committed to providing excellent care; they are experts in managing uncertainty. There is concern GPs are becoming de-skilled in maternity care. GPs need to demonstrate that they are competent in maternity care by updating their knowledge and skills to manage the emergency situations they may have to face.

Although some GPs and registrars no longer consider pregnancy care a routine part of general practice, as primary care specialists we are ideally placed to provide this service and women highly value the continuity of care that is characteristic of general practice. As clinicians, we have a responsibility to practise high-quality medicine. By engaging in continuing professional development, being familiar with current evidence-based clinical practice guidelines and reflecting on reports by organisations such as CMACE, we will be able to provide high-quality maternity care to our pregnant patients into the future.

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Cardiac disease

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While the maternal mortality rate in Australia is gratifyingly low, cardiac disease remains a major cause of morbidity and mortality in young mothers.

Precise data relating to Australia are difficult to obtain, but the most recently published data from the UK show that heart disease associated with, or aggravated by, pregnancy is the commonest indirect cause of maternal death.[1] Some of the more common causes of maternal morbidity and mortality are discussed below.

Congenital heart disease

The treatment of congenital heart disease (CHD) in children has improved and increasing numbers of women with CHD are reaching childbearing age (there are now more adults with CHD than children). Thus, CHD is commonly encountered in pregnant women. In most cases, significant CHD will be known before pregnancy and pre-pregnancy assessment and counselling is strongly recommended. Fortunately, most women with CHD, tolerate pregnancy well. However, the risks are increased and depend on the specific cardiac lesion and associated factors, such as left and right ventricular function, severity of valvular stenosis or regurgitation, functional status and the presence of cyanosis or pulmonary hypertension.

Pulmonary artery hypertension, whether primary or secondary to congenital heart disease (Eisenmenger syndrome), is generally considered a contraindication to pregnancy and, if pregnancy occurs, termination should be considered. The maternal and fetal mortality rate is extremely high and, if pregnancy proceeds, these patients should be managed in a tertiary facility.

Acquired heart disease

Coronary artery disease and myocardial infarction

Atherosclerotic coronary artery disease was rarely seen in pregnant women several decades ago. However, ischaemic heart disease has now become a common cause of death in pregnancy. It is likely that increasing maternal age, together with an increase in the prevalence of traditional risk factors such as obesity, smoking, hypertension, hypercholesterolaemia and diabetes, has contributed to the increased incidence of myocardial infarction during pregnancy.

Women with pre-existing coronary artery disease are at significantly increased risk during pregnancy and the immediate postpartum period, and should be monitored closely by a cardiologist.

Pregnant women may also present with quite atypical features of ischaemia, such as shortness of breath, vomiting or dizziness. There should be a low threshold for further cardiac investigations, including serial electrocardiograms (ECGs), troponin levels and stress testing in women who present with ischaemic-sounding symptoms, especially in those with cardiac risk factors.

In patients presenting with an acute coronary syndrome (ischaemic pain together with ECG changes), early recognition allows acute coronary intervention to be performed, usually with coronary stenting. An aggressive interventional approach in acute coronary syndrome will also facilitate the diagnosis of coronary artery dissection, a rare, but often fatal, condition that is more likely to occur during pregnancy or the postpartum period. This condition may also be successfully treated by coronary stenting.

Aortic dissection

Aortic dissection is a rare, but lethal, condition in pregnancy. Risk factors for aortic dissection include Marfan syndrome and other connective tissue disorders, bicuspid aortic valve, coarctation of the aorta or a previous coarctation repair, hypertension and pre-eclampsia. Women with Turner Syndrome appear to be at significantly increased risk of aortic dissection, even in the absence of associated cardiac anomalies, such as bicuspid aortic valve or coarctation of the aorta. This risk appears to be further increased if pregnancy has resulted from in-vitro fertilisation, which is usually the case. Aortic dissection may also occur in apparently normal pregnant women, usually near term.

Aortic dissection usually presents with very severe chest, abdominal or back pain, requiring opiate analgesia for relief. In any patient with such pain, requiring strong analgesics, there should be a high index of suspicion for the diagnosis and further investigations

Summary

- Heart disease in pregnancy is becoming an increasing problem – it encompasses a wide spectrum of disorders.
- Ischaemic heart disease, previously rarely seen in pregnancy, is becoming more common. Patients with ischaemic-sounding symptoms or risk factors for coronary artery disease, should be assessed and investigated rapidly.
- Women with pre-existing cardiac lesions should be evaluated and counselled with respect to the risk they encounter with pregnancy.
- Contraindications to pregnancy include severe pulmonary hypertension or Eisenmenger's syndrome, cardiomyopathy with severe symptoms, severe uncorrected valvular stenosis, unrepaired cyanotic CHD and a dilated thoracic aorta. A history of PPCM is a relative contraindication.
- High-risk patients should be managed in specialised centres by a multidisciplinary team.

should be performed, including a chest x-ray and a computed tomography scan of the chest, magnetic resonance imaging of the chest or echocardiography (transthoracic or transoesophageal). Even when the diagnosis is made in a timely manner, and urgent surgery performed, maternal and fetal mortality remain high.

Peripartum and other cardiomyopathies

Peripartum cardiomyopathy (PPCM) occurs in the latter stages of pregnancy or within six months postpartum, with an incidence that varies from 1:300 to 1:4000 pregnancies, depending on the population studied. Risk factors include multiparity, ethnicity, smoking, diabetes, hypertension or pre-eclampsia and advanced age of mother or teenage pregnancy. Women with PPCM present with symptoms of breathlessness, orthopnoea and peripheral oedema, sometimes very acutely. These symptoms may be difficult to differentiate from symptoms often noted in normal pregnancy and, therefore, a chest x-ray and an echocardiogram should be performed.

Patients with a prior history of PPCM should be assessed by a cardiologist pre-pregnancy for risk-assessment and pre-pregnancy counselling. Any residual left ventricular function indicates an increased risk of heart failure during a subsequent pregnancy. Even in patients with normalised left ventricular function, a subsequent pregnancy may be associated with deterioration in ventricular function. Thus, patients with a prior history of PPCM should be closely monitored, with regular echocardiograms throughout pregnancy.

Women with known dilated or hypertrophic cardiomyopathy also have an increased pregnancy risk and should have expert pre-pregnancy assessment and counselling.

Rheumatic valvular disease

Rheumatic heart disease (RHD) continues to decline as a major cause of maternal mortality in the Western world. However, it remains a major cause of morbidity and mortality in developing countries and also in Indigenous Australians. Continued migration from countries with a high incidence of rheumatic fever means RHD will remain an issue in Australia. Most patients with RHD tolerate pregnancy well, but patients with severe valvular lesions, especially stenotic lesions, often deteriorate in the third trimester or during labour and delivery. Women with known severe rheumatic valvular disease should have cardiac intervention (percutaneous or surgical) prior to pregnancy, since intervention during pregnancy is performed at high maternal and fetal risk.

Table 1. Modified WHO classification of maternal cardiovascular risk: principles.[2]

Risk class	Risk of pregnancy by medical condition
I	No detectable increased risk of maternal mortality and no/mild increase in morbidity.
II	Small increased risk of maternal mortality or moderate increase in morbidity.
III	Significantly increased risk of maternal mortality or severe morbidity. Expert counselling required. If pregnancy is decided upon, intensive specialist cardiac and obstetric monitoring needed throughout pregnancy, childbirth and the puerperium.
IV	Extremely high risk of maternal mortality or severe morbidity: pregnancy contraindicated. If pregnancy occurs termination should be discussed. If pregnancy continues, care as for class III.

Modified from Thorne et al. [3]

Table 2. Modified WHO classification of maternal cardiovascular risk: application.[2]

Conditions in which pregnancy risk is WHO I
Uncomplicated, small or mild • pulmonary stenosis • patent ductus arteriosus • mitral valve prolapse
Successfully repaired simple lesions (atrial or ventricular septal defect, patent ductus arteriosus, anomalous pulmonary venous drainage)
Atrial or ventricular ectopic beats, isolated
Conditions in which pregnancy risk is WHO II or III
WHO II (if otherwise well and uncomplicated)
Unoperated atrial or ventricular septal defect
Repaired tetralogy of Fallot
Most arrhythmias
WHO II–III (depending on individual)
Mild left ventricular impairment
Hypertrophic cardiomyopathy
Native or tissue valvular heart disease not considered WHO I or IV
Marfan syndrome without aortic dilatation
Aorta <45mm in aortic disease associated with bicuspid aortic valve
Repaired coarctation
WHO III
Mechanical valve
Systemic right ventricle
Fontan circulation
Cyanotic heart disease (unrepaired)
Other complex congenital heart disease
Aortic dilatation 40–45mm in Marfan syndrome
Aortic dilatation 45–50mm in aortic disease associated with bicuspid aortic valve
Conditions in which pregnancy risk is WHO IV (pregnancy contraindicated)
Pulmonary arterial hypertension of any cause
Severe systemic ventricular dysfunction (left ventricular ejection fraction <30%, NYHA III–IV)
Previous peripartum cardiomyopathy with any residual impairment of left ventricular function
Severe mitral stenosis, severe symptomatic aortic stenosis
Marfan syndrome with aorta dilated >45mm
Aortic dilatation >50mm in aortic disease associated with bicuspid aortic valve
Native severe coarctation

Adapted from Thorne et al.[3]

Infective endocarditis

Infective endocarditis is rare in pregnancy (1:100 000 pregnancies), but is associated with a very high maternal and fetal morbidity and mortality, mainly owing to heart failure and thromboembolic complications (up to 33 per cent in one study). Predisposing conditions include congenital heart disease, acquired valvular disease and intravenous drug use, with the highest risk occurring in those with prosthetic heart valves or prosthetic material used for previous repair. Infective endocarditis should be treated the same way as in non-pregnant patients, bearing in mind the potential risk of certain antibiotics. Cardiac surgery is associated with a high risk of fetal loss and delivery should be performed prior to cardiac surgery, if possible.

Mechanical prosthetic valves

Mechanical prosthetic valves represent a major risk to successful pregnancy, mostly as a consequence of valvular thrombosis. The safest option for the pregnant woman is to remain on warfarin throughout pregnancy. However, warfarin, particularly at doses greater than 5mg/day, may cause an embryopathy. There are several strategies used for managing the problem of anticoagulation during pregnancy, including continuing warfarin if the daily dose is <5mg/day, changing to low molecular weight heparin (LMWH) injections from weeks five to 15, to minimise the risk of warfarin embryopathy, or using LMWH throughout pregnancy. Careful monitoring of LMWH dose, guided by anti-Xa levels and good compliance is extremely important in obtaining a good outcome. Whatever strategy is used, these women are at extreme risk of complications and should be managed in a multidisciplinary tertiary facility.

Maternal risk assessment

The European Society of Cardiology Guidelines on the management of cardiovascular diseases during pregnancy recommend that maternal risk assessment is performed using the modified World Health Organisation (WHO) risk classification.[2] This risk classification includes all known cardiovascular risk factors, including the underlying cardiac abnormality. Table 1 depicts the general principles of this classification. Table 2 outlines the application of the principles in specific conditions.

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Massive haemorrhage in early pregnancy

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Massive haemorrhage in early pregnancy is a rare, but potentially life-threatening condition.

In the Centre for Maternal and Child Enquiries (CMACE) report, Saving Mothers' Lives, five deaths owing to uterine haemorrhage in early pregnancy were reported in the UK, while in Australia ectopic pregnancy was the most common cause of first trimester maternal deaths, accounting for two per cent of maternal deaths from 1997–2005.[1,2]

Massive haemorrhage in obstetric practice should be regarded as blood loss >1500ml, approximately 25 per cent of the patient's blood volume in later pregnancy.[3] It is associated with a number of serious sequelae including hypovolaemic shock, disseminated intravascular coagulation (DIC), renal failure, hepatic failure, adult respiratory distress syndrome (ARDS), psychological trauma and loss of fertility.[3] Recognition of haemorrhage, adequate resuscitation and early surgical management are essential in the safe management of women with early pregnancy haemorrhage.

Physiology in early pregnancy

Uterine placental blood flow increases in a gradual and linear fashion early in pregnancy from 20–50ml/min up to 450–800ml/min at term in singleton pregnancies. This is achieved through a number of mechanisms both uterine and systemic. Uterine changes include an expansion of the uterine vasculature, vasodilatation resulting in reduced vascular tone and the development of the placenta. On a systemic level, maternal cardiac output increases and there is an expansion of maternal vascular volume.[4]

Maternal cardiovascular changes start as early as four weeks gestation. Red blood cell mass increases from four weeks and plasma volume expansion increases by 10–15 per cent by 12 weeks gestation.[5] Cardiac output increases by 30–50 per cent during pregnancy and half of this increase occurs by eight weeks gestation. Systolic and diastolic blood pressure falls early in pregnancy to around 5–100mmHg below baseline and remains lower until the third trimester.[6] Young healthy women can often compensate due to these changes until there has been substantial blood loss.

Table 1. Causes of haemorrhage in early pregnancy.

Common	Rare
• Spontaneous miscarriage • Retained products of conception • Ruptured ectopic pregnancy	• Molar pregnancy • Haemorrhagic ruptured corpus luteal cyst • Uterine abnormality – arteriovenous malformation • Complication of dilation and curettage • Placenta accreta • Spontaneous uterine rupture/ scar rupture • Cervical ectopic pregnancy • Thrombotic thrombocytopenic purpura

Clinical presentation

Haemorrhage in early pregnancy can present with obvious vaginal bleeding, vague abdominal symptoms or haemoperitoneum, depending on the site of pregnancy (see Table 1).

Spontaneous miscarriage is the most common cause of uterine haemorrhage in early pregnancy, but molar pregnancy should always be considered if unusually heavy bleeding occurs. Arteriovenous malformation can be a cause of haemorrhage at the time of dilation and curettage and rarer causes of vaginal bleeding, such as a cervical ectopic pregnancy, should be considered.[7,8]

Signs of haemoperitoneum can present with abdominal pain, bloating, vomiting and/or diarrhoea, signs of peritonism or collapse. All women of fertile age should have a pregnancy test, if presenting with these symptoms or undifferentiated abdominal pain, to exclude pregnancy.[2] Ruptured ectopic pregnancy is the most likely cause of haemoperitoneum, but there are increasing case reports of uterine rupture in first trimester pregnancy, secondary to accreta or molar pregnancy implanted into a previous scar. Although rare, a history of previous uterine surgery in a woman presenting in early pregnancy with signs of haemoperitoneum may warrant laparoscopy, despite ultrasound confirming an intrauterine pregnancy.[9-12]

Management

The management of haemorrhage in early pregnancy differs according to the location of the pregnancy and site of haemorrhage. Assessment of the woman needs to determine if she is haemodynamically stable, and whether imaging is appropriate, or if she should be taken directly to theatre for surgical management.

There is little evidence-based literature in regards to management of acute uterine haemorrhage in early pregnancy. The approach to haemorrhage in early pregnancy should be similar to that in postpartum haemorrhage (PPH): determine the cause of the bleeding, assess extent of haemorrhage, examination, resuscitation, medical and surgical management.

Women presenting with heavy vaginal bleeding should have a speculum examination to remove any product from the os; this is an important part of the resuscitation process.[13] If heavy bleeding occurs during dilation and curettage, examination of the cervix should

be performed in case of cervical damage requiring repair, uterine perforation should be considered and diagnostic laparoscopy may need to be performed to exclude perforation.[14,15]

The therapeutic aims in massive haemorrhage are to maintain tissue perfusion and oxygenation, stop bleeding and prevent coagulopathy. The first goal in massive haemorrhage is resuscitation and adequate volume expansion. A (airway) B (breathing) C (circulation) is the initial step in resuscitation and often women with haemorrhage are not adequately resuscitated at the outset. Two large-bore cannulas (16g) should be sited and a full blood count (FBC); group and hold; and antibody screening should be sent. Baseline electrolyte, renal function and coagulation screen (including fibrinogen and thrombin time) should also be collected.[16] All women should have a blood group test and rhesus-negative women given anti-D at an appropriate time when stable.

Regular observations should be recorded and, ideally, a member of the team should be assigned to the task of scribing to record all observations and medications given. The importance of clear communication between clinicians, team members and pathology (blood bank) as well as accessing early senior-level assistance in maternal haemorrhage has been emphasised in multiple reports into maternal deaths.[3,16,17]

Volume resuscitation

Delay in attempting to restore circulating volume can result in serious complications and prolonged hypotension; increases morbidity and mortality; and increases the risk of DIC. Crystalloid solution, such as 0.9 per cent normal saline or Hartmann's solution, should be the first-line therapy in early resuscitation and should be infused as a bolus until systolic blood pressure is restored to 80–100mgHg. The risks of aggressive fluid resuscitation include pulmonary oedema, exacerbation of thrombocytopenia and coagulopathy secondary to haemodilution, and this must be balanced with the need to maintain tissue perfusion with adequate blood pressure.[18] It is recommended that crystalloids be used for resuscitation and, in situations where colloids are used, at a maximum volume of 1000–1500ml in 24 hours, as larger volumes affect haemostatic function.[3] Hypothermia increases the risk of end-organ damage and, in cases where large volumes of fluid are required, the use of warm air blankets and pre-warmed fluids helps prevent hypothermia and the risk of coagulopathy.[16]

Blood transfusion

Transfusion of red blood cells (RBCs) should not be used as a volume expander, but to provide oxygenation to tissue and contribute to haemostasis by contributing to platelet function.[16] Red cells are usually not indicated with an Hb >100 and are usually always indicated for an Hb <60. In massive haemorrhage where a crossmatch is unavailable, O (Rh) D-negative blood cells can be administered but should be swapped for ABO-specific blood as soon as possible. Each hospital should have a massive transfusion protocol and the suggested criteria for activation of this protocol is in patients who will need four units of RBC in less than four hours (actual or anticipated) and/or are haemodynamically unstable with or without ongoing bleeding.[18] Blood should be given through a blood warmer, with regular monitoring of FBC, coagulation studies, ionised calcium and arterial blood gases (see Table 2).[18] Coagulopathy is often present early in obstetrics patients and the triad of acidosis, hypothermia and coagulopathy increases the mortality rate.[18]

Table 2. Parameters in massive blood transfusion: monitor every 30–60 minutes.[18]

Aim for	
Temp	>35°
pH	>7.2
Base excess	<-6
Lactate	<4mmol/l
Ca_2+	>1.1mmol/l
Platelets	>50 x 10^9/l
Partial thromboplastin time/activated partial thromboplastin time	<1.5x normal
International normalised ratio	≤1.5
Fibrinogen	>1.0g/l

Once four units of RBCs have been administered, other blood components should be considered and administered (see Table 3). Fresh frozen plasma (FFP) should be given to maintain a partial thromboplastin time (PTT) or activated partial thromboplastin time (APTT) ≤1.5 x normal. A suggested regime is four units FFP per four units of RBC and a single adult dose of platelets, with appropriate correction of calcium.[19] Thrombocytopaenia will be present when 1.5–2 times blood volume has been transfused due to dilution and increasing consumption. The platelet count should be kept above 50x10^9/l and should be kept above 80–100x10^9/l if surgical intervention is required. If the platelet count drops below 50x10^9/l and bleeding is not under control during massive haemorrhage, then a bolus of FFP followed by 8–12 units of platelets should be rapidly transfused.[3] Fibrinogen may not be corrected by FFP alone and cryoprecipitate should be administered if fibrinogen is <1g/l.[18] If haemorrhage continues – despite adequate stabilisation, massive transfusion protocol and surgical or radiological management – consideration of Factor VIIa should be made in consultation with a haematologist (see Table 3). Patients who receive Factor VIIa are at high risk of thromboembolism and calf compressors and TEDs should be started immediately with consideration of prophylactic doses of unfractionated or low molecular weight heparin within 24 hours after haemorrhage.[19] Early DIC should always be considered in obstetric patients and cryoprecipitate should be administered early and consultation with a haematologist about the role of antithrombin (Thrombotrol-VF) should be considered.[18]

Medical management

Medical management includes syntocinon, ergometrine and misoprostol or prostaglandin $F_{2\alpha}$. There is little evidence to support a certain regime or dosing in early pregnancy and generally the doses used are the same as for postpartum haemorrhage.[14] Bimanual massage can be used in conjunction with uretotonics; this may be while the patient is being stabilised, is waiting for theatre or during theatre when bleeding continues despite suction dilation and curettage.

Myometrial oxytocin receptors are present at low levels up until week 17 when they increase dramatically until term, with maximal receptors being found in the myometrium during labour.[20] A small randomised controlled trial study of 64 women undergoing suction curettage at ≥9 weeks gestation showed that 1ml intravenous syntocinon resulted in a statistically significant reduction in blood loss, although not a clinically significant reduction in loss as both groups of women lost <100ml during the procedure, demonstrating there is some response to syntocinon despite low levels of myometrial receptors being present in early pregnancy.[21]

Surgical management

Much of the literature concerning surgical techniques for control of haemorrhage in early pregnancy is case studies. Suction dilation and curettage is first-line surgical management for uterine haemorrhage and, again, surgical techniques are aligned closely with clinical practice for PPH (see Table 4). The use of a balloon catheter to tamponade bleeding in PPH is well documented and appears to have increasing evidence in the treatment of bleeding owing to post-abortion haemorrhage, arteriovenous malformations and first and second trimester haemorrhage.[22,23] This method is effective and also available in all settings (especially rurally with limited resources) as a Foley's catheter can be used, volumes of balloon catheter varied in studies from 30ml to 150ml.

Table 4. Surgical management of uterine haemorrhage in early pregnancy.

Suction dilation and curettage
Packing uterus
Balloon catheter
Radiological intervention – embolisation uterine artery
Laparotomy – uterine or internal iliac arty ligation, haemostatic suture (B-Lynch suture)
Hysterectomy

Radiological intervention (uterine artery embolisation – UAE) is associated with less morbidity and mortality then laparotomy and hysterectomy, but is not available in all centres. It has been used successfully in arteriovenous malformations, post-abortion haemorrhage and PPH.[22,24] UAE has also been used in conjunction with laparoscopy and laparotomy to control bleeding while repair of uterine perforation is performed.[14] UAE is also fertility preserving in women wishing to have future pregnancies.

If bleeding continues despite full medical management and the preceding surgical/radiological techniques, laparotomy may need to be performed. Haemostatic sutures, such as the B-Lynch suture, have been reported in the literature and it is reasonable to attempt this before hysterectomy, which is definitive treatment for bleeding.[25]

Grief/trauma counselling

Management for haemorrhage can be a traumatic and distressing experience for the woman and her family. In addition to this, they are also dealing with a pregnancy loss and it is important, as clinicians, we recognise both the experience of a life-threatening event and also the loss of a pregnancy and offer debriefing and counselling as appropriate.[26]

Table 3. Blood components in massive haemorrhage and indications in massive transfusion.[27]

Therapy	Indication and dosing
Red cells	• Massive transfusion protocol indicated if ≥ 4 units RBC in 4 hours (actual or anticipated) + haemodynamically unstable +/- ongoing bleeding • Uncrossmatched group O, RH (D) negative red cells if group unknown; change to group-specific blood when crossmatch available • To avoid hypothermia, give through blood warmer
Fresh frozen plasma	• Give FFP to maintain PT & APTT ≤ 1.5x normal • Recommend 4 units fresh frozen plasma for every 4 units red blood cells • Usual dose 15ml/kg • If blood group unknown – give AB fresh frozen plasma • 30 minutes thawing time
Platelets	• Normal adult dose (1 bag) • Recommend 1 bag per 4 units red blood cells and 4 units fresh frozen plasma • Aim to keep platelet count >50x10^9/l and 80–100x10^9/l if surgery indicated • Thrombocytopaenia (<50x10^9/l) can be expected at 1.5–2x blood volume replacement
Cryoprecipitate	• If Fibrinogen <1g/l despite FFP replacement cryoprecipitate is indicated • Early use of cryoprecipitate is indicated in DIC • Dose 3–4g of fibrinogen (consult with transfusion lab) • 30 minutes thawing time
Recombinant factor VIIa	• Ongoing haemorrhage despite: – blood component therapy with adequate coagulopathy correction – failed surgical or radiological intervention to control bleeding – pH >7.2 and temp >34° – Consult with haematologist – 90μg/kg (rounded to nearest vial) as a single bolus over 3–5 min • Check dose at 20 mins and optimise calcium, pH, temp, platelets and fibrinogen, if no response administer second dose[19]
Calcium correction[19]	• Correct calcium as appropriate • Optimise temperature and acidaemia
Cell salvage	• Consider use where appropriate where anticipated blood loss > 1500ml • May be appropriate for patients who refuse allogenic blood transfusion (eg. Jehovah's Witness patients)[28]

Conclusion

In the report, Maternal Deaths in Australia 2003–05, no deaths from massive uterine haemorrhage in early pregnancy were identified, but massive haemorrhage can occur suddenly and be life threatening, therefore anyone treating women in early pregnancy should be prepared. Management follows the basic principles of postpartum haemorrhage, but often occurs in settings not used to dealing with significant haemorrhage. Recognition of haemorrhage, early help and adequate resuscitation should be implemented as well as early medical and surgical management.

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Induced abortion and maternal death

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The complications of induced abortion continue to be a cause of maternal death, albeit an uncommon one, even in developed countries in the 21st century. In the developing world, induced abortion – often unsafe illegal abortion – is still a major contributor to maternal mortality rates, although there has been some improvement in death rates over the past two decades.

The Eighth Report of the Confidential Enquiries into Maternal Deaths (CEMD) in the UK, published in 2012, contains details of two maternal deaths following termination of pregnancy (TOP) in the period 2006–08.[1] A total of 2 291 493 births (live and stillbirths after 24 weeks gestation) were recorded in the UK in this time, giving a maternal mortality rate for abortion of less than one death per one million maternities.

Significantly, these deaths are included in the chapter dealing with genital tract sepsis, from which the key message is: 'Be aware of sepsis – beware of sepsis'. The first death was related to staphylococcal toxic shock syndrome following surgical TOP, the second to sepsis from *Clostridium septicum* after a medical TOP. Prophylactic antibiotics were not prescribed in either case. Of course, clostridial infection is a rare, but recognised, cause of maternal death following vaginal and caesarean births as well as miscarriage and is probably related to the vaginal carriage of the organism, which ascends into the uterus and the exposed placental site.

Figures of one or less than one death per 100 000 abortions are widely quoted for early procedures, surgical and medical, in developed countries.[2] In New Zealand, in 1980–2005, there were almost 250 000 TOPs, the vast majority of which were surgical, and no recorded mortality.[3] Figures for surgical abortion in Australia, although not formally available for recent years, are likely to be similar. Goldstone and colleagues in 2012, reporting on more than 13 000 early medical abortions performed in the Marie Stopes group of clinics from 2009–11, using mifepristone/misoprostol, recorded one maternal death.[4] Few details of the circumstances of this death are available, but it appears that the woman concerned developed an infection with Group A streptococcus several days after medical TOP; other family members also had upper respiratory Group A strep infections. The woman declined to seek treatment initially, which undoubtedly contributed to her death nine days following the abortion process. Prophylactic antibiotics were not administered. This is the only death known to have occurred in what must now be more than 50 000 medical abortions performed using mifepristone/misoprostol in Australia.

The recommendation of the Royal College of Obstetricians and Gynaecologists (RCOG) is that abortion services 'should offer antibiotic prophylaxis effective against *Chlamydia trachomatis* and anaerobes for both surgical abortion (evidence grade A) and medical abortion (evidence grade C).'[5] The reason for the lower level of evidence for medical termination is simply that the randomised controlled trials have not yet been done. Given that there is clear evidence for the use of prophylactic antibiotics after surgical TOP, when in most cases the woman is going home with the uterus completely emptied of products of conception that might provide a nidus for infection, it seems logical to give the same antibiotics to a woman who is undergoing the expulsion of products of conception either in a clinic or at home, particularly when this process may occur over a period of days. The RCOG guidelines recommend metronidazole one gram rectally at the time of the abortion plus, commencing on the day of the abortion, either doxycycline 100mg twice daily for seven days or azithromycin one gram orally. The alternative to such prophylaxis, screening women for pathogens and treating those with demonstrable infection, can be difficult to carry out meticulously in the context of abortion provision.

It is important also that women undergoing induced abortion are aware of the need of reporting immediately for medical assessment if they develop fever, pain or other symptoms of infection. In this regard, practitioners providing abortion should ensure that women who have travelled away from home to access abortion will feel comfortable accessing post-abortion care locally if they need it once they have returned home.

Haemorrhage was not a cause of death associated with abortion in the UK Report. Where surgical abortion is performed in suitable clinical surroundings by experienced practitioners who can proceed to laparoscopy/laparotomy if needed, the risk of such deaths is likely to remain very small, as shown by the New Zealand figures. However, the Report does caution about the risk of abnormal placentation at the site of previous caesarean section scars, which can lead to catastrophic haemorrhage even in the first trimester, and certainly in the second and third, no matter by which route the pregnancy ends.[1] In this respect, it would be prudent for practitioners contemplating offering home medical TOP to a woman with a history of several caesarean births to consider whether such a woman may be better managed with medical TOP within a clinic or with surgical TOP.

There has been a welcome decrease in death rates from abortion in developing countries over the past 20 years.[6] Increasing contraceptive use and the availability of more effective forms of contraception have cut the number of maternal deaths in such countries by 40 per cent in this time, simply by reducing the number of unplanned and unwanted pregnancies.[2,6] In addition, since misoprostol has become widely, if illegally, available in

Africa, Asia and South America, induced abortion using this drug has to a large extent taken the place of unsafe abortion in which untrained practitioners used unsterile instruments to perforate the membranes and/or extract products of conception.[7,8] While misoprostol and other drugs recognised as suitable for medical abortion should ideally be used, at appropriate gestations, under the supervision of a health professional and with access to emergency care if needed; self-administered misoprostol, especially in early pregnancy, is relatively safe, and much less likely to lead to major haemorrhage, organ damage or infection than the sticks or other sharp objects often used previously in places where legal safe abortion is unavailable.

However, each year, according to the WHO, nearly 42 million women faced with an unplanned pregnancy have an abortion, and about 20 million of them do so unsafely, either inducing abortion themselves or obtaining abortion clandestinely. It is among the latter group that most of the deaths occur that constitute the 13 per cent of worldwide maternal mortality attributable to abortion. More effective sex education, more accessible effective contraception, better access for all women to safe abortion when needed, must all play a part if we are to reduce these figures.[9]

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2013 RANZCOG Shan S. Ratnam Young Gynaecologist Award (YGA)

RANZCOG is pleased to announce that the following members have been awarded the 2013 RANZCOG Shan S. Ratnam Young Gynaecologist Awards (YGA) to attend the 23rd Asian and Oceanic Congress of Obstetrics and Gynaecology (23rd AOCOG 2013) to be held 20 – 23 October 2013, Bangkok, Thailand

- Dr Christopher Smith YGA for Australia
- Dr Christina Tieu YGA for New Zealand

Congratulations Christopher and Christina!

For further information on the 23rd AOCOG 2013 in Bangkok, Thailand, visit http://www.aocog2013.org/

Venous thromboembolism and pregnancy

A search for clarity in an uncertain world.

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Venous thromboembolism (VTE) is a relatively uncommon complication of pregnancy and the postpartum, with an incidence of around one in 1000–1500 pregnancies.[1] It remains an important cause of maternal death in developed countries and was the second most-common cause of direct maternal death in the Australian maternal mortality report from 2003–05[2] and third most common in the UK.[3] In New Zealand, the Maternal Mortality Working Group of the Perinatal and Maternal Mortality Review Committee (PMMRC) reported only two deaths from VTE from a total of the 21 direct maternal deaths between 2006 and 2011.[4] Most women who develop pregnancy-associated VTE (PA-VTE) do not die, but studies suggest that around one in 40 of pulmonary emboli (PE) are fatal.[5-7] One-third to one-half of PA-VTE occur postpartum[5,8,9], most within the first six weeks, with the daily risk of VTE in the six-week postpartum period greater than during the antenatal period.[6, 9,10] At least half of the antepartum events occur in the first two trimesters[7,11-13], emphasising the importance of pre-conception counselling in high-risk women as well as an assessment of risk factors early in pregnancy.

Risk factors for PA-VTE

Pregnancy predisposes to thrombosis, as all three components of Virchow's triad are present in pregnancy: venous stasis, induced by venous dilation and obstruction to venous return; increases in procoagulant factors and reduction in natural anticoagulants; and vessel wall injury during labour and following caesarean section (CS). Pregnancy is also a pro-inflammatory state, with activation of endothelial cells. Additional maternal factors further increase the risk of VTE and recognisable risk factors were identified in as many as 75 per cent of women who died from PE in the UK.[14] Risk factors for PA-VTE are summarised in Table 1. Some risk factors, such as obesity, immobilisation and VTE, are common to many patient populations, whereas others, such as CS and pre-eclampsia, are specific to pregnancy. Increased BMI is an important and consistent risk factor for PA-VTE.[15,16] In the UK, two-thirds of women who died from PE had a BMI >25 and almost 20 per cent were morbidly obese (BMI >40).[14] Increased BMI combined with immobilisation significantly increases the risk of PA-VTE.[16]

Prior history of VTE

Previous VTE is one of the most important risk factors for PA-VTE. The risk of recurrence is higher following previous unprovoked (no identified risk factors) than provoked (associated with a risk factor) events.[17] In pregnant women with a prior VTE associated with a major (surgical or traumatic) provoking factor, the risk of antepartum recurrence appears to be low, with no recurrent events seen in this patient group in two studies.[17,18] The risk of recurrence in women with either a prior unprovoked or hormonally provoked VTE (pregnancy or oral contraceptive associated) is higher, with antepartum recurrence risk ranging from two to five per cent and two to ten per cent, respectively.[18-20] Some women have risk factors that place them at an increased risk of VTE throughout pregnancy and the postpartum period and warrant extended thromboprophylaxis. Others will develop complications during pregnancy or will require thromboprophylaxis only if they are hospitalised.

Inherited thrombophilias

The inherited thrombophilias have gained much attention in the last couple of decades as risk factors for VTE. Factor V Leiden (FVL) and the prothrombin mutation are relatively weak in terms of their prothrombotic risk, but are found in five per cent and two to three per cent of Caucasians, respectively, whereas deficiencies of

Table 1. Clinical risk factors for PA-VTE

Standard risk factors	Adjusted OR
Previous VTE	24.8
Age >35	1.4-1.7
Obesity (BMI > 30kgm^2)*	1.7-5.3
Active medical illness	2.1-8.7
Smoking	1.7-3.4
Family history VTE	2.9-4.1
Immobility	7.7-10.1
Varicose veins	2.4
Pregnancy-specific risk factors	
Multiparity (>2)	1.6-2.9
Multiple pregnancy	1.6-4.2
Preeclampsia	3.0-5.8
Assisted reproduction technology	2.6-4.3
Hyperemesis	2.5
Postpartum risk factors	
Planned caesarean section	1.3-2.7
Emergency caesarean section	2.7-4.0
Placental abruption	2.5-16.6
Postpartum infection	4.1-20.2
Postpartum haemorrhage	1.3-12.0

Adapted from McLintock et al.[1]

antithrombin, protein C and protein S are more thrombogenic, but are rare each with a prevalence of around 0.1 per cent.[21] While these inherited clotting tendencies are associated with an increased risk of development of an initial VTE, they are less important risk factors for recurrent VTE.

Thromboprophylaxis – who should get it?

Prevention of PA-VTE by offering thromboprophylaxis to women at highest risk of an event is clearly a sensible clinical approach. However, just how to identify these high-risk women is hampered by the paucity of evidence from clinical trials with wide estimates of risk that are likely to be subject to various sources of bias. This means that decision-making in this clinical setting and recommendations for thromboprophylaxis are 'eminence based' rather than evidence based. An individual's appreciation of risk is also personal and for these reasons it is difficult to base recommendations for prophylaxis on arbitrary quantitative estimates of risk. Decisions should therefore be made after explaining the available evidence to the patient and taking into account her perception of the balance of risk and benefit in thromboprophylaxis.

An assessment of risk of thromboembolism should be carried out in all pregnant women. Limited evidence suggests that risk factors are synergistic[16] therefore a patient with multiple risk factors warrants discussion with regard to thromboprophylaxis, even in the absence of a personal or family history of VTE. There is a higher daily risk of PA-VTE in the postpartum period so the threshold for recommending thromboprophylaxis is lower during this period.

Guidelines have been published by the Royal College of Obstetricians and Gynaecologists in the UK[22] and more recently Australian and New Zealand (ANZ) specialists in haematology, obstetric medicine, anesthesia, obstetrics and maternal fetal medicine who are actively involved in this field have published ANZ recommendations for thromboprophylaxis[1] and also for diagnosis and management of acute VTE in pregnancy.[23] The ANZ guidelines were endorsed by the Australasian Society of Thrombosis and Haemostasis and the Society of Obstetric Medicine of Australia and New Zealand. The National Health and Medical Research Council of Australia has also developed guidelines for thromboprophylaxis for inpatients, including obstetric patients.[24] One key difference between the RCOG guidelines and the ANZ recommendations for thromboprophylaxis relates to the importance of the inherited thrombophilias in risk assessment. In the absence of a personal or family history of VTE, the ANZ recommendations do not consider the presence of an inherited thrombophilias, such as heterozygosity for FVL or the prothrombin gene mutation, protein C or protein S deficiency, to be an indication for antenatal thromboprophylaxis if a woman has no other risk factors. The ANZ recommendations also take the prothrombotic potential of the inherited thrombophilias into account, recognising that higher rates of VTE are reported in patients with deficiencies of antithrombin, protein C, protein S or who are homozygous for FVL or the prothrombin gene mutation or compound heterozygotes for these mutations. The RCOG also stipulated that higher doses of LMWH for thromboprophylaxis should be used in woman who weigh more than 90kg. In contrast, the ANZ group did not consider that there was sufficient evidence to make this recommendation.

In the absence of evidence, what should we do?

Development of guidelines or recommendations in the absence of high-level evidence from clinical trials opens the authors to the criticism that thromboprophylaxis will be given to large numbers of women whose risk of developing PA-VTE is relatively low.[25] Some groups[26] have attempted to develop estimates of the number-needed-to-treat to prevent an thromboembolic event. However, in the absence of randomised clinical trials in pregnancy cohorts, accurate statistics are not available and extrapolating efficacy rates with thromboprophylaxis from other patient groups is problematic.

So where does that leave clinicians? Very few guidelines or recommendations would be published in medicine if we had to wait for incontrovertible truths to be available to support them. In our imperfect world, most clinicians are happy to have pragmatic, carefully considered recommendations that provide guidance for clinical decision-making, while clearly identifying the limitations of the data that support these recommendations and acknowledging the lack of certainty. Development of eminence-based recommendations should not close the door to continuing to conduct more robust clinical research. To answer the important clinical questions relating to prevention of PA-VTE will require international multicentre trials – no easy feat, but hopefully attainable.

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Perinatal mood and anxiety disorders

How to recognise these disorders and the effect they have on families.

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When we consider the maternal mortality rate, it is important to recognise that mental health issues are one of the most preventable causes of maternal (and infant) mortality.

Risks are not just limited to suicide or infanticide (which are both thankfully rare). Perinatal mental health disorders not only impose a huge burden on mothers, their health and parenting capacity, but may also affect infant development and the social and emotional well-being of the entire family.

Psychiatric conditions in the perinatal period

Despite the cultural expectation that women should 'bloom' in pregnancy, the prevalence of depression and anxiety is about 12 per cent.[1] Antenatal depression can lead to poor self-care, even to the extent of self-injuring behaviours. There is also a growing body of evidence that if a mother is stressed or anxious while pregnant, the child is substantially more likely to be anxious, to have cognitive problems and language delay, and to face an increased risk of attention deficit and hyperactivity disorder, regardless of postnatal anxiety or depression.[2] This is thought to relate to increased fetal exposure to cortisol in utero. Conditions such as hyperemesis gravidarum are more common in women with anxiety and depression.[3] Antenatal depression is one of the strongest predictors of postnatal depression and it does not often spontaneously remit after childbirth.

Postnatal depression has a general prevalence of 15 per cent.[4] Depressive symptoms such as fatigue and poor sleep are hard to disentangle from the normal disruptions of pregnancy and early parenthood. Experiencing pervasive sadness, hopelessness, feelings of inadequacy and guilt, and suicidal thoughts are indicative of mental illness.

Postnatal depression is often referred to as a smiling depression, as it is common for women to have a level of suffering worse that appearances might suggest. Because symptoms may be minimised by the patient, it is important to maintain a high index of suspicion. Diagnostic clues include a decrease in self-care, a 'wooden' facial expression and slowed movements, the mother appearing to be excessively anxious about her baby, or unresponsiveness to her increasingly distressed baby.

Postnatal anxiety is as common as depression. The symptoms of anxiety are very distressing and can significantly impact on parenting confidence and the mother-infant relationship. Symptoms include feelings of apprehension and dread, panic; and physical symptoms such as vomiting, bowel upset, tachycardia, hyperventilation, inability to relax, sleeplessness and exhaustion. Obsessional thoughts are intrusive and upsetting, often involving thoughts of the baby being harmed or, more distressingly, unwanted thoughts of the woman harming the baby herself. These conditions need urgent and careful assessment, to distinguish obsessional thoughts (which are seen as abnormal and distressing to the mother) from similar thoughts in depression and psychosis that may be associated with risk to the baby.

> 'Although the rate is low, suicide is one of the most common and preventable causes of maternal mortality in Australia, the UK and New Zealand.'

Risk factors for perinatal depression and anxiety are varied and include being a very young mother, poverty, stressful life events, grief and loss, domestic violence and low levels of social support, past psychiatric or substance abuse, history of childhood abuse or a perfectionistic personality style. Obstetric factors such as a past history of termination, miscarriage, unwanted pregnancy or a traumatic delivery can also add 'fuel to the fire'.

Post-traumatic stress disorder (PTSD) may have a perinatal prevalence of between two and eight per cent.[5] PTSD was first recognised in soldiers involved in horrifying events where they were threatened with serious injury or death. Despite all the advances in safety of obstetrics, difficulties in childbirth can still be very terrifying, traumatic and lead to PTSD. In some cases, the PTSD may be pre-existing. Symptoms include re-experiencing through flashbacks, intrusive memories or nightmares; feeling detached and numbed emotionally; and having intense anxiety responses. PTSD is highly co-morbid with depression, anxiety and substance use. In clinical practice, it can be strongly associated with bonding difficulties. Perinatal PTSD requires specialised psychiatric or psychological treatment.

Puerperal psychosis occurs in one to two per 1000 births, most often a few weeks after childbirth.[6] Puerperal psychosis can begin insidiously. The initial symptoms may include quiet confusion, a delirious state, restlessness, excessive anxiety or inability to sleep. Mood symptoms are common, with erratic or inappropriate emotions, manic symptoms of excessive energy and activity,

disinhibition, pressured thoughts and speech, loss of contact with reality and hallucinations. Puerperal psychosis is a psychiatric emergency requiring urgent assessment and usually hospitalisation, as the risks of self-harm or harm to the baby are high.

Consequences for the infant and family

Fathers and family

Postnatal depression may have serious financial implications on the family if the father has to take extra time off work. It can severely affect the marital relationship, in many cases becoming a factor in separations and divorce.

Depression is recognised to affect about five to ten per cent of new fathers.[7] It is often hard for men to disclose their symptoms and seek help. Depression may be reflected in an increased risk of substance abuse and domestic violence. Paternal depression may have a detrimental effect on children's emotional and behavioural development, even after controlling for maternal depression.[8]

Infant development

Overwhelming research data have shown that infant development occurs in the context of the caregiver relationship. If the caregiver has a mental illness or is severely distressed, it is more difficult for them to see their child's emotional and physical needs. This may have consequences on the infant's social, emotional, cognitive and language development.

Depression may impact on mothers' perception of, and behaviour towards, their infants and children. Depressed mothers can be disengaged, withdrawn and unresponsive to their infants, or they may be overly intrusive. Babies of depressed mothers may have learned to avert their gaze from their mother (while giving other people, such as the clinician, big smiles), or indeed seem depressed themselves. Depressed mothers have told me, 'my baby doesn't want me, he won't look at me', indicative of their distorted perception of the infant and perpetuating the cycle of avoiding contact and the mother's low mood.

Unfortunately, and likely as a result of these repeated unsuccessful interactions, children of depressed mothers are more likely to have difficulties in social-emotional, behavioural and cognitive functioning, and are at greater risk for later psychopathology, particularly if the postnatal depression becomes chronic.[9]

It is important to recognise that postnatal depression is not the only factor impacting on infant outcomes – risks accumulate. They may include: developmental disorders; prematurity or medical illnesses; social adversity, including domestic violence; substance abuse; maternal stressors, such as grief; and relationship difficulties. However, when a mother is thriving and not depressed, she is more able to buffer these risks.

Suicide and infanticide

Having a baby does not protect against suicide. Although the rate is low, suicide is one of the most common and preventable causes of maternal mortality in Australia, the UK and New Zealand.[10,11,12] Owing to data-collection issues, suicide is likely under reported.

The majority of suicides by mothers with young children have occurred by violent means. Many of these women have had a previous documented history of mental illness issues. These are generally not just a cry for help, but a determined effort to die.[13] Even if unsuccessful, the consequences of an attempted suicide are devastating for the mother and infant.

Intimate partner violence is an associated risk with suicidal ideation and completed suicides worldwide[14] and homicide is also a significant cause of maternal mortality.[10]

Infanticide is also rare, but also likely to be under-reported, meaning research is limited. It appears that maternal neonaticide (murder of a baby in the first 24 hours after birth) is often associated with unwanted pregnancy, pregnancy denial and a young, poorly educated woman, most likely still living at home. Maternal infanticide (defined as murder of an infant less than 12 months old) is more likely associated with mental illness, in particular, psychosis.[15]

Steps to treatment

The first and most challenging step in treating perinatal psychiatric disorders is recognition. The stigma of mental illness, or concerns about being forced into taking medication, may lead women (or their families) to be reluctant to discuss their difficulties. Depression and anxiety do not spontaneously resolve, indeed they often worsen or become chronic. There are significant risks to women, their families and their infants' emotional, cognitive and physical development if not treated. These risks need to be considered against the patient's worries of potentially harming their baby through taking antidepressant medication, or being reluctant to engage in psychological therapy.

It is important to give mental health issues time and space. Asking a patient how they are faring and showing concern about mental health will set expectations that this is an important aspect of obstetric care, and gives permission for discussion and disclosure. The Edinburgh Postnatal Depression Rating Scale (EPDS) is a useful self-report screening tool that may help break the ice on this sometimes difficult topic.[4] The website www.beyondblue.org.au has a wide variety of patient education-resources on perinatal depression and anxiety.

Delayed bonding and mothers' feelings of 'this could be anyone's baby' need to be pursued. Infant safety red-flags include violent thoughts, hostility, irritability and parental delusions about the infant, or severe confusion and disorganisation of the unwell mother. The severely depressed or psychotic mother, especially if those delusions involve the child, will need to be hospitalised.

Although the specifics of treatment are beyond the scope of this article, it is important to emphasise that excellent treatment options are available. Pregnancy and breastfeeding are not contraindications to many pharmacological treatments, and psychotherapy and support services are becoming more widely available in each state.

Obstetricians, as trusted experts in physical health and intimate issues, have a tremendous opportunity to open the dialogue regarding mood and stress and reduce the risks to mothers and their families of mental illness.

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Amniotic fluid embolism

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Amniotic fluid embolism is a rare and potentially catastrophic, but poorly understood condition that is unique to pregnancy. It may range from a relatively minor subclinical episode through to one which is rapidly fatal.

Despite being first reported over 70 years ago, the underlying pathophysiology of amniotic fluid embolism (AFE) is still not well understood. Even the name AFE has been criticised as not being representative of the underlying condition, with suggested alternatives being 'anaphylactoid syndrome of pregnancy' or 'sudden obstetric collapse syndrome'.

The incidence of AFE in Australia has recently been reported as 6.1 per 100 000 deliveries (95 per cent CI 5.2–6.9) with a case fatality rate of 14 per cent.[1] Despite its rarity, AFE is a major contributor to maternal mortality in developed countries. It is currently the leading cause of direct maternal death in both Australia and New Zealand, while in the UK triennial reports it is consistently in the top four.

Traditionally, AFE was associated with a very high morbidity and mortality rate. Early published data from Clark[2] and Morgan[3] documented a mortality of between 61 and 85 per cent, with poor outcomes in those women who did survive. However, more recent data would suggest the mortality is much lower than this, ranging from 11–43 per cent (see Table 1). In addition, the outcomes in women who do survive the initial insult also appear encouraging. This improvement in morbidity and mortality is likely to be secondary to a number of factors, including greater awareness of the condition (such that less severe cases are now reported), developments in resuscitation and intensive care, as well as multidisciplinary training for the management of the collapsed obstetric patient.

The outcomes for the neonate, if the AFE occurs in utero, are unfortunately not as positive compared to the mother. Fetal distress may be an initial presenting sign of AFE and, if an in utero reaction does occur, the neonatal mortality may be up to 40 per cent.[1]

Pathophysiology

The pathophysiology of AFE is poorly understood and no reliable animal model exists for AFE. For this reason, most of the theories surrounding the pathophysiology of AFE are derived from clinical cases. The term 'embolism' itself is a common source of confusion. While some of the typical features of an AFE may be similar to the mechanical obstruction of pulmonary blood flow seen with a traditional pulmonary embolism, a number of other features do not fit with this model. For this reason possible immune-mediated mechanisms have also been suggested.[4]

Despite the lack of understanding as to why amniotic fluid can trigger a reaction, what is well accepted is that amniotic fluid must first enter the maternal circulation. Amniotic fluid is normally separated from the maternal circulation by the intact fetal membranes and AFE is thought to occur when there is a breach in this barrier. A breach may occur at a number of sites, including the endocervical veins, the area of placentation and sites of uterine trauma. Conditions that increase intrauterine pressure may contribute to the passage of amniotic fluid into the maternal circulation (for example, augmentation of labour with oxytocin) and go some way to explaining some of the identified risk factors for AFE.

However, potentially confusing the current understanding of AFE is the observation that amniotic fluid may enter the maternal circulation as a relatively normal aspect of childbirth and not trigger any adverse reaction. Evidence supporting this includes studies whereby components of amniotic fluid have been found in the maternal circulation without any other evidence of an AFE reaction[4], in addition to the failure of AFE to be reliably reproduced by the injection of amniotic fluid into the circulation in animal models. This would suggest that amniotic fluid only triggers an AFE in a small proportion of women.

It is unclear what components of amniotic fluid or meconium lead to the clinical syndrome that is seen with AFE. Amniotic fluid contains a number of potentially vasoactive substances as well as substances that may interfere with coagulation.[5] In addition, there may be immune-mediated processes that contribute. This is supported by a number of observational findings, such as AFE being more common in women carrying a male fetus, as well as decreased complement levels

Summary
AFE is currently a leading cause of maternal mortality in the developed world and it cannot be either predicted or prevented. However, there would appear to be a significant improvement in the both the morbidity and mortality of AFE. Severe cases of AFE are likely to present with a combination of cardio-respiratory compromise and coagulopathy, although the exact mechanism for the presenting signs and symptoms is unclear. The management is essentially supportive following the principles of care for unwell obstetric patients. Coagulation abnormalities may be significant and expert assistance may be required. The greater availability of echocardiography may help guide haemodynamic therapy in severely unwell women. In Australia and New Zealand, AFE is the leading cause of direct maternal death, it is currently unclear why this is the case and further research, such as that being conducted by Australasian Maternity Outcomes Surveillance System, is required.

(suggesting complement activation). An elevated mast cell tryptase, usually a finding in cases of anaphylaxis, is not routinely seen with AFE, suggesting that anaphylaxis does not play a significant role. However, it has been suggested that an anaphylactoid process (which is not an IgE-linked reaction) may contribute to AFE and hence one of the suggested alternative names.[2]

Risk factors

A large number of risk factors for AFE have been identified, including advanced maternal age, placenta praevia, placental abruption, operative delivery and induction of labour.[6] However, given the rare and unpredictable nature of AFE, these risk factors are only useful for retrospective analysis. Identified risk factors should not currently be used to alter the clinical management of individual woman (for instance, avoidance of induction of labour or caesarean delivery) as the baseline risk is still very low.

Table 1. Incidence of AFE and case fatality rates in published series.

Author	Year Published	Incidence (per 100 000 maternities)	Case fatality rate (%)
Knight[1]	2012	1.9-6.1	11–43
Knight[10]	2010	2.0	20
Abenhaim[6]	2008	7.7	21.6
Tuffnell[11]	2005	not reported	29.5
Gilbert[12]	1999	4.8	26.4
Clark[2]	1996	not reported	61
Burrows[13]	1995	3.4	22
Morgan[3]	1979	not reported	86

Table 2. Signs and symptoms of AFE.

Signs or symptoms	Frequency (%)
Hypotension	100
Fetal distress	100
Pulmonary oedema or ARDS	93
Cardiopulmonary arrest	87
Cyanosis	83
Coagulopathy	83
Dyspnea	49
Seizure	48
Uterine atony	23
Bronchospasm	15
Transient hypertension	11
Cough	7
Headache	7
Chest pain	2

This data, while dated and reflecting the presentation of more severe cases of AFE, demonstrates the wide range of signs and symptoms of AFE. Adapted from Clark.[2]

Clinical presentation

The majority of episodes of AFE are reported to occur in the intrapartum and immediate postpartum period, although cases have been described during amniocentesis, termination of pregnancies, abdominal trauma and post-caesarean delivery. The classic description is of a sudden and severe deterioration that is rapidly fatal. With greater recognition of the syndrome, less-severe cases may present with more subtle signs and symptoms. Table 2 shows the wide range of features that AFE may present with and, while these data are comparatively dated and reflect more serious cases, it does highlight the significant cardio-respiratory involvement in the condition as well as the likelihood of a compromised fetus.

Hypotension is a common sign in severe episodes of AFE, although the exact mechanism is unclear and may vary between patients. Case reports have documented a variety of contributing factors, including severe pulmonary vasospasm and pulmonary hypertension; impaired left ventricular filling secondary to severe right ventricular failure; and myocardial ischaemia. It has also been suggested that either amniotic fluid or meconium may contain substances that have a direct myocardial depressant effect, or that a substance with direct myocardial depression is released as part of the episode.

Respiratory signs and symptoms may range from shortness of breath to hypoxia or respiratory arrest. The underlying mechanism for the hypoxia is likely to be multifactorial. Initially, severe ventilation and perfusion mismatching may occur; then, as the episode progresses, cardiogenic pulmonary oedema may then develop secondary to left ventricular failure. In addition, non-cardiogenic pulmonary oedema, secondary to capillary damage from amniotic fluid, may also develop.

Coagulation disturbances can occur rapidly in AFE and, in some cases, may be the initial presenting sign. Amniotic fluid contains both tissue factor that acts as a pro-coagulant as well as plasminogen activation inhibitor-1, which is involved in fibrinolysis. Thus, both a consumptive coagulopathy as well as massive fibrinolysis may occur.[7]

Diagnosis

The diagnosis of AFE is a clinical one, based on the presence of cardiovascular and respiratory compromise and coagulopathy after the exclusion of other causes.[4] There is currently no widely available diagnostic test that can be used in survivors. The diagnosis can be made at autopsy by examining for the presence of fetal material in the maternal pulmonary circulation. However, given that fetal material may be present in otherwise normal women, the sensitivity of this finding may not be as high as first thought.

Management

The initial management of a suspected episode of AFE should be tailored to the severity of the presentation. In severe cases, management is essentially supportive, following the principles of basic and advanced life support, with modifications made for the

pregnant or recently pregnant state (uterine displacement and early securing of the maternal airway).[8] If the presentation occurs prior to delivery then consideration should be given to urgent delivery of the fetus. While this may limit hypoxic damage to the fetus, its main benefit is for the mother, with potentially improved cardiac output by the limitation of aortocaval compression as well decreasing oxygen consumption.

Maternal oxygenation is likely to be significantly compromised in severe cases such that intubation and positive pressure ventilation will be required. Given the potential difficulties associated with intubation in the pregnant patient as well as the compromised maternal state, expert assistance should be obtained where available. Haemodynamic support is likely to be required and echocardiography can be a useful modality to help guide appropriate therapy.[7]

Coagulation abnormalities can develop rapidly; the potential for major haemorrhage should be anticipated and coagulation studies performed as soon as possible. Point-of-care coagulation monitoring (for example with thromboelastography [TEG] or rotational thromboelastometry [ROTEM]) may allow more specific targeting of coagulation factors, compared with traditional tests such as the international normalised ratio (INR) and activated partial thromboplastin time (aPTT). In addition to conventional blood products, antifibrinolytics, such as tranexamic acid, may also be of benefit, while the use of recombinant activated factor VII has been potentially linked to poorer outcomes.[9]

A number of novel therapies have also been described in case reports for AFE. These include the use of cardio-pulmonary bypass, extracorporeal membrane oxygenation, inhaled nitric oxide and prostacycline and haemofiltration or plasma exchange. The consideration of such therapies will be dependant on the local expertise available as well as the clinical condition of the patient.

Future pregnancies in survivors of AFE

A common issue faced by survivors of AFE is whether it will recur in future pregnancies. This may be a significant source of anxiety when the next pregnancy does occur. Currently, there is no evidence to suggest these women are at a higher risk of recurrence, with no documented cases of women having suffered a subsequent AFE.

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RANZCOG

2013 ANNUAL SCIENTIFIC MEETING

Evidence in O&G

Food for thought or recipe for disaster?

call for abstracts

Abstracts are invited for Free Communication Oral and Electronic (E-Poster) Presentations for the Royal Australian and New Zealand College of Obstetricians and Gynaecologists 2013 Annual Scientific Meeting (RANZCOG 2013 ASM) being held in Sydney on 8-11 September 2013. Abstracts are welcome on any topic relevant to women's health. The deadline for submission is Friday 17 May 2013.

Abstracts will be reviewed by the Organising Committee and will be selected using criteria such as:

- Presentation of new research findings
- Utilisations of new technology in obstetrics and gynaecology
- Papers that challenge current thinking in obstetrics and gynaecology

Abstracts should be submitted by the presenting author. Only one author per abstract can be nominated as the 'presenting author'.

Please note that all presenters (Free Communications and E-Posters) must register for the meeting and pay the appropriate registration fee by the early bird deadline **Friday 12 July 2013**. It will be assumed any presenter not registered by this date has withdrawn their abstract from the program.

8-11 September 2013

Sydney Convention
& Exhibition Centre

Awards and Prizes

Best Free Communication and Best Poster

During the meeting prizes will be awarded to the Best Free Communication presented and Best Electronic (E-Poster) Presentation displayed.

RANZCOG Early Career Researcher Award

A prize of A$1,000.00 will be awarded to the Best Free Communication presented by an Early Career Researcher. To be eligible the presenting author must be a FRANZCOG Trainee or a RANZCOG Fellow five years or less post-Fellowship at the time of the meeting.

RANZCOG Diplomate Researcher Award

A prize of A$1,000.00 will be awarded to the Best Free Communication presented by a Diplomate Researcher. To be eligible the presenting author must be a Diploma Trainee or a DRANZCOG / DRANZCOG Advanced holder.

Finalists of the RANZCOG Early Career Researcher Award and RANZCOG Diplomate Researcher Award will be asked to make an eight (8) minute presentation followed by a two (2) minute question time. Remaining applicants may be invited to submit e-poster presentations. To be considered for either researcher award, please indicate on the online submission form your eligibility when submitting an abstract and prepare your abstract in accordance with the *Abstract Preparation and Submission Guidelines*.

Presentation Format

Authors will be required to indicate the preferred presentation format when submitting an abstract.

- Please select Free Communication if you wish for your abstract to be considered for Free Communication Oral Presentation only.
- Please select Electronic (E-Poster) Presentation if you wish for your abstract to be considered for an Electronic (E-Poster) Presentation only.
- Please select Free Communication or Electronic (E-Poster) Presentation if you wish for your abstract to be considered firstly for Free Communication Oral Presentation and secondly for Electronic (E-Poster) Presentation (if not accepted for Free Communication).

Presentation Types

Free Communication Oral Presentations

Free Communications will be allocated an eight (8) minute presentation followed by a two (2) minute question time. Abstracts not selected for Free Communications may be eligible for presentation as Electronic (E-Poster) presentations. Please note that following acceptance of an abstract for Free Communication, the abstract shall not be presented to any other scientific body before the RANZCOG 2013 ASM.

Electronic (E-Poster) Presentations

All Poster presentations will be in Electronic (E-Poster) format and will be prominently displayed in the meeting venue. Moderated E-Poster sessions may be structured on the program for which presenting authors will need to be available to answer questions about their posters. Full details of the format and display of E-Posters will be detailed within acceptance notifications sent to nominated presenting authors.

RANZCOG

2013 ANNUAL SCIENTIFIC MEETING

Evidence in O&G

Food for thought or recipe for disaster?

Preparation Guidelines

1 Length and Structure

- A maximum of 250 words excluding title will be accepted.
- Abstracts should be structured to contain an objective, statement of findings or supporting information and key conclusions or recommendations.

2 Spelling and Grammar

- All abstracts should be written in English and thoroughly checked for spelling and grammar before submission.

3 Layout and Format

- Each abstract must be prepared in Microsoft Word. No other formats will be accepted.
- The title of the abstract should be written in sentence case i.e. initial capital letter, followed by lower case.
- List details of the author/s name/s (surname followed by initial of first name with a comma separating each author). Name of the presenting author must be asterisked (*).
- List author/s affiliation/s (organisation, city, state, country), separated by commas. Author/s and affiliation/s to be linked by a superscript number.
- Please note that double spacing should separate the title, author/s name/s, affiliation/s and body of the text.
- Please refrain from using printed enhancements such as italics, underlining, bold text etc. Italics may be used for non-English words or scientific names where necessary.
- Please note abstracts may be re-formatted for publication in the program handbook.

Abstract sample:

Obstetric solutions

Phillips J[1]*, Thomas D[2]

[1] Women's and Childrens' Hospital, North Adelaide, South Australia
[2] Royal Brisbane and Women's Hospital, Brisbane, Queensland

Abstract text should be typed in English not exceeding 250 words.

Submission Guidelines

- Closing date for abstract submission is **Friday 17 May 2013**.
- All abstracts must be submitted electronically via the online submission facility (Presenter Portal) accessed on the meeting website www.ranzcog2013asm.com.au
- Authors wishing to submit more than one abstract may do so by clicking 'New Abstract' in the Presenter Portal.
- Abstracts should be submitted by the presenting author. Only one author per abstract can be nominated the 'presenting author'.
- All submissions must be accompanied by a maximum 80 word biographical paragraph of the presenting author. Biographical details will be used by the session chair for introductions purposes and may also be published in meeting literature.
- Submission of abstracts implies the author's agreement to publish the abstract in the meeting program handbook and other meeting literature as applicable.
- Abstracts accepted for Free Communication Oral and Electronic (E-Poster) Presentations may be provided upon request to media outlets promoting a specific presentation. Please note that submission of abstracts implies the author's consent.
- Please note authors of the abstracts accepted for Free Communication Presentations may be invited to submit the abstract for publication in The Australian and New Zealand Journal of Obstetrics and Gynaecology (ANZJOG). In this instance, presenting authors will be required to complete a copyright form prior to publication.

8-11 September 2013

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Key Dates

Deadline for receipt of abstracts
Friday 17 May 2013

Authors notified whether abstracts have been accepted
Monday 11 June 2013

Authors to confirm participation
Monday 24 June 2013

Deadline for presenting authors to register
Friday 12 July 2013

Early bird registration closes
Friday 12 July 2013

RANZCOG 2013 ASM
8-11 September 2013

- Please note that all accepted presenters (Free Communications and E-Posters) must register for the meeting and pay the appropriate registration fee by the early bird deadline **Friday 12 July 2013**.

Please ensure you have the following required information available before beginning the online abstract submission process:

- Your contact details.
- The title of your abstract.
- Preferred presentation type (Free Communication and / or E-Poster Presentation).
- Name and organisation of your co-author/s, if applicable.
- Your (the presenting author's) brief biography (maximum 80 words). Biographies are to be written in full sentences (not dot points) and in third person.
- Your abstract prepared in the format specified under Abstract Preparation Guidelines.

Failure to meet the specified deadlines or guidelines may result in exclusion from the program.

Presenter Terms and Conditions

Submission of your abstract/s implies you have read, understood and comply with the Presenter Terms and Conditions. These are available to view within the Presenter Portal (online submission facility) at the time of submitting your abstract.

Double Check Your Submission

A confirmation e-mail will not be sent when an abstract has been submitted and you are required to double check your abstract has uploaded correctly during the upload process. To do this you should log back into the Presenters Portal (online submission facility) and check your submission status on the "Show Abstracts" page. If you have any concerns, please contact the meeting Secretariat immediately, by email to ranzcog2013asm@wsm.com.au or by phone on +61 3 9645 6311.

Acceptance Notification

Acceptance or otherwise is at the discretion of the Organising Committee. Notification will be sent to the presenting author by **Monday 11 June 2013**. Full instructions for accepted presenters will be forwarded on acceptance of the abstract.

Further Information

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Postpartum haemorrhage

Dr Katherine Mckenzie
FRANZCOG

A typographical error in lecture notes that described postpartum haemorrhage as 'one of the moist testing situations for the labour ward team' has since seemed to me a very appropriate summary of postpartum haemorrhage.

Globally, it is estimated that half a million women die annually from causes related to pregnancy and childbirth and that half of these deaths are related to obstetric haemorrhage. The risk of death from obstetric haemorrhage in Australasia and the UK is rare, and has declined in the UK over the past two triennia from rates of 0.85/100 000 to 0.39/100 000. Unfortunately, surveys have shown more than two-thirds of cases of severe maternal morbidity, or near misses, are attributable to haemorrhage and the incidence of major obstetric haemorrhage seems to be increasing. Furthermore, substandard care was cited as a major contributor to the nine deaths in the UK from haemorrhage in the most recent Centre for Maternal and Child Enquiries (CMACE) report.[1,1a,19]

WHO defines primary postpartum haemorrhage (PPH) as genital tract blood loss greater than or equal to 500ml within 24 hours after birth, while secondary PPH occurs from 24 hours to 12 weeks postpartum.[2] This is a very arbitrary definition that fails to take into account the subjective nature and inaccuracy of visual estimations of blood loss. It fails to acknowledge differences in losses between vaginal and caesarean birth or that losses of this magnitude rarely compromise maternal wellbeing in our population, although this is dependent on pre-existing medical conditions. Perhaps a more clinically relevant (separate from data-collection requirements and audit) definition is blood loss that causes haemodynamic instability (even if loss is less than 500ml), is in excess of 1000ml or necessitates red cell transfusion.[3,6,7,9,10,13]

So what makes a difference to outcome in PPH?

Table 1. Risk factors PPH

Antenatal	Intra/peripartum
Polyhydramnios Multiple pregnancy Fibroids Past PPH Previous retained placenta Previous Caesarean Section/ uterine surgery Placenta praevia/percreta/ increta APH High parity Maternal Age Obesity Drugs e.g. Nifedipine/$MgSO_4$/ salbutamol Hypertensive disorders Pre-existing coagulation disorder e.g. Von Willebrand's Therapeutic anticoagulation Anaemia	Fetal demise in utero Abruption Induction/augmentation of labour Prolonged labour Pyrexia Prolonged ruptured membranes Instrumental delivery Episiotomy Retained placenta/membranes Physiological third stage Drugs e.g. inhaled anaesthetic agents Therapeutic anticoagulation/ DIC

Prevention

There are many risk factors that can be identified at booking.[2a,12,13,18,19] Health professionals must be aware of specific antenatal risk factors for PPH (see Table 1) and should take these into account when counselling women about place of delivery and type of care. Care plans must be modified when risk factors are identified. Anaemia identified antenatally should be treated. Women with bleeding disorders should have a clear plan of intrapartum management documented after consultation with an obstetric physician or haematologist. Any woman with a previous caesarean section should have an ultrasound scan to identify placental site and, if it is praevia or of any concern, referred to a tertiary centre.[1,2a,6,7,9,13] Women with placenta accreta/percreta are at very high risk of major PPH. If placenta accreta or percreta is diagnosed antenatally, there should be consultant-led multidisciplinary planning for delivery.

More often overlooked are those risk factors which develop intrapartum (see Table 1). There is clear evidence from randomised controlled trials that active management of the third stage (including use of a uterotonic) results in reduced blood loss and therefore reduced risk of PPH. Plans for active management of the third stage should be documented for those identified at risk antenatally and consideration given to routine use of preventative measures in intrapartum situations known to increase risk, for example, pyrexia or assisted delivery.

WHO recommends active management should be offered to all women attended by a skilled practitioner.[2] Prophylactic use of oxytocics reduces the risk of PPH by 60 per cent.[3,4,11,12]

Recognition

You would think it should be easy to identify a PPH, but minor blood loss can gradually become major haemorrhage, visual estimations frequently underestimate loss[5,17] and haemorrhage may be concealed. It is vital to take and react to patient observations. The most common site of concealed haemorrhage is the uterus, hence the importance of monitoring uterine tone and fundal height postnatally.

By the time a woman drifts into unconsciousness she will have lost around 40 per cent of her circulating volume. It is the hypovolaemia, rather than anaemia, that kills women during an acute haemorrhage. It seems appropriate that PPH protocols should be instituted at an estimated blood loss well below this figure, as the

Table 2. The 4 Ts (after ALSO).

Tone	Abnormalities of uterine contraction
Tissue	Retained products of conception
Trauma	Genital tract trauma
Thrombin	Abnormalities of coagulation

aim of management is to prevent haemorrhage escalating to the point where it is life threatening.

Prompt, appropriate intervention

An empty contracted uterus will not bleed in the absence of a coagulopathy. The four Ts, developed by ALSO (see Table 2), are a good aide mémoire when managing PPH and thinking of cause. By far the most common cause of PPH is uterine atony, with genital tract trauma and retained products of conception often co-existing. A clotting diathesis is rarely primary. Disseminated intravascular coagulation (DIC) is usually secondary to significant blood loss and loss of clotting factors secondary to a condition such as abruptio placantae or pre-eclamptic toxaemia, which is the trigger for DIC.

Accepting that antenatal risk assessment at best identifies only 40 per cent of those who have a PPH and that delay(s) in initiating appropriate management is the major factor resulting in adverse outcomes after PPH, it is essential to have a clear, logical sequence for management.[1,2,18] Protocols and flow charts may be helpful. In the seventh Confidential Enquiry into Maternal Deaths report, failure of identification and management of intra-abdominal bleeding, uterine atony and placenta percreta were the main reasons for substandard care (Confidential Enquiry into Maternal and Child Health, 2006). Furthermore, a reduction in length of postgraduate training programs and reduced hours have led to less practical experience, which may result in failure to recognise even the clear signs and symptoms of intra-abdominal bleeding.[1] The UK Royal College of Obstetricians and Gynaecologists and the Royal College of Midwives have thus recommended annual 'skill drills', including maternal collapse. RANZCOG is also encouraging development of multidisciplinary obstetric emergency courses (for example, PRactical Obstetric Multi-Professional Training) to ensure that everyone knows how to work together to ensure swift and efficient treatment in such an emergency.

Management of the acute presentation of PPH requires multiple tasks to be performed simultaneously. The basic principles can be grouped under the following principles of assessing the maternal condition and extent of the bleeding, arresting the bleeding and replacing her circulating volume and blood products.[7]

Once a PPH has been identified, appropriate help should be called. Early involvement of appropriate senior staff, including laboratory specialists, is fundamental to the management of major PPH. It is vital that trainee obstetricians and anaesthetists do not perceive calling for senior colleagues as involving 'loss of face'. Senior staff must be receptive to concerns expressed by juniors and midwives.

A primary survey of a collapsed or severely bleeding woman should follow a structured approach of simple 'ABC' (airway, breathing, circulation), with resuscitation taking place as problems are identified; that is, a process of simultaneous evaluation and resuscitation. Simple measures such as laying the patient flat, keeping her warm and administering oxygen should not be overlooked. The urgency and measures undertaken to resuscitate and arrest haemorrhage need to be tailored to the degree of shock.

It is important to assess both the extent of prior bleeding and ongoing loss. Blood loss is usually visible at the introitus and this is especially true if the placenta has been delivered. If the placenta remains in situ, then a significant amount of blood can be retained in the uterus behind a partially separated placenta, the membranes or both. Loss may be concealed in kidney dishes on the delivery trolley, in linen or beneath the patient.

The cause of bleeding should be established as best able (see Table 2). The most common cause of primary PPH is uterine atony. Fundal massage should expel clot, improve uterine tone and provide an assessment of contracted fundal height. An in-dwelling catheter will keep the bladder empty and may later be useful for fluid management.

A further ecbolic should be given: 5iu syntocinon as a slow intravenous (IV) infusion is recommended as first-line treatment. In addition, Ergometrine, a syntocinon infusion or Carboprost (prostaglandin $F_{2\alpha}$) may be needed.[2,3,9,10,13]

If pharmacological measures are failing to control the haemorrhage, arrangements to transfer to theatre, then interventional/surgical measures should be instituted until the bleeding stops.

Contemporaneously, fluid resuscitation should be underway. The aim is to rapidly restore circulating blood volume. Two large-bore IV catheters should be sited and bloods drawn for complete blood count, coagulation studies, baseline renal and liver function and cross match.

Fluid resuscitation in obstetrics is often overly conservative either owing to underestimating loss, delay in symptoms of hypovolaemia in women with good compensatory mechanisms or because of concern that over resuscitation will cause pulmonary oedema. Until blood is available, infuse up to 3.5 litres of warmed fluid solution, crystalloid and/or colloid, as rapidly as required. The best equipment available should be used to achieve rapid, warmed infusion of fluids. Special care needs to be taken in patients with pre-eclampsia. Once adequate clear fluids are given, think O_2 carrying capacity.

Compatible blood should be given as soon as available, but if 3.5 litres clear fluid and bleeding is massive and ongoing then un-cross-matched blood should be given. Fresh frozen plasma, platelets and cryoprecipitate should be given if the clinical situation or clotting results warrant this. After six units of red cells, clotting is likely to be altered. Clinicians and blood transfusion staff should liaise at a local level to agree on a standard form of words (such as 'we need compatible blood now' or 'group-specific blood') to be used in cases of major obstetric haemorrhage and also a timescale in which to produce various products.[6,7,8,13] Most large units will have a massive transfusion protocol that, when activated, will ensure appropriate blood and products are delivered to theatre.

Once in theatre there should be a systematic exploration of the genital tract, repairing tears and identifying any factors contributing to ongoing loss. The judgment of senior clinicians, taking into account the individual woman's future reproductive aspirations, is required in deciding the appropriate sequence of interventions. Balloon tamponade has replaced uterine packing in the management of PPH secondary to atony. A variety of hydrostatic balloons are available with similar efficacy. Studies suggest successful avoidance of hysterectomy in up to 78 per cent of cases using a balloon. Haemostatic sutures (B-Lynch, Hayman, Cho and so forth)[14,15] placed at laparotomy have been shown to be effective in controlling severe PPH from atony, reducing the need for hysterectomy. Uterine artery ligation may significantly reduce bleeding and is relatively simple to perform; while internal iliac ligation is best left to those comfortable operating in the pelvic sidewall and not undertaken by generalists 'occasionally' during an emergency. While there are no studies making direct

comparisons, it would seem balloon tamponade and haemostatic uterine sutures are equally effective as internal artery ligation in reducing the need for hysterectomy and may be more appropriate as first-line procedures. In large centres, there may be facilities for radiological intervention and selective uterine or internal iliac embolisation in the stable patient with ongoing loss. In the face of life-threatening PPH, recombinant factor VIIa may have a useful adjuvant role to surgical treatment.[13]

Most literature supports early recourse to hysterectomy, particularly in situations of uterine rupture or accreta/percreta. Ideally, the decision should be made by a senior obstetrician and surgery performed by a surgeon.[1,6,8,13,16] Once the bleeding has been controlled and initial resuscitation has been completed, continuous close observations in either a high-dependency unit on the labour ward or an intensive care unit is required. The recording of observations on a flowchart helps in the early identification of continuous bleeding.

It is also important that once the bleeding is arrested and any coagulopathy is corrected, thrombo-prophylaxis is administered, as there is a high risk of thrombosis.

Debriefing of patients and relatives is an important aspect of management that is often overlooked. Complications following massive PPH are diverse and may not be immediately apparent. They range from surgical complications (synechiae, uterine necrosis, sepsis) through to delayed lactation (Sheehan's syndrome) and post-traumatic stress disorder.

Stalin said: 'One death is a tragedy. One million is a statistic.' By preventing one, we may improve the other.

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Severe pre-eclampsia: its recognition and management

Dr Barry N J Walters
FRANZCOG (ad eundem)
FRACP

Of all serious medical complications of pregnancy, pre-eclampsia dominates as relatively common, dangerous, unpredictable in its course and difficult to manage. In all formal reports of maternal and perinatal mortality, it features prominently.

This article deals with certain aspects of the disorder that have not been as well covered in the literature as other elements. The clinical manifestations are manifold, variable and sometimes, if unrecognised, can have unfortunate results. The first truism to accept is that there is no such entity as 'mild' pre-eclampsia. All cases have the potential to undergo a transition to a life-threatening illness for mother and baby and, therefore, close monitoring as an inpatient is essential.

Pre-eclampsia is a disease of the placenta and its vasculature. The placenta cannot be directly examined and its function is poorly assessed by current techniques. The derangements that come to clinical attention are protean, variable and, in the early stages of pre-eclampsia, may not include hypertension, the usual diagnostic entry point.

For these reasons, every woman, particularly those in the first pregnancy, should be regarded as being at risk. It is largely to detect pre-eclampsia that repeated visits to the obstetrician are necessary, since it may be absent at an antenatal visit yet present in a severe form within days.

Symptoms

A perplexing feature of pre-eclampsia is the fact that it may present with symptoms before any clinical signs appear. In other cases, there may be some clinical signs, but minimal or absent hypertension, and even these cases may progress undetected and lead to fetal death, eclampsia, abruption or other severe maternal complications. Many women who ultimately develop severe pre-eclampsia experience suggestive symptoms in the days or weeks before diagnosis, including headaches, visual disturbance, swelling, dyspnoea, nausea or vomiting, reduced fetal activity and, particularly in the worst cases, a severe pain in the upper abdomen or lower chest that has distinctive diagnostic characteristics that are defined in a paper detailing many cases.[1]

The term chosen for this pain is 'pre-eclamptic angina', angina being used in its original sense as signifying a severe cramp-like pain. The presence of pre-eclamptic angina signifies a grave prognosis for mother and baby, as it is a feature of only the worst type of pre-eclampsia. It is a sign of imminent calamity and terrible complications may be seen in those women who manifest pre-eclamptic angina. Its genesis lies in hepatic infarcts and haemorrhages, which were well documented by Sheehan in his landmark text[2] describing the pathology at postmortem of many cases of severe pre-eclampsia and eclampsia. This severe, and often recurring, symptom (sometimes first experienced a week or more before presentation) is a marker of a dangerous and unstable state. Those with pre-eclamptic angina are in need of urgent delivery. Women with any of the above symptoms require careful evaluation, with attention to pre-eclampsia as the possible underlying cause.

Signs and laboratory features

The classical sign of pre-eclampsia is hypertension, but it is usually episodic and may be entirely absent when the woman is seen in the morning, but severe and dangerous by the late afternoon or at night. This reversal of the diurnal rhythm of blood pressure in pre-eclampsia was described decades ago.[3] The presence of hyperactivity of the deep tendon reflexes or clonus is of great concern and suggests nervous system involvement and risk of progression to complications, even eclampsia.

> '...medicine has not yet mastered the prediction or management of this enigmatic disorder and has no cure other than delivery'

Tenderness of the liver accompanies the pain mentioned above and should be taken as a worrying feature. Oedema is often seen, but is not invariable and many women develop substantial oedema in normal pregnancy so careful evaluation is necessary. Proteinuria is a sign of severity and is not required to diagnose pre-eclampsia as in many pre-eclamptic women it is not present until the disease reaches a late stage. Thus many with the disease do not have proteinuria at presentation and its absence cannot be taken as reassuring, while its presence indicates a more advanced stage and those with it are at greater risk. In years past, heavy proteinuria was taken as an absolute indication for delivery. Now, with better fetal assessment and monitoring, pregnancy may be allowed to proceed in the presence of proteinuria, but only at very early gestations where it is felt that prolongation is essential for fetal reasons, as continuation of pregnancy in this situation is always at the cost of risk to the baby and mother. Proteinuria is a reliable indicator of severity signifying the need for intensive repeated monitoring of the baby as old studies confirmed that proteinuria correlates with higher risk of fetal demise.[4]

The laboratory features of pre-eclampsia are well known, but the presence of normal tests does not exclude the disorder and should not provide reassurance. The most reliable test, although even it may mislead in some cases, is plasma uric acid[5] that rises in most cases,

but not until the disease is entering a more severe state. It should not exceed 0.33 in any pregnant woman (unless there is renal disease or dehydration and it is not reliable with twins) and should not exceed the same numerical value as the gestational age before 34 weeks – thus at 28 weeks, it should be no higher than 0.28.

Thrombocytopenia, any evidence of haemolysis or a high haemoglobin (indicating plasma volume contraction), elevation of alanine aminotransferase and any elevation in creatinine are all worrying features. In 1954, the combination of some of these abnormalities was described[6] in very advanced cases, but this classic description went largely ignored until the acronym 'HELLP' was applied to this clinical subgroup many years later[7] and this has benefited women and babies as it is a memorable term and has increased the recognition of a severe variant of pre-eclampsia that sometimes does not manifest hypertension, but is nevertheless very dangerous.

Complications and their prevention

Pre-eclampsia is a progressive disorder that worsens always, sometimes gradually and sometimes with fulminating rapidity. Therefore, repeated assessment of mother and baby is necessary. Ideally, this should be in hospital – so volatile and unpredictable is pre-eclampsia that it demands in-patient observation and care. It cannot safely be dealt with as an outpatient and neither can it be assessed reliably in a period of a few hours as its worst signs may not be present until later.

I believe that there is no place for the recently developed 'maternal fetal assessment unit' in the evaluation of cases of suspected pre-eclampsia, as a reliable predictive assessment cannot be made in a short period of time. Ongoing inpatient care is essential for these women.

The aim of continuing care in hospital is to assess maternal and fetal fitness to continue the pregnancy, and to control hypertension. Pre-eclampsia worsens until delivery, and even for some time afterwards. For this reason it is always in the mother's best interests to deliver the baby, but in very early cases it is usually necessary to attempt prolongation for fetal benefit, even though this exposes the mother to the hazards of eclampsia, abruption, hypertensive cerebral haemorrhage, pulmonary oedema, retinal detachment, hepatic haemorrhage and renal impairment or failure. Such attempts at prolongation require the most assiduous and diligent of monitoring, and should only be undertaken at a tertiary centre with the best available modalities of fetal and maternal surveillance and, ideally, assistance from a physician trained in Obstetric Medicine.

When the baby is mature, there is no benefit that justifies the risk in deferring delivery. A recent study[8] randomised women with pre-eclampsia between 34 and 37 weeks to either immediate delivery or attempted prolongation. This valuable work showed no difference in fetal outcome, but more maternal complications in those where delivery was not undertaken promptly. For this reason, after 34 weeks it is difficult to justify continuing the pregnancy in women with definite pre-eclampsia. Moreover, when the decision is taken to deliver the baby, it should be accomplished without delay. Where betamethasone administration is required for fetal lung maturation it is reasonable to wait, provided the fetus is constantly monitored. Otherwise, even overnight delay may expose the baby and mother to unnecessary risk such that if delivery is determined to be necessary it should not wait until the next morning. Many cases have deteriorated in that interim period.

However, delivery alone is insufficient management of pre-eclampsia. It must always be combined with control of abnormalities and preparation of the patient including any or all of the following: control of severe hypertension by use of oral or intravenous agents, correction of disordered fluid status, correction of coagulopathy, and prophylaxis against eclampsia by means of magnesium sulphate. Not all those with pre-eclampsia require magnesium. The indications are uncontrolled hypertension, persistent headache or vomiting, altered conscious state, tremor or agitation, hyperreflexia with or without clonus and, of course, eclampsia itself. Any woman requiring magnesium must be delivered as soon as possible and the magnesium continued for 24 hours.

When is delivery necessary?

As mentioned above, after 34 weeks there is little benefit and considerable hazard in continuing the pregnancy with definite pre-eclampsia. The decision to terminate the pregnancy earlier than 34 weeks is always difficult, and involves consideration of a number of factors. Prediction of progression in pre-eclampsia is imprecise and unreliable, although the recently available test 'Placental Growth Factor' has been shown of reasonable predictive value in studies elsewhere.[9] This test may well provide a useful enhancement to the current care of women with pre-eclampsia.

The following factors represent maternal endpoints. Once reached, any one of these indicates that delivery is necessary or that if continuation (to achieve fetal viability) is to be attempted it carries high risk:

- failure of blood pressure control despite use of any two drugs in standard doses;
- worsening thrombocytopenia;
- pre-eclamptic angina or liver test abnormalities;
- rising creatinine, oliguria despite adequate hydration, or heavy proteinuria;
- pulmonary oedema;
- haemolysis;
- persistent neurological symptoms, any alteration of conscious state, confusion or persistent headache;
- antepartum haemorrhage; or
- other persistent symptoms such as vomiting or accumulating oedema.

Fetal indications often constitute the reason for delivery. These include the following:

- abnormal fetal CTG monitoring or flow aberrations on ultrasound;
- subnormal growth between ultrasound examinations;
- reduction in amniotic fluid volume; and
- achievement of sufficient maturity, namely 34 weeks, but earlier in severe cases.

In many cases, a combination of subcritical factors may decide the need for delivery, even though no single factor, by itself, would constitute an endpoint. When pre-eclampsia complicates diabetes, renal disease, lupus or any other medical or obstetric (for example, intrauterine growth restriction) disorder, the prognosis is worse and the decision for delivery should be made at the slightest sign of deterioration, usually earlier than in cases not complicated by a second disorder.

It should also be recognised that the whole situation of being under surveillance for a life-threatening condition is so stressful for the patient that she may decide she cannot tolerate further

uncertainty and, in these cases, it is entirely correct to agree to her request for delivery, even if things may seem medically stable. It is the patient and her family who suffer most and her opinion must be taken seriously.

After delivery, the disease remains active for at least a week and sometimes longer. The woman's blood pressure commonly reaches severe levels three to seven days postpartum[10] and eclampsia has been seen several days after delivery. This postnatal hypertension may need ongoing drug therapy. Thus, continued surveillance of the mother is well justified and she should not be sent home less than a week after delivery. The blood test abnormalities slowly return to normal, but it may take many days for the platelet count to rise, liver enzymes to fall and renal function to recover.

It is a great pity, but nevertheless the truth, that medicine has not yet mastered the prediction or management of this enigmatic disorder and has no cure other than delivery. This situation may change with future advances, but in the meantime the best that can be offered is diligent and insightful care and meticulous monitoring, bearing in mind that, if the pregnancy is continued too long and warning features not heeded, a serious complication is very likely to occur in every case of pre-eclampsia, and this may include fetal or maternal death.

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Sepsis

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Recent data show that cases of maternal sepsis are increasing. Heightened awareness and rapid action will save lives.

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Since their inception in the 1920s, the UK Confidential Enquiries into Maternal Deaths have provided useful information and reflection on why mothers die and what we can do to prevent it. The most recent report, published in 2011, contains some old enemies, such as eclampsia and thromboembolism, but of concern is that maternal sepsis seems to be making a reappearance.[1] Australian figures place sepsis sixth in the list of direct causes of maternal mortality. Data on maternal deaths from 2003–05, attributed one of 29 cases directly and four of 26 cases indirectly to infection.[2] The WHO estimates that puerperal sepsis accounts for 15 per cent of maternal mortality globally.[3] In high-income settings, maternal death rates are so low that morbidity data may yield more useful information, such as case-fatality rates for maternal sepsis. Incidence of serious acute maternal morbidity (SAMM) from sepsis in such countries is 0.1–0.6 per 1000 deliveries.[4] However, uniformity of definitions, management protocols and data collection across countries and regions are necessary to enable meaningful comparisons.

In 1795, Alexander Gordon's Treatise on the Epidemic of Puerperal Fever in Aberdeen suggested spread of infection by healthcare workers.[5] This was followed by Semelweiss's seminal work, in 1847, demonstrating a drop in puerperal fever from 18 per cent to 1.27 per cent with simple handwashing.[6,7] Since then, antisepsis, infection-control measures, improved socio-economic conditions and antibiotics made death from puerperal fever in high-income countries close to a thing of the past. This most recent report, however, indicates an increase in maternal deaths owing to sepsis and, in particular, a resurgence of beta haemolytic Group A streptococcus (GAS) as a cause. In the report, several organisms were identified and many infections were polymicrobial, but GAS was responsible for 13 of the 29 deaths described, followed by *E.coli* and *Staphylococcus aureus*. Deaths occurred from early pregnancy through to the postnatal period. Most of those affected by GAS had complained of an antecedent upper respiratory tract infection and/or had contact with young children, and the majority of cases occurred between December and April (winter/early spring in the UK).

Streptococci are gram-positive cocci that are identified by various laboratory tests including microscopy, biochemical tests, growth and haemolysis on blood agar. Many species pathogenic for humans, including *Streptococcus pyogenes*, show a zone of complete lysis of blood surrounding their colonies (beta haemolysis) on blood agar. In 1933, Rebecca Lancefield published her method of differentiating beta-haemolytic streptococci into different serogroups, based on group-specific cell wall (carbohydrate) antigens; this is the basis of the serogrouping system still in use. Strains of GAS are further classified on the basis of a cell surface molecule, the M protein, a major virulence factor of GAS that displays a variety of properties including antiphagocytic activity. Type-specific acquired immunity to streptococcal infection is based on the development of opsonising antibodies to the antiphagocytic moiety of the M protein. Serotyping of strains based on antigenic differences between M proteins yields many different M types, although more precise information is provided by nucleotide sequence data of the emm gene that encodes the M protein. Our understanding of this important protein has recently been described as 'fragmented' and more detailed study of the full-length of M proteins from different isolates is required to help understand pathogenesis of GAS infections and sequelae (such as rheumatic fever), and to enhance vaccine development.[15]

S.pyogenes (GAS) is a pathogen specific to humans, being found mainly on the skin or in the throat, but also occasionally isolated from the female genital tract and rectum. It causes a range of different infections from impetigo and sore throat through to toxic shock syndrome and necrotising fasciitis.[4] With respect to GAS and maternal sepsis, a prospective study in North America of postpartum invasive streptococcal infections, conducted between 1995 and 2000, showed bacteraemia with no clearly identified focus of infection and endometritis to be the most common presentations, with peritonitis, toxic shock and necrotising fasciitis individually occurring in less than ten per cent of cases.[8]

For unwary practitioners and patients, typical symptoms and signs of sepsis may be lacking and clinical suspicion may be all that directs the attending clinician. Intrinsic physiological changes of pregnancy (for example, hyperdynamic circulation and changes in peripheral vascular resistance) may cloud interpretation of clinical and laboratory findings. An apparently minor skin and soft tissue infection, including post-operative wound infection, with pain out of proportion to the clinical appearance should be regarded as necrotising fasciitis until proven otherwise. Seemingly mild gastrointestinal symptoms may be due to sepsis. Feeling generally unwell with no localising signs, hypothermia, agitation and confusion and low peripheral white blood cell count may all herald sepsis. Persistent tachycardia and tachypnoea and/or a raised C-reactive protein should suggest sepsis as a possible diagnosis.

For most patients, the first interaction is likely to be with a midwife, general practitioner, emergency department doctor or other health professional who may never have seen a case of maternal sepsis owing to GAS. Failure to recognise the possibility of serious maternal sepsis and implement appropriate investigations and early empiric antibiotic treatment increases the risk of death. There is a point in sepsis beyond which antimicrobial treatment cannot prevent death, owing to a variety of factors such as,

inter alia, exotoxins produced by GAS, an exaggerated host immune response, cytokine activation and haemodynamic decompensation. Some of the documented cases illustrate the alarming speed with which GAS sepsis can take hold and inexorably progress to death. Some GAS strains possess particular M proteins that are associated with greater virulence than others; for example, M28, which has been blamed for a resurgence of severe GAS-associated puerperal sepsis.[4]

The Surviving Sepsis Campaign guidelines are endorsed by the Royal College of Obstetricians and Gynaecologists and adherence to them can optimise outcomes.[9,10] Key features include obtaining swabs (of wounds, perineum and throat), urine and blood cultures promptly and commencing broad-spectrum antibiotics within an hour of the diagnosis of septic shock. Blood cultures should be collected even if fever is not present. Prompt and adequate resuscitation with volume replacement and inotropes or vasopressors, where needed, should be instituted. Pregnant women are vulnerable to fluid overload, so support from an intensive care unit should be considered at an early stage. Teamwork is essential, with involvement of experts from key disciplines. Early consultation with a clinical microbiologist or infectious diseases physician is also recommended. Although GAS remains susceptible to penicillin, the preferred antibiotic is intravenous clindamycin (600–1200mg every six or eight hours) as it inhibits GAS exotoxin production and is immunomodulatory.[11] Because the possibility of gram-negative septicaemia may be difficult to exclude clinically, empiric therapy often involves using clindamycin in combination with agents such as piperacillin-tazobactam (or ticarcillin-clavulanate) and metronidazole. These should be modified based on clinical progress and the microbiology results.

Although trials have been small, there is some evidence of benefit from use of intravenous immune globulin (IVIg) for toxic shock and necrotising fasciitis, especially that owing to GAS, and for septic shock in general.[12,13] A recent Cochrane Review concluded: 'Polyclonal IVIg reduced mortality among adults with sepsis, but this benefit was not seen in trials with low risk of bias….Most of the trials were small and the totality of evidence is insufficient to support a robust conclusion of benefit.'[14] Despite this, and although supplies are limited and expensive, in patients severely ill with GAS-associated toxic shock and necrotising fasciitis, early consideration of IVIg use is advisable.

Timely surgical intervention is essential in the management of necrotising fasciitis. Surgical involvement may also be warranted as antibiotic therapy alone is not adequate if a focus of infection persists (for example, retained products of conception). Timing of surgery can obviously be problematic, but in a severely ill patient, high operative risk may be acceptable when balanced against the need for likely life-saving surgery.[1] Unfortunately, even with rapid response by an expert team, deaths will still occur, as was noted in the UK report.

New laboratory methods can assist investigation of clusters of infection. Pulsed-field gel electrophoresis and rapid amplified polymorphic DNA analysis have been used to determine whether a particular M type is the cause of a cluster of infections. Investigation of a cluster of GAS infections in a puerperal sepsis cluster in 2010, in NSW, by whole-genome sequencing permitted finer epidemiological discrimination of closely related strains (in this case M28 strains).[15]

Preventative measures can also be undertaken. Antiseptic practice and infection control measures are common sense, but should be reinforced. Education of mothers regarding personal hygiene, to avoid colonisation of the perineum with GAS and other bacteria, can prevent infection. Caesarean section, particularly as an emergency procedure, confers a five- to 20-fold risk of developing sepsis.[7] Use of prophylactic antibiotics, in both elective and emergency caesarean section, has been shown to reduce the incidence of endometritis by two-thirds to three-quarters and also causes a reduction in wound infection. The effects on the baby are unknown, but there is clear benefit to the mother.[16]

Maternal deaths owing to GAS infection will continue to occur. Education and training of frontline staff to recognise this rare, but potentially rapidly fatal, infection is important because, with early recognition, consultation, appropriate management and teamwork, morbidity and mortality can be kept to a minimum.

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Emergency training

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Good teamwork is a key contributing factor to successful emergency obstetric outcomes. There are three courses available within the field of obstetrics that aim to enhance teamwork and leadership.

Two courses from the UK have changed the landscape in emergency training in obstetrics in the last few years: Managing Obstetric Emergency and Trauma (MOET) and PRactical Obstetric Multi-Professional Training (PROMPT). These join the Advanced Life Support in Obstetrics (ALSO) course that originated in the USA and has been in Australia since 2001.

Most RANZCOG Fellows and Trainees will be familiar with ALSO, an entry-level emergency obstetric course that is well suited to obstetric senior residents and junior registrars. ALSO is a multidisciplinary course, bringing together obstetricians, midwives and general practitioners.

The ALSO course is owned by the American Academy of Family Physicians and overseen in Australia by an ALSO Board. Held over two days, it incorporates lectures and scenario training. The course covers basic obstetric material, such as shoulder dystocia and postpartum haemorrhage, as well as basic resuscitation. The course uses mnemonics – such as HELPERR for shoulder dystocia – that have become a feature on many a delivery suite wall.

The strength of the ALSO course is the multidisciplinary nature of its candidates and teachers. Maternal resuscitation needs to be regularly practised in order to maintain skills. All obstetricians will benefit from attending this course, although senior obstetricians may find they are not greatly challenged by some of its obstetric components.

PROMPT is an intermediate-level course that is relatively new to Australia. This course has been largely designed and promoted by a former MOET instructor. It is also multi-disciplinary and uses a variety of teaching modalities to teach core knowledge and skills in maternal resuscitation and obstetric emergencies.

The PROMPT course has been shown to produce better outcomes for obstetric units and is an essential course for anyone working on a delivery suite. Its great strength is the fact that standardised training can be delivered locally within a unit.

The focus of the PROMPT course is very much on optimising the function of the team. Since substandard care and poor outcomes often have their roots in poor communication, the teamwork nature of this course is one of the reasons RANZCOG has accepted the task of rolling it out to maternity units throughout Australia. There is, however, less focus on the team leader role and senior obstetricians may again find they are not greatly challenged by the course.

The final course that I wish to mention is the one with which I am most familiar. The MOET course was developed in the UK to equip participants with the knowledge, confidence and skills to manage serious obstetric and anaesthetic complications in pregnant women. It was designed by Charles Cox and Richard Johanson, along the same lines as the Emergency Management of Severe Trauma and Battlefield Advanced Trauma Life Support courses.

The MOET course draws on its military origins to promote a systematic and structured approach to trauma and emergency obstetric management, using the standard basic life support and advanced life support algorithms. Like the other two courses, MOET is multidisciplinary; however, it is aimed principally at obstetricians and anaesthetists at the level of consultant or senior registrar. Midwives attend as observers and participate as assistants in the scenarios, as in real life.

MOET is run under the auspices of the Advanced Life Support Group, an educational charity that is based in Manchester, UK. The course is supported by a course manual, which is a standalone teaching text that is an invaluable resource to anyone working, teaching or interested in this area.

The course and course manual underwent a major revision in 2007, which provided a greater emphasis on obstetric emergencies and less on trauma. Trauma remains, however, an excellent vehicle for teaching a structured approach. It is useful for clinicians to start with A (airway) B (breathing) C (circulation) and to focus on all aspects of resuscitation, based on the principle that adequate resuscitation of the mother is paramount. It also incorporates major and recurring recommendations from the Confidential Enquiries into Maternal and Child Health.

The great strength of the MOET course is that it challenges everyone, from candidates to instructors, each and every course. The focus is very much on learning the role of the individual as the team leader, rather than the function of the team as a whole. Using a diverse range of teaching modalities the course covers basic and advanced resuscitation to both obstetric and anaesthetic emergencies.

The MOET course started in Australia in 2009. Initially supported by Advanced Pediatric Life Support, the course is now administered by Mayhem, the organisation that also administers the ALSO courses. There are usually two MOET courses per year in Australia and one in New Zealand. Like other courses, the MOET course is regularly updated. Current plans for the course include a section on human factors and in-utero transfers. Just one of the great benefits of the MOET course is the greater understanding of obstetric anaesthesia gained by obstetricians and of obstetrics by anaesthetists. If you want a course to stimulate and challenge you, then this may be the course.

In summary, we have a variety of courses that all incorporate multidisciplinary, multi-modality and evidence-based teaching to improve obstetric care in Australia and New Zealand.

Further information
ALSO: http://www.also.net.au
MOET: http://www.moetaustralia.com.au
PROMPT: http://www.promptmaternity.org

A bold suggestion: a history of treating ectopic pregnancy

Prof Caroline de Costa
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The idea that ruptured ectopic pregnancy might be successfully treated by laparotomy was initially greeted with ridicule.

In the 21st century, ectopic pregnancy continues to make a significant contribution to maternal mortality rates. While figures are improving in developed countries, women in less-developed parts of the world frequently die from ruptured ectopic pregnancies that are either untreated or inadequately treated. Yet recognition of the condition and its clinical presentation, and definitive treatment, were well researched and documented more than a hundred years ago.

The first description of the mechanism of ectopic gestation comes from French physician Pierre Dionis, who in 1718 wrote, both accurately and poetically:

> 'If the egg is too big, or if the diameter of the tuba Fallopiana is too small, the egg stops and can get no farther, but shoots forth and takes root there; and, having the same communication with the blood vessels of the tuba that it would have had with those of the womb, had it fallen into it, it is nourished, and grows big to such a degree that the membrane of the tuba being capable of no such dilatation as that of the uterus, breaks at last, and the foetus falls into the cavity of the abdomen; which occasions the death of the mother by breaking open its prison.'

During the 18th and much of the 19th centuries, there was a great deal written about the possible underlying causes of ectopic pregnancy. Classifications were elaborated that usually separated continuing abdominal pregnancy from ovarian, tubal and interstitial sites of ectopics. There were a few reported cases of advanced abdominal pregnancies being successfully removed by abdominal incision, including two performed by Virginia surgeon William Baynham in 1791 and 1799; in both cases the fetus had died a considerable time before. However, everything known about major intra-abdominal haemorrhage from ruptured ectopic pregnancy came from postmortem specimens, since there was no known effective treatment for this condition.

In 1876, Philadelphia surgeon John Parry wrote of ruptured ectopic that: 'Here is an accident that may happen to any wife in the most useful period of her existence, which good authorities have said is never cured; and for which, even in this age when science and art boast of such high attainments, no remedy either medical or surgical has been tried with a single success.' Parry made the very radical suggestion that a possible remedy might be to open the abdomen, by the new technique of laparotomy, and then either tie the bleeding vessels or remove the gestational sac entirely, but this idea was considered ludicrous by his colleagues.

Five years later, the same suggestion was made to the eminent English gynaecological surgeon Lawson Tait of Birmingham, by a general practitioner, Mr Hallwright. Tait later wrote that '…in the summer of 1881 I was asked by Mr Hallwright to see a patient… in a condition of serious illness diagnosed by him as probably haemorrhage into the peritoneal cavity from a ruptured tubal pregnancy….I agreed with Mr Hallwright as to the nature of the lesion. This gentleman made the bold suggestion that I should open the abdomen and remove the ruptured tube. The suggestion staggered me and I am ashamed to say I did not receive it favourably….I declined to act on Mr Hallwright's request and a further haemorrhage killed the patient. A postmortem examination revealed the perfect accuracy of the diagnosis. I carefully inspected the specimen which was removed and I found that if I had tied the broad ligament and removed the ruptured tube, I should have completely arrested the haemorrhage and I now believe that had I done this the patient's life would be saved.'

Robert Lawson Tait pioneered surgical treatment for ectopic pregnancy.

Eighteen months later, in 1883, Tait was referred a similar case, and decided to operate at once; this was the first recorded case of such an attempt being made to save a woman dying from a ruptured ectopic. Unfortunately, the woman was already in extremis. Tait wrote: 'We got her to bed alive, and that's all that can be said…I thought very much about this case for it was a bitter disappointment.' He decided that in any future such case he would try immediately to find the source of the bleeding and arrest it, and only then remove intra-peritoneal blood and clots.

A suitable further case was referred to him later in 1883, by a Birmingham GP: a woman with a three month history of amenorrhoea, in great pain and with marked tachycardia. Tait wrote: 'I advised abdominal section and found the abdomen full of clot. The right fallopian tube was ruptured and from it a placenta was protruding. I tied the tube and removed it, searched for, but could not find, the foetus and I suppose it got lost among the folds of intestine and there was absorbed…The patient made a protracted convalescence but she is now perfectly well.'

Five years later, in 1888, Tait was able to report his results for 42 cases of laparotomy for ruptured ectopic pregnancy, with only two deaths, including that of his first case. In several of these cases the pregnancy had clearly been discharged from the tube into the peritoneal cavity after some weeks gestation, but had continued to grow until symptoms and signs demanded intervention. In another paper in 1888, Tait established clearly from a postoperative specimen that this course of events, hitherto only suspected, could indeed occur.

News of Tait's successes spread rapidly and his principles, of first arresting the haemorrhage then performing meticulous peritoneal lavage, were widely adopted in Europe and America. In his major 1890 textbook, A Treatise of Gynaecology, Samuel Pozzi, dubbed the 'father' of French gynaecology, acknowledged Tait's 'remarkable success', adding that 'there is no longer any question that when a haemorrhage (of this kind) threatens a woman's life it is essential to seek at once for the source of the bleeding. To procrastinate…in the great majority of cases, leads to the woman's death.' Furthermore, said Pozzi, Tait's methods were by 1890 being widely followed 'in America and Germany' with excellent results. Pozzi devoted more than 30 pages of his textbook to the diagnosis and treatment of ruptured ectopic pregnancy; by 1890, advances in anaesthesia and widespread understanding of the principles of antisepsis meant that if the diagnosis was made early enough, in most cases the prognosis was favourable. However, the possibility of sudden unexpected collapse without previous symptoms was also widely recognised. From the beginning of the 20th century, the management of ectopic pregnancy, with early laparotomy in suspected cases, was part of every gynaecological textbook.

Lawson Tait remained a formidable pioneer in the area of surgery for women. He helped develop the surgery of ovarian cysts and tumours, in 1886 reporting 137 consecutive cases of successful ovariotomy (ovarian cystectomy) performed without a single death. In 1890, he made the suggestion that placenta praevia might be usefully treated by the relatively recently developed operation of caesarean section, with the possibility of both mother and infant surviving. Like the idea of laparotomy for ectopic pregnancy, this proposal was greeted with ridicule by his colleagues, who believed that pushing aside the placenta once labour had started and performing internal podalic version with vaginal breech delivery (with death of the fetus and often the mother) was the correct management. However, in 1898 Tait successfully performed a Porro caesarean (caesarean followed by subtotal hysterectomy) in a case of central placenta praevia, with both mother and baby surviving. Sadly, Tait died the following year, at the relatively young age of 54, probably depriving gynaecological history of more remarkable performances.

Laparotomy remained the mainstay of treatment – and indeed of diagnosis – of ruptured ectopic pregnancy until the last two decades of the 20th century. Diagnostic ultrasound, quantitative measurement of ß-HCG levels and laparoscopic surgery now mean that in most cases of ectopic laparotomy can be avoided and often medical management using methotrexate is curative in early cases. However, the sudden catastrophic intraperitoneal haemorrhage can still occur and the diagnosis and management of acute ruptured ectopic pregnancy should be familiar to all doctors practising abdominal surgery, especially in remote or less-developed regions of the world. Women continue to die from this condition, even in 2013, because treatment is too little, too late, or not accessible.

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Q&a

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.

Q *A 38-year-old multigravid woman presents to the delivery suite with severe headache at 30 weeks gestation. She is type 2 diabetic and has a history of migraine. What must you not miss when assessing her and how would you manage a case of severe headache in the third trimester of pregnancy?*

Dr Rehena Ahmed
RANZCOG Trainee

a Headache is extremely common during pregnancy. As many as ten per cent of cases of primary headache syndrome initially present, or are first diagnosed, during gestation.[1] Among pregnant women with new or atypical headache: one-third have migraine, one-third have pre-eclampsia/eclampsia-related headache and the remaining one-third have a variety of other causes of headache, such as intracranial haemorrhage, cerebral venous thrombosis and so forth.[2,3] The incidence of subarachnoid haemorrhage (SAH) is 1–5/10000 pregnancies. Maternal mortality is 30–40 per cent; however, rates as high as 80 per cent have been reported.[4] Cerebral venous thrombosis is rare, but occurs more commonly in association with pregnancy.[5]

Even though headache is common in pregnancy, it could be the first manifestation of a life-threatening condition. Hence, very careful and prompt assessment to lead to proper diagnosis is of utmost importance in the management of these patients.

The aim of assessment of severe headache in third trimester is to rule out the uncommon, but potentially sinister, causes while establishing a diagnosis. One should be mindful of the so-called red flags[6]:

- sudden onset of severe headache;
- significant changes in the pattern of chronic headache;
- new-onset migrainous headaches;
- neurologic signs and symptoms;
- changes in patient's level of consciousness, personality or cognition;
- headache precipitated or exacerbated by Valsalva's manoeuvres;
- associated with fever;
- meningeal signs;
- history of recent trauma to head/neck;
- hypertension or endocrine disease; and
- immunosuppression.

When assessing headache, as with any pain, the quality, location, severity, time, course and exacerbating or relieving factors should be fully evaluated. Ask for any associated neurological symptoms, such as numbness, tingling, loss or alteration in sensations or movements. Enquire about systemic disturbances, such as fever, nausea, vomiting, skin rash; and rule out medication overuse/withdrawal, any drug being given for pre-existing headache. Clinical examination should start with blood pressure, a complete neurological examination and a brief general physical examination, with particular attention paid to: throat and sinuses; stiffness of neck; fever; ear, nose and throat; and eye examination, including fundoscopy. The level of consciousness and cognitive ability should be assessed during history taking and examination. Evaluation should always include the clinical assessment of fetal wellbeing while investigating a cause for the mother.

With a history suggestive of migraine headaches, a normal neurological examination and resolution with simple measures, the patient may be followed clinically.[4] The nature and extent of investigation is tailored to the clinical possibilities revealed during detailed history taking and clinical examination. Blood should be taken for complete blood count, renal and liver function tests and coagulation screening; and urine for protein creatinine ratio if clinical assessment points towards pre-eclampsia/eclampsia. An imaging study of the brain is an essential if an intracranial pathology is suspected. At this stage, simultaneous consultation with a neurophysician and a transfer to tertiary hospital either for diagnostic imaging facilities and/or for fetal consideration is essential. A noncontrast head computed tomography (CT) scan is typically the first diagnostic study. However, magnetic resonance imaging (MRI) is safe during pregnancy. In general, MRI is preferable to CT for assessing non-traumatic or non-hemorrhagic

Table 1. Causes of headache relevant in pregnancy from the international classification of headache disorder (ICHD-II).

Primary headache	Migraine, tension headache
Secondary headache	Post head and neck injury
	Vascular disorder (imminent eclampsia, SAH, acute ischaemic stroke)
	Non-vascular intracranial disorders (idiopathic Intracranial hypertension, tumours)
	Drug withdrawal headache (substance abuse: alcohol, caffeine, cocaine, tobacco)
	Disorder of homeostasis (hypoglycemia, hypoxia)
	Disorders of cranial structures (toothache, jaw pain, sinusitis)
	Psychiatric (anxiety, depression, insomnia)
	Neuralgias (trigeminal, Bell's palsy)

craniospinal pathology, such as oedema, vascular disease, mass lesions or local infection. MR venogram is the standard for detecting venous thrombosis. In a review of pregnant patients receiving neuroimaging, the most common imaging studies obtained were MR brain without contrast (87 per cent) and MR angiography head without contrast (73 per cent). The majority of patients (96 per cent) delivered in the third trimester without significant complications.[7] Radiation exposure to the fetus with head CT, cerebral angiography and chest x-ray is approximately 50, 10, and 1mrad, respectively and these levels are considered safe.

A lumbar puncture should be performed following neuroimaging if increased intracranial pressure or infection is suspected. It may become necessary if a small subarachnoidal haemorrhage (SAH) is missed by CT/MRI brain. Further investigations that may be required are echocardiography, carotid doppler studies, peripheral blood smear, HIV screen, antinuclear antibody and laboratory evaluation for inherited or acquired thrombophilia.

Migraine is often unilateral, throbbing or pulsatile quality can be associated with nausea, vomiting, photophobia or phonophobia during attacks. Most women (60–70 per cent) with a history of migraine have improvement over the course of pregnancy, approximately five per cent describe worsening, and the remainder have no change. There is also increasing body of evidence supporting association of migraine, pregnancy induced hypertension and pre-eclampsia. When primary headache such as migraine presents as severe headache, pharmacological treatment generally includes use of simple agents such as acetaminophen, narcotics for severe cases or short course adjuvant glucocorticoids for refractory cases. Nonsteroidal anti-inflammatory drugs should generally be avoided in the third trimester. Although use of triptan is generally avoided, it can be given in severe cases not responding to other treatments. Human experience with triptan exposure during pregnancy has been generally reassuring.[8]

The cause of headache in severe pre-eclampsia/eclampsia is not known, but may be related to increased cerebral perfusion pressure (for example, hypertensive encephalopathy)[9], cerebral ischemia from vasoconstriction, posterior reversible encephalopathy syndrome (PRES), cerebral oedema, or microhaemorrhages. Maternal mortality rates for eclamptic women have been reported to be 0–14 per cent in last few decades, and are higher in poor countries. The most common cause of death in eclamptic women is brain ischemia or haemorrhage.[6] The goals of management of severe pre-eclampsia/eclampsia or HELLP syndrome are to stabilise the mother, prevent recurrent convulsions, treat severe hypertension to reduce or prevent cerebral oedema and haemorrhage, and initiate prompt delivery. Plasma exchange is the treatment of choice for thrombotic thrombocytopenic purpura-hemolytic uremic syndrome (TTP-HUS) occurring during pregnancy.

Stroke in pregnant or postpartum women is rare, however, risk is increased compared with non-pregnant age-matched controls, especially in late pregnancy and early puerperium. Recent evidence suggests that the rate of strokes occurring during pregnancy and postpartum are increasing, substantially for intracranial haemorrhage and cerebral venous sinus thrombosis.[6] The primary cause of intracranial haemorrhage/SAH during gestation is ruptured cerebral aneurysms (most commonly occurs in the third trimester) or arteriovenous malformations.[4,10] The classical presentation of SAH is the sudden onset of sever incapacitating headache, neck rigidity and collapse. Management is by interdisciplinary care with neurosurgeons and indications for neurosurgery are as for non-pregnant patients.

The incidence of pregnancy-associated ischemic stroke is 3–4/100,000/yr.[4] Women with hypertension, diabetes mellitus, tobacco use, hyperlipidemia, sickle cell disease or antiphospholipid antibody syndrome are at risk. Management is by multidisciplinary team care, using aspirin and anticoagulation with heparin. The efficacy and safety of thrombolytic therapy for acute ischemic stroke in pregnant women is unknown.

The incidence of cerebral venous sinus thrombosis is 0.7–24/100,000 deliveries[6] and accounts for three to 57 per cent of pregnancy-related stroke reported by small retrospective studies. Clinical manifestations consist of diffuse, often severe headache, vomiting, focal or generalised seizure, confusion, blurred vision, focal neurologic deficits, and altered consciousness. The mainstay for the treatment is anticoagulation with heparin.

For women who have a stroke at between 28 and 32 weeks gestation, antenatal glucocorticoids can be administered to accelerate fetal lung maturation. A multidisciplinary approach in consultation with neurology, neurosurgery, anesthesia, neonatology and perinatology should be undertaken to stabilise the mother and assess fetal status. As long as maternal and fetal wellbeing are not deteriorating, plans can be made to continue the pregnancy with a scheduled controlled delivery between 34 and 39 weeks gestation to optimise fetal outcome.

Key points

Despite headache being a common symptom in pregnancy, in the case of severe headache in the third trimester potentially catastrophic medical conditions associated with maternal and perinatal mortality and morbidity always need to be excluded. The obstetric care providers need to be vigilant of red-flag signs to ensure prompt diagnosis and safe management of these patients.

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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.

Paediatric labial adhesions

Labial adhesions are a common finding in prepubertal girls, with presentation most commonly associated with difficulty in urination or vaginal irritation. The most common initial treatment for labial adhesions is topical oestrogen, with some cases – those with scarring or fibrosis – eventually progressing to surgical separation. In recent years, topical corticosteroid (0.05 per cent betamethasone) has also been found to be successful in separating the adhesions. In both cases, initial treatment typically ranges from 4–6 weeks. In the longer term, the adhesions generally resolve at puberty as oestrogen levels increase. The authors suggest that there may be concerns with topical oestrogen, with labial pigmentation and premature breast budding being reported.

These two retrospective studies compare the results of oestrogen and betamethasone in prepubertal labial adhesions. The earlier study of 151 girls (Mayoglou et al) reported a success rate of 71 per cent for oestrogen and 78 per cent for betamethasone. Eventually, 27 per cent of the girls treated with oestrogen required surgical separation, with 16 per cent in the steroid group requiring surgery. Unfortunately, 26 per cent of the girls requiring surgery had a recurrence of the adhesions, with 73 per cent of those subsequently treated successfully with topical therapy. Side-effects seen in the oestrogen group were rash (three per cent), breast development (five per cent) and vaginal bleeding (one case). Betamethasone treatment resulted in one case of pubic hair development and one case of erythema and pain. The second study of 131 girls (Eroglu et al) compared topical oestrogen, betamethasone and a combination of the two. They reported that there was no statistically significant differences in the separation rates between the oestrogen only (15 per cent separation), betamethasone only (16 per cent) and a combination of both treatments (28 per cent). It is notable that the success rates in the second study are much lower than the first, possibly due to a younger average age and shorter treatment duration in the second study, but quite possibly an indication of the difficulties of retrospective studies. These two studies both suggest that betamethasone may be a useful alternative to topical oestrogen in the medical treatment of prepubertal labial adhesions.

Mayoglou L, Dulabon DO, Martin-Alguacil N, et al. Success of treatment modalities for labial fusion: A retrospective evaluation of topical and surgical treatments. *J Pediatr Adolesc Gynecol*, 2009, 22:247-250.
Eroglu E, Yip M, Oktar T, et al. How should we treat prepubertal labial adhesions? Retrospective comparison of topical treatments: Estrogen only, betamethasone only, and combination estrogen and betamethasone. *J Pediatr Adolesc Gynecol*, 2011, 24: 389-391.

Joint hypermobility and prolapse

Benign joint hypermobility syndrome (BJHS), also known as Ehlers-Danlos III, is a syndrome of increased joint mobility, resulting from an abnormality in collagen. This case-control study recruited 60 women from an English hypermobility clinic and compared them to 60 non-hypermobile age-, parity- and ethnicity-matched women. Participants completed self-reported prolapse and sexual function questionnaires, and were physically assessed using the Pelvic Organ Prolapse Quantification System (POP-Q).

Women with BJHS reported significantly more prolapse symptoms than the control group, including dragging and heaviness, defecatory problems and backache. POP-Q scores showed a significantly greater degree of prolapse in the BJHS group compared to the control group, with significant differences at a number of levels. The authors suggest that many women with BJHS have an increased risk of pelvic organ prolapse; it is an association that gynaecologists should consider when taking a history from women with prolapse.

Mastoroudes H, Giarenis I, Cardozo L, et al. Prolapse and sexual function in women with benign joint hypermobility syndrome. *BJOG* 2013, 120:187-192.

Maternal posture in occipitoposterior labour

Persistent occipitopostior (OP) position is associated with longer labour, an increased rate of instrumental or caesarean delivery rate and lower Apgar scores. Various methods may be used to reduce the rate of persistent OP presentations, including oxcytocin augmentation, manual rotation and maternal posture.

This French study is a randomised controlled trial of 220 women in labour with a single fetus in a cephalic OP position. Women were randomised to two groups, the control group laboured in a dorsal recumbent position, while the intervention group adopted postures depending on the station of the fetal head. With the station at -5 to -3, women were on hands and knees with their arms supported on a fitball; from -2 to 0, the women were on their side, with the fetal spine down, and from station 0 to delivery they were on their side with their upper leg supported. Any fetal heart abnormality or maternal hypotension would result in a temporary move to a lateral recumbent position.

The authors reported no significant difference in head position at delivery, duration of first or second stage of labour, rate of instrumental or caesarean deliveries, perineal trauma or Apgar score. The authors concluded that no posture should be imposed on women with an OP position in labour as position had no significant effect on maternal or fetal outcomes.

Desbriere R, Blanc J, Le Du R, et al. Is maternal posturing during labor efficient in preventing persistent occiput posterior position? A randomized controlled trial. *Am J Obstet Gynecol.* 2013 Jan;208(1):60.e1-8.

Letter to the editor

Tibolone and libido

I would like to express concerns regarding the content of the article 'Tibolone and libido: it's not a trivial pursuit' *O&G Magazine* Vol 14 No 4 Summer 2012. My concerns pertain to comments that have been made about hormone replacement therapy (HRT) in general, tibolone and testosterone therapy.

That 'HRT may have the effect of improvement in cognitive information processing' is a highly controversial and potentially misleading statement. As summarised by Maki in her recent review, the effects of HRT vary with the mode of administration and formulation of oestrogen used with the use of progestin and type of progestin being also a major determinant of any positive or negative cognitive effect.[1]

Observational data suggest micronised progesterone may be less harmful in terms of breast cancer risk than other progestins[2], although this remains uncertain. In contrast, there is uncertainty as to whether the Mirena intrauterine device is or is not associated with breast cancer risk when used as HRT, with one large observational study indicating that it does carry a risk.[3] Therefore, the information given in paragraph three of the article is not accurate.

The effects of tibolone on mood are not significantly greater than those seen with conventional oestrogen therapy. One RCT comparing tibolone to transdermal oestradiol plus norethisterone in women with female sexual dysfunction (FSD) showed comparable effects of the two therapies. Based on this and other studies, it is completely incorrect to suggest preferred use of tibolone over other regiments for its effects on mood, libido or menopausal symptoms.[4] There is no research that supports the suggestion that tibolone is more effective for mood and libido in postmenopausal women without FSD.

On the second page of the article, the author states: 'Tibolone with HRT and daily oral DHEA provided significant improvement in sexual function…' This statement indicates a lack of understanding of the published literature. Firstly, it suggests tibolone can be combined with HRT, which is incorrect. Secondly, the study that has been referenced for DHEA reported a within group improvement in sexual function with DHEA therapy from baseline, but no greater efficacy when compared with placebo (between group comparison) and the study numbers were small.[5] It was a negative study. Consistent with this, no robust RCT has demonstrated a benefit of DHEA on libido in postmenopausal women.[6]

The table listing contraindications to tibolone is incorrect. Being over 60 years of age is not a contraindication to the prescription of tibolone. Several studies have been conducted in women aged 60 to 79 years. In this age group a 1.25mg dose of tibolone is more effective than raloxifene for prevention of bone loss.[7] In addition, 1.25mg of tibolone daily is associated with a significantly lower rate of breast cancer, a significantly low rate of colon cancer, a significant reduction in fractures, no increased risk of thrombosis and no increase in cardiac events.[8] There was a small but significant increase risk of ischaemic stroke, but the absolute number was so small that in terms of prevention of fracture, tibolone lines up extremely well against other therapeutic options in this population. I commonly recommend women age 60+ take 1.25mg (a half tablet) daily.

No study has ever demonstrated an increase in venous thrombo-embolic events with tibolone. Therefore, a past history of VTE is not an absolute contraindication and, in fact, tibolone is usually the safest option for symptomatic women with this medical history.

With respect to the use of AndroFeme one per cent, it is wrong to recommend 'transdermal testosterone therapy should only be used when testosterone levels are measured and low levels identified'. This has not been an inclusion criterion for any of the large RCTs that have shown significant benefit with testosterone therapy in postmenopausal women for low libido, for example APHRODITE[9], ADORE.[10] Considering how inaccurate testosterone measurements are in women, this statement has the potential to prevent women who would benefit from therapy from being treated.

Measurement of testosterone should not include measurement of 'free testosterone' as the available assays to measure free testosterone lack the precision to enable them to be of clinical use. It is acceptable for a laboratory to calculate free testosterone from a recognised equation such as Sodergard[11] if testosterone has been measured with precision. The free androgen index is not a useful guide for testosterone therapy and women and should really only be applied to assessment of conditions of androgen excess such as polycystic ovarian syndrome.

That 'androgen value should be in the lowest quartile of normal ranges from reproductive-age women' in order for someone to be treated, contradicts the existing evidence and is not an appropriate clinical recommendation.[12] It was a suggestion made a decade ago based a consensus of opinion (including my own)[13] that has been refuted by published data.

Transdermal testosterone cream should not be applied to the arm for two reasons: first, application to the arm commonly interferes with subsequent measurement of testosterone levels for safety monitoring, inappropriately high levels from either a reservoir in the skin of the arm or contamination of the blood draw are common; and, second, the venous drainage from the arm involves breast vasculature and there is no knowledge as to the resultant effects. Transdermal testosterone therapy should only be applied to the lower torso or upper outer thigh (not the inner thigh).

The final conclusion that tibolone should be avoided in women over the age of 60 will deny women what should be considered optimal therapy for this age group – it is probably the safest therapeutic option for women 60+ who continue to experience menopausal symptoms or who have osteopenia or osteoporosis without fracture.

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Monash University

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RANZCOG Research Foundation

Scholarships and Fellowships in 2013

Prof Jonathan Morris
Chair, Grants and Scholarships Committee

As in past years, the RANZCOG Research Foundation offered a number of research scholarships for application in 2012 for research commencing in 2013. The Foundation's selection process is closely modelled on that of the National Health and Medical Research Council, and each year an increasing number of highly competitive applications are received.

The recipients and the research they are conducting in 2013 are set out below.

Ella Macknight Memorial Scholarship, 2013–14

Recipient: Dr Kjiana Elkje Schwab

Project: Gene Profiling Endometrial Stem/Progenitor Cells In Eutopic Endometrium From Women With Endometriosis

Institution: The Ritchie Centre, Monash Institute of Medical Research

Dr Schwab is a Research Fellow and her project will examine the gene profile of endometrial stem cells (epithelial progenitors and mesenchymal stem cells) and their non-stem cell counterparts in women with and without endometriosis to identify gene pathways that confer survival or self-renewal of stem cells shed into the pelvic cavity. It is hoped that this may provide new molecular targets in endometrial stem cells for new medical treatments; leading to changes in the way endometriosis is treated and minimising the need for invasive procedures.

Glyn White Research Fellowship, 2013–14

Recipient: Dr Mary Tolcos

Project: Using Diazoxide To Promote Oligodendrocyte Differentiation And Myelination In The IUGR Brain

Institution: The Ritchie Centre, Monash Institute of Medical Research

Dr Tolcos is a Senior Research Officer at the Ritchie Centre, Melbourne, and was awarded the Glyn White Research Fellowship for her project Using Diazoxide To Promote Oligodendrocyte Differentiation And Myelination In The IUGR Brain. Intrauterine growth restriction (IUGR) is associated with a delay in the development of oligodendrocytes, the myelin producing cells in the brain. Using their established sheep model of IUGR, Dr Tolcos' team will test the ability of diazoxide, a drug currently used in infants with high insulin, to promote oligodendrocyte development and restore myelin within the developing brain.

Luke Proposch Perinatal Research Scholarship, 2013

Recipient: Dr Ratana Lim

Project: Sirtuin 1 (SIRT1) As A Therapeutic Target To Prevent Preterm Birth

Institution: Department of Obstetrics and Gynaecology, University of Melbourne

Dr Lim is a Postdoctoral Research Scientist at the University of Melbourne and has been awarded the Luke Proposch Perinatal Research Scholarship for her project which will look at the role of sirtuins (SIRTs) in human preterm labour. The expression of, and effect of altering, SIRT proteins in human gestational tissues will be investigated, along with whether SIRT activators can delay preterm birth in a mouse model and improve neonatal outcome.

Taylor-Hammond Research Scholarship, 2013

Recipient: Dr Antonia Shand

Project: Maternal Motor Vehicle Driving Capacity After Birth

Institution: Department of Obstetrics, Royal Hospital for Women, Randwick, NSW

Dr Shand, a RANZCOG Fellow and Maternal Fetal Medicine subspecialist, is undertaking a prospective cohort study determining the capacity of women to drive a motor vehicle after giving birth (either vaginally or by caesarean section), either early (2–3 weeks post birth) or late (5–6 weeks post birth). Women's driving capacity will be tested in a driving simulator and recovery after birth will be assessed by questionnaires and actual driving experience.

RANZCOG Fellows' Clinical Research Scholarship, 2013

Recipient: Dr Wan Tinn Teh

Project: Genomic Determinants of Uterine Receptivity

Institution: Obstetrics and Gynaecology, The Royal Women's Hospital; University of Melbourne

Dr Teh is a RANZCOG Trainee and has been awarded the RANZCOG Fellows' Clinical Research Scholarship for her project titled Genomic Determinants of Uterine Receptivity, to be undertaken at the Royal Women's Hospital, Melbourne. The ability of the uterus to accept an embryo is one of the key determinants for a successful pregnancy and the receptivity of the uterus is influenced by the interplay of many genes and environmental factors. Dr Teh plans to study the gene expression of the uterus in women with reduced uterine receptivity, with the aim to identify genes that could be developed into a clinical test to help women suffering from infertility

ASGO National Travelling Fellowship, 2013

Recipient: Dr Vivek Arora

Purpose: To gain further experience in radical debaulking surgery and work within a unit specialising in this surgery and post-operative management of patients

Institution: Department of Gynaecology, Charite Hospital, Berlin (Vircho-Kinkum Campus), Germany

Dr Arora is a RANZCOG Fellow and current gynaecological oncology subspecialty Trainee who will visit the Charite Hospital in Berlin to study surgery treatment for the management of ovarian cancer.

Brown Craig Travelling Fellowship, 2013

Recipient: Dr Poonam Charan

Purpose: To learn about the role of fetal surgery and acquire firsthand knowledge of the latest developments in the field of fetal medicine in Europe

Institution: Department of Fetal Medicine, St George's Hospital NHS Trust, London, UK

Dr Charan is a RANZCOG Fellow and current obstetric and gynaecological ultrasound subspecialty Trainee who is to visit St George's Hospital, UK, to learn more about the role of fetal surgery and acquire firsthand knowledge about the latest developments in the field of fetal medicine in Europe.

Scholarships continuing in 2013

Arthur Wilson Memorial Scholarship, 2012–13

Recipient: Dr Clare Whitehead

Project: Measuring Hypoxic-induced mRNA Transcripts in Maternal Blood to Identify the Hypoxic Growth-Restricted Fetus

Institution: Mercy Hospital for Women, University of Melbourne

Dr Whitehead is a RANZCOG Trainee and was awarded the scholarship for her project, Measuring Hypoxic-induced mRNA Transcripts in Maternal Blood to Identify the Hypoxic Growth Restricted Fetus. Fetal growth restriction is a major cause of stillbirth and current methods to monitor the wellbeing of a growth restricted baby are sub-optimal. Her group proposes developing a new method to monitor the wellbeing of the baby using a molecule in the mother's blood. Over the next two years they will collect blood samples from mother's carrying either growth restricted or well babies to develop this test. If successful, this test may reduce the number of babies lost due to fetal growth restriction.

Fotheringham Research Fellowship, 2012–13

Recipient: Dr Phillip McChesney

Project: A Randomised, Single Blind Controlled Study Assessing the Effect of Endometrial Injury on Live Birth Rate in Women Who are Undergoing an IVF/ICSI Cycle

Institution: Fertility Associates New Zealand

Dr McChesney is a RANZCOG Fellow and current reproductive endocrinology and infertility subspecialty Trainee and was awarded the Fellowship for his project A Randomised, Single Blind Controlled Study Assessing the Effect of Endometrial Injury on Live Birth Rate in Women Who are Undergoing an IVF/ICSI Cycle. The study aims to determine whether a single luteal phase biopsy influences the live birth rate in women under 40 years of age who have failed to conceive a clinical pregnancy despite having undergone at least two embryo transfers of reasonable quality embryos.

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:
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College Statements Update

November 2012

A/Prof Stephen Robson
FRANZCOG
Chair, Women's Health Committee

The Women's Health Committee (WHC) reviewed the following statements in November 2012, which were subsequently endorsed by Council. College statements can be viewed on the College website.

New College statements

The following new statements were endorsed by the RANZCOG Council and Board in November 2012:

- (WPI 18) Fatigue and the Obstetrician Gynaecologist
- (C-Gyn 28) Combined Hormonal Contraceptives
- (C-Gen 21) Driving after Abdominal Surgery including Caesarean Delivery

Revised College statements

The following statements were re-endorsed by the RANZCOG Council and Board in November 2012 with significant amendments:

- (C-Obs 3a) Pre-pregnancy Counselling (REWRITE)
- (C-Gen 14) Guidelines for performing Robotic Surgery
- (C-Gen 15) Evidence-based Medicine, Obstetrics and Gynaecology
- (C-Gyn 25) Managing the Adnexae at the time of Hysterectomy for Benign Gynaecological Disease
- (C-Obs 12) The use of Misoprostol in Obstetrics
- (C-Obs 13) Rotational Forceps
- (C-Obs 16) Instrumental Vaginal Delivery
- (C-Trg 1) Guidelines for Training in Advanced Endoscopic Surgery

The following statements were re-endorsed by the RANZCOG Council and Board in November 2012 with minor or no amendments:

- (C-Gyn 17) Termination of Pregnancy
- (C-Obs 10) Neonatal Male Circumcision
- (C-Obs 19) Maternal Group B Streptococcus in Pregnancy: Screening and Management

New College statements under development

- Management of Obesity in Pregnancy
- Postpartum Bladder Management
- Screening and Management of STIs in Pregnancy
- Substance Use in Pregnancy
- Joint Australian and New Zealand Guidelines for Management of Gestational Trophoblastic Disease

Other news

College statements are no longer be published in full in *O&G Magazine* as the comprehensive list is available online on the College website at: http://www.ranzcog.edu.au/womens-health/statements-a-guidelines/college-statements.html?showall=1

RANZCOG Women's Health Services

Should you have any queries for the Women's Health Committee or the Women's Health Services department, please use the following phone number:
(t) +61 3 9412 2920

College website

College statements

Can be viewed at: http://www.ranzcog.edu.au/womens-health/statements-a-guidelines/college-statements.html . Should you have any difficulties with any documents from the webpage, please phone the College (t) +61 3 9412 2920.

Resources for Fellows

This section includes local and international guidelines and articles of interest such as links to new titles on ACOG Committee Opinions and Practice Bulletins, SOGC Clinical Guidelines, National Institute of Clinical Excellence (NICE) guidelines and Department of Health and Ageing reports. Access at: http://www.ranzcog.edu.au/members-services/fellows/resources-for-fellows.html .

News from the Historical Collections

Gifts (July 2012 to January 2013)

We wish to thank the following Fellows and Friends who have kindly donated items as listed to the Historical Collections during the last seven months:

- Barnett, Mrs Susan (Vic) — Pamphlet
- Bates, Dr John (WA) — Books, original drawings, print
- Beaton, Dr Chris (Vic) — Rubin's test kit
- Black, Dr Jules (Qld) — MRCOG case records
- Cope, Mrs Joan (NSW) — Microscope (belonged to late Dr Ian Cope)
- Crowe, Dr Peter (NSW) — MRCOG case records, FRCOG gown
- Eizenberg, Dr David (NSW) — Portable doppler, Sonicaid, 1975
- Furber, Mrs H M (NSW) — FRACOG gown (belonged to late Dr James Furber)
- Hewson, Dr Alan (NSW) — Culdoscope
- Houston, Mrs M (NSW) — Personal papers, books from library of her father Dr S Robertson
- Hyslop, Dr Ray (NSW) — Personal papers, books
- Keszai, Ms Irma (Hungary) — Book
- Leeton, A/Prof John (Vic) — FRACOG gown, FRCOG gown
- Renou, Dr Peter (Vic) — FRACOG gown
- Roche, Dr James (NSW) — Rare books
- Monash Medical Centre — Fetal monitor; Set 13 anatomical O&G teaching models (from former Prince Henry's Hospital); Series 6 models of birth process (from former Queen Victoria Memorial Hospital)
- Weerasinghe, Dr S (NSW) — MRCOG case records
- Wren, Dr Barry (NSW) — FRACOG gown, books, pamphlets

Donations to the Friends of the College Collection – (July 2012 to January 2013)

We are grateful to the following Fellows and Friends who have generously contributed financial donations to the Friends of the College Collection during the last seven months, to the amount of $2 650.

- Barry, Dr Chris (WA)
- Bishop, Dr Geoffrey (Vic)
- Charters, Dr Deryck (Qld)
- Farrell, Dr Elizabeth (Vic)
- Howell, Dr Euan (Vic)
- Jalland, Dr Mark (Vic)
- Jenkins, Dr Gregory (NSW)
- Kirsop, Mr Wallace (Vic)
- Miller, Dr Eric (Vic)
- Officer, Dr Colin (Vic)
- O'Malley, Dr Terry (NSW)
- Roche, Mrs Mary (NSW)
- Ross, Mr Ian (Vic)
- Svigos, A/Prof John (SA)
- Swift, Dr Gary (Qld)
- Wallace, Dr Gilbert (NSW)
- Weaver, A/Prof Ted
- Zipser, Dr Gabriel (NSW)

Australia Day Honours Awards

Officer (AO) in the General Division

- Dr Colin Douglas Matthews, Walkerville, SA (FRANZCOG)
  For distinguished service to reproductive medicine, particularly through the establishment of donor insemination and in vitro fertilisation programs, through contributions to research and as an academic.

Member (AM) in the General Division

- Prof Jonathan Mark Morris, Longueville, NSW (FRANZCOG)
  For significant service to maternal and infant health as a clinician, educator, patient advocate and researcher.
- Prof Roger Smith, Newcastle, NSW (Honorary Fellow ad eundem)
  For significant service to medical research and development in the Hunter region and in the field of maternal health.

Medal (OAM) in the General Division

- Prof Ajay Rane, Thuringowa, Qld (FRANZCOG)
  For service to medicine in the field of urogynaecology.

Staff news

New appointments

Andreana Newson recently joined RANZCOG as Senior Assessment Coordinator. In this role, she will manage the Assessment Services team, with responsibility for educational assessments of the College. Andreana has a Master of Education degree from Deakin University and a Bachelor of Music Education degree from Melbourne University. She comes to RANZCOG from the independent school sector, where she has been faculty head, year level coordinator and deputy principal. Andreana is a VCE and International Baccaulerate Examiner.

Bridget Anderson started with the College in a temporary capacity at the end of November, assisting in the Finance Department. During this time a permanent position became available and Bridget was successful in the application process and commenced in the position of Graduate Accountant in January. Bridget has more than ten years' accountancy experience and has worked for a range of organisations in Australia and the UK, including Science Teachers' Association of WA and ICRT, the International Secretariat established to facilitate joint testing between the Consumer Associations around the world. Bridget continues to study accountancy and her goal is to complete the Institute of Public Accountants Program.

Angie Spry provides the administrative support for the Rural Obstetric and Anaesthetic Locum Scheme (ROALS), organising locum relief for regional doctors, and promoting the program. She has 11 years' administrative experience, previously working for a not-for-profit member organisation in the mining industry. Angie has a familiarity with rural issues having grown up in regional NSW and worked in rural areas in NSW and Victoria. Before starting at RANZCOG she volunteered at a crèche in the favelas of Rio de Janeiro, Brazil.

Caroline Schmid joined RANZCOG in February as the Women's Health Coordinator. In this role, she looks after the Women's Health Committee and, in particular, the College statements. She completed her Masters degree in Marketing at RMIT University in June 2012. Before joining RANZCOG, she worked as an administrator for a small food start-up business. She has a Bachelor degree in Business Administration and has worked for a number of multinational companies such as Bosch in Germany.

Departures

Shamila Kumar left her role in Women's Health Services in early March to commence a midwifery course. We wish her every success in her studies.

Dominique Ottobre left the College in January to commence a placement in the Victorian Public Service graduate program. We wish her all the best for her new career.

Notice of Deceased Fellows

The College was saddened to learn of the death of the following Fellows:

Dr Russell Linton Millard, of Strathfield, NSW, on 9 Jan 2013
Dr Beryl Overton Howie, of Auckland, NZ, on 1 December 2012
Dr Kenneth David Richardson, of Balmain, NSW, on 27 November 2012
Dr Richard John Reynette Lewis*, of Wagga Wagga, NSW, on 24 November 2012
*An obituary appears on page 76 of this issue of *O&G Magazine*.

RANZCOG Women's Health Award 2012

Julia Serafin
Media and Communications
Senior Coordinator

The Royal Australian and New Zealand College of Obstetricians and Gynaecologists has been proud to present the RANZCOG Women's Health Award for the past eight years, to outstanding university students in O and G from medical schools across Australia, New Zealand, Papua New Guinea and Fiji.

Committed to promoting the specialty of O and G as an exciting and valuable career option, the College is confident this award helps foster awareness of the specialty among medical students.

The RANZCOG Women's Health Award 2012, valued at 500 AUD, was received by the following successful awardees:

- Charlotte Rea, University of Auckland;
- Julia Whitby, School of Clinical Medicine, Australian National University;
- Lucy Pitney, Bond University;
- Guy Reynolds, Faculty of Health Sciences, Flinders University;
- Amanda Ie, Griffith University;
- Shampavi Sriharan, Lauren Prictor, Althea Askern, Laura MacAulay, Aisling Duff, Linda Abenthum and Tamara Turnbull, who shared an award, School of Medicine, James Cook University;
- Alexandra Cussen, School of Medicine, University of Melbourne;
- John Albert Lawson, University of Newcastle;
- Lileane Xu, University of NSW;
- Logan Walker, School of Medicine, Dunedin Medical School, University of Otago;
- Thomas Du and Angela Hemetsberger, who shared an award, University of Papua New Guinea; and
- Troy Wooding, School of Women's and Infants' Health, University of Western Australia.

Obituary

Dr Richard John Reynette Lewis
1921 – 2012

Richard John Reynette Lewis was born on 23 August 1921, in Townsville, QLD. His father, on returning from war service, had taken up the lease of Tattersall's Hotel in Winton, Central QLD. When Richard was nine, and with the Depression at full force, the family, with little income, had to abandon the hotel. The possibility of better prospects drew them to Newcastle, NSW, and then to Hurstville in Sydney. Richard attended Canterbury Boys High, where he completed his Leaving Certificate in 1938. War came and Richard enlisted, joining the 7th Field Ambulance. It was in this unit that he met his later mentor, Lt Colonel Stanley Devenish Meares, in peacetime, an obstetrician on the staff of the Women's Hospital, Crown Street, in Sydney.

Richard began the medical course at Sydney University at the beginning of 1945. Graduating with second class honours, he spent his intern year at the Royal Prince Alfred Hospital. With general practice in mind, he applied for obstetric training at Crown Street with a session at the Children's Hospital to follow. At Crown Street, where he spent two and a half years, he met two influential people. Renewing his association with Stan Meares directed his interest towards obstetrics; meeting Marcia Dodd, a charge sister in one of the hospital's departments, led to a life-long partnership and five sons.

After a year at the Children's Hospital and now feeling equipped for general practice, he began work in Armidale, NSW, purchasing a practice from Dr Bert Child, father of former RANZCOG President, Andrew. The mid-1950s was a time of accelerating childbirth rates in Australia and Armidale provided Richard with much work in this area. By 1961, and with a family of five, he decided that he would travel to the UK to sit the MRCOG. At the end of 1963, he returned to Australia to set up in specialist practice, with a country location in mind. He sought advice and was directed towards Wagga Wagga.

Arriving in Wagga Wagga in early 1964, Richard was immediately confronted with the problem of convincing the local general practitioners that having a specialist obstetrician available was to the benefit of their patients. Not all agreed with this, having been used to handling all of the town's obstetrics up to that time. At first, work and clientele were slow to build up. It was the support of out-of-town doctors that kept up the supply of obstetric cases. Then, quite suddenly, his skill and expertise were recognised by the region's women and they began to beat a path to his door. He became so busy that, by 1966, he took on the first of four partners who would work with him until his retirement.

The importance of Richard Lewis' example is twofold. Firstly, as the first holder of the MRCOG to set up in independent specialist obstetric practice in regional NSW, his pioneering work paved the way for many to follow, not only to Wagga Wagga, but also to all major regional centres. He proved that one did not have to do general practice in addition to a specialty to be able to fit in to the rural medical scene, although his previous experience in that area of medicine would have given him insight into how general practitioners thought and acted. Secondly, his welcoming approach to those he invited to join him and the stable partnership practice that he maintained through the 1970s and 80s served as strong refutation that specialist associations are always fragile and fraught.

He is survived by Marcia, his wife of almost 60 years, his five sons, Richard, Philip, David, Robert and Peter, a brother and a brace of grandchildren. He will be remembered by his patients and colleagues as the consummate gentleman.

Dr Ian Stewart
FRANZCOG
Wagga Wagga, NSW

Asia Pacific Committee

Involved in a developing country?
We'd love to hear from you!

The APC is keen to be kept informed about activities and involvement of our Fellows in all developing countries, but particularly the Asia Pacific region. From this information we will be able to increase valuable networks and build a more comprehensive picture of the involvement of College Fellows in the region, either under the auspices of the College or via other avenues or personal connections you may have.

Please send one paragraph outlining details of any activities/projects/consultations you have been involved in over the past year or details of activities you will be involved in for the coming year to:

Carmel Walker
Coordinator Asia Pacific Services
(e) cwalker@ranzcog.edu.au

Author Index

Volume 14 – 2012
Volume numbers will appear first in index entries, issue numbers in brackets afterwards and page numbers following: , e.g. 10(1): 18 = Vol 10, No 1, page 18
Articles A, In and The are ignored in filing entries

The following abbreviations have been used:
O and G = obstetrics and gynaecology

Series Names and editorials have been placed in square brackets [ ]
Eg. Cervical cancer, postcoital bleeding [Q&a] 11(3): 54
[Q&a] refers to an article from the Q&a series

Subject Index

Volume 14 2012
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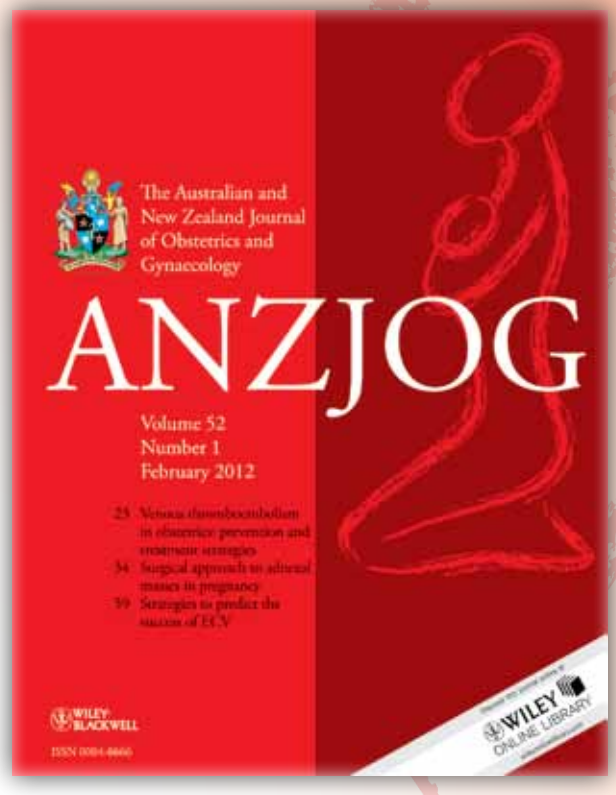

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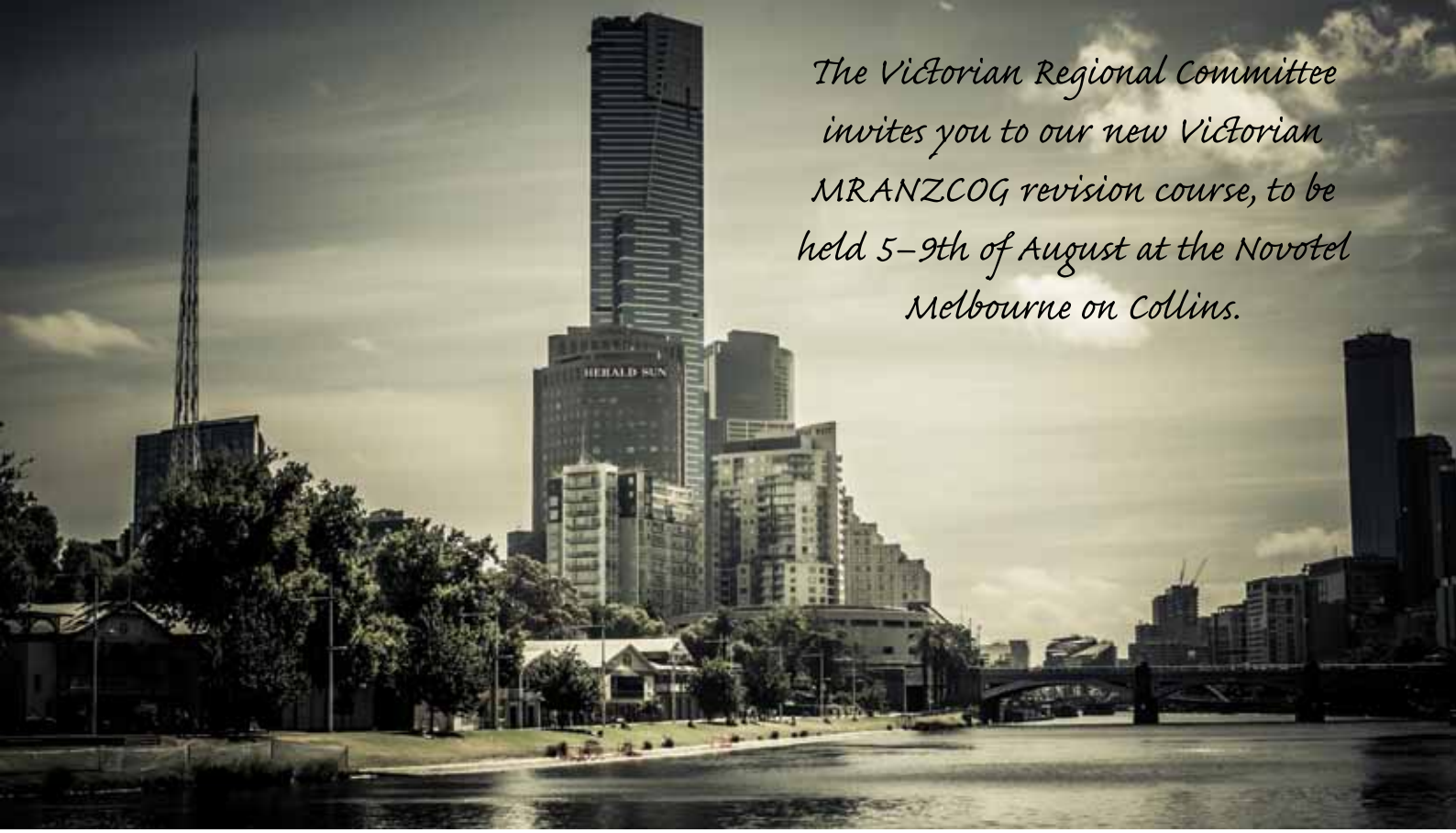

The Victorian Regional Committee invites you to our new Victorian MRANZCOG revision course, to be held 5–9th of August at the Novotel Melbourne on Collins.

Key Highlights:

- Pitched at MRANZCOG level, to maximise use of lecture time.
- Carefully selected speakers who are experts in their fields.
- Inclusion of topics that are difficult to grasp by book study alone and coverage of topics that have featured prominently in previous examinations, including:
  - Primary/secondary amenorrhea
  - Key medical disorders in pregnancy: cardiac/infectious/renal/thyroid
  - New advances in monitoring monochorionic twins
  - Updates on PCOS
  - Updates on the new technologies of prenatal diagnosis, including free fetal DNA to diagnose T21
  - Genetics (relevant to antenatal care)
- Internationally eminent speakers presenting topics on which they are acknowledged as global leaders:
  - Professor Martha Hickey: Menopause
  - Professor Euan Wallace: Preeclampsia and fetal growth restriction
  - Professor Susan Walker: Fetal surgery
  - Dr Lachie Hayes: Haematology
  - Special guest, Dr Michael Rasmussen, will present on exam format and technique.
- Conference dinner (included in the registration) where our special guest will be the RANZCOG President, Prof Michael Permezel

Conveners:

A/Prof Stephen Tong (Education Officer VRC, FRANZCOG, PhD) and
Dr Alison Fung (Chairperson VRC, FRANZCOG, DDU, CMFM)

Costs:

Early Bird Registration (before 30 June)	$1200
General Registration (1 July onwards)	$1300
Day Registration	$300

Further information is available on the website or contact

Mr Mathew Davies, Executive Officer
VIC/TAS Regional Committees, RANZCOG
t: +61 3 9412 2998
e: mdavies@ranzcog.edu.au

Registration numbers are strictly limited.