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Nutrition

The Royal Australian and New Zealand College of Obstetricians and Gynaecologists

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Nutrition

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From the President

Dr Rupert Sherwood
President

Welcome to the Spring issue of *O&G Magazine* themed 'nutrition'. We are all aware of the health implications directly related to over-eating and the resultant high body mass index (BMI). Equally, we all remain stymied with respect to a sustainable and affordable solution. We recognise the value of bariatric surgery in its varied guises, but at the same time acknowledge the limitations of applying this to more than a small number of those needing intervention to reverse the ill-effects of excess body mass.

As clinicians with daily opportunities to counsel women with respect to preventative health, it behoves us to offer help without criticism, utilising the tried and effective methods of increased physical activity in combination with sensible food intake, both in quantity and quality. This opportunistic intervention can be effective and the extra time spent worthwhile.

True prevention is probably only available to the very young, as eating habits, exercise patterns and body mass are largely set before puberty. Again, through sensitive and supportive care of women who are embarking on or already practising parenting, we can make some inroads into this problem.

When this issue goes to print I will have presided over my sixth and final Council meeting – July 2012. The Eighth RANZCOG Board and Council take office in November under the leadership of Prof Michael Permezel. While each President and Council set their own strategic plan and agenda for their two-year term, much of the work and output of our College is a continuum – Trainees are selected, training programs run, assessments set and marked, and Fellowship services such as CPD and various scientific meetings organised, delivered and analysed for future improvements.

The theme of my term of office has been engagement. Recognising that any project upon which we embark requires for its success the input of the membership, I have striven to make Fellows, Diplomates, Trainees and other membership categories take some personal ownership of 'the College' and the ideals to which we aspire. Nevertheless, there remains much to be done in engaging those outside our immediate collegial group, such as the various jurisdictional representatives with whom we interact on a daily basis, but with whom we equally need to have ongoing dialogue at a leadership level to ensure the message of ongoing delivery of the highest standard of reproductive healthcare is maintained across both nations.

On the international stage, RANZCOG is recognised as a leader and we should not ignore the opportunities that this envied reputation affords our organisation with respect to our ability to influence health outcomes in the less-advantaged nations with whom we enjoy strong ties.

This month I received a publication from the Colleges of Medicine of South Africa (CMSA). The CMSA President, Anil Madaree, commented in his editorial on the impact of the new media and the various electronic communication methods on the practice of medicine as we move further into the 21st century. Certainly, this has been a feature of my term as President and I have no doubt that we are yet to reach the right equilibrium in relation to how best to harness the worlds of the internet, Facebook and the ubiquitous Twitter to further the aims of the College. Our new RANZCOG website is a definite improvement, online CPD is almost a reality and e-Learning portals will be widely available to not only Trainees, but also Fellows and Diplomates in the very near future.

> '...I have striven to make Fellows, Diplomates, Trainees and other membership categories take some personal ownership for 'the College' and the ideals to which we aspire..'

Communication between the College and its members has traditionally been via 'snail mail', a time-consuming and relatively expensive modality of writing to all Fellows, Diplomates or Trainees with an important update, information about a meeting or changes in College regulations with potential impact on practice. Although the College is increasingly using electronic means, recent legal advice on our Privacy Policy with regard to universal email messaging to all members has indicated a need for members to 'opt-in' to receive electronic messaging (email or SMS). Accordingly, the membership will shortly receive a request to this effect and I would strongly encourage all members to agree to the use by the College of electronic communication. I can assure the membership that this use will be controlled and used only for matters directly related to our business. The College mailing list is carefully protected from misuse and will continue to be so.

The Personally Controlled Electronic Health Record (PCEHR) is another example of the challenges of conducting the complex business of healthcare through an electronic format. As important as what gets included in an e-Health record is that which is omitted, either by patient request under privacy provisions or by inadvertent omission by various providers. Our College has a working group of Council charged with keeping abreast of this technology and how we should best apply it in our discipline, and the College statement on the PCEHR is available online: www.ranzcog.edu.au/component/docman/doc_download/1045-wpi-22-the-personally-controlled-electronic-health-record-pcehr.html .

An essential part of both our engagement with the international O and G scientific community and raising RANZCOG's international profile is active participation in international meetings. While attendance by Fellows provides good collegial and networking opportunities, the provision of speakers at these meetings is an excellent way of keeping our College at the forefront of the world O and G stage. The next congress of the Asia Oceania Federation of O and G will be held in Thailand in March 2013, and I have received a request for speakers. Any Fellows interested in this opportunity can email the organisers directly at: speakers_aocog2013@kenes.com .

The July Council meeting reviewed and passed an important statement relating to a new category of membership, the 'Semi-retired Fellow'. The Board has responded to requests from members of the College whose work and scope of practice had changed such that they felt the current requirements for CPD and annual subscriptions payable by Fellows needed revision. Some Fellows whose scope of practice had retracted with progress towards full retirement, but who were not ready to formally retire via the existing Retired Fellows Declaration, requested that we consider a new 'semi-retired' category of Fellowship. Eligibility for this category relates to scope of practice, sessions worked per quarter and length of College membership. Details can be viewed on the RANZCOG website: www.ranzcog.edu.au/component/docman/doc_download/1084-semi-retired-ranzcog-fellow-statement-wpi-24-jul12.html?Itemid=341 .

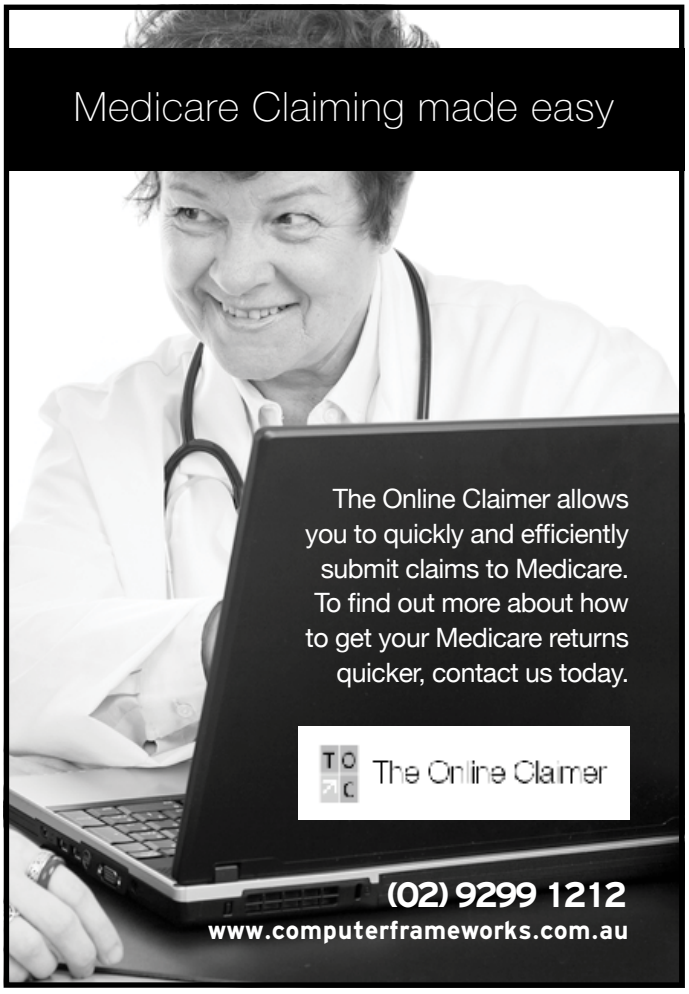
Two telephone calls with Fellows made while writing this report serve to exemplify issues relevant to our specialty. The first was in response to a query with regard to the College role in credentialing Fellows (in other words, defining scope of practice in a particular facility with respect to current qualifications and appropriate current competencies). With a noticeable trend for Fellows who have practised only gynaecology for some, sometimes many, years deciding to supplement their income with locum tenens appointments (often a lifestyle decision with respect to flexibility and ability to balance work and leisure), the question of currency of obstetric competency arises when a locum position entails both parts of the specialty. RANZCOG does not have a direct role in credentialing – this is the function of an appropriately convened credentialing committee, which should form part of the good governance structure of every healthcare service – but does, however, provide guidance on it, and the Board has recently endorsed a statement to this effect (see: www.ranzcog.edu.au/component/docman/doc_download/1054-wpi-23-credentialing-in-obstetrics-and-gynaecology.html and www.ranzcog.edu.au/component/docman/doc_download/987-wpi-06-guidelines-for-the-assistance-of-hospital-committees-in-the-delineation-of-credentials.html). I would strongly encourage all Fellows to actively engage in the credentialing process within their organisation as this is one of the most effective affirmative actions that a Fellow can take to ensure the highest standard of care is provided to women in Australia and New Zealand.

The second telephone call was to a Fellow in an isolated regional centre. This practitioner works in solo specialist practice and spoke to me regarding his concerns over the problems facing rural and regional specialist O and G practice, and the fears he shares with many provincial specialists about the ability to maintain a viable service into the future. Taking the carrot-and-stick approach, I believe only the former will be effective in the long term. Firstly, we must do all we can to encourage new graduates with a clear intent and passion for a regional-based career to apply for our training programs. Secondly, we will continue to mandate that all Trainees spend a minimum of six months in a rural rotation during the first four years of their Fellowship training, with the intent that some will be inspired to return to the country setting for at least part of their O and G consultant career and all will gain, at the very least, an appreciation of the challenges of isolated specialist practice.

In my response, I outlined two new initiatives. The first is to introduce a 12-month Advanced Training Module (ATM) in rural/regional O and G as part of the Advanced Training (post MRANZCOG years 5–6) of the revised training program. This module will concentrate on those 'generalist' skills that are needed in situations where a subspecialist colleague is not available and transfer to a tertiary centre is not necessarily an option. Advanced (open) pelvic surgery, endoscopic skills beyond that required at ITP (core training) level, caesarean hysterectomy competency, continence and prolapse management, ultrasound and other skills that the Provincial Fellows Committee (charged with the design of this unit) deems appropriate will be considered by the Board for inclusion in this module. Secondly, and perhaps of more importantly with respect to potential long-term recruitment into non-urban specialist practice, is the concept of the rural-based core training. Designing the first four years of the FRANZCOG program around a regional base, with terms spent 'away' in the tertiary centres to acquire the requisite subspecialist exposure would attract those graduates with genuine intent to spend at least part of their consultant careers in a rural/remote location. Work on this is to be progressed by the Provincial Fellows Committee and a proposal put to the Board for consideration.

The question 'what does the College give me for my subscription?' is both common and very reasonable, given the annual fee we charge Fellows for ongoing membership of RANZCOG. Actively increasing the services specifically designed to improve the knowledge, audit and practice review capabilities of a Fellow, and hopefully the ability to adapt and evolve their clinical (and, if relevant, administrative) scope of practice to meet the constantly changing environment in which we work, has been earmarked for inclusion in the strategic plan of the next Board and Council. Funding this increase in Fellowship services requires a partial re-balancing of some budgetary areas within the College's areas of activity. Historically, we receive more income from Fellows than we spend, while the converse applies to Trainees, where the outgoings (the cost of administering training and assessment of over 550 trainees across two countries) far exceed the income. The Board has approved a significant increase in the annual training fee to partly redress this imbalance. Recognising that Trainees will, in due course, progress to Fellowship and thus access benefits available through an expanded range of Fellowship services, the Board and Council believe this is the most equitable approach to maintaining the Fellowship as a 'value for money' product. We are also aware that, under current AHPRA guidelines for ongoing specialist registration, the RANZCOG CPD program may well have to compete in an open market in the not-too-distant future.

In closing, I would like to thank the members of the current Board and Councillors of the Seventh RANZCOG Council for all their support and hard work during my term of office as President. As always, I am indebted to the CEO, Dr Peter White, and all the College staff, without whom I would achieve only a fraction of the output we need to maintain our position as the leading voice in standards, education and advocacy in reproductive health across the two nations. I congratulate those elected to the incoming Board and wish Prof Permezel all the best as he takes over the wheel of what former President Dr Ted Weaver was wont to call 'the good ship RANZCOG'.



From the CEO

Dr Peter White
CEO

This issue of *O&G Magazine* is the last to be published during the term of the current RANZCOG Board and Council. This column was written immediately following meetings of the College Board, Council and a wide range of the committees that underpin the work of the College. As part of the meeting of Council, elections were conducted for positions on the RANZCOG Board that will take office from the next Annual General Meeting of the College to be held in November. In addition to the election of the College President for that term, Prof Michael Permezel, which was decided at the meeting of Council held in March, my congratulations to board members: Prof Ajay Rane (Vice-President); A/Prof Stephen Robson (Vice-President); Dr Sarah Tout (Vice-President); Dr Martin Ritossa (Treasurer); Dr Gino Pecoraro; and Dr Vijay Roach. Nominations for election of Councillors are currently open and I thank all those who nominate for election. As all at RANZCOG are aware, the willingness of members to serve is extremely important to the health of the organisation.

Arrangements for the RANZCOG 2012 Annual Scientific Meeting (ASM) in Canberra are now approaching the final stages. Indeed, by the time readers receive this issue of *O&G Magazine*, the ASM will be occurring or have taken place. With a range of eminent speakers, an interesting scientific program and a social program including dinner in the Great Hall at Parliament House, the meeting promises much to registrants. The success of these meetings is due to the work of the Organising and Scientific Committees, as well as College staff and others involved in the organisation. I take this opportunity to thank all involved for their work.

Readers will be aware that the College's bid to host the 12th International Scientific Meeting of the RCOG in Brisbane in 2015, to be held in conjunction with the RANZCOG 2015 ASM, was successful. A preliminary meeting with representatives of the RCOG was held and further details will be provided as they become known. This has the potential to be an outstanding meeting in all respects and the College looks forward to working with the RCOG to showcase the specialty and the destination during the meeting.

In June, the College hosted a meeting of the Asia-Oceania Federation of Obstetrics and Gynaecology (AOFOG) Council in Lautoka, Fiji. This was the first time the AOFOG Council had met in Fiji and an Educational Forum on O and G conducted by RANZCOG and the Fiji National University College of Medicine, Nursing and Health Sciences, provided a valuable opportunity for Councillors to see firsthand the challenges those living and working in the Pacific, and Fiji in particular, face. I am sure that all who attended will follow with interest the implementation of the associated actions and their progress over time. At the same time, a meeting of the Executive of the Pacific Society for Reproductive Health (PSRH) was held and the Fiji Obstetrics and Gynaecology Society (FOGS) launched. The College supports and works actively with PSRH on initiatives aimed at improving maternal health in the Pacific region and looks forward to developing relations with FOGS.

The 2012 Congress of the International Federation of Obstetrics and Gynaecology (FIGO) will take place in Rome in October. RANZCOG is supporting former College President, Dr Kenneth Clark, who is standing for President-Elect of FIGO. Dr Clark's nomination will be put before the General Assembly of the FIGO 2012 Congress in Rome, with candidates from China, India and Malaysia also standing. Elections for society membership of the FIGO Executive Board will also be held at the Congress and, as the College's current term is due to conclude, RANZCOG has again nominated for membership.

Arrangements for the 2013 reaccreditation process conducted by the Australian Medical Council (AMC) for the Medical Board of Australia (MBA) continue. Recommendations previously approved by the RANZCOG Board to ensure the College meets the accreditation standards guided activities at a recent accreditation planning meeting. Chairs of committees worked with College staff to plan work for the coming months; as was evident in the activities progressed during the recent Council week. One such matter is establishing more formal evaluation frameworks for RANZCOG training programs. For instance, in relation to the FRANZCOG Training Program, in addition to the surveys of Year 2 and Year 4 ITP trainees currently undertaken, there is a need to gather feedback in relation to the adequacy of preparation for specialist practice from newly elevated Fellows as well as the perceptions of Training Supervisors of the Trainees they supervise. The challenges will, however, lie in ensuring that the evaluation methodology enables the College to respond to information gathered as it strives to continually improve the way in which it conducts its programs.

Workforce is another area in which there is much activity at present, both within the College and externally; appropriately, it was the theme of the July Council Forum. The President and I recently met individuals from Health Workforce Australia (HWA) to discuss their workforce projections and Dr Ruth Kearon from HWA spoke at the Council Forum on the O and G statistics and demographics. These specialty-specific figures and projections form part of the Health Workforce 2025 Report (Volume 3), which is due to be considered by the HWA Board, AHMAC and Health Ministers in the latter part of this year.

As part of the Workforce Committee's work, a survey of current FRANZCOG Trainees and recent Fellows with regard to workforce intentions was conducted earlier in the year, and the Board is currently considering the completed report ahead of any further promulgation of the results. Dr Will Milford, Chair of the Trainees' Committee, presented some results of the survey during the Council Forum. The Workforce Committee is also looking at the interactions of the College at regional level with health jurisdictions. It is envisaged that in the coming months meetings will be held with stakeholders responsible for workforce matters in individual jurisdictions, and that these meetings will continue at regular intervals to ensure meaningful and adequate dialogue. This is a dynamic and increasingly important area of relevance to the College's activities and it is important that constructive dialogue is maintained.

The Training Review Implementation Working Party (TRIWP) met face-to-face during Council week. While arrangements for the implementation of the revised training program are progressing, the Board has, in consultation with the Chair of the TRIWP, taken the decision to delay implementation of the revised training program until 1 December 2013/January 2014. All involved are cognisant of the need for effective communication in relation to the revised program, as well as the demands that are placed on Training Supervisors, ITP coordinators, Regional Training Accreditation Committee members and others, and the additional 12 months will

allow for workshops to introduce the revised program. The core documents that pertain to the revised program are, however, in the final stages of drafting, including the regulations that will underpin the program and its requirements.

Arrangements for the revised CPD program continue to advance, with a staged implementation from early 2013 planned. This program, using a framework modelled on the Profile of an O and G Specialist contained in the RANZCOG Curriculum, enables Fellows to tailor CPD activities to reflect their practice profile and educational needs as their scope of practice and/or area(s) of interest change over time. Online facilities are also being finalised for use alongside the new program. Overall requirements will remain at 150 hours of CPD across a three-year period, with 25 hours required in the category of Practice Review and Clinical Risk Management (PR&CRM). In order to better reflect activities that meet the requirements, the College's CPD Committee recommended altering the terminology to Practice, Audit and Reflection (PAR). This recommendation was accepted and the new terminology will be incorporated into the revised program.

With the assistance of Marsh Risk Consulting, the College has recently completed a review of the organisational risk profile. The exercise enables the identification of risks relevant to College activities, as well as associated mitigation strategies that are in existence or need to be developed. Relative to the previous iteration (conducted in 2009), the exercise identified an overall increased number of risks associated with activities, but a lower number of risks considered to be 'Extreme' or 'High'. This reflects the increased complexity of the activities being undertaken by the College, the environment in which the activities are being undertaken and also the increased understanding the College has developed in evaluating the organisation's actual risk. Commensurate with this is the degree to which the review is now able to inform aspects of future College activity, including business activity (budgeting and insurance) and staff education.

Ms Ann Robertson, Director of Women's Health, has indicated a desire to scale back her commitments, with a view to retiring at the end of 2012. The College is currently recruiting for a Director of Women's Health and, following an appointment to this position, Annie will continue to manage the Nuchal Translucency program until the end of the year. Annie has made an enormous contribution to the College over a number of years and on behalf of the Board, Council and other Fellows with whom she has worked, I wish to thank her for all that she has done in her time with the College. At the same time as appointing a new Director of Women's Health, the decision has been taken to combine Women's Health and CPD at an organisational level, acknowledging the importance of ensuring that CPD is underpinned by quality clinical information, combined with the acknowledged need to offer high-quality CPD activities to the Fellowship. The College has recently appointed a guideline developer as part of this strategy, as it seeks to ensure that College membership continues to provide value and is relevant to individual members and their practice in the specialty.

Under the College Constitution, the ability exists to introduce different categories of College membership as need arises. The Board has recently approved the introduction from the 2013 membership renewal (effective for the 2013–14 financial year) of a category of membership for 'semi-retired Fellows'. Eligibility criteria are as follows and involve the completion of a declaration attesting that the Fellow meets these criteria:

- is actively practising as a Fellow of RANZCOG and has done so for more than 25 years;
- is a current financial member of RANZCOG;
- will not perform surgery as the primary surgeon;
- may assist another surgeon in the role of assistant surgeon;
- will consult for no more than 13 days per quarter;
- may be involved in teaching and training of medical students, RANZCOG Trainees or other healthcare professionals;
- may be involved in acting as an examiner of medical students, candidates for the AMC or examinations for other health professionals; and
- may be involved in preparing legal reports as long as they comply to RANZCOG guidelines (refer to RANZCOG Expert

Witness Register and WHC statement) and the incidents relate a time period when they were in active practice.

In recognition that the annual completion of 50 hours of CPD is required to maintain general medical registration with the Medical Board of Australia, the Board has maintained the requirement of 150 hours of CPD for Fellows opting to join this category of membership; however, given the nature of the practice involved, has removed the requirement of completion of a specified minimum number of PR&CRM points. The subscription rate for semi-retired Fellows will be set at 50 per cent of that applicable to Fellows in active practice.

The new CLIMATE eLearning platform was made available to all RANZCOG Trainees, Fellows and Diploma candidates on 10 May 2012. User groups were notified by direct email, banner and news item alerts from the College website and the redirection of existing links to eLearning resources. A follow-up advertising and email campaign is planned.

The previous Flexible Learning Program (FLP) modules remained accessible until the second week of June 2012; however, they are no longer available to any users. All eLearning support resources are now accessed via CLIMATE. The 17 Core CLIMATE modules map resources and learning activities directly to the RANZCOG Curriculum via common learning outcomes. These modules are in an ongoing state of development. The following resources are carried by CLIMATE:

- Core CLIMATE Modules;
- CWH/Diploma Support Modules;
- MCQ bank for Membership and Diploma;
- Online Research Project;
- Clinical Educator Training Modules;
- Online Lecture Series;
- 2011 ASM Webcast Presentations;
- 2012 New Zealand Committee ASM Presentations;
- 2011 Provincial Fellows Clinical Teleconference Podcasts; and
- 2012 CREI Symposia Webcast Presentations.

Many existing CLIMATE eLearning resources have been rewritten for access via mobile and tablet devices (iPad and Android). New resources, case scenarios and learning sequences are in development.

In closing, I take this opportunity to assure College members of a committed staff, who are acutely aware of the *pro bono* efforts of members with whom they work to improve the College as an organisation. The reports and recommendations from committees that were considered by the Board at their most recent meetings served again to reinforce the size and diversity of activity that is occurring. For some Councillors, the meeting of Council just completed will have been their final meeting in that capacity. I take this opportunity to thank all such members of Council for their contributions to the College, and, as always, wish all those who are seeking to return to the next Council the best with their election.

Editorial

Dr John Schibeci
DRANZCOG

'Our food should be our medicine and our medicine should be our food.'
Hippocrates

On a rather frigid morning recently, I was walking past our aviary and noticed the finches lined up on the sunward side, trying to get what they could from the miserly offerings. I wondered whether they were there for the warmth or did they have the instinct that they needed vitamin D. A quick 'Google' on the subject revealed that they needed 1000IU/kg/day, 90 per cent from sunlight. Not only do vitamin D-deficient birds get rickets, but they also produce soft-shelled eggs, low clutch sizes and suffer from egg binding and low hatchability. So in our avian friends there is even an 'obstetric' function for vitamin D.

Vitamin D is truly the vitamin of the decade and new functions for it seem to be found all the time. How things have changed. In my 1976 physiology textbook, calcium and bone metabolism are only mentioned. Ten years ago one didn't think of vitamin D in day-to-day practice and now we find a whole new medical industry has sprung from it, hopefully for our betterment. In this issue A/Prof Glyn Teale tries to sort out the fact from the fiction in a wonderfully erudite fashion.

We couldn't let vitamin D run away with all the attention so the other important micronutrients iodine, folate and iron also get guernseys. The discussion of nutrition would be incomplete without mentioning eating disorders, hyperemesis and the management of the vegetarian. For those interested in the menopausal patient, a review of the management of osteoporosis is likewise excellent.

'We are what we eat' and, in many circumstances, rather what we *don't* eat, so hopefully with the help of this issue we will be able to make some sense of the potential for deficiencies particularly in our pregnant patients. As you can see this issue of *O&G Magazine* is more about depletion than repletion, which was covered in our Summer 2008 issue. However, there are also articles on infant nutrition, about which we should be conversant, therapeutic uses for complementary medicines and even chocolate – who would have ever thought? Perhaps there is hope for Australian company Darrell Lea yet!

Once again our authors have done themselves and us proud and we thank them for their considerable time and effort in allowing us to be more aware of the aforementioned sentiment of the legendary Hippocrates.

Vitamin D: miracle panacea or quackery?

A/Prof Glyn Teale
BSc, MRCP, MRCOG, MD, FRANZCOG

'There are known knowns; these are things we know that we know. There are known unknowns; that is to say there are things that we now know we don't know. But there are also unknown unknowns – there are things we do not know, we don't know.'
Donald Rumsfeld, 2002

Just over a hundred years ago, rickets posed a frequent challenge in obstetric practice with reports of approximately three per cent of deliveries in Scotland requiring manoeuvers, including the revolutionary procedure of caesarean section, to deal with a contracted rachitic pelvis.[1] The identification of a steroid compound able to prevent rickets led to the award of the 1928 Nobel Prize for Chemistry to Adolf Windaus. As the fourth 'vital mineral' to be identified, it was labelled 'D'. Its biochemistry is now understood in great detail and the vital roles in calcium homeostasis and prevention of rickets and osteomalacia are well characterised. Encountering a rachitic pelvis is now rare, but a recent report confirms that childhood rickets has not disappeared from Australia, with over 850 cases notified annually.[2]

More recently, non-skeletal actions of vitamin D have been identified and over 900 genes are now recognised to be activated through the ubiquitous vitamin D receptor. It appears that vitamin D is vital to the adequate functioning of the immune system and deficiency is associated with increased risks of multiple sclerosis, rheumatoid arthritis, diabetes and an expanding list of cancers. A recent meta-analysis involving over 50 000 subjects found that all-cause mortality is reduced by seven per cent in vitamin D supplemented groups.[3] Such data have led to a general perception that vitamin D is a 'cure-all'. This thinking has also crept into obstetric practice, following the realisation that deficiency is linked to pregnancy complications such as preeclampsia. It is, however, important to remember the distinction between association and causation and to ensure there are no unexpected consequences on neonatal development before widespread supplementation can be endorsed.

Biochemistry of vitamin D and defining deficiency

Most vitamin D is derived from sunlight-stimulated conversion of provitamin D in the skin. The ability to synthesise vitamin D from sunlight is reduced in those with darker skin and at latitudes moving away from the equator. About ten per cent of vitamin D may be derived from the diet, in particular from certain fish (salmon, herring and mackerel) as well as the result of margarine supplementation. Subsequent liver metabolism results in the production of 25-OH vitamin D (25(OH)D), the index of vitamin D status in serum assays. Thereafter, activation occurs mainly in the kidney, although this process is now recognised to occur in every organ system in the body; with the placenta the most prominent site of extra-renal activation of 25(OH)D in pregnancy.[4]

Over the last decade, the 25(OH)D level that is thought to represent a healthy level of vitamin D has increased. Somewhat confusing terminology abounds, with various definitions for adequacy, insufficiency and deficiency. A recent Australian multidisciplinary position statement defines deficiency in adults as:

- Mild vitamin D deficiency: 30–49nmol/l
- Moderate vitamin D deficiency: 12.5–29nmol/l
- Severe vitamin D deficiency: <12.5nmol/l

It is worth noting the caveat that seasonal variation is best managed by targeting a level 10–20nmol/l higher at the end of summer to compensate for the natural decrease in most areas in winter. The statement also reiterates concerns over the reliability of some assays for 25(OH)D level, which are addressed by ensuring laboratories participate in external quality assurance programs.[5]

A pregnancy and childhood consensus group position statement is currently in preparation and seeks to recommend the same levels (personal communication). While some authorities, including the American Academy of Pediatrics, suggest pregnancy levels over 80nmol/l to be desirable, there is insufficient evidence of safety at the present time to support this target.

Vitamin D deficiency in pregnancy

There is a strong relationship between maternal and fetal/neonatal vitamin D levels such that deficient mothers are more likely to give birth to deficient neonates and extreme deficiency may even cause rickets in utero. Breastmilk is a poor source of vitamin D, with breastfed babies of deficient mothers being considerably more likely to become deficient than formula-fed babies. Therefore, ensuring adequate vitamin D levels in pregnant women is important, particularly among darker skinned and veiled women, who are often severely deficient and also over-represented in the groups whose children develop rickets. Studies from Melbourne, Sydney and New Zealand have identified between 50 and 94 per cent of women from these high-risk groups to be severely vitamin D deficient. Therefore recommendations to routinely screen and supplement these high-risk women and to supplement their children should be followed.[6]

Interestingly, recent reports have shown high levels of deficiency among groups traditionally thought to be at low risk. Thirty five per cent of pregnant women in Northern Victoria and in Canberra and 25 per cent of pregnant women in Campbelltown, New South Wales, have been found to be vitamin D deficient, many of these women being Caucasian.[7,8] Given that obesity is a risk

factor for low vitamin D, it is possible that the rapid rise in obesity levels explains some of the findings. Whatever the explanation, these are prevalence rates of a deficiency state with potentially serious neonatal consequences that are higher than any of the other conditions that are routinely screened for antenatally. Not all studies, however, have reported such widespread deficiency with a recent report from Far North Queensland, on 116 women, finding no women to have a vitamin D level below 50nmol/l.[9] This finding makes a single Australia-wide recommendation on screening for vitamin D deficiency difficult.

Non-skeletal associations with vitamin D deficiency

Heightened interest in the detection and prevention of pregnancy vitamin D deficiency has resulted from the increasing list of associated diseases. Adding weight to the role of vitamin D in immunity has been the discovery that maternal hypovitaminosis D increases neonatal and childhood risk of eczema, asthma and wheeze as well as viral infections, including respiratory syncytial virus and bronchiolitis.

In obstetrics, a 2007 paper identifying an association of low vitamin D with preeclampsia was met with great excitement. Not only did the discovery potentially explain the long-recognised increased risk in African-Americans, whose darker skin is associated with lower vitamin D levels, but there was also a discernable 'dose-response' effect with a quantifiable reduction in preeclampsia risk with increasing 25(OH)D.[10] To add to the tantalising possibility that an easily preventable vitamin deficiency might reduce the risk of a significant cause of perinatal mortality, came data linking vitamin D deficiency with increased risk of preterm labour, gestational diabetes and even caesarean delivery.[4] As obstetricians, had we found the holy grail?

Routine testing and supplementation in pregnancy

Despite the excitement, the potential for significant reduction in pregnancy complications by vitamin D supplementation is, currently, unproven. A 2012 Cochrane review, utilising data from randomised controlled trials involving 1023 women, was not encouraging.[11] While supplementation did increase maternal levels of 25(OH) D and there was no difference in overall incidence of adverse effects between groups, the preeclampsia risk was not significantly reduced (RR 0.67; 95 per cent CI 0.33 to 1.35) and there was only a borderline benefit in terms of reducing low birth weight (RR 0.48; 95 per cent CI 0.23 to 1.01).

So, where to now? It is difficult to dismiss the very strong associations of reduced pregnancy risks with increased levels of vitamin D. However, if there is the possibility of pregnancy benefit, but it is currently unproven, are there disadvantages and dangers to testing and supplementation that warrant a cautious approach?

The economic burden of the sudden rise in 25(OH)D assays in the context of unproven benefit has been questioned. If the aim is the prevention of childhood skeletal complications the potential benefit/risk ratio of routine testing will be influenced by prevalence; given the finding that deficiency is rare in Far North Queensland, routine screening is unlikely to be economically sensible. With much higher rates of deficiency in Victoria, Canberra, New South Wales and New Zealand, there may be more benefit. An alternative approach would be to consider routine supplementation rather than testing. This would avoid the laboratory costs, but would risk supplementing some women with already adequate vitamin D levels. It is possible with oral supplementation to raise serum 25(OH)D to potentially toxic levels and there have been reports of increased rates of eczema and asthma in offspring of women with 25(OH)D levels over 75nmol/l. This finding has recently been refuted, but the potential for a U-shaped benefit and risk curve remains.[12] This warrants a cautious approach. Interestingly, sunlight exposure cannot lead to toxic levels and so skin exposure advice may be appropriate before conception and antenatally.[5]

On the basis of current evidence, screening is best undertaken in the context of risk factors such as dark skin colour, limited sun exposure, raised body mass index and a local assessment of prevalence. Supplementation should be undertaken to target a 25(OH)D level of 50nmol/l in the hope that this will prevent neonatal bone complications. Current recommendations support supplementing with 600–1000IU daily, but the utility of these doses has been questioned. Recent reports suggest that 4000IU/day is safe but, once again, the longer term consequences are unclear and so caution is necessary as the effects are clarified.[13]

Vitamin D and gynaecological cancers

There is potential for vitamin D to offer benefits in gynaecology.[14] The wide range of cancers associated with vitamin D deficiency include breast, ovarian and endometrial. Teasing out the association is complicated by the recognised link between obesity and both hypovitaminosis D and cancer, but some studies suggest strong links even after accounting for body mass index. There is also evidence to suggest that survival after a diagnosis of breast or vulval cancer is higher in vitamin D replete individuals. Recent animal data

have shown that vitamin D dramatically reduces the development of endometrial cancer in obese mice.[15] Human trials are ongoing.

Conclusions

There is the potential that the correction of vitamin D deficiency could improve pregnancy outcomes, reduce gynaecological cancers and reduce a long list of autoimmune diseases. Unfortunately, these attractive possibilities must be counterbalanced by the, as yet, uncertain potential for unexpected adverse effects. There is good evidence that preventing maternal vitamin D deficiency is important for the prevention of neonatal rickets, but the hope that significant pregnancy complications can be prevented is unclear. This is a developing field, but at present a degree of caution is required before we can recommend routine supplementation without question. *Primum non nocere.*

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The north-south divide

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Vitamin D screening and supplementation in pregnancy in Australia; is it safe to say one size fits all?

Vitamin D and its physiological role in pregnancy have been topics of much interest in recent years. Insufficient levels of this vitamin have been implicated in numerous obstetric and perinatal complications. The optimal level of serum vitamin D in pregnancy and the value and appropriate dosage of supplementation are subjects of controversy in current literature. Stemming from evidence gained in recent research, there have been proposals that the introduction of routine screening for vitamin D deficiency and/or routine supplementation should be undertaken nationwide in Australia.

Vitamin D is a fat-soluble steroid hormone that has long been known to play an integral role in bone metabolism and calcium homeostasis. In recent times, however, there has been emerging evidence that vitamin D has important non-skeletal functions, including in reproductive health.[2,4,5,6,9,10]

Deficiency/insufficiency has been associated with an increased risk in adults of malignancy, autoimmune disorders, certain infections and poor glycaemic control.[2,4,5,7,9] Suboptimal levels have also been linked to a variety of adverse obstetric and perinatal outcomes, including gestational diabetes, pre-eclampsia, gestational hypertension, small for gestational age (SGA), caesarean section and preterm labour.[2,4,5,7] As a logical progression from the discovery of these associations, it has been suggested that screening for suboptimal vitamin D levels in pregnancy and supplementation of women found to be deficient/insufficient may improve pregnancy and neonatal outcomes.

Vitamin D exists in the human body in two physiologically active forms, ergocalciferol (vitamin D2) and cholecalciferol (vitamin D3). It can be ingested in its natural form in the diet in foods such as fatty fish (mackerel, salmon, tuna), mushroom and egg yolk or in supplemented foods such as margarine, milk and yoghurt. The more important source of vitamin D for humans, however, is the conversion of 7-dehydrocholecalciferol, a cholesterol derivative, to vitamin D3 in the skin, a reaction produced by ultraviolet (UVB) light.[2,5,7,9] Subsequently, anything that reduces the penetration of UVB into the skin will predispose to suboptimal vitamin D levels. For example, a high density of melanin (such as in dark-skinned people), a more obtuse incident angle of the sun, such as that found at extreme latitudes or in winter, sun protection as promoted in recent decades by sun-safe campaigns and practices of reducing skin exposure for cultural or religious reasons have all been shown to increase the prevalence of vitamin D insufficiency/deficiency.[2,4,7]

Supplementation of vitamin D can be in the form of vitamin D2 or D3. Both vitamin D2 and D3 are hydroxylated in the liver to form 25 hydroxy vitamin D (25(OH)D) and then hydroxylated in the kidney to 1,25 dihydroxyl vitamin D (1,25(OH)2D or calcitriol), which is the active form of the vitamin. Calcitriol (1,25(OH)2D) travels in the bloodstream to target receptors throughout the body where it carries out its various physiological functions, including regulation of up to 900 genes.[9]

Vitamin D has a short half-life (24 hours for vitamin D, two to three weeks for 25(OH)D and four to six hours for 1,25(OH)2D) and thus ongoing intake or in vivo production is necessary to ensure sustained circulating levels.[2,7]

Vitamin D toxicity leads to hypercalcaemia and its attendant consequences. This generally does not occur until levels of 220nmol/l (88ng/ml) are reached.[4]

Vitamin D levels are estimated by measuring 25(OH)D levels in serum. As demonstrated by a 2012 Cochrane review, supplementation of vitamin D during pregnancy leads to increased vitamin D levels at term[2], however, there exist widely disparate opinions about the optimal serum level in pregnancy and appropriate dosage of supplementation during pregnancy. The US Institute of Medicine considers levels of 25(OH)D of 20ng/ml (50nmol) to be the lower limit of normal.[5] They recommend intake of 400–600IU per day to achieve these levels, which they claim can be obtained solely from the diet without supplementation. The Endocrine Society suggests, however, that normal levels in pregnancy are above 30ng/ml (75nmol) and recommend an intake of 1500–2000IU day.[4] A recent randomised controlled trial (RCT) by a group from the Medical University of South Carolina suggests higher levels still are optimal. This group undertook a multi-year RCT which showed that the optimal level of 25(OH)D in pregnancy is 32ng/ml (80nmol) and that this can be achieved with a dosing regimen of 4000IU per day.[5,9]

According to the recent Cochrane review on the subject[2], there has been only one RCT analysing the link between Vitamin D deficiency and pre-eclampsia, which did not support this association. Three trials analysed in the review demonstrated that women who received supplementation had a reduced risk of having a low birth weight baby compared to those who received placebo or no treatment; however, the statistical significance was low. (It is suspected that vitamin D status may influence placentation and thus fetal growth.) The Cochrane review demonstrated no statistically significant increase in stillbirths or neonatal deaths in women who did not receive vitamin D supplementation compared to those who did. There have been no randomised control studies on preterm birth, maternal mortality, admission to NICU or APGAR scores and the review did not look at studies on gestational diabetes.

There has been a recent retrospective cross-sectional study, however, demonstrating that vitamin D levels were inversely proportional to blood glucose levels and HbA1c.[6] There have also been observational studies demonstrating an association between vitamin D deficiency and preterm labour and caesarean section.

Overall, it appears that while there is biological plausibility to the hypothesis that vitamin D deficiency in pregnancy can increase the risk of a variety of pregnancy and neonatal complications – and there is lower level evidence in the form of observational, cross-sectional and case-control studies – this has yet to be proven with high-level evidence. This paucity of good-quality, high-level evidence highlights a need for further RCTs in this field.

The prevalence of vitamin D deficiency in pregnancy obviously depends on the definition used. According to the IOM definition of <20ng/ml (<50nmol/l), 100 per cent of Somali immigrants living in Sweden, 89 per cent of urban Japanese women, 54.7 per cent of UK women in the first trimester and 46 per cent of labouring women in Pakistan are vitamin D deficient.[2] Using a definition of deficiency <37.5nmol/l, one study found that 29 per cent of black pregnant women and five per cent of white pregnant women in the US were deficient.[2]

Three recent Australian studies done in Victoria and NSW demonstrated high levels of vitamin D deficiency in the populations tested. In a study of 147 women attending the GDM Clinic in Westmead hospital, 41 per cent of 147 women had vitamin D insufficiency or deficiency (as defined by levels <25nmol/l and 25–50nmol/l, respectively).[6] In a 2010 cross-sectional study, it was found that the rate of suboptimal vitamin D levels in pregnant women was 35 per cent in Canberra and 25.7 per cent in Campbelltown, Sydney. Definitions for deficiency and insufficiency were <25nmol/l and 26–50nmol/l, respectively. In this study it was found that skin exposure (attire), ethnicity, season and body mass index were determinants of vitamin D levels.[7] In a third study, done in rural Victoria in 2008–09, 65.5 per cent of subjects had suboptimal levels of vitamin D (<75nmol/l). This study also found that ethnicity and season affected rates of deficiency.[9] The authors of all three studies suggest that these rates of vitamin D inadequacy warrant routine screening for vitamin D levels antenatally and/or routine supplementation.

The proposal that universal screening is warranted throughout Australia is questioned by the findings of a small 2011 study from Northern Queensland. The results of this study were that none of the subjects in a study of 116 women booking into the Cairns Base Hospital Antenatal Clinic were vitamin D insufficient, using a definition of insufficiency of <50nmol/l.[1] If a threshold of 75nmol/l was used to define insufficiency, 6.9 per cent had insufficient levels. The results of this study suggest that nationwide universal screening may not be indicated. While the incidence of vitamin D deficiency in southern states has been shown to be significant, the incidence in more northerly populations is likely to be much lower. Therefore, it may be more prudent in these areas to use targeted screening in groups known to be at risk such as women with skin that has a high melanin content, those with gestational diabetes and those who have limited skin exposure due to cultural/religious reasons. Further research is required in other regions of northern Australia before we are able to confidently conclude that universal screening is not needed in each of these areas.

In addition, it is evident from the aforementioned study that routine supplementation of pregnant women, prior to screening for vitamin D levels, as suggested in some previous studies, could potentially lead to toxic levels in pregnant women in the northern regions. Of women in this study, ten per cent had serum vitamin D levels of 151–200nmol/l. Given that the toxic level of vitamin D is over 220nmol/l, routine supplementation could easily lead to hypercalcaemia in these women.

A standard definition of vitamin D deficiency in pregnancy and appropriate dose for supplementation is needed to guide clinical practice. While it does appear that vitamin D deficiency may be associated with adverse pregnancy and neonatal outcomes, this has not been proven by RCTs, with the exception of the outcome of low birth weight. That is not to say that vitamin D deficiency is not an issue of public health significance. On the contrary, the results of trials done to date suggest that pregnancy and neonatal outcomes are likely to be significantly improved if the population is vitamin D replete. Supplementation of women with proven deficiency is unlikely to cause harm and is likely to lead to benefit; however, the introduction of universal screening may not be either cost effective or necessary. An argument may be made that it is more prudent to bring in screening for high-risk populations, such as those in colder climates (the southern states where the prevalence of vitamin D deficiency has been shown to be significantly higher than in northern Australia), with dark skin and little sun exposure for cultural/societal reasons.

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Iodine deficiency

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A public-health issue from the 1950s is becoming increasingly relevant to today's Australia.

Iodine deficiency causing endemic goitre was a well-recognised public-health problem, particularly in the eastern states of Australia and Tasmania, up until the 1950s. The widespread domestic use of iodised salt, supported by iodine supplements given to children in various parts of the country, coupled with the ingestion of abundant quantities of iodine leaking into milk from iodine-containing sanitisers (iodophores) used in the dairy industry, saw the problem disappear. Things changed in the 1990s when the dairy industry phased out iodophores, the food industry ignored the inclusion of iodised salt in food manufacture and consumers, for whatever reason, used less iodised salt in the home.

The developing brain of the fetus and infant requires optimal circulating concentrations of thyroxine for full maturation and that is directly dependent on maternal iodine intake. Until well into the third trimester the fetus receives thyroxine transported across the placenta from the maternal circulation and failure to get enough is the commonest worldwide cause of preventable intellectual impairment. The recommended daily intake (RDI) of iodine is approximately 100μg in the neonate increasing to 150μg in adults and stepping up even further to 250μg for pregnant and breastfeeding women. Extra iodine is needed during pregnancy to ensure that the thyroid gland can increase thyroid hormone production early in the first trimester by up to 50 per cent to meet the demands of pregnancy. Similarly, the breastfeeding mother needs an additional 100μg secreted daily into breastmilk to provide for the needs of the baby.

Australia-wide surveys of schoolchildren conducted in 2004 confirmed that Australian children are mildly iodine deficient. Several surveys in New South Wales, ACT, Victoria, Tasmania and South Australia have confirmed that iodine deficiency is widespread in pregnant women and that the average intake is approximately half the pregnancy RDI. These data have convinced State and Commonwealth Health Ministers to mandate the use of iodised salt in all bread baked in Australia, but this initiative is insufficient to correct the deficiency in pregnant women, leading NHMRC to recommend that all pregnant women take an iodine supplement of 150μg each day, with the proviso that women with pre-existing thyroid conditions should seek advice from their medical practitioner prior to taking the supplement.

Iodine supplementation

NHMRC How much iodine do pregnant and breastfeeding women need?

Iodine is an essential nutrient that humans need in very small quantities. A small gland in the neck, known as the thyroid, uses iodine to produce thyroid hormones.[1] These hormones are vital to ensure normal development of the brain and nervous system before birth, in babies and young children.[2,3] For this reason, it is very important that pregnant and breastfeeding women get enough iodine. Humans store iodine in the thyroid.[1] As only small amounts can be stored, any excess is excreted.[4]

The National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health recommend that women who are pregnant have 220μg of iodine per day. Women who are breastfeeding should have 270μg per day.[1] The World Health Organization (WHO) recommends women who are pregnant or breastfeeding take a daily oral iodine supplement so that the total daily intake is 250μg.[5] Pregnant and breastfeeding women need to top up their dietary iodine intake because of the increased requirements during pregnancy and breastfeeding and the likelihood that they won't get enough from their diet and mandatory fortification.

What foods contain iodine?
Most foods in Australia contain only small amounts of iodine, making it difficult for pregnant and breastfeeding women to get enough iodine through food alone. The amount of iodine varies greatly based on factors such as changes in season and processing practices. Fortified bread, dairy and seafood are the main dietary sources of iodine in Australia.

Why do pregnant and breastfeeding women need more iodine than other population groups?
Humans store iodine in the thyroid. In pregnancy, the thyroid is particularly active, producing about 50 per cent more thyroid hormones than usual.[3] To produce enough thyroid hormones to support the mother and fetus, the mother needs to increase her iodine intake.

If iodine intake is inadequate before pregnancy, the mother's stores may run low and be inadequate to support the unborn baby in later stages of pregnancy.[6] The production rate of thyroid hormone returns to normal when breastfeeding. However, NHMRC recommends that breastfeeding women also take an iodine supplement because breast fed infants are completely dependent on milk as a source of iodine and need around 90 to 100μg of iodine per day.[1] Infants use this iodine to build their own reserves of thyroid hormones.[7,8]

Are pregnant and breastfeeding women getting enough iodine?
No. The weighted results from the National Iodine Nutrition Survey suggest the median urinary iodine concentration in Australia is 96μg/l which, according to WHO, suggests that the Australian population is mildly deficient.[9] There are limited studies specific to the iodine status of pregnant women in Australia, but those available prior to fortification suggest it was inadequate.[9]

Why has this occurred?
Reasons suggested for the recurrence of iodine deficiency in Australia include reduced use of iodine-based cleaning products by the dairy industry and reduced household use of iodised salt (caused by a gradual increase in consumption of commercially processed foods containing non-iodised salt).[10]

What happens if pregnant and breastfeeding women do not get enough iodine?
The main health concern of mild iodine deficiency during pregnancy and breastfeeding is its negative effect on the brain and nervous system of unborn children and infants, in particular reduced intelligent quotient (IQ).[11,12,13,14]

When iodine intake falls below recommended levels the thyroid cannot produce enough thyroid hormones and a range of iodine deficiency disorders can occur.[1,15] Iodine deficiency is of particular concern during pregnancy because abnormal function of the mother's thyroid has a negative impact on the nervous system of the unborn baby, and increases the risk of infant mortality.[3] Adverse effects on early brain and nervous system development are generally irreversible and can have serious implications for mental capacity in later life.[16]

I know bread has recently been fortified with iodine. Do pregnant and breastfeeding women need a supplement as well?
Yes. Mandatory fortification is where food manufacturers are required to add a vitamin or mineral to a food. Mandatory iodine fortification has been introduced in Australia and New Zealand as an initiative to improve public health. Bread made in Australia, where salt is used, except bread represented as organic, is now required to contain iodised salt, instead of non-iodised salt. 'Bread' includes not only bread but also rolls and hamburger buns and other bread products.

Through mandatory fortification, most of the Australian population will get enough iodine[17], meaning women of child-bearing age should enter pregnancy with adequate iodine intake. However, the extra iodine available through bread is not enough to meet the additional needs of pregnancy and during breastfeeding.[18] Therefore, NHMRC recommends that women who are pregnant, breastfeeding or considering pregnancy take an iodine supplement of 150μg each day. Supplements of 150μg/d of iodine are safe and effective for pregnant and breastfeeding women.

Recommendations
- The NHMRC recommends that all women who are pregnant, breastfeeding or considering pregnancy, take an iodine supplement of 150μg each day.
- Women with pre-existing thyroid conditions should seek advice from their medical practitioner prior to taking a supplement.

Are iodine supplements safe for all women?
Women with pre-existing thyroid conditions should seek advice from their medical practitioner prior to taking a supplement.

Is it possible that by taking an iodine supplement pregnant and breastfeeding women will have too much iodine?
No. In Australia, where the population is only mildly iodine deficient, taking an iodine supplement at the recommended dose of 150μg/day will not lead to an excessive iodine intake. However, monitoring of intake to ensure levels don't exceed the recommendation is included in the mandatory iodine fortification monitoring framework and will be conducted by the Australian Institute of Health and Welfare.

Are there any benefits of taking supplements with more than 150μg iodine?
No. NHMRC recommends supplementation of 150μg/day to ensure that all women who are pregnant, breastfeeding or considering pregnancy have adequate iodine status. As only small amounts of iodine can be stored, any excess is excreted.

How was the level of supplementation determined?
The level of supplementation was determined using data on average iodine intakes post mandatory bread fortification, based on knowledge that the Australian population is now classified by WHO as mildly iodine deficient.[19,20]

Are there some iodine supplements that women who are pregnant, breastfeeding or planning pregnancy should not take?
Yes. Women should not take kelp (seaweed) supplements or kelp based products because they contain varying levels of iodine[21] and may be contaminated with heavy metals such as mercury. In addition, a survey of 15 prenatal supplements available in Australia found iodine contents of between 150 and 270μg, with one leading brand containing no iodine.[22] Pregnant and breastfeeding women are advised to check that their vitamin and mineral supplements include the recommended amount of iodine and should consider taking supplements designed specifically for pregnancy and breastfeeding.

When should women start and finish taking iodine supplements?
Women should take iodine supplements from the point of planning pregnancy through the full duration of pregnancy and breastfeeding. If pregnancy is not planned, women should start taking an iodine supplement as soon as possible after finding out that they are pregnant.

How was this statement developed?
NHMRC developed this Statement via a review of the literature and consultation with an expert group. The review, Iodine supplementation during pregnancy and lactation, is available at www.nhmrc.gov.au . The evidence supporting this Statement will be reviewed in time and the Statement revised as necessary. Members of the expert group were Professor Katrine Baghurst, Professor Colin Binns, Professor Peter Davies, Professor Creswell Eastman and Dr Dorothy Mackerras.

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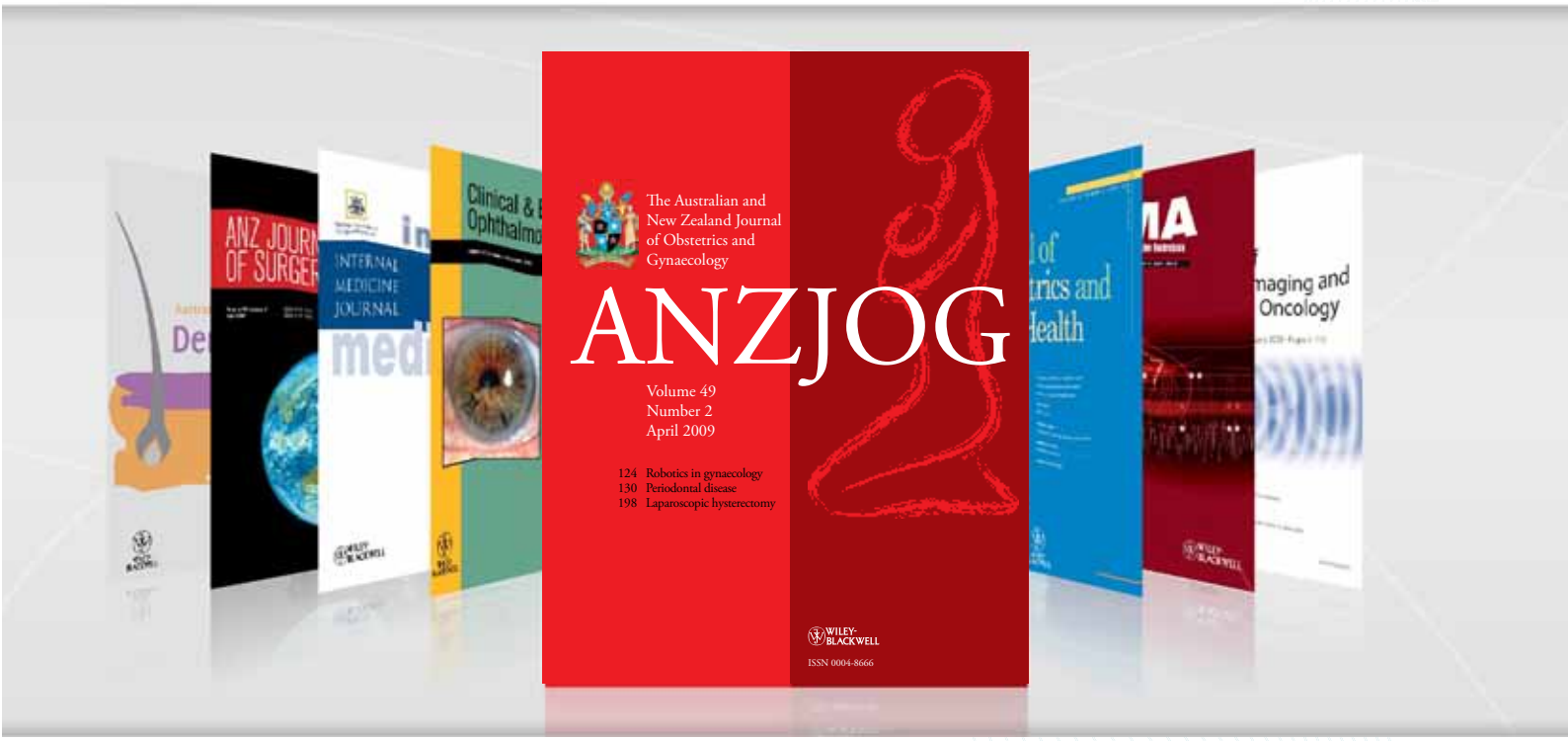

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Osteoporosis

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Osteoporosis is a disease characterised by reduction in bone mass and disruption of skeletal architecture, which ultimately leads to fragility fractures.

Based on the 2007–08 National Health Survey (NHS), it is estimated that 3.4 per cent of the Australian population have osteoporosis as diagnosed by a medical practitioner.[1] However, this is almost certainly an underestimate of the true prevalence, as investigation for osteoporosis generally only occurs after a fracture. Moreover, two-thirds of spinal fractures are silent and thus mostly not detected. Fracture results in significant morbidity, cost and premature mortality that is not just limited to hip fractures. The associated mortality risk increase is greatest in the first five years post-fracture before returning to that of the general population.[2] However, not only are less than 20 per cent of women and fewer men with minimal trauma investigated for underlying causes, but also less than 30 per cent of women and ten per cent of men are on treatment once they have had a fracture.[3]

Risk factors for osteoporosis can be classified as modifiable and non-modifiable (see Table 1). All individuals should be investigated after a minimal trauma fracture, but individuals with risk factors listed in Table 1 should be investigated earlier.

Bone mineral density

The most common method of measuring bone mineral density (BMD) is by dual energy absorptiometry (DXA). There are different DXA manufacturers (GE-Lunar, Norland and Hologic) and comparisons of actual BMD values are not valid between the different systems. However, BMD results are also reported as T-scores (number of standard deviations below that of a young normal individual) and Z-scores (number of standard deviations below an age-, sex- and, in some cases, weight-adjusted individual) and comparisons of these scores can provide an idea of trend.

Table 1. Risk factors for osteoporosis.

Non modifiable	Potentially modifiable
Age	Sex hormone deficiency (oestrogen in females, testosterone in males)
Sex (female)	Smoking
Family history of osteoporosis	Alcohol excess
Previous minimal trauma fracture	Medical conditions (eg malabsorption, coeliac disease, chronic lung disease)
	Low body weight
	Prolonged low dietary calcium intake
	Medications (eg glucocorticoids, anti-epileptics, aromatase inhibitors)
	Endocrine disorders (Cushing's syndrome, hyperprolactinaemia, hyperthyroidism)

The WHO definition of osteoporosis is based on a T-score ≤ -2.5. A T-score between -1 and -2.5 is defined as osteopenia, while scores ≥ -1 are considered normal. Serial BMD measurements can be used for monitoring changes with age or treatment response. Secondary causes of osteoporosis should be investigated for particularly in individuals with low Z-scores. A recommendation of the laboratory tests has been summarised in Table 2.

Management

Exercise

Regular weight-bearing exercise and resistance training improves muscle strength and may help to preserve bone density. Strategies for fall prevention – such as balance training and provision of vision aids – are also beneficial. It should be noted that there is no randomised controlled trial (RCT) evidence for exercise having a direct effect in preventing fractures.

Calcium intake

The recommended daily intake of calcium is 1000–1300mg (three to four serves of dairy products). Ideally, the RDI of calcium should be achieved through dietary means; however, this is often not possible. A calcium supplement may be taken to make up the shortfall. There has been recent controversy surrounding calcium supplement (but not dietary) intake and possible increase rate of heart attacks in some[4-6], but not all studies.[7] Despite the controversy, we would recommend taking a calcium supplement with food if there was inadequate dietary intake but not to exceed the RDI.

Vitamin D

Adequate vitamin D level is essential for calcium absorption. Formation of vitamin D occurs after exposure to ultraviolet light.

Table 2. Recommended laboratory tests.

Investigation	Reason
Serum biochemistry	Exclude hyper/hypocalcaemia, renal, liver dysfunction
Serum 25-hydroxy vitamin D	Exclude vitamin D deficiency
Serum parathyroid hormone	Exclude hyperparathyroidism
Protein electrophoresis	Exclude multiple myeloma
Anti endomyseal anti-tissue transglutaminase antibodies and Ig A level	Exclude coeliac disease
Urinary free cortisol (24 hour)	Exclude Cushing's syndrome if clinical suspicion
Serum prolactin	Exclude prolactinoma if clinical suspicion

The recommended skin exposure is five to 15 minutes of sunlight, depending on time of year and latitude, four to six times a week, longer for people with darker skin. Individuals with limited sunlight exposure – for instance, institutionalisation, long indoor working hours and cultural dress – are more likely to be vitamin D deficient. A serum level >75nmol/l is considered as optimal for skeletal health. A recent meta-analysis showed that vitamin D supplementation of greater than 800IU was associated with a 30 per cent reduction in hip fractures and 14 per cent reduction in non-vertebral fractures.[8]

Levels below 25nmol/l are considered as severely deficient, 25–50nmol/l are deficient and 50–75nmol/l are insufficient. Supplementation should be commenced at 3000–5000IU of cholecalciferol for 12 weeks for the moderate to severely deficient, while 1000 to 2000IU is likely to be adequate for insufficiency. The 25(OH)D level should be rechecked three months after commencement of supplementation.

Pharmacological therapies

Available pharmocological therapies are primarily anti-resorptive with one anabolic therapy (teriparetide) available. Most therapies have been shown to reduce vertebral fractures by up to 50 per cent and peripheral fractures by 15–40 per cent.[9-19]

Table 3. Current PBS indications.

Bisphosphonates	Primary prevention: Aged ≥ 70 years and T score ≤ 2.5 for alendronate, others T score < 3.0 Secondary prevention: Patients with minimal trauma fractures at any T score Corticosteroid induced osteoporosis: T score ≤ 1.5
HRT	Not PBS listed for fracture prevention
Tibolone	Not PBS listed for fracture prevention
Denosumab	Only PBS listed for use in women for the following: Primary prevention: Aged ≥ 70 years and T score ≤ 3.0 Secondary prevention: Patients with minimal trauma fractures at any T score
Strontium ranelate	Only PBS listed for use in women for the following: Primary prevention: T score ≤ 3.0 Secondary prevention: Patients with minimal trauma fractures at any T score
Teriparatide	In patients with established osteoporosis: Three criteria: T score ≤ 3.0 and two minimal trauma fractures and one year of continuous anti-resorptive therapy Can only be initiated by specialists

Bisphosphonates

Alendronate (Fosamax) and risedronate (Actonel) are the two oral bisphosphonates available in Australia.[9-12] Both agents are available as daily and once-weekly doses, with risedronate being available as a once-monthly preparation as well. These agents are also available in various combination packs with Vitamin D and calcium.

Bisphosphonates are poorly absorbed enterally (less than one per cent) and must to be taken after an overnight fast with a glass of water in an upright position, at least 30 minutes before food or other drink. The relatively new enteric-coated delayed-release risedronate (Actonel EC) overcomes some of these restrictions as it can be taken with or immediately after breakfast. However, it must not be taken with other tablets, particularly calcium, and subjects should remain upright for 30 minutes after taking it.

Zoledronic acid (Aclasta or Zometa) is a long-acting intravenous bisphosphonate approved as a once yearly infusion. As well as reducing fracture incidence, it also decreased mortality following hip fractures.[20] Although approved as an annual infusion, its action can last 18 months or more in many people.

A common side-effect of oral bisphosphonates is gastrointestinal irritation and with intravenous bisphosphonates flu-like symptoms are associated with the first infusion. This commonly lasts not more than 48 hours and should settle with paracetamol.

Osteonecrosis of the jaw (ONJ) is a serious, but very rare, side-effect. The estimated incidence in people taking bisphosphonates for osteoporosis is one in 10,000 to 100,000 person-years and has mainly been reported after major dental work such as tooth extraction. Other risk factors include poor dental hygiene, corticosteroids and diabetes. ONJ is more common in cancer patients on much higher bisphosphonate doses.

Atypical femoral stress fractures have been rarely reported since 2005 in long-term bisphosphonate users.[21,22] However, the risk reduction in typical hip fractures far outweighs the risk of atypical fractures.[23]

Raloxifene

Raloxifene (Evista) is a Selective Estrogen Receptor Modulator (SERM) that has similar effects on bone as oestrogen, inhibiting bone resorption. Its use can exacerbate menopausal symptoms such as hot flushes. There is evidence for reduction in vertebral fractures however there is limited evidence for peripheral fracture reduction.[13]

HRT

Hormone replacement therapy (HRT) is an option for women peri-menopausal and early post-menopause. HRT is effective in preserving BMD and reducing vertebral and peripheral fracture risk.

Tibolone

Tibolone (Livial) is a selective tissue oestrogenic activity regulator that is an alternative to HRT for postmenopausal women. Tibolone has been shown to reduce the risk of vertebral and peripheral fractures without the increased breast cancer or clotting risk seen with traditional HRT.[14] However, it was associated with a small risk of stroke in older women.[14]

Densosumab

Denosumab (Prolia) is a fully human monoclonal antibody to the nuclear factor kB ligand. By blocking the binding of RANK to the ligand, it inhibits the development and activity of osteoclasts. It is administered subcutaneously every six months. Denosumab has been shown to reduce vertebral, non-vertebral and hip fractures.[15] The effects of the drug wear off after six months thus must be given regularly or bone loss will ensue. Denosumab can be safely used in people with stage I to IV chronic kidney disease without dose reduction. There was some initial concern with people commencing on denosumab having a higher incidence of skin infections, however, this has been negated on longer follow-up.[16]

Strontium

Strontium ranelate (Protos) is an oral form of strontium taken as a powder mixed with water.[17,18] It needs to be taken two hours after the evening meal for maximal absorption. Side-effects include nausea, diarrhoea, headache and rashes. These should resolve quickly. Thromboembolism is a rare, but serious, side-effect thus consideration should be given in patients with history of thromboembolic disorders.

> ' It is no longer acceptable that only a small proportion of people with osteoporosis and fracture are actively treated.'

Teriparatide

Teripartide (Forteo) is a recombinant form of human parathyroid hormone, given as a subcataneous injection daily. Teriparatide is an anabolic agent that stimulates bone formation.[19] It is approved for a once-only lifetime course of 18 months. Upon completion of the 18-month course, patients should be commenced on an anti-resorptive therapy.

In conclusion, osteoporosis is a common disorder that is associated with significant cost, morbidity and increased mortality risk. Treatment has been shown to decrease fracture risk with minimal side-effects. There are a variety of treatment options available; hence treatment can be tailored to an individual's needs. It is no longer acceptable that only a small proportion of people with osteoporosis and fracture are actively treated.

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Herbal essentials

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An introduction to popular herbs and natural supplements frequently used in women's health.

Herbs and natural supplements are also known as complementary medicines by many people, although the line is blurring between complementary medicine and just good old medicines. For instance, how do we classify vitamin C or fish oil supplements? For this reason, I will sometimes refer to them as herbs and natural supplements in acknowledgement of the fact they are found in nature (not first created in a pharmaceutical laboratory).

Australians have embraced herbs and natural supplements as a consistent part of their approach to healthcare. A national survey of over 1100 pharmacy customers found over 70 per cent use these medicines and the majority describe them as effective or effective enough. Surveys consistently report that women are the main users of complementary medicines, in particular, women with a higher education and socioeconomic background.

Selecting medicines from a shelf in the community pharmacy or supermarket, via the internet or from the garden shows initiative and meets people's personal need for autonomy and control in their healthcare management. In addition, herbs and natural supplements fill a gap in practice when pharmaceutical treatments have limited effectiveness, are associated with undesirable side-effects or potential long-term consequences such as an increased risk of cancer. They are sometimes viewed as a 'softer' option to address mild symptoms or minor health issues and as something to try before bringing out the big guns and their potential safety issues.

So, what is being used and does it work?

Chasteberry, also known by the Latin name *Vitex agnus castus* is a popular herbal extract used in the treatment of premenstrual syndrome. It has been prescribed by herbalists and naturopaths for years, partly based on traditional evidence but also on scientific evidence indicating benefits for a range of PMS-related symptoms. Major nutraceutical companies also manufacture commercial preparations that are available over-the-counter in pharmacies.

Chasteberry has been the subject of numerous clinical trials over the last decade.[1-5] It is taken orally, either as capsules or liquid drops and found to decrease symptoms commonly associated with PMS such as mastalgia, oedema, constipation, irritability, mood alterations and headache in some women.[6] Clinical studies have compared chasteberry to placebo or vitamin B6 for treatment of PMS and compared it to fluoxetine (20–40mg/daily) for premenstrual dysphoric disorder (PDD).

A recent randomised, double-blind, placebo-controlled trial involving 128 women found 40 drops of Vitex taken for six days before menses over six cycles was significantly more effective than placebo ($P<0.0001$) for alleviating mild to moderate symptoms of PMS.[2] In 2010, another randomised, double-blind, placebo-controlled trial of 67 women with PMS found one Vitex tablet daily (40mg) effectively reduced sum symptom scores by at least 60 per cent by the third cycle of treatment. Negative effect, water retention and sum symptom scores were all significantly reduced in the group taking Vitex compared to placebo thereby showing active treatment was effective.[7] Older randomised studies report similar results using either liquid drops or other oral dose forms. Overall, studies indicate that chasteberry extract produces symptom relief rather than reducing the duration of symptoms and benefits tend to occur after three cycles of treatment. When one considers that 40 per cent of women with PMS do not respond to the currently available treatments, Vitex extract presents a novel treatment option worthy of consideration.[8]

Migrainous women with PMS present a challenge in practice and may also benefit from chasteberry extract. Results from a recent study suggest that some migrainous women with PMS also experience a reduction in frequency of migraine attacks and number of migrainous days in the month without associated side-effects.[1] The open label study involved 100 women and now needs replication in a double-blind study to confirm these promising results.

Evening primrose has been a popular treatment for PMS in the past. Back in the 1990s, the entrance of most pharmacies were littered with dump bins filled with large containers of evening primrose capsules that were being sold cheaply as a means of enticing women shoppers to walk through the door. While early reports suggested benefits, the subsequent clinical trials didn't stack up and it can't be confidently recommended as a treatment for PMS.[9] However, there are more promising results from clinical studies in the treatment of mastalgia where it is sometimes recommended in combination with pyridoxine. The main caution here is to make sure that the dose of pyridoxine remains lower than 100mg daily to avoid inducing peripheral neuropathy, a side-effect associated with chronic use.

Ginger (*Zingiber officinale Roscoe*) is one of my favourite herbal medicines because it works quickly, is effective for nausea and enabled me to continue working and lecturing throughout my three pregnancies back in the 1990s. Ginger is available from most supermarkets in the vegetable aisle as the raw root, in the vitamin aisle in tablet form and in the beverage section in the form of herbal tea. A versatile and cheap substance, ginger has been used for thousands of years by most ancient systems of medicine in Europe and Asia and is one of the most commonly consumed condiments in the world. The oily resin from the roots contains many bioactive components that have been the subject of numerous scientific studies. Increasingly, science has sought to verify traditional uses of the herb as a preventative or therapeutic treatment and help us further understand its pharmacological and physiological actions.

In O and G, the most common use of ginger is as a treatment for nausea and morning sickness. Studies have compared various ginger extracts and other medicinal forms to placebo and standard pharmaceutical treatments such as metoclopramide. A review last year concluded that evidence is emerging for ginger as an effective herbal medicine for nausea and vomiting in pregnancy.[10]

A recent randomised, double-blind study of three arms, placebo, ginger extract and metoclopramide demonstrated that ginger

Black cohosh can be used to treat the symptoms of menopause.

treated was superior to placebo ($p < 0.05$) for treatment of nausea and vomiting in pregnancy, however, the metoclopramide was more effective overall.[11] From personal experience, I can definitely recommend it in cases of mild to moderate nausea as it takes the edge off symptoms about 30–45 minutes after ingestion.

The herb black cohosh (*Cimicifuga racemosa*) is one of the better known herbs used in the treatment of menopausal symptoms, in particular hot flushes. It was first used medicinally by the Native Americans many centuries ago as a treatment for female reproductive problems, but also for fatigue, snakebite and arthritis.[6] It was widely adopted by European settlers, eventually becoming a very popular treatment in Europe for various gynaecological conditions, including menopausal symptoms. Most recently, the popularity of diet, lifestyle and herbal treatments such as black cohosh were given a boost after the release of results from the Women's Health Initiative and the Million Women Study.

As with all herbal medicines, multiple active components give rise to a medicine with myriad actions. Whether black cohosh exerts oestrogenic activity remains uncertain, but overall evidence suggests this is unlikely to be a key mode of action. It is generally agreed that the herb reduces luteinising hormone secretion as a result of at least three different active phyto-constituents acting synergistically. Animal studies have revealed an anti-inflammatory action and other tests identified compounds in a black cohosh methanol extract that were capable of strong binding to the 5-HT(1A), 5-HT(1D) and 5-HT[7] receptor subtypes.[6] All this preliminary evidence suggests biological plausibility, so what of the clinical trials?

A 2010 review of systematic reviews, randomised trials and epidemiological studies concluded that black cohosh appears to be effective therapy for relieving menopausal symptoms, primarily hot flushes, in early menopause in contrast to phytoestrogen extracts, including isoflavones and lignans, which appear to have only minimal effect on hot flushes but have other positive health effects, for example on plasma lipid levels and bone loss.[12] Other reviews about the benefits of black cohosh for menopausal symptoms are less positive because not all studies produce consistent results. This begs the obvious question, why do results vary so much?

Two answers were provided in an editorial in *Maturitas* this year by Wuttke.[13] The first is that many different Cimicifuga species exist; three North American and seven Asian species that naturally have different chemical compositions and tests on commercial products in North America indicate significant product-to-product variability in the amounts of specific triterpene glycosides and phenolic constituents. The second answer is even more important and relates to the dosage used in various studies. The two most commonly used German preparations contain between 2.8 and 4mg of Cimicifuga extract from well-characterised, standardised, field-grown *Cimicifuga racemosa* whereas several other studies producing negative results have used far higher doses, up to 160mg, of extract. Keeping in mind the concept of the bell-shaped response curve used in pharmacology, it might be that very low and very high doses are ineffective and medium range doses are most effective.

This example highlights some of the key differences between herbal and pharmaceutical medicines, whereby herbal medicine are chemically complex and can have several mechanisms of action owing to the presence of multiple active components. It also highlights the need to consider chemical variation between preparations and that vigilance must be used when interpreting data.

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An invitation to join PSRH to support reproductive health in the Pacific

and advance notice of

PSRH Biennial Reproductive Health Meeting

The Pacific Society for Reproductive Health (PSRH) is a Charitable Trust, registered in New Zealand in 2007. Our aim is to improve reproductive, neonatal and perinatal in the Pacific by developing the Pacific workforce. Our objectives are:

1. To establish a supportive network of health professionals involved in reproductive health in the region
2. To use this network as a support mechanism for exchange of ideas, knowledge and experiences, and other resources thereby contributing to the improvement of reproductive health services and programs
3. To foster continuing professional development for Pacific Island health professionals in order to enhance quality of care in reproductive health and workplace satisfaction.
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It's not just morning sickness

How should we manage nausea and vomiting of pregnancy?

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Nausea and vomiting of pregnancy (NVP) ranges from the occasional wave of nausea while trying to cook, to severe hyperemesis gravidarum. It affects up to 70 per cent of all pregnancies and persists beyond 20 weeks in 13 per cent. True hyperemesis gravidarum, characterised by severe vomiting, dehydration, significant weight loss and electrolyte disturbance, is relatively rare (0.3–3 per cent).

Women suffering from NVP need a coordinated, evidence-based approach to their illness that recognises the immense toll exacted from being 'seasick' for weeks or months on end. By the time these women seek medical attention, they are often physically exhausted, depressed and psychologically distressed by the ambivalence they may feel towards the pregnancy that is causing their illness. Women with recurrent NVP are particularly susceptible if their previous experience of pregnancy was similar. The recurrence risk for hyperemesis gravidarum has been estimated around 70 per cent.

There are many theories for why women experience NVP (see Table 1). HCG receptors are present in area postrema of the brain stem, an area associated with nausea and vomiting reflexes, which is not protected by the blood-brain barrier and freely accessible to HCG. The tendency to recover by either 12 weeks (60 per cent) or 16 weeks (90 per cent) is consistent with the biology of HCG secretion in pregnancy. The association with molar pregnancy and multiple gestations supports this theory.

Apart from the discomfort and misery of NVP, potential maternal complications include hypokalemia, hyponatremia, metabolic alkalosis, gestational hyperthyroxinemia, Mallory-Weiss tear, reflux oesophagitis and the devastating neurological sequelae of Wernicke's encephalopathy or central pontine myelinolysis.

In women with nausea during pregnancy, the probability of miscarriage is decreased and this is directly linked with the severity of symptoms. There is an increased incidence of preterm delivery and fetal growth restriction limited to women with a poor maternal weight gain during pregnancy suggesting that malnutrition could play a role in these associations.

Investigation

At the first presentation with significant NVP, investigations should include full blood count, urea, electrolytes, creatinine, calcium, liver function tests, thyroid stimulating hormone, urine culture and pelvic ultrasound. This should identify other possible causes of significant nausea and vomiting in pregnancy (see Table 2). The latter is helpful to diagnose molar pregnancy, multiple gestation and to accurately date the pregnancy. During treatment, electrolytes, urinary ketones and liver function tests should continue to be monitored regularly.

NVP and thyroid dysfunction

Biochemical thyrotoxicosis has been reported in up to 60 per cent of women with hyperemesis gravidarum. HCG levels are highest in the first trimester of pregnancy and through cross-reactivity with the Thyroid Stimulating Hormone (TSH) receptor may cause stimulation of the thyroid. The typical finding is a low TSH (<0.5 mIU/l) with or without an elevation of free T4. In most cases, the thyrotoxicosis is asymptomatic but it may cause palpitations, sweating or excessive weight loss. The degree of hyperthyroidism correlates with the severity of vomiting. The course is self-limiting and rarely needs specific therapy.

In most cases, the thyroid function tests normalise by 16 weeks, although differentiation from other causes of thyrotoxicosis may not be possible until after pregnancy. Referral for specialist assessment may be considered if the patient has persistent symptoms of thyrotoxicosis or the free T4 is very high, for example, more than 1.5 times the upper limit of the normal range. Occasional patients may experience resolution of their nausea and vomiting following treatment with antithyroid drugs.

Table 1. Possible mechanisms for NVP.

Gastric dysrhythmia	
Excess free beta Human Chorionic Gonadotrophin (HCG)	
Hormonal sensitivity	T4/T3, estradiol, leptin
Infectious	Helicobacter Pylori
Genetic	Increased risk in daughters, sisters; defects in taste sensation; glycoprotein hormone receptor defects; or latent disorders in fatty acid transport or mitochondrial oxidation suggest a contribution from the maternal but probably not the paternal genotype

Table 2. Possible causes of NVP.

Gastrointestinal	Infection, gastritis, cholecystitis, peptic ulceration, hepatitis, appendicitis, pancreatitis
Urinary tract infection	Cystitis, pyelopnephritis, nephrolothiasis
Vestibular	Labyrinthitis, Ménière's disease
Drugs	Including withdrawal eg marijuana
Endocrine disorders	Hypercalcaemia, Addison's disease, uremia, thyrotoxicosis, porphyria
Neurological	Migraine, raised intracranial pressure

Management
Therapy for NVP should always include sympathetic reassurance and close follow up. A simple algorithm for treatment is described in Figure 1. For mild NVP, dietary modification based on the woman's own appetite and tastes may be all that is required. Avoiding food preparation and food smells with careful food selection, cold foods or even frozen foods can be helpful. Fluids of any kind should be encouraged, even soft drinks, as these can provide much-needed calories. Pregnancy multivitamins are often poorly tolerated and should be suspended while maintaining adequate intake of folate and Vitamins B1, B6 and B12. Hypnosis and acupuncture may help some women, although they have not been effective in randomised trials.

Where to care for the woman with NVP
The development of Pregnancy Day Stay Units and Early Pregnancy Assessment clinics has allowed a more practical model of care for women affected by NVP. Rather than multiple admissions to hospital or long waits in hospital emergency rooms, a management plan should be developed at first presentation or even preconceptually for women who have previously experienced significant NVP. This should include a staged approach to antiemetic therapy that can commence as soon as symptoms develop.

Antiemetic therapy
A range of antiemetics have been used for NVP, all of which are effective when used in appropriate form and dosage. Extensive studies and/or clinical experience have shown that these agents have no apparent teratogenic effects and in fact may reduce fetal malformations through their beneficial effects on maternal nutrition. Similarly, there is no evidence that one antiemetic is superior to another and hence drug selection should be based on those drugs with the longest safety record and fewest side-effects.

Pyridoxine (Vitamin B6) and ginger may be helpful for mild nausea in pregnancy, but have little role once vomiting has commenced. Most antiemetics (with the exception of ondansetron) can cause drowsiness. Extrapyramidal side-effects, including agitation and oculogyric crisis, may result from administration of metoclopramide and prochlorperazine. This complication may be dose-related or idiosyncratic. The major adverse effect of ondansetron is constipation. Laxatives, for example, coloxyl 2-4/day should always be given if ondansetron is commenced. As this drug is not PBS listed for NVP, it is also very expensive, although a grateful patient will happily make this investment. The sublingual wafers can be helpful, but absorption still requires the drug to be swallowed as mucosal absorption does not occur.

The timing of antiemetic therapy is critical and should be based on the pattern of the woman's symptoms, aiming for maximum drug levels when symptoms are worst.

Intravenous fluids and supplements
Women who are severely dehydrated and ketotic need to be carefully assessed and appropriate fluid and electrolyte replacement commenced. Normal saline or Hartmann's solution are appropriate fluid replacement choices. Dextrose-containing fluids are potentially dangerous and may precipitate Wernicke's encephalopathy. Hyponatremia requires the infusion of sodium-containing fluids ensuring that hyponatremia is not corrected too rapidly because this can lead to central pontine myelinolysis.

Fluid and electrolyte balance must be reassessed frequently and management modified accordingly. Intravenous vitamins and antiemetics may be co-administered if oral or rectal dosage is

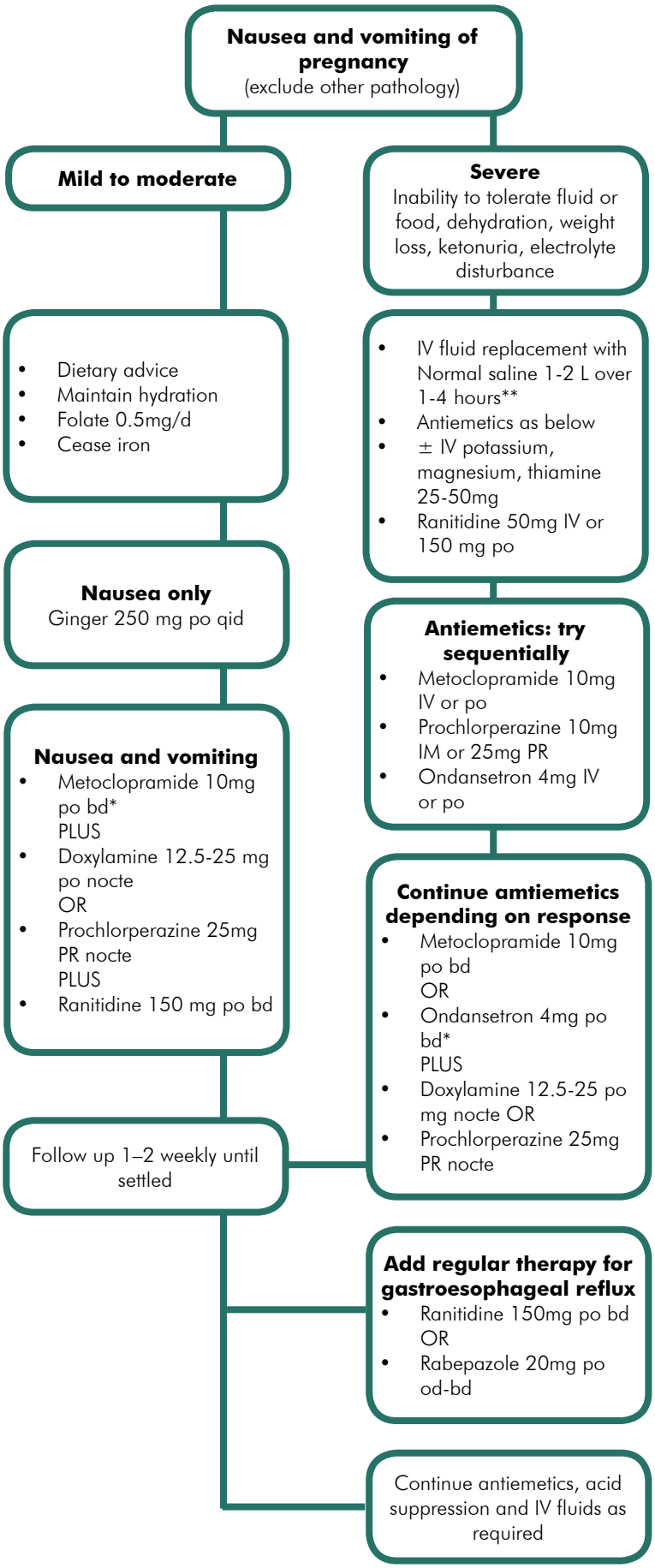

*Figure 1. Suggested management for NVP. *administer first antiemetic of the day 20-30 minutes before getting out of bed, second dose 1-2pm. ** Day assessment service OR emergency room OR inpatient.*

not suitable. Continuous subcutaneous infusions of, for example, ondansteron are used in some countries to avoid hospital admission.

Corticosteroids
The use of steroids should be limited to women with intractable NVP. There is still concern that administration of corticosteroids prior to ten weeks gestation may increase the risk of oral clefts. Although cohort studies using corticosteroids for treatment of NVP have been enthusiastic, RCTs have been less convincing. If steroids are used, usual commencement is with either hydrocortisone 100mg IV bd or Prednisone 50mg daily, reducing to the lowest dose that controls symptoms (usually 5–10mg/day) over 7–10 days. This dose may need to be maintained until the natural resolution of NVP.

Additional measures
Once NVP has been present for more than a few weeks, there is inevitably associated gastro-esophageal reflux and inflammation. These women will benefit from aggressive acid suppression with either an H2 receptor blocker or proton pump inhibitor at high dose. Persistent NVP beyond 16 weeks gestation often reflects undertreatment of this associated problem.

Diazepam and mirtazapine have also been used as adjunctive therapy in some studies. Very occasionally, a woman will fail to respond to these measures. In those cases, admission to hospital for nasojejunal feeding, percutaneous endoscopic gastrostomy feeding, total parenteral nutrition or even termination of pregnancy may be required. Early aggressive treatment can usually prevent these sequelae.

Conclusion
The management of NVP is part of the brief of every clinician caring for the pregnant woman. Early aggressive management, including preconceptual planning, is often more effective than trying to rescue a despondent, dehydrated woman requesting termination. Appropriate treatment expectations need to be defined. In general, the aim is to control symptoms to allow adequate oral intake of fluid and food. Complete resolution of nausea and vomiting cannot always be achieved. An important aspect of therapy includes returning a sense of control to the woman and her family, even if her condition is difficult to manage.

References and further reading are available upon request.

Dr Kenneth Clark
Candidate for President-Elect

International Federation of Gynecology and Obstetrics (FIGO)

I am standing for the position of President-Elect of FIGO, and seek your support and endorsement.

I have been nominated by the Royal Australian and New Zealand College of Obstetricians and Gynaecologists (RANZCOG).

I have extensive experience in College and Society affairs both internationally and within Australia and New Zealand. In addition, I have acknowledged expertise and experience in clinical and organisational governance and in the management of health services. My career to date has been based upon service to my patients and the profession, and on striving to maintain and promote all the tenets of ethical practice.


Dr Kenneth Clark

My vision is to see FIGO maintain and increase its activities, sphere of influence and its reputation – all in the interests of improving women's health across the world, and in building the capacity and capability of Obstetricians and Gynaecologists (and all other health professionals working in women's health) to achieve improved health outcomes.

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Diet during pregnancy

Nutrition guidelines for a growing baby and a glowing mother.

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As dietitians, we always hear stories from patients who are struggling to cut out 'bad foods', with negative connotations for their relationship with food, rather than making positive choices of foods that are delicious, to be enjoyed and shared. When people are encouraged to listen to their hunger and fullness signals and choose to eat what they want, enjoy and need, (guided by common sense) good nutrition often follows.

Pregnancy is no exception. The majority of women's questions are about 'danger foods' and 'foods to avoid'. The list often focuses on mercury-containing fish (with other, healthy, non-affected fish caught up 'in the net') and some, but not all, listeria-at-risk foods (usually some deli meats and soft cheese (see Box 1). While these are important issues – and if advice is not followed, it may have serious consequences – many dietitians are starting to suggest a refocus of priorities may be required.

The rate of listeriosis in Australia is miniscule compared with the high percentage of women not achieving healthy diet and lifestyle recommendations.[1] Over 90 per cent of women do not meet fruit and vegetable guidelines for pregnancy[2] and over 80 per cent consume excess amounts of 'extra' foods that are often high in saturated fats and sodium.[3] Increasing numbers of women are starting pregnancy above a healthy body mass index (BMI) (30–50 per cent), often resulting in excessive gestational weight gain (50–100 per cent), depending on BMI range.[2] These behaviours have serious outcomes during pregnancy and also contribute to lifelong risks for chronic disease.

Accuracy and completeness of pregnancy diet information is required and there are some simple strategies that can be followed to ensure food safety and hygiene to limit adverse reactions to foods. However, as women enter pregnancy with a range of dietary behaviours and beliefs, unfortunately not all of them healthy for themselves or their baby, we can draw on studies that have shown us how to deliver important messages to influence how likely it is that a woman will be able to incorporate this guidance into her lifestyle and food preferences. Encouragingly, women are interested in receiving antenatal nutrition education, information and support.[2]

What determines diet quality?

While the recommended number of serves from the groups outlined in Table 1 has recently been the focus of a large review process (to be finalised in late 2012) the final message will still be the same: meals and snacks should be based on fruit, vegetables/legumes and

Food safety

1. Fish consumption guidelines to reduce mercury consumption (http://www.foodstandards.gov.au/consumerinformation/mercuryinfish.cfm). Food Standards Australia New Zealand has set the following guidelines for safe fish intake in pregnancy.

Pregnant women and women planning pregnancy (1 serve = 150 g)
1 serve per fortnight of shark (flake) or billfish (swordfish/ broadbill and marlin) and NO other fish that fortnight
OR
1 serve per week of Orange Roughy (Deep Sea Perch) or catfish and NO other fish that week
OR
2–3 serves per week of any other fish and seafood not listed above

2. Minimising listeria at-risk foods (http://www.foodstandards.gov.au/consumerinformation/listeria/). It is easy to make safe choices by following these tips:

- Freshly prepared and cooked foods have low levels of bacteria. Bacteria grow over time, so avoid eating food if it has been made more than 24 hours since being prepared. Always reheat to steaming hot.
- Raw fruit and vegetables should be washed thoroughly before eating.
- Listeria is destroyed in normal cooking, so freshly cooked hot food is safe if eaten straight away
- Even at-risk foods risk can be eaten safely if heated above 74 degrees Celsius for over two minutes.

Foods that might carry listeria include:

- raw/uncooked/smoked meat and seafood, ready to eat chilled seafood;
- deli meats, cooked cold meat, pate, meat spreads;
- leftovers (more than 24 hours after cooking);
- pre-prepared salads, smorgasbords, buffets;
- unpasteurised milk and soft-serve ice cream;
- soft cheeses (brie, camembert, ricotta, feta, blue);
- unwashed raw fruit and vegetables; and
- raw eggs or foods containing raw or partially cooked eggs.

Hard cheeses, such as cheddar, are safe. Processed cheese, plain cream cheese and plain cottage cheese are fine if purchased sealed and stored in the fridge.

Table 1. Core food group serves for non-pregnant and pregnant women. (NHMRC, 2003)

Women	Core food groups					
	Cereals (including breads, rice, pasta and noodles)	Vegetables and legumes	Fruit	Milk, yoghurt, cheese	Lean meat, fish, poultry, nuts and legumes	Extra foods
Aged 19–60 years (non-pregnant)	4–9	5	2	2	1	0–2
Pregnant women (all ages)	4–6	5–6	4	2	1–1.5	0–2

Breads/cereals: 1 serve = 2 slices of bread, 1 medium bread roll, 1 cup cooked rice, pasta, noodles; vegetables/ legumes: 1 serve =1/2 cup (75g) cooked vegetables or legumes, 1 cup salad vegetables, 1 small potato; fruit: 1 serve = 1 medium, piece (150g) fruit, 1 cup diced pieces or canned fruit, ½ cup fruit juice; Milk: 1 serve = 1 cup (250ml) fresh milk, 2 slices (40g) cheese, 1 small carton (200g) yoghurt; Lean meat: 1 serve = 65- 100g cooked meat or chicken, 80–120g cooked fillet fish, 2 small eggs, 1/2 cup cooked legumes, 1/3 cup nuts, 1/2 cup sesames seeds; extras: foods that do not fit into the five food groups such as soft drink, crisps and chocolate.

wholegrain breads/grains groups, with moderate amounts of dairy products and meats (or alternatives), and small amounts of healthy fats and oils. A balanced diet that meets the Australian Dietary Guidelines/Guide to Healthy Eating is recommended in pregnancy.

Australian studies show women consume less than half the recommended levels of fruit and vegetables for pregnancy, impacting on a woman's intake of folate, fibre and iodine – essential nutrients in pregnancy.[2,4] Practitioners can start by simply asking a woman how many serves of fruit and vegetables she eats each day (see Table 1 for serve guides). Encourage her to increase her consumption towards her goal with simple, achievable changes. Fruit (fresh, tinned or dried) can be a great snack and frozen fruits are good for dessert. Soon to be released Pregnancy Lifescripts will also provide a useful guide to including these questions in a consult session.[5]

A woman's gestational weight gain (GWG) goal depends on her pre-pregnancy weight. The Institute of Medicine[6] (IOM) 2009 guidelines outlined in Table 2 are based on a systematic review of evidence that evaluated the optimal outcomes for both mother and baby. GWG outside these recommendations is associated with adverse outcomes during pregnancy, delivery and into the future beyond the immediate postpartum period. Women not advised of GWG guidelines are more likely to fall outside the recommended weight-gain range[7] and following the 5As (assess, advise, assist, agree, arrange) framework outlined in a previous issue of *O&G Magazine*[8] has had demonstrated reductions in overall GWG in a tertiary health service.[9]

Starting those 'difficult conversations'

Recent research[10] has highlighted confusion and uncertainty among maternity professionals regarding the most appropriate approach to take when counselling their patients about dietary quality and weight management. Concerns were also articulated about being effective, but not offensive, in the way clinicians convey information. The societal stigma associated with obesity has relevance for all health professionals striving to maintain good patient relationships, while providing evidence-based care.[11] Terminology acceptable to patients may differ from that routinely used by health professionals; terms such as 'weight' and 'BMI' are likely to be received more positively than references to 'fat' and/or (morbidly) 'obese'.[12] When advising women about weight gain goals (informed by pre-pregnancy BMI), avoiding direct reference to BMI category names (such as 'underweight', 'overweight', 'obese' and so forth) and replacing with a more appropriate sentence, such as 'based on your pre-pregnancy weight, you should aim to gain xx-xx kg for the healthiest pregnancy possible' is more likely to (respectfully) promote healthy GWG.

Swap it, don't stop it

Pregnancy is a time where some nutrient needs increase significantly, with only a small increase in energy needs, rendering the quality of the diet imperative. One of the most common traps people can fall in to is non-hungry eating (NHE). NHE is eating when you are not physically hungry for food. Some NHE is normal and many people do it. When we end up doing a lot of NHE it can be hard to achieve a balanced diet.[13] We encourage women to achieve a balance between eating to appetite, meeting their food group requirements

Tips for a more nutritious diet

If women are not gaining weight at the correct rate, recommend nutritious snacks. Good snacks include:

- fruit or grain toast with margarine, jams or nut spreads;
- dried fruit, nuts and seeds;
- yoghurt, custards and fromage frais;
- muesli bars, choose wholegrain with dried fruit and/or nuts;
- hard cheese and crackers; and
- milk drinks (Milo and milk, milkshakes, fruit smoothies).

If women are gaining weight too quickly, they need to make sure they are not 'eating for two'. It is also important to include regular exercise most days. To help manage weight during pregnancy:

- choose fruit – fresh, tinned or frozen instead of higher calorie snacks;
- use minimal fat in cooking;
- choose low-fat milk, yogurt and cheese in place of full-fat products;
- trim all the fat off your meat before cooking and remove the skin from chicken;
- limit high-fat takeaway foods (or opt for salad alternatives);
- drink water and limit fruit juice to one serve a day;
- consider diet or low-joule products in place of sweetened soft drinks or cordial;
- limit intake of desserts and puddings; and
- minimise snacking on 'extras' outside of core food groups.

and tracking their weight gain. See Box 2 for more tips.
Practical tips to help women implement these changes:

- Encourage women to make positive changes to meet their requirements for core foods.
- Pre-planning helps with positive choices – nutritious back-up meals and snacks are cheap and quick substitutes for takeaway. Frozen pre-chopped vegetables, tinned soups with added beans, pasta with bottled sauces, or scrambled eggs with grated cheese and washed salad are quick-and-easy meal choices.
- Start the conversation about dietary variety and recommended gestational weight gain.
- Monitor your patient's weight following the 5As (assess, advise, assist, agree, arrange).[7]
- Encourage food safety (see Box 1), but encourage suitable omega-3-rich choices such as salmon and sardines. Vegetarian omega 3 sources include walnuts, canola oil and margarine, and linseed bread.
- Encourage women to seek expert dietary advice from an Accredited Practising Dietitian (APD). To access an APD in Australia, contact the Nutrition Department of your facility directly or go to: www.daa.asn.au and click on 'Find an APD', or call the toll-free APD hotline on 1800 812 942.

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Supplementation in pregnancy

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With many different commercial supplement products on the market, what should pregnant women be taking and why?

The dietary intake of women of childbearing age in New Zealand and Australia is relatively poor. Dietary surveys from both countries show female nutritional intake characterised by high total and saturated fat, high sodium, low calcium, low fibre, low dietary folate and low iron. This is indicative of not enough wholegrain carbohydrates, lean protein foods, and fruit and vegetables, too many fast foods and unhealthy snack-type foods. Poor dietary habits appear to continue during the preconception and early gestational period; even in women with a planned pregnancy.[1] Improving nutritional intake prior to and during pregnancy should be encouraged by all antenatal health professionals.

Nutritional supplement use is common during pregnancy. There is strong scientific evidence for nutritional supplementation during the preconception and gestational period for folic acid, iodine and iron (if deficient). However, there is little evidence to support supplementation with other micronutrients or herbs. Until further research is conducted, it is safer to recommend dietary improvements instead.

Folic acid

Taking 0.4mg folic acid in the preconception period and the first trimester of pregnancy reduces the risk of neural tube defects (NTD) by 70 per cent. Women are recommended to take 0.8mg (New Zealand) or 0.4mg (Australia) per day for at least one month prior to conception and for the first three months of pregnancy.[2,3] Choosing foods high in folate, such as leafy green vegetables, wholegrain carbohydrates and folate-fortified foods should also be encouraged. Women with a family history of NTD, a previous NTD-affected pregnancy or who take certain medications should be prescribed the larger dose of 5mg folic acid/day. Obese women (body mass index >30) have reduced folate levels and are at higher risk of an NTD-affected pregnancy, therefore these women should also be prescribed 5mg of folic acid.[4,5]

Iron

Iron deficiency and anaemia is common in women of childbearing age; seven per cent of females in Australia and New Zealand are iron deficient, and approximately four per cent have iron-deficiency anaemia.[6,7] Women at risk of iron deficiency include those following a vegetarian or vegan diet, who have heavy menstruation, a low intake of red meat or exercise heavily. Requirements for iron increase in pregnancy; improving iron status during the preconception period is important as it is difficult to raise iron levels during pregnancy. Appropriate dietary changes should be recommended to improve dietary iron intake and absorption. This includes regular consumption of red meat, chicken, fish, seafood, eggs, as well as vegetarian iron sources such as leafy green vegetables, dried beans, peas, lentils and iron-fortified foods. Vitamin C increases iron absorption; women should be encouraged to have colourful vegetables with meals, or a piece of fruit immediately after main meals. Conversely, phytates (found in unprocessed bran), coffee and tea decrease iron absorption and should be avoided for 90 minutes either side of main meals.

Many women will require an iron supplement during pregnancy. Iron supplements available in both countries are Ferro-Tab (65mg elemental iron), Ferro-Gradumet (105mg elemental iron) and Ferrograd C (105mg elemental iron with 500mg Vitamin C). Iron supplements commonly cause constipation and some pregnant women are reluctant to take them. Taking one iron tablet every second day and increasing dietary fibre and water consumption can help ease symptoms.

Iodine

Adequate maternal iodine is important for thyroid function and fetal cognitive development. A high proportion of the Australian and New Zealand population have iodine intakes below nutritional recommendation levels. A 150mcg iodine supplement (Neurokare) is recommended by the New Zealand Ministry of Health to be taken for the duration of pregnancy and breastfeeding. Similarly, women in Australia are recommended to take 150mcg iodine per day[8], though there is no specific 150mcg iodine supplement available for prescription/purchase. Instead, over-the-counter prenatal multivitamin products are commonly used to obtain iodine in Australia. Kelp and seaweed tablets should be avoided as their iodine content varies widely. It is unclear at this stage whether there is a benefit to iodine being taken during the preconception period.

Multivitamins

Pregnancy multivitamin supplements have not been demonstrated conclusively to improve pregnancy outcome and are therefore not generally recommended.[2,3] Instead, women are advised to take specific micronutrients according to their personal nutritional status and to consume a wide variety of foods to improve their nutritional intake. Multivitamins often contain micronutrients that compete for absorption in the body, such as iron, zinc and calcium. Additionally, many multivitamins do not contain the recommended amounts of folic acid and iodine and, therefore, additional supplements are frequently necessary.

Other micronutrients

There are a range of other micronutrients that are important during pregnancy, but for which nutritional supplementation is not recommended owing to a lack of conclusive evidence and/or that adequate levels can be obtained without supplementation.

For women who have low calcium intake, taking a calcium supplement (1000–2000mg elemental calcium) may reduce the risk of developing pre-eclampsia by around 50 per cent.[9]

Women following a strict vegan diet may require an intramuscular vitamin B12 injection before or during pregnancy (hydroxocobalamin 1000mcg/ampule).

A clear guideline for Vitamin D is yet to be decided; there is controversy over what constitutes a low vitamin D status, who should be prescribed supplements and what dose should be taken. The main source of Vitamin D is sunlight; however, there is no set level of sun exposure that generates adequate vitamin D without increasing the risk of skin cancer. In summer, care should be taken between 10am and 4pm when UV levels are highest. Skin exposure to sunlight outside these hours in summer, and around midday in winter, should safely generate adequate vitamin D for most women.

Long-chain polyunsaturated fatty acids (omega-3s) are commonly taken as fish oil supplements. There is little evidence of benefit for this specific population group, aside from a small reduction in preterm birth.[10] Omega-3 supplements vary widely in quality and purity. In general, women should be recommended to obtain omega-3s from oily fish rather than supplements; one small serving of oily fish provides one-to-two weeks' worth of omega-3s.

In conclusion, care should be taken with nutritional supplementation in pregnancy. Women should be recommended to take adequate folic acid, iodine, iron (if necessary) and to consume a wide variety of foods.

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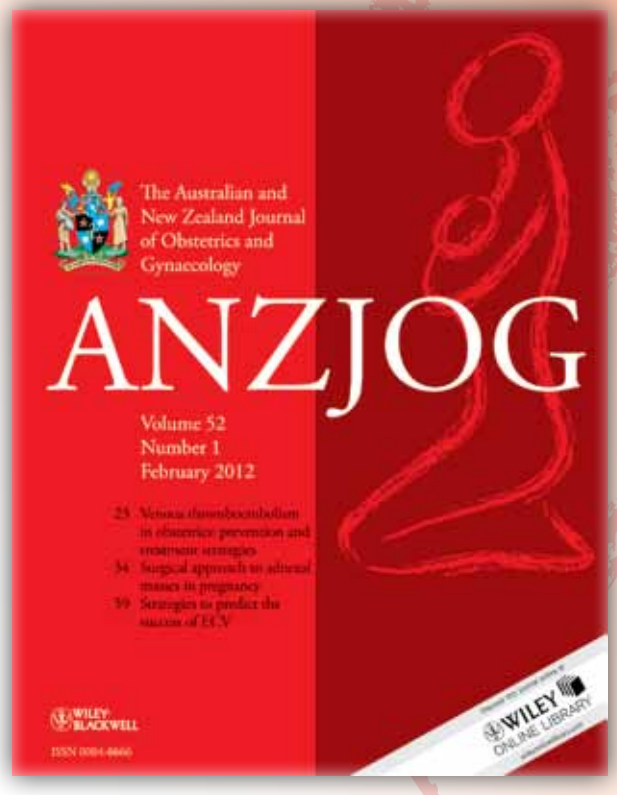

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Chocolate and pregnancy

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Who would have thought that something so outrageously decadent as expensive chocolate might actually be good for you?

When Hernando Cortés and about 600 Spanish soldiers, the *conquistadores*, landed on the Yucatan peninsula of modern-day Mexico, the Aztecs (under the leadership of their King Montezuma) controlled all of the area. The Spanish wanted to convert the Aztecs to Christianity, but had also come to find gold and return it to Europe. Though vastly outnumbered, Cortés' men had a terrible advantage that almost wiped out the Aztecs – viral disease. The Mesoamericans had no herd immunity to the diseases the conquistadores brought with them, likely to have been smallpox and measles, and so succumbed to the Spanish. Legend has it that the Aztecs valued the humble cocoa bean more highly than gold, and when the Spanish eventually broke into the treasure stores expecting to find gold, they found stores of cocoa beans instead.

The cacao tree grows in Central America, flourishing in the climate of persistent heat, humidity and high rainfall (not unlike Cairns). The Mexican climate was too dry, and the Aztecs traded to obtain the prized cocoa beans from the Mayans. Cacao is a Mayan word, meaning 'God food', and 'cocoa' is the European corruption of that word. Indeed, the Latin species name for the cocoa bush is *Theobroma Cacao*, literally 'the food of the Gods'.

The Mayans and Aztecs consumed chocolate as a drink, after pulping and cooking the beans then mixing it with chili powder and some maize flour. The resulting mixture was subsequently allowed to ferment in ceramic containers. The residue of chocolate drinks has been found in Central America by archaeologists in earthenware dating more than a thousand years before the birth of Christ. Cortés and his men, when they first encountered 'chocolate drinks', were horrified by the brew, describing it as 'unpleasant' and 'loathsome'. The Aztec name for the malodorous brew was *xocolatl*, which was pronounced as 'chocolate' by the Spanish. It was thought that xocolatl was an aphrodisiac, hence its prized place in Aztec society.

To make the mixture drinkable for the Europeans, the Spanish modified the recipe and mixed the pulped cocoa beans with sugar and vanilla beans. Once back on Spanish soil, the fermented beans were roasted then ground up like flour and thereafter exported. The fact that cocoa beans provided the raw material for cocoa powder was kept secret, much like the Coca Cola recipe or Colonel Sanders' 11 secret herbs and spices are today. The story goes that buccaneers seized a heavily protected Spanish vessel, assuming it to be loaded with treasure. The holds were full of cocoa beans, which they mistook for sheep manure, prompting the raiders to scuttle the vessel in frustration, unaware that, by weight, cocoa beans were more valuable than silver at the time!

The spread of chocolate to the new world

Chocolate powder exported from Spain had spread around Europe and arrived in England in the 16th century. A splinter group from the Puritans of the English Civil War, the so-called Quakers, took to working with chocolate. The Quakers were a strictly pacifist group and preferred to work with food rather than in industries that could contribute to war. Many of the great names associated with chocolate manufacture – Rowntree, Cadbury and Fry – were Quaker families. The Quakers had a strong sense of social responsibility and, in contrast to many other industrialists of the era, took their responsibility to employees very seriously. The Cadbury family built an entire town, complete with a church, library, school and good-quality housing for their employees. The town was named Bourneville, a name we associate with cocoa powder to this day.

Many of the original colonies of North America – eventually becoming Canada and the USA – were established by conservative puritan Christians, who took the cocoa beans with them. At the time, processes for extracting the 'bitter-tasting fat' or cocoa butter were developed, to improve the palatability of the cocoa. It was not until the mid-19th century, though, before the first solid chocolate was manufactured by mixing sugar with cocoa powder in a cocoa butter base. This method of making solid chocolate was developed by Fry and Sons of Bristol in England.

What's good for you?

It has long been recognised that foods such as red wine and green tea contain antioxidants with positive health effects. Cocoa beans also contain plentiful polyphenols, of which the sub-class of flavanols are particularly abundant in dark chocolate (chocolate that is rich in cocoa). However, it was not until the turn of the new century that researchers began to investigate whether the antioxidants in dark chocolate might impart the same health benefits as other antioxidant foods. Studies of dark chocolate yielded remarkable results, with a systematic review concluding that levels of chocolate consumption seem to correlate with a substantial reduction in the risk of cardiometabolic disorders.[1]

Flavonoids such as flavan-3-ol and their oligomeric derivatives, procyanidins, are present in high concentration in cocoa and dark chocolate products. Consumption of dark chocolate by healthy subjects has been associated with 'a beneficial effect on endothelial function in healthy adults.'[2] Similar studies in smokers have shown that dark chocolate induces a rapid and significant improvement in endothelial and platelet function in healthy smokers up to eight hours after ingestion.[3] Those authors attributed these effects to the antioxidant effect of dark chocolate, including reduction in platelet

activation. A similar study by Engler and colleagues reported that flavonoid-rich dark chocolate improved endothelium-dependent vasodilatation in healthy adults.[4]

The flavanols in dark chocolate have been shown to deactivate free radicals and also stimulate release of nitric oxide (NO), a potent vasodilator, from vascular endothelium. It also seems that they reduce low density lipoprotein (LDL) oxidation by neutralising free radicals, and may help prevent endothelial injury that is associated with plaque formation. Dark chocolate has also been found to have a similar effect to low-dose aspirin on arterial place thrombus formation.

Chocolate and pre-eclampsia

Although the pathophysiological basis of pre-eclampsia (PE) remains incompletely understood, a number of key physiological abnormalities have been identified and these will be of interest to chocolate lovers. In the first instance, PE is more common in pregnancies with increased placental oxygen demand (for example, multiple pregnancy, rapidly growing hydatidiform moles and hydropic placenta). Similarly, when there is decreased oxygen transfer the incidence of PE increases (microvascular diseases such as chronic hypertension, diabetes and collagen-vascular diseases). It is important that the primigravid uterus has a less well-developed vasculature than in multigravid women.

Food of the gods it might well be, but can eating high-quality dark chocolate during pregnancy help prevent pre-eclampsia?

In normal pregnancy, the end of the first trimester is characterised by invasion of cytotrophoblastic tissue through the basal decidua to the inner myometrium. There are marked morphological changes to the spiral arterioles in the intervillous spaces. The arterioles are converted to large, tortuous sinusoidal vessels. The endothelium and internal elastic lamina is replaced by trophoblast and amorphous matrix containing fibrin.[5] The morphological changes mark conversion of the placental vascular supply to a 'low-pressure, high-flow' system. In PE, these arteriolar changes either do not occur or are limited to vessels located in the decidua. This 'failure of endovascular trophoblastic infiltration' results in the uteroplacental arteries maintaining their musculo-elastic architecture and responsiveness to vasomotor influences.[6]

The endothelium is an epithelial monolayer, in direct contact with the blood. The endothelium has many known functions, including modulation of vascular smooth muscle reactivity in response to stimuli. The endothelial cells synthesise various substances. These include small molecules such as NO, platelet activating factor (PAF), endothelin and prostacyclin (PGI2). Nitric oxide and PGI2 are particularly important, as these normally inhibit platelet adhesion to the endothelium.[7]

Pre-eclampsia is associated with abnormal endothelial cell function, with the consequences of activation of the coagulation cascade and fluid extravasation (resulting from breakdown in the endothelial barrier) and increased pressor effects of vasoactive substances. Endothelial synthesis of PGI2 and NO is reduced in women with PE.[8] It is postulated that dysfunctional endothelium activates platelets with consequent release of thromboxane (TXA), resulting in an abnormal ratio of TXA:PGI2 that would enhance relative or absolute vasoconstriction.[7] Taken together with results of studies of a large number of other vasoactive substances, it is likely that 'endothelial cell dysfunction may be the cause rather than the consequence of the increased blood pressure observed in pre-eclampsia.'[5] Indeed, since endothelium is ubiquitous, it would explain the multisystem nature of the manifestations of PE.[9] There is evidence for impairment of endothelium-dependent relaxation of myometrial arteries in PE. As well, platelet activation is increased in PE, associated with relative thrombocytopaenia and increased mean platelet volume (as younger platelets are released into the circulation).[9]

With these physiological mechanisms in mind, a number of attempts have been made to reduce the incidence of PE. These are reviewed in detail by Visser and Wallenburg[10] and include lifestyle interventions, and dietary restrictions (salt, for example). Systematic review and meta-analysis of pooled data from trials of aspirin suggests that 'low-dose aspirin is mildly beneficial in the prevention of pre-eclampsia in high-risk women, but no beneficial effect was observed in women at low risk of developing pre-eclampsia.'[11]

Since PE is associated with widespread endothelial dysfunction, would consumption of flavonoid-rich dark chocolate (which has a broad effect to improve 'endothelial dysfunction' and reduce platelet activation) be expected to have a similar, or indeed superior, effect to that shown in trials of low-dose aspirin in reducing the primary incidence of PE in low-risk populations? A recent prospective cohort study examined the amount of chocolate consumed by pregnant women and found that increased chocolate intake in the first trimester was associated with reduced odds of gestational

hypertension, with an adjusted odds ratio of 0.65 (95 per cent CI: 0.45, 0.87), which seems to be a dramatic decrease.[12]

With these findings in mind, we are currently undertaking a pilot study of dark chocolate in early pregnancy. Among the issues we are trying to iron out are whether women will be too nauseated to eat 25mg of dark chocolate each day and how to stop women in the control arm from simply going to a supermarket and buying chocolate.

A guilty pleasure

How delightful it is that something so outrageously decadent as dark chocolate might have positive benefits. From its bloody history in the jungles of Central America, to the antenatal clinics of Cairns and Canberra, the lure of the 'food of the Gods' continues.

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Are you doing O&G in China?

RANZCOG has recently been approached by RACS, who have run a project in China called Project China over the last 20 years. RACS is now in the early stages of establishing the China-ANZ Project, which seeks to develop a more strategic approach to relationships between specialty colleges and institutions in Australia, New Zealand and China.

RACS is undertaking, in the first instance, a stocktake of existing programs, both formal and informal, undertaken by Australasian individuals and academic institutions in China, with a view to better coordination of activities between colleges in China.

RANZCOG does not currently have any formal programs in China, however, it would be useful to hear from any Fellows or members who have an interest in, or do independent work in China under another umbrella or as individuals; with a view to information exchange/networking/collaboration with our RACS colleagues and the potential for forming a working relationship with RACS in China in the future.

Please email our Asia Pacific Senior Coordinator, Carmel Walker (cwalker@ranzcog.edu.au), for further discussion.

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Eating disorders in O and G

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Eating disorders, encompassing anorexia nervosa and bulimia, affect approximately five per cent of women and have profound medical and psychiatric ramifications.

Eating disorders are associated with significant morbidity and mortality rates and, therefore, early recognition is imperative. Due to the ubiquity of these conditions and the growing number of patients diagnosed at increasingly young ages, it is probable that the majority of O and Gs will encounter such patients in their practice under various guises. Initial presentations may be to an O and G, with the patient not having a prior documented history of eating disorders.

According to the American Psychiatric Association Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM V), the proposed diagnostic criteria of anorexia nervosa are associated with a restriction of energy intake relative to requirements leading to a significantly low body weight in the context of age, sex, developmental trajectory and physical health. Anorexia is further associated with an intense fear of gaining weight or becoming fat, or persistent behaviour that interferes with weight gain, despite the fact that the patient has significantly low weight. A common feature of anorexia is a disturbance in the way in which one's body weight or shape is perceived or persistent lack of recognition of the seriousness of the current low body weight. Finally, in postmenarchal women, amenorrhoea in at least three consecutive menstrual cycles is characteristically described in this condition. Previous criteria defined the weight in anorexic patients to fall below 85 per cent of their expected weight. It is postulated that moving away from this rigid criteria would allow inclusion of adolescents, allowing for normal variability in development, and women with partial syndromes who may still suffer adversely as a result.

Two types of eating disorder are described, a food restricting and a binge eating or purging type. Bulimia nervosa is defined as recurrent episodes of binge eating. An episode of binge eating is characterised by eating an amount of food that is larger than most people would eat during a similar period of time under similar circumstances and associated with a sense of lack of control over eating during the episode. Compensatory measures are then taken in order to prevent weight gain. Typically these include self-induced vomiting; misuse of laxatives, diuretics or other medications; fasting; or excessive exercise. The binge eating and inappropriate compensatory behaviours both occur, on average, at least once per week for three months.

Female reproduction resulting in pregnancy requires an investment of a large amount of energy. Energy stores are diverted to the baby while in utero and, subsequently, energy is required during parturition, lactation and child rearing. This comprises the largest expenditure of energy in a woman's lifetime.[1] As such, survival mechanisms exist in undernourished women to protect them against the additional energy demands of an ensuing pregnancy. This is achieved by interplay between the reproductive axis and the nutritional status, leading initially to a reduction and ultimately cessation of ovulation when energy stores are deemed insufficient to support a pregnancy. Energy balance appears to be a more important factor than absolute weight or body mass index (BMI); clinicians should bear in mind that anovulation may precede significant weight loss.[2] Conversely, moderate improvements in nutritional status may restore fertility in most cases.

> 'Infertility is a common presentation in patients with eating disorders; indeed, primary infertility may be the initial presenting manifestation of eating disorders.'

Malnourishment has profound medical ramifications. The Dutch famine, which affected the western parts of the country between October 1944 and May 1945, resulted in a fall of an average daily calorific intake from 1500 kilocalories per capita to less than 700 kilocalories per capita. Subsequently, the birth rate nine months after the onset of the famine decreased. It has been postulated that a maturation process of the hypothalamic-pituitary-gonadal axis in children can be undermined by periods of starvation and may have reproductive sequelae in adulthood. From the Dutch experience it was retrospectively seen that the reproductive lives of girls experiencing the famine when aged between three and 13 years old had a 1.88 fold increase (95 per cent CI = 1.29-1.74) of having no or fewer children than desired.[3]

Eating disorders predominantly first develop in adolescence, with 90 per cent presenting before 25 years of age. Clinical sequelae of anorexia and bulimia depend on timing of the disease in relation to puberty. Presentations may include pubertal delay, primary or secondary amenorrhoea, pubertal arrest, cold intolerance, constipation or diarrhoea, fatigue, frequent fractures, nerve compression, easy bruising and scalp hair loss and/or dental problems associated with enamel erosion.[4,5] The earlier these conditions are recognised, and treatment initiated, the higher the chance of successful remission. In the case of anorexia, full recovery is anticipated in a third of patients; while with bulimia, resolution is seen in 74 per cent. However, achieving these recovery rates requires a protracted period of follow up and surveillance and, despite this, relapse occurs in a third of patients.[6]

Fertility

Anorexia nervosa and bulimia nervosa affect up to five per cent of women of reproductive age causing amenorrhoea, infertility and, in those who do conceive, an increased likelihood of miscarriage.[7] Menstrual periods often cease after a 10–15 per cent decrease in normal body weight. Amenorrhoea may occur in normal BMI bulimic women, indeed amenorrhoea may persist in 30 per cent of patients who have recovered from anorexia and regained a normal weight.[8]

Infertility is a common presentation in patients with eating disorders; indeed, primary infertility may be the initial presenting manifestation of eating disorders. Identification of this is imperative both for the successful treatment and in order to deal with potential deleterious ramifications during pregnancy and later on in the woman's life. In the fertility clinic setting, a disproportionately high number of patients have eating disorders. Of patients presenting with amenorrhoea or oligomenorrhoea, it has been estimated that over 50 per cent may have an underlying eating disorder. It is worth keeping in mind that patients may not recognise that their behaviour constitutes an eating disorder or be in denial of this. Sensitively broaching eating habits, exercise, nutritional intake and examination in combination of body weight and height should be routinely completed with all patients presenting to an infertility clinic. Infertility may be due to a combination of anovulation, sub-physiological levels of sex hormones that may result in reduced libido and, in some, a rejection of sexual activity. Restoration of the hypothalamic-pituitary-ovarian axis depends on weight gain and resolution of underlying psychological issues. Persistence of amenorrhea despite appropriate weight gain is in some cases related to the duration of anorexia; the longer the duration of the illness the longer it may take for resumption of menses. Underlying anxiety disorder or abnormal eating habits may also contribute to a persistence of amenorrhoea.[9,10,11,12] Interestingly, several studies have demonstrated that live birth rates in patients with low BMI are not reduced in comparison with normal BMIs. In a Norwegian study of patients receiving ART, those with BMIs $<18.5 kg/m^2$ had identical live birth rates to those patients with BMIs ranging from 18.5 to $25 kg/m^2$.[13] This may reflect the fact that BMI is not a sensitive measure of energy stores as described above.

Pregnancy

Early symptoms of pregnancy may mimic symptoms associated with eating disorders. These include delay in menstrual cycle, hyperemesis and fatigue. As such, pregnancy may on occasion present late. Hyperemesis gravidarum may be caused by a variety of factors. It is however worth noting that intractable, severe hyperemesis may be associated with increased frequency in those with eating disorders.[14,15] Indeed, some women with eating disorders have increased incidence of impaired weight gain during pregnancy and of delivering infants with lower birth weights, microcephaly and intrauterine growth-restricted babies.[16,17] Women's response to pregnancy varies, some studies report an improvement in symptoms as a direct consequence of their pregnancy, with the motivation of maintaining their health and health of their babies.[18,19] This is especially seen in women who have previously been treated and have a supportive network. Others find it difficult to distinguish their pregnancy with weight gain. With a profound fear of uncontrolled weight gain, pregnancy is perceived as adopting poor body image and as such concomitant anxiety and depression are commonly. The postpartum period is a stressful time associated with profound physiological and hormonal changes. These are coupled with great emotional upheavals coping with a newborn that can precipitate dysfunctional eating habits. A large proportion of women who report an improvement in eating disorder symptoms during pregnancy, regress in the postpartum period. As such, pregnant women with a current or past history of eating disorders should be monitored closely postpartum so that potential issues with either mother or baby may be addressed.

Understanding the nature of these conditions and the various presentations in an O and G context is imperative. Adopting a non-judgemental, holistic, multidisciplinary view is undoubtedly advantageous in management of these patients

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Vegetarians and vegans

Dr Lindsay Edwards
FRANZCOG Trainee

A vegetarian for 20 years, my decision to stop eating meat had more to do with a love of animals than the associated health benefits. Now, I also view vegetarianism as having numerous health and environmental benefits.

The term 'vegetarian' is applied to a number of different diets, all based on a consumption of fruit, vegetables, legumes, whole grains and nuts; and an elimination of meat and/or all animal products. Vegetarian diets differ in their popularity and composition around the world. An estimated five per cent of Australians identify as vegetarian, but only two per cent abstain from eating any meat, fish or poultry.[1] This compares to 2.5 per cent in the USA, three per cent in the UK and four per cent in Canada.[2]

A lacto-ovo vegetarian diet is one that excludes meat (including poultry and seafood), but includes dairy products and eggs, and is the most common type of vegetarian diet. A vegan diet eliminates all animal products, including dairy. Those who follow a vegan lifestyle also avoid animal products, such as leather, altogether. Some 'vegetarians' (pescatarians/vegaquarians) eat a small amount of seafood and, increasingly, non-vegetarians are reducing their red meat consumption, as a 'vegetarian-influenced' lifestyle is sought for health, economic or environmental reasons.

Reasons to be vegetarian

There are a number of reasons why people choose to be vegetarian. For many, the concerns for animal welfare are of the utmost importance; with 2011's media exposure of the realities of live export a reminder of the life and death that many farmed animals have to endure. Worldwide each year, 56 billion land animals are reared and slaughtered for human consumption[3], with many of these animals having lived, been transported or killed in inhumane or substandard conditions.

Factory or 'intensive' farming is becoming more common in countries such as the USA and China, as the demand for meat increases at an alarming rate. Animals, commonly pigs and poultry, are held in small cages, crates or stalls, in an artificial warehouse-like environment, devoid of natural stimuli and adequate space.[3] Australia, unfortunately, is not immune to this practice, with 75 per cent of eggs sold coming from battery (cage) hens and an even greater number of 'broiler' hens (grown rapidly for 30–60 days prior to slaughter) reared in cramped conditions. Annually in Australia, 470 million broiler hens are produced, with the average person consuming 38kg of chicken per year.[4]

In these 'factory farms,' debeaking is not an uncommon practice. It is used to prevent 'cannibalism', a behaviour that occurs when too many birds are in a confined space and they end up pecking and attacking one another due to their stressful environment. In Australia, 5.5 million pigs are killed for meat each year, with 95 per cent raised indoors in intensive 'farming' conditions. One recent, though small, advance has been the move by the pork industry to phase out 'sow stalls' (by 2017), in which pregnant and lactating pigs are housed in concrete pens, with little room to move.[5]

There are over 16 million beef cattle in Australia, the majority of which graze in pastures, but intensive feedlots have increased in numbers, with cattle being fattened by grain in confined lots prior to (often stressful) transport and slaughter. Up to 50 per cent of the beef in supermarkets comes from feedlots.[5]

Environmental impact

Many people are turning to a vegetarian diet to reduce their carbon footprint. A lacto-ovo vegetarian diet requires significantly less energy, land and water resources than a meat-based diet.[6] Production of 1kg of animal protein requires 100-times more water than 1kg of grain protein. In the USA, the nine billion animals of the livestock trade consume more than seven-times the amount of grain consumed directly by the human population each year.[6] The impact of the livestock industry on the environment is enormous. The Food and Agricultural Organization of the United Nations reports the livestock sector as one of the most significant contributors to serious environmental problems, such as land degradation, climate change, air pollution, water shortage, water pollution and loss of biodiversity.[7] Over 18 per cent of greenhouse gas (GHG) emissions (measured in carbon dioxide equivalent) are attributed to the livestock trade, a similar share to industry and a higher share than transport.[7, 8] The livestock industry is the leading offender in the production of other GHGs, responsible for 37 per cent of the methane emissions (mostly from enteric fermentation in ruminants, such as cattle and sheep) and 65 per cent of the nitrous oxide emissions caused by humans.

Livestock production, for grazing or feedcrop, accounts for 30 per cent of the planet's land surface. Around the world, deforestation has been devastating, with up to 70 per cent of land previously covered by rainforest in the Amazon cleared for pastures or for the plantation of livestock feed.[7] In Australia, the dairy industry is the highest user of irrigated water in the Murray-Darling Basin and livestock consumes over eight per cent of total global water use.[9]

With the shift toward a Western diet in developing countries, along with general population growth, the global production of meat is expected to more than double from 229 million tonnes in 1999/2001 to 465 millions tonnes in 2050.[7] Experts are calling for a substantial reduction in meat consumption in developed countries, and constrained growth in developing countries.[8] Worldwide, the 'Meatless Monday' movement, which began in 2003, has gained popularity in an effort to decrease the demand and flow on effects from a meat-based diet.

Health benefits

Vegetarian diets include lower amounts of saturated fats, cholesterol and animal protein; and increased amounts fruits, vegetables, whole grains, nuts, soy, fibre and phytochemicals, with increased levels of magnesium, folate, vitamin C, vitamin E

and antioxidants, thought to be beneficial in reducing the risk of chronic disease. Vegetarians tend to have lower body mass indices and are less likely to be overweight or obese; they have lower low-density lipoprotein cholesterol levels, lower blood pressure, and less risk of diabetes and the metabolic syndrome.[9-11] Death from ischaemic heart disease (IHD) is also reduced in vegetarians compared with non-vegetarians.[10] In some studies, a vegetarian diet has been associated with lower overall cancer rates and high red meat intake has been linked with an increased risk of colorectal cancer.[10] Vegetarians have also been shown to have a longer life expectancy.

Women's health and nutrition

The American Dietetic Association and the Dietitians of Canada endorse appropriately planned vegetarian (and vegan) diets as healthy and nutritionally adequate.[2] It is their position that a well-planned plant-based diet is suitable in all stages of life, including pregnancy, lactation, infancy, childhood and adolescence.[11]

Most people in the developed world consume more protein than is necessary.[12] The recommended dietary intake (RDI) of protein per day is 46g for women aged 19–70 years, 58–60g in pregnancy, and 63–67g during lactation.[13] In a study of Australian women aged 18–45 years, vegetarians (including vegans) had a mean protein intake of 54g per day, lower than the 67g per day for omnivores, but still meeting the RDI.[14] A variety of protein sources (soy and mixed vegetable) best ensures protein and individual amino acid requirements are met. Nuts, seeds, soy and legumes are important sources of protein in a vegetarian diet. Consuming protein from plant sources, rather than animal sources (which are also higher in saturated fat and cholesterol), may be one of the reasons why vegetarians have a lower risk of obesity and IHD.[12]

Vegetarian diets contain as much iron as diets containing meat.[15] The iron from a plant-based diet is non-haem, while both non-haem and haem iron are obtained from a mixed diet. The bioavailability of non-haem iron is largely determined by the body's need for iron – when iron stores are low, absorption increases and excretion decreases. Vegetarian diets are also higher in vitamin C, which aids in the absorption of non-haem iron.[14] In a European study of 43 000 women, vegetarians and non-vegetarians had similar iron intakes and haemoglobin concentrations[16], and a number of other studies have demonstrated that vegetarian women are no more likely to have iron deficiency anaemia than non-vegetarians.[14] Obesity, a by-product of the Western, affluent meat-based diet, is more strongly associated with iron deficiency anaemia than a vegetarian diet is.

The current RDI for iron for women aged between 19 and 50 years is 18mg/day.[13] The RDI in pregnancy is 27mg/day. Pregnancy sees an increase in intestinal iron absorption by up to 60 per cent in the third trimester as nutritional demands increase. In a prospective cohort of 1274 pregnant women, vegetarians were less likely to have low dietary iron intake, and more likely to take iron supplements during pregnancy, with a positive effect on fetal weight.[17]

Well-planned, plant-based vegetarian diets have also been shown to meet current recommendations for most omega-3 fatty acids, zinc, iodine, calcium, vitamin D and vitamin B12.[11] Vitamin B12 deficiency may occur in vegan diets, as animal-based products are almost exclusively the source of this vitamin. It is recommended that vegans (and vegetarians who do not eat an adequate amount of eggs or dairy) have their vitamin B12 levels checked regularly, and take supplements, or fortified foods to reduce the risk of deficiency, which can lead to neurological symptoms and disease. Vitamin B12 in this way is highly bioavailable and deficiency can be easily prevented. Infants born to vegan mothers are also at risk of significant vitamin B12 deficiency, if the mother has not had adequate supplementation in pregnancy and lactation.[18]

It is important to consider where your food comes from, to make ethical and responsible decisions regarding your next meal and, if possible, reduce the amount of meat in your diet. International Vegetarian Week (1–7 October) is a good place to start. As Albert Einstein said, 'Nothing will benefit human health and increase chances of survival for life on earth as much as the evolution to a vegetarian diet.'

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The first superfood

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Breastmilk, breastfeeding and optimal nutrition: nutrition is only half the story.

Breastmilk is species-specific, living, biodynamic and synergistic. Biodynamic because breastmilk varies from woman to woman, by gestational age of the infant, from day to night, from the beginning to the end of the breastfeed and also changes flavour depending on what the mother eats. Vanilla, garlic and carrot have been described as discernible taste sensations in breastmilk and some research suggests that these flavour changes are significantly important as primers for infants when they start to eat other foods.[1] Another significant aspect of breastfeeding described by Ball and Klingaman is that humans are, 'a low solute, frequent suckling species,' which explains why breastfeeding works best when mothers sensitively respond to infant cues for feeding.[2] Based on evidence evaluated by the World Health Organisation, breastfeeding is recommended for around six months at which point developmentally ready infants should be offered appropriate complementary foods. As pointed out by epidemiologist Adriano Cattaneo,[3] developmental readiness for solid food has a distribution range like any other biological variable, a bell-shaped curve that has a mode at six months and is skewed to the right, which means more infants are ready after than before six months. It is recommended that breastfeeding continues after solid foods are started for up to two years and then beyond if the mother wishes to continue.[4]

Breastfeeding has established itself within research as the optimal method of feeding babies. The American Academy of Pediatrics (AAP) first released a policy statement on breastfeeding and the use of human milk in 1997[5] that was revised in 2005, owing to 'significant advances in science and clinical medicine.'[6] In 2012, an updated policy was released stating that 'recently published research and systematic reviews have reinforced the conclusion that breastfeeding and human milk are the reference normative standards for infant feeding and nutrition.'[7]

The AAP statements identify health, nutritional, immunologic, developmental, psychological, social, economic and environmental advantages to breastfeeding. The protective effects of breastmilk against sudden infant death syndrome (SIDS), insulin-dependent diabetes mellitus, allergic diseases, Crohn's disease, ulcerative colitis and other chronic digestive diseases are recognised. Research also shows strong evidence that breastmilk feeding decreases the incidence and severity of diarrhoea, lower respiratory tract infection, otitis media, bacteraemia, bacterial meningitis, botulism, urinary tract infection, necrotising enterocolitis and late-onset sepsis in preterm infants. There are also significant health advantages for mothers that become more robust with increased breastfeeding duration.

Half a story – optimal nutrition

Breastfeeding provides optimal nutrition to the human infant. Milk from a human mother contains a wide range of nutrients and also hormones such as ghrelin, a metabolic modifier that assists with the coordination of food intake, energy expenditure and nutrient utilisation.[8] The carbohydrates in human milk include lactose and oligosaccharides as major components. Oligosaccharides may provide a good source of energy for infants. Milk production and the gross composition of breastmilk are largely independent of maternal diet and there is little variation between population groups in amounts of milk produced.[9]

The average calorie content of human milk is 22kcal/30ml, but this varies not only throughout each feed, but also changes depending on the time of day. The fat range is 22.3–61.6g per litre, independent of breastfeeding frequency.[10] It is, however, not the

Only 35 per cent of the world's infants are as lucky as this little one. Frank Medworth Mother and Child II, 1929 (Mother feeding baby) Ink and colour pencil. The drawing depicts the artist's wife, Muriel, and daughter, Diana. Inscribed with date Oct 17 1929. From the RANZCOG Historical Collections. Purchased with Funds from the Friends of the College Collection, 2003.

amount of fat in the breastmilk that correlates with infant growth, but the volume of breastmilk intake.[11]

As Goldman pointed out in 2000, 'the notion that human milk supplies only nutrients to the infant was still in vogue four decades ago'[12], which is why there is another half of the story.

The other half of the story

A large part of human milk is unable to be digested by the infant and serves an entirely different purpose to nutrition. Human milk has been described as an 'astonishing product of evolution'[13] shaped by natural selection and designed to ensure to the maximum extent possible the survival of the mother and her infant. Akre points out that '200 million years or so of mammalian evolution should be worth something in terms of our default position being intrinsically weighted in favour of breastfeeding.'[14]

There are many components of breastmilk designed for infant defence against infection, including large amounts of Secretory IgA antibodies that migrate from the mother's gut to her breasts. Oligosaccharides are thought to provide a food source for a healthy microbiome and they may also attach themselves to viruses and other pathogens and safely take them out of the infant's system. Oligosaccharides have been described as '130 reasons to breastfeed'[15] and not only do they stimulate an increase in the number of good gut bacteria in breastfed babies and a significant decrease in the number of potentially bad bacteria, but they are also important for brain development, nerve transmission, memory formation, cell-to-cell communication and resistance to infection.

The immune system of human milk provides a continuum of the maternal immune protection that extends from the transfer of immunoglobulin G (IgG) from the mother via the placenta to the infant in utero, and then from the mother via breastfeeding and breastmilk to the infant after birth.[16] Cregan et al, describe how this continuum of support for the immature human infant/child unfolds with a new mother's mammary glands taking over from the placenta to provide developmental guidance.[17] Breastfeeding actively stimulates the infant's immature immune system and modulates development; studies have found that the thymus gland of an exclusively breastfed infant is twice the size of a non-breastfed infant.[18]

The complex components of human milk are irreproducible and include lactoferrin, a single polypeptide chain glycoprotein that forms two lobes, both of which bind iron.[19] Lactoferrin is quite resistant to degradation in the gut and the stools and urine from a breastfed baby contains significant amounts of lactoferrin, including large fragments. Special receptors in the baby's gut uptake lactoferrin and these large lactoferrin fragments. Lactoferrin is bactericidal for many gram negative and gram positive bacteria and also has antiviral and antifungal properties. Lactoferrin destroys microbes without inducing tissue engagement and inflammatory responses and prevents production of several pro-inflammatory cytokines.[19] It is thought that lactoferrin protects breastfed babies against urinary tract and bowel infections.

A happy ending to the story?

The breastfeeding and breastmilk information just presented remains only a fraction of a much bigger and remarkable true story. Despite all the accumulating evidence that clearly shows the risks of not breastfeeding, only 35 per cent of the world's infant population are exclusively breastfed at three months of age. It is critical that all health professionals incorporate actions that protect, promote and support breastfeeding into their everyday work. Alongside this some system changes are also essential to enable women to establish breastfeeding, breastfeed for longer and avoid breast-milk substitutes. These include support for the Baby Friendly Hospital and Community Initiatives, longer paid parental leave, family- and breastfeeding-friendly workplaces and some meaningful regulation of the infant formula industry to reduce or preferably eliminate aggressive marketing and misleading health claims. The loss of breastfeeding has significant negative implications and as a researcher recently stated, '…every component of [human] milk probably has a special role. It's all there for a purpose though we're still figuring out what that purpose is, so for God's sake, please breastfeed.'[13]

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Gastroenterological disorders in pregnancy

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A guide to the recognition and management of new-onset or existing disorders affecting the liver and digestive organs supplements the optimal care provided during pregnancy.

Pregnancy is a unique milestone in a woman's life, marked by significant physiological and sentimental developments. In this article, gastrointestinal and hepato-biliary disorders in pregnancy are reviewed, with a particular focus on inflammatory bowel disease (IBD) and gastro-oesophageal reflux disease.

IBD

IBD, manifesting as either Crohn's disease or ulcerative colitis, requires particular attention in pregnancy. A high proportion of women with IBD are diagnosed around the time of their reproductive years. There can be significant associated complications and complex therapeutic treatment requirements that can cause substantial emotional strain, particularly in those with a recent diagnosis of the condition.

Fertility

Female fertility rates are relatively equivalent to the general population for both Crohn's disease and ulcerative colitis (UC), although poorly controlled inflammatory bowel activity can adversely impact on fertility rates.[1,2,3] The higher voluntary infertility rate among IBD patients is typically related to concerns regarding the effect of the disease or treatment associated adverse effects on pregnancy outcomes. Surgical procedures such as ileal pouch anal anastomosis (IPAA) following procto-colectomy for severe ulcerative colitis have resulted in significant reduction in fertility.[4] When possible, restorative surgery is postponed until completion of pregnancy in order to preserve fertility. Sulfasalazine causes reversible reduction in male sperm count and motility, which returns to normal around three months following drug cessation.[5,6]

Heredity

Genetic predisposition, mucosal immune system defects and environmental factors are thought to have contributed to the development of IBD. Having a family member with IBD can increase the comparative risk of disease by up to 20 times that of the general population. Crohn's disease and ulcerative colitis are heterogeneous polygenic disorders with underlying genetic predisposition in a non-Mendelian fashion as established in familial studies showing monozygotic concordance rate of around 37–58 per cent for Crohn's disease and 6–17 per cent for UC.[7,8]

Pregnancy effect on disease

IBD activity can generally flare in the first trimester and postpartum. If conception occurs during quiescent disease state, then the risk of relapse during pregnancy is similar to those non-pregnant patients with IBD.[9] Conversely, of those with active disease at conception, two-thirds will have continued or worsening disease activity during pregnancy. This underscores the importance of disease control prior to conception.

Disease effect on pregnancy

There have been variable and conflicting effects on pregnancy outcomes in population studies. However, overall, pregnancies in IBD patients seem to be associated with poorer outcomes. A Northern Californian population study found IBD patients had an increase in the rate of spontaneous abortion (OR: 1.65; 95 per cent CI: 1.09-2.48); adverse pregnancy outcome (stillbirth, preterm birth or small for gestation age infant OR 1.65; 95 per cent CI: 1.00-2.38); and labour complications (OR 1.78; 95 per cent CI: 1.13-2.91).[10] Studies from Denmark and Sweden showed higher prevalence of preterm birth (OR 1.77), caesarean section (OR 2.01), small for gestational age (OR 2.78) and neonatal death (OR 1.93) in patients with UC and the risks correlated with disease severity.[11] Recent meta-analysis by Cornish et al also found increased premature birth, low birth rate and caesarean section particularly in Crohn's patients and more congenital abnormalities in UC patients.[12]

Strong focus has been placed on disease control during pregnancy, as there is evidence to suggest that disease activity has closer association with poor disease outcome than the diagnosis itself.[13] Therefore, optimal disease control would have the highest impact in ensuring uncomplicated pregnancy course.

Endoscopic evaluation

Unless required to make the initial diagnosis, an endoscopic evaluation of IBD is rarely indicated in pregnancy; nevertheless, it

Key points

- Optimal disease control of inflammatory bowel disease prior to conception is essential in minimising pregnancy complications.
- Common therapeutic agents in inflammatory bowel disease are generally safe for pregnancy and breastfeeding. Any potential minimal adverse effects are negated by the risks associated with disease flare.
- Hepatic diseases intrinsic to pregnancy can result in significant poor maternal and fetal outcomes which require intensive monitoring, supportive management and expedient delivery.
- Reflux symptoms are common during pregnancy and they usually respond well to lifestyle modifications and simple therapeutic agents.

generally can be performed safely. When possible it is postponed to the second trimester and often a limited sigmoidoscopy with minimal sedation can provide adequate information. Care is taken to monitor fetal wellbeing, particularly in relation to potential hypoxic effects from sedatives.[14]

Therapeutic options

Medication safety is frequently a major cause of apprehension among pregnant IBD patients and the primary care providers who lack familiarity with biologic drugs and immunomodulators. Pharmacotherapy in IBD generally involves the use of corticosteroids for induction followed by maintenance therapy with aminosalicylates and immunomodulators such as thiopurine analogues. Biological agents in the form of anti-tumour necrosis factor-alpha (TNF-α) can also be used as induction and maintenance therapy for both Crohn's and UC.

Corticosteroids are generally safe to be used for induction therapy during pregnancy. There is a possible minor association with cleft palate and reduction in birthweight.[15,16] Long-term adverse steroid effects are similar to those in non-pregnant patients.

Aminosalicylates (sulfasalazine/mesalazine/balsalazide/olsalazine) do not incur significant adverse risk in pregnancy or for breastfeeding.[17] Reports of diarrhoea in breastfeeding infants of women taking rectal 5-ASA has prompted monitoring the infant's stool consistency. The sulfapyridine component of sulfasalazine does cross the placenta and may affect folic acid metabolism. The usual supplementation with folic acid is recommended. Owing to limited evidence, the concerns regarding aminosalicylates causing premature closure of the ductus arteriosus has not led to established restrictions in the third trimester.

Thiopurine analogues – azathioprine and 6-mercaptopurine – have historically been assigned as Category D drugs in pregnancy; however, this was based on extrapolation from animal data and high-dose usage in oncology settings. With an increasing duration of experience, recent evidence suggests they are well tolerated when used for IBD in pregnancy and any minimal increase in adverse outcomes is negated by the risk associated with poor disease control.[18] Furthermore, potential adverse effects are lowered in the fetus due to a deficit of the enzyme inosinate phosphorylase required to convert thiopurine analogues to harmful active metabolites.

The use of biologics has revolutionised IBD management in recent years, with significant improvement in clinical response and remission rates. Two anti-TNF-α agents, infliximab and adalimumab, are approved for use in Australia. Administration of these agents during conception and pregnancy are considered to be low risk.[19] High levels of infliximab can be found in infants owing to placental transfer and thus treatments during the third trimester are sometimes withheld due to potential risk. A minimal amount of infliximab is secreted in breastmilk and breastfeeding is not contraindicated, however the data on adalimumab are limited.

Cyclosporine is a Category C drug that can be used as rescue induction therapy for those with fulminant ulcerative colitis. It is not strongly associated with adverse pregnancy outcomes, although breastfeeding is contraindicated owing to high levels present in breastmilk.[20] Methotrexate is contraindicated in pregnancy as it is a proven abortifacient and causes congenital defects.

Requirement for surgery in pregnant IBD patients is similar to non-pregnant patients and usually relates to complications of poorly controlled disease. Indications include bowel perforation, drainage of abscesses, intestinal obstruction and management of enterocutaneous fistulae. There are very limited data examining the role of surgery in pregnancy.

Mode of delivery

The mode of delivery in an IBD patient is based on obstetric considerations. Caesarean section is preferred in those with peri-anal or rectal disease.[21] Similarly, concerns about anal sphincter disturbance with vaginal delivery may prompt caesarean section in patients who already have borderline continence from altered anatomy following ileal-pouch anal anastomoses.[22] Use of episiotomy is minimised, if possible, with the risk of perineal involvement with Crohn's leading to delayed healing.

Liver disease unique to pregnancy

Intrahepatic cholestasis of pregnancy

This disorder occurs in around 0.5–1.5 per cent of European pregnancies (15 per cent in Chilean population) and seems to have some genetic predisposition.[23] Cholestasis typically develops during the second to third trimester, with higher prevalence in twin pregnancy is presumed to be related to the effect of elevated oestrogen levels. The usual manifestations of this disease include pruritus, particularly in soles and palms, along with elevation of serum bile acid and aminotransferases. High maternal bile acid levels are associated with poor fetal outcomes including premature birth, fetal distress and intrauterine death.[24]

Cholestasis commonly resolves after delivery and does not affect the long-term maternal prognosis. Ursodeoxycholic acid is safe and well tolerated for alleviating marked pruritus and biochemical abnormalities.[25] Some obstetric units electively induce labour at around 37 weeks of gestation to prevent fetal death; however, there are no randomised controlled trials to support this intervention and management should be considered on an individual, case-by-case basis.[26]

Pre-eclampsia / HELLP

Pre-eclampsia is a multi-system disorder of unclear aetiology characterised by hypertension, oedema and proteinuria along with transaminitis as a hepatic component of multi-organ dysfunction. The HELLP syndrome (haemolysis, elevated liver enzymes and low platelets) is considered to be part of a spectrum of disease that complicates 20 per cent of severe pre-eclampsia during pregnancy.[27] Presenting in the second and third trimester and sometimes postpartum, patients may develop abdominal pain, nausea and vomiting, occasionally jaundice or may be completely asymptomatic. They may also have other features of pre-eclampsia such as hypertension and proteinuria. Severe cases may progress to develop acute liver failure, hepatic haematoma and rupture along with eclamptic complications.[28] Fetus prematurity and intrauterine growth retardation are common adverse outcomes seen in HELLP. Treatment of pre-eclampsia and HELLP mainly revolves around intensive monitoring and support, including correction of coagulopathy, hypertension management and electrolyte normalisation, until expedited delivery. Risk of recurrent HELLP is increased in subsequent pregnancies; however, long-term complications are generally uncommon.

Acute fatty liver of pregnancy

Acute fatty liver of pregnancy (AFLP) is a rare disorder occurring in late pregnancy with significant maternal and neonatal mortality. Patients present with non-specific symptoms including right upper quadrant pain, nausea, vomiting, jaundice and encephalopathy. They will typically demonstrate transaminitis, hyperbilirubinaemia, leucocytosis and elevated serum creatinine.[29]

Often it may be difficult to distinguish between HELLP, AFLP and other acute liver disorders such as viral hepatitis. AFLP seems to be

associated with more severe hypoglycaemia, hyperuricaemia and coagulopathy or disseminated intravascular coagulopathy (DIC). Delayed management leads to fulminant hepatic failure and life-threatening complications.

An association has been found between AFLP and deficiency in long-chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD).[30] This enzyme is involved in glucose production through fatty acid oxidation during times of fasting. An increased incidence of AFLP is reported in those women who are heterozygous for LCHAD and babies with homozygous LCHAD deficiency. Although the mechanism of AFLP is yet to be elucidated it has been postulated that there is increased fetal fatty acid cross over to the maternal circulation resulting in microvesicular steatosis and liver injury. Nevertheless, neonatal screening for LCHAD deficiency is recommended following development of AFLP. Once again the management of AFLP focuses on expedient timely delivery to minimise long-term maternal and neonatal complications.

Hyperemesis gravidarum

This does not result in any significant hepatic disorder; however, some patients may have non-specific elevation of aminotransferases. Some of these patients may require hydration and electrolytes support. Most conventional anti-emetics – including dopamine agonists (metoclopramide/domperidone), phenothiazines (prochloperazine) and H1 antagonists – are safe to use in pregnancy.

Concurrent or existing liver disease

Viral hepatitis

Acute viral hepatitis can occur at any time during pregnancy. Hepatitis A is the most common pathogen; however, hepatitis B, hepatitis C, Epstein-Barr and cytomegalovirus can also be involved. Of note, herpes simplex virus infections rarely result in fulminant hepatitis. Generally only supportive measures are required as treatment. Acute Hepatitis E, although rarely seen in Australia, can also cause fulminant hepatitis during pregnancy, with a maternal mortality rate of 15–25 per cent as well as resulting in poor obstetric and fetal outcomes.

Chronic hepatitis B patients with highly active disease or advanced liver disease are encouraged to receive anti-viral therapy prior to conception.[31] Until the recent introduction of Tenofovir, there have been no agents with proven safety in pregnancy. Pegylated interferon is contraindicated during conception and pregnancy. Those with mild disease but high viral load can be treated with lamivudine in the third trimester to minimise vertical transmission. Furthermore, hepatitis B vaccination and immunoglobulin should be provided to neonates with an HBsAg-positive mother as these are highly effective ways to reduce vertical transmission. Mode of delivery and breastfeeding do not affect the rate of transmission.

Vertical transmission of chronic hepatitis C occurs in five per cent of those with positive hepatitis C virus (HCV) RNA and is accentuated by high viral load and co-infection with HIV. Currently, there is no proven method of risk reduction other than HCV eradication. Pegylated interferon-based regimens once again are contraindicated in pregnancy. There is no clear evidence-based consensus on the mode of delivery in reducing transmission risk.[32]

Autoimmune / Wilson / Haemochromatosis

Autoimmune liver diseases, including primary biliary cirrhosis and primary sclerosing cholangitis along with metabolic hepatic disorders, often affect patients during childbearing years. Control of disease prior to conception with multidisciplinary management between obstetricians and gastroenterologists is essential in lowering complications during pregnancy.

Biliary disease

Risk of biliary lithogenesis increases during pregnancy and pregnant women may present with biliary and pancreatic complications such as cholecystitis, cholangitis and pancreatitis. Abdomen ultrasound and magnetic resonance cholangio-pancreatography (MRCP) are useful non-invasive diagnostic imaging modalities. Endoscopic retrograde cholangio-pancreatography (ERCP) can be safely performed for extra-hepatic biliary obstructions, provided care is taken to minimise fetal radiation exposure.[33] If necessary, laparoscopic cholecystectomy can be undertaken in the second trimester.

Cirrhosis

Pregnancy in cirrhotic patients is generally considered high risk with significant maternal morbidity and mortality resulting from potential hepatic decompensation as well as gastrointestinal tract bleeding.[34] It is also associated with increase in spontaneous abortions, preterm deliveries and stillbirth. Management of pregnant cirrhotic patients is similar to those non-pregnant patients with optimal medical optimisation. Patients should receive endoscopic variceal surveillance and prophylaxis. Close monitoring of antenatal liver function is recommended. Coagulopathy may need to be corrected prior to labour.

Gastro-oesophageal reflux disease

Reflux in pregnancy

Gastro-oesophageal reflux disease (GORD) is a common disorder in pregnancy and may be present in up to 50 per cent of all pregnancies. There is evidence to suggest that the lower oesophageal sphincter pressure is abnormally low during pregnancy and it has been postulated that this is the result of the hormonal influence of progesterone.[35] Animal studies have shown altered sphincter response to physiological stimuli with relaxation of sphincter tone. Acid reflux symptoms are similar to those in non-pregnant patients, with heartburn, regurgitation and dyspepsia being the most common complaints. Symptoms generally become more prevalent through to the third trimester and generally settle postpartum.[36]

Reflux disease can be reliably diagnosed based on clinical history alone. Endoscopic evaluation is rarely required unless there are severe symptoms not responding to maximal medical therapy. Contrast barium studies should be avoided due to radiation exposure.

Non-pharmacological therapy

Lifestyle and dietary modifications are generally recommended as first-line treatment for reflux disease in pregnancy. This includes elevation of the upper torso during sleep, small frequent meals and avoidance of alcohol and acid-inducing food groups.

Antacids

Over-the-counter antacid formulations are mostly safe and effective in pregnancy. Calcium- and magnesium-based compounds are favoured as there are some data suggesting possible risk reduction of hypertension/pre-eclampsia and eclampsia, respectively. These compounds can result in constipation with long-term use. Excess sodium bicarbonate formulation should not be used as it may lead to metabolic alkalosis.

H2-receptor antagonist

For those with troublesome reflux symptoms, histamine receptor antagonists such as ranitidine and cimetidine can be trialled. Most

of the studies did not find any significant increase of maternal complication or fetal malformations in pregnant women exposed to these agents.[37] The FDA classifies all of them as category B drugs. Use during lactation is acceptable except for nizatidine.

Proton pump inhibitors

Caution should be exercised when using proton pump inhibitors (PPI) in patients with severe dyspeptic and acid reflux symptoms not amenable to above therapy. Omeprazole may have some fetal risks in animal studies; however large observation studies did not conclusively show significant risk of malformation.[38] It is currently assigned class C by the FDA. More data are required to fully assess the safety of other PPI agents and although the overall risk is probably low this class of drugs are only used in carefully selected cases. In light of their low molecular weight properties, high concentration can be found in breastmilk and hence avoidance during lactation is recommended.

Conclusion

Common gastroenterological disorders may be associated with a multitude of pregnancy-related issues and potentially significant adverse pregnancy outcomes. Emphasis placed on prompt recognition and management in a multidisciplinary approach in consultation between obstetric and gastroenterology teams will result in improved maternal and fetal outcomes.

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Antibiotics for caesarean

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Antibiotic prophylaxis for caesarean section: is pre-incision better than post cord clamping?

Rates of caesarean section (CS) are rising in the developed world and Australia is certainly no exception. Rates vary from state to state, but nationally CS now accounts for around 30 per cent of hospital births in Australia.[1] While the reasons for these statistics are numerous and much discussed, CS rates are likely to continue at, or above, current levels.

Of significant concern is the five- to 20-fold increased risk of post-operative infection with CS compared to vaginal birth.[2] Since its initiation by Burke on animal models in the 1960s, antibiotic prophylaxis for CS has become established practice worldwide, and this has been shown unequivocally to reduce the incidence of post-operative infection.[2] However, the timing of prophylactic antibiotics for CS delivery has not yet been standardised across Australian hospitals.

With all surgical procedures there is the potential for post-operative infection. This is particularly the case for CS, due to the direct anatomical connection of the vagina with the operation site, allowing normal vaginal and bowel flora and pathogens to ascend intra- and post-operatively and colonise both the placental site and the wound site.[2] The major infective morbidities associated with caesarean delivery are endometritis and surgical site infections (SSI); rates vary, depending on whether the surgery was scheduled or emergency, but there are also disparities in reporting of rates owing to variations in practice of post-discharge surveillance among institutions.[2,3,4,5] Both SSI and endometritis are associated with considerable morbidity, longer hospital stays and in some cases re-admission. Reducing such post-operative complications following CS is highly desirable, given that women are coping with both the post-operative period and a new baby.

In all other surgical procedures administration of antibiotics prior to skin incision is standard, to ensure blood levels of antibiotic are optimal at the time of surgery, resulting in less exposure to pathogens.[2] Historically, post-cord clamping antibiotic prophylaxis was routine for CS, owing to the perceived risk of fetal exposure that might mask neonatal infections and increase the need for sepsis workup in newborn infants. There was also considered to be the potential for maternal anaphylaxis to the prescribed antibiotic that might compromise the fetus. However, recent overseas studies have established that antibiotic prophylaxis given prior to skin incision has a much lower incidence of SSI following CS, without affecting neonatal outcomes.[2,3,4,5]

A 2008 retrospective cohort study from the USA showed a significant decrease in post-CS SSI (from 6.4 per cent to 2.5 per cent) when hospital policy for antibiotic prophylaxis was changed from post-cord clamping to pre-incision.[6] A large systematic review, published in 2009, reached the same conclusion.[7] A meta-analysis published in 2008 included 15 studies (three of which were randomised controlled trials) and evaluated the timing of antibiotic prophylaxis for CS as well as the type of antibiotic used. This study concluded that cephazolin alone before skin incision was associated with a significant reduction in postpartum endometritis and total infectious morbidities, without affecting neonatal outcome.[5] Cephazolin was the antibiotic used in the three RCTs; it has a half-life of 1.8 hours, and has been shown to be as effective as cefoxitin, but 80 per cent less expensive.[5]

> '... the change in timing of prophylactic antibiotic..resulted in the most significant improvement in health outcomes for women following CS.'

Cairns study

As a medical student I took part in a prospective study at Cairns Base Hospital, Queensland, in 2010–11, assessing rates of SSI following CS (emergency and elective). An initial cohort comprised all women (numbering 203) undergoing CS in the hospital over a three-month period in 2010; hospital protocols at the time included prophylactic antibiotics following cord clamping. Rates of SSI post-CS were found to be 10.8 per cent. Subsequently, protocols were changed to 2g cephazolin given 30 minutes before skin incision. An otherwise identical study was then conducted on all women undergoing CS for a three-month period in 2011; the rate of SSI in this cohort of 181 women was 2.8 per cent. Demographic, medical and obstetric characteristics of the two study populations were similar.

We considered a number of other risk factors noted as significant in other studies including emergency rather than elective CS, prolonged duration of the surgery, use of staples rather than sutures, obesity and diabetes, but found no association with increased rates of SSI. It was the change in timing of prophylactic antibiotic that resulted in the most significant improvement in health outcomes for women following CS. Nevertheless, antibiotics are not a substitute for all other factors that aid in reducing infection levels such as: hair clipping opposed to shaving, effective antisepsis of patient and staff, air ventilation, maintenance of sterile surgical fields and post-operative wound care.[3]

In 2010, following recent study findings, the Society of Obstetricians and Gynaecologists of Canada (SOGC) Infectious Disease Committee made a recommendation that all women undergoing CS should receive a single dose of a first-generation cephalosporin

15–60 minutes before skin incision.[3] Other bodies that have adopted this change include the American College of Obstetrics and Gynaecology (ACOG) and the American Academy of Paediatricians. In December 2011, the National Institute for Health and Clinical Excellence (NICE) followed suit with amendments to its guidelines, now recommending administration of antibiotic prior to skin incision.[2,8]

RANZCOG guidelines

The current RANZCOG statement on antibiotic prophylaxis for caesarean section, C-Gen 17, November 2011, is as follows:

> For caesarean section: there is evidence that antibiotics are beneficial for prophylaxis of wound sepsis as well as endometritis for all caesarean sections, elective or non-elective. Use: cephazolin 1g (adult 80kg or more: 2g) IV.
>
> Administration after the cord is clamped has been common practice to avoid exposing the neonate to antibiotics, and to avoid compromise to the fetus in the event of maternal anaphylaxis. These considerations need to be weighed against lower maternal infection rates if prophylaxis is administered before skin incision.

Given the very considerable weight of evidence now in favour of pre-incision antibiotics, the apparent lack of harm to mothers and neonates, and the changes in national guidelines in the UK and North America, it may be appropriate to consider a more decisive recommendation for Australia and New Zealand.

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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 25-year-old primigravida has asymptomatic bacteriuria found on her booking MSU at ten weeks of pregnancy. What implications does this have for the pregnancy and how is it best managed?*

Dr Morven Crane
DRANZCOG Trainee

a Asymptomatic bacteriuria (ASB) is usually defined as isolation of >105 colony-forming units of a single bacterial species per millilitre of urine, in the absence of urinary symptoms.[1] Pregnant women will certainly benefit from treatment, unlike the general population in whom there is usually no indication to treat. The benefits of treatment during pregnancy have been recognised to such an extent that screening for ASB is found in many countries' guidelines and is considered a standard of antenatal care.[2,3] Estimates of the prevalence of ASB during pregnancy range from two to ten per cent, depending on the population sampled and the inclusion criteria used.[4]

Being sexually active is a recognised risk factor for bacteriuria in young women.[5,6] Other risk factors include low socioeconomic status, anatomical abnormalities of the urinary tract, diabetes, or a personal or family history of recurrent urinary tract infection.[4]

ASB is associated with pregnancy complications. Of women with ASB in early pregnancy, 30 per cent go on to develop pyelonephritis, compared with 1.8 per cent of controls without bacteriuria.[7] Treatment of ASB detected in early pregnancy has been consistently shown to reduce the incidence of pyelonephritis. A recent Cochrane systematic review showed treating women for ASB led to approximately a 75 per cent reduction in the incidence of pyelonephritis during pregnancy. Seven women need to be treated in order to prevent one episode of pyelonephritis.[4]

There is evidence that ASB is associated with low birthweight and preterm birth, if left untreated.[8] The evidence surrounding the effect of treatment is less consistent. If ASB is merely a marker for low socio-economic status, which is also associated with low birthweight, then one would expect there would be no effect of treatment of ASB on low birthweight. A Cochrane systematic review found a statistically significant reduction in the incidence of low birthweight; however, the poor methodological quality of the studies means conclusions for this outcome should be drawn cautiously. The same review included three studies comparing the effect of treatment of ASB on preterm birth with no treatment and found no significant reduction in incidence of preterm birth.[4]

In Australia, screening is usually conducted by sending a urine sample at the booking visit. Internationally, there is controversy about the ideal timing of the screening urine culture. Most studies include screening before 20 weeks gestation, but one study suggested a single culture before 20 weeks may miss more than half of women with ASB.[9] There is also controversy about whether one specimen is adequate to make the diagnosis. Some guidelines suggest sending specimens at two consecutive visits, reserving treatment for women in whom a pathogen is consistently identified.[10] If two consecutively voided cultures yield the same organism, there is a probability of 95 per cent that the woman has true bacteriuria whereas one positive culture gives a specificity of 80 per cent.[11] Sending one specimen for culture provides a practical and effective alternative.

Screening for ASB appears to be cost-effective in most populations. An American analysis concluded that, unless the prevalence of ASB is less than two per cent, screening is cost-effective during pregnancy.[12, 13] After a woman has had one positive urine culture, repeat screening at every visit has been suggested by some clinical guidelines.[2] This is based on management in large multinational clinical trials.[14] The cost-effectiveness of this approach has not been assessed.

A clean catch midstream specimen has traditionally been viewed as the ideal. Some evidence suggests this technique may not reduce the number of contaminated specimens compared with giving no specific instructions to the patient.[15] A culture is the gold standard as it is much more effective in detecting bacteriuria when compared with reagent strip dipstick testing. Reagent strip testing has the advantage of being quick and inexpensive when compared with urine culture. The sensitivity of reagent strip testing ranges from 8.18 to 50.0 per cent, and the specificity has ranged from 47 to 92 per cent. This means the maximum performance using reagent strip testing will be detection of half of the women with ASB.[2] If reagent strip testing is performed, women who test positive should have urine sent for culture and sensitivity testing.

Antibiotics are the mainstay of management of ASB. E. coli accounts for the majority of positive cultures.[4] Empirical treatment is only recommended in the setting of symptomatic urinary tract infection. Culture and sensitivity testing should be used to guide antibiotic selection for eradication of ASB. Antibiotic resistance appears to be increasing, so the use of broad spectrum agents is discouraged. Nitrofurantoin and cephalexin regimens are recommended in the current therapeutic guidelines. The use of amoxicillin alone is recommended if the organism is known to be sensitive. Clavulanic acid should be avoided as it has been associated with an increased incidence of necrotising enterocolitis in neonates whose mothers given antibiotic prophylaxis in the setting of preterm premature rupture of membranes, however, there was no difference at long-term follow up.[16-18]

Due to the heterogeneity in the body of evidence, analysis in relation to the optimal duration of antibiotic therapy has been

difficult. A systematic review that compared single-dose antibiotic treatment with a four- to seven-day course of antibiotic treatment for ASB showed no difference in the prevention of preterm birth or pyelonephritis. Longer duration of treatment, however, was associated with increased reports of adverse effects.[19]

If group B streptococcus (GBS) is found in a woman's booking urine, it is indicated to treat this with antibiotics, both at that time and later intrapartum.[3] This recommendation comes from a retrospective cohort study in which 82 per cent of women with group B streptococcal bacteriuria were treated with intrapartum antibiotics, and no association was found between GBS bacteriuria and an increased risk of early-onset disease. The authors concluded that this represented successful prevention.[20] A much older randomised controlled trial compared the treatment of GBS bacteriuria with penicillin to treatment with placebo. Results indicated a significant reduction in rates of premature rupture of membranes and preterm delivery in the women who received antibiotics.[21]

For this patient, I would recommend and prescribe appropriate antibiotic treatment, based on culture and sensitivity testing. The patient should be counselled about the role of treatment to prevent adverse pregnancy outcomes such as low birthweight and pyelonephritis. It should be emphasised that despite being asymptomatic at the present time, compliance with antibiotic treatment is important. She should also be re-screened once the antibiotic course is complete to ensure adequate treatment has occurred. If the pathogen was GBS, intrapartum antibiotic prophylaxis would be indicated.

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Cancer treatment in Laos

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For a large proportion of the women in the world, current advances in cancer care are of little benefit.

In 2000 there were an estimated ten million new cases of cancer and seven million deaths worldwide, with 53 per cent of the incidence and 56 per cent of the deaths in developing countries. It is projected that, by 2020, the incidence of cancer will increase by a staggering 73 per cent in developing countries. Cancer mortality rates in developing countries are expected to be five times higher than in the developed world.[1] About 80 per cent of cervical cancers occur in developing countries that lack effective screening, detection and treatment programs.[2] It can be misleading to group all developing countries together as development occurs at different rates. Nevertheless, an overview of the issues we see in the country in which I work, Laos, gives some idea of the issues that affect cancer prevention and treatment for women in low-resource settings.

A huge problem in Laos is the lack of health awareness among the population. This is hardly surprising, given the rudimentary education systems and low standards of teacher training. The adult literacy rate for women is said to be around 60 per cent and in a recent study relating to cervical screening in rural areas where about 80 per cent of Lao women live, 11 per cent had no schooling, while 52 per cent had only completed primary school.[2] The study found women's main sources of information on health issues were the television, 47 per cent; healthcare providers, 30 per cent; and friends 23 per cent. An unpublished study by the same authors demonstrated that, among healthcare providers, only 20 per cent had ever advised a woman to seek cervical cancer screening and over one-third believed cervical cancer was untreatable. Of the female providers surveyed, less than one-quarter had ever sought cervical screening. Their level of knowledge regarding reproductive cancers was very poor. For these reasons, most gynaecological cancers are in advanced stages at the time of diagnosis.

Where cancers are diagnosed at an advanced stage, cure rates with the best treatments are low and it is easy for an attitude of hopelessness to develop. Soon after I arrived in Laos I gave a series of talks to workers from rural health centres. Their own education was very poor, with most having no more than a year of formal training and about half of them having only completed primary education. One of the participants said, 'It seems pointless to treat cancer, because all the women I see with cancer die, regardless of treatment.' This is a not unreasonable observation, given the advanced stage of disease. Another important factor is that the families of those treated are considerably poorer after their death, given the cost of healthcare relative to the income of the population.

The cost of healthcare in Laos must take into account more than the fees charged and the cost of diagnostic procedures, drugs and disposables. The cost of transportation to a healthcare facility can be considerable, especially if the visit is to one of the central hospitals in Vientiane. If inpatient care is required, relatives are needed in the hospital at all times to undertake basic nursing care, visit the pharmacy for drugs and buy and give food to the patient. While one relative can usually sleep on the floor of the ward, for a second person the cost of accommodation must be factored in. In a country where 80 per cent of the population survive on US$2 per day, often from a farm or family business, the loss of productivity caused by having the patient and carers off work is a heavy burden.

In the villages, where the majority of Lao live, basic drug kits are available, but there is no trained healthcare provider. There are about 750 health centres where primary care is provided by health professionals with basic training: each health centre serves 1000–7000 people. While access to these centres is relatively easy, the services offered are limited. There are around 150 district hospitals, most served by medical assistants, nurse assistants and sometimes by trained nurses with or without doctors. These personnel are poorly trained, paid and supported in terms of professional development. Provincial hospitals, of which there are 13, have trained nurses and sometimes trained midwives and specialists. The five central hospitals in Vientiane have the highest proportion of staff with specialist training, but their knowledge and skill levels, especially in relation to malignant disease, are very poor.

As in Australia, there is a maldistribution of medical services in favour of the major cities. Pay rates are very low and so doctors run private clinics to supplement their income, often charging high fees for their services. While a great deal of effort is put into improving medical, midwifery, nursing and allied health training at both undergraduate and postgraduate levels, there is a long way to go. There is no reward within the government service for excellence and very little for advanced training. Nevertheless, women often seek care in private clinics because of the convenience of being seen immediately compared to the long waits often experienced in hospitals. It is not uncommon to see women who have postcoital and intermenstrual bleeding arriving with two or three pelvic ultrasounds showing either no abnormality or 'cervical fibroids' when a clinical examination would have revealed a cervical cancer.

Pathology services are rudimentary. Anatomical pathology and cytology are based in Vientiane, posing major problems of access for the rural population. When a specimen is sent, and even in the major teaching hospital it is often not, it is carried by the patient's relatives to the laboratory, the processing fee is paid and the relative told to return on a set day, collect the results and take them to the doctor. The same is true for Pap smears. There are many problems with this system. It is not uncommon for the relatives to discard the specimen owing to a failure to understand the importance of the examination or the cost. Pathology reports are written in either French or English by pathologists, and referring doctors often have no real understanding of the significance of the report, so problems may be overlooked.

A good example of difficulties with the system is provided by a malignant germ cell tumour in a 15-year-old girl that was reported as an immature teratoma, grade 1 on the basis of four blocks from a 20cm tumour. The diagnosis was subsequently found to be incorrect, but the referring doctor mistook 'immature teratoma' for a mature teratoma or dermoid cyst and mistakenly reassured her and her family that all was well when a 10cm pelvic mass was found six months later.

Two young women referred for very advanced recurrent germ cell tumours about to get BEP chemo after reoperation. The younger is 15, the older 18. The father of the older girl will manage the drips after the nurse departs.

It was a further five months before she visited a central hospital with a tumour extending from the pelvis to the xiphisternum. Tumour markers suggested a mixed germ cell malignancy that, fortunately, was resectable with conservation of ovarian tissue and after chemotherapy (paid for by a charitable trust) her hair has regrown and her periods returned with apparent cure.

Many patients choose to travel to neighbouring countries for diagnostic or therapeutic services that are very expensive by Lao standards, but perceived to be of a better standard. Often, patients run out of money before they can get treatment there and return home, poorer but untreated, to seek care locally. Radiotherapy is not available in Laos, so women with advanced or recurrent cervical cancer have to travel out of the country for this treatment. Patients frequently complain of poor communication on the part of the foreign doctors, which is hardly surprising given differences in language. Payment is required prior to treatment and can rapidly exhaust the resources of the family. Furthermore, radiation requires the patient to be near to the treating hospital for about six to eight weeks, imposing a further burden for food, accommodation and lost productivity.

Palliative care is almost unheard of in Laos. The problems experienced by women dying of advanced cancer are rarely seen by doctors because when patients determine that cure is not possible they return home to die – to ensure that their spirits do not wander searching for home and for social and economic reasons. One doctor has been campaigning to have oral morphine preparations made available in addition to the injectable forms, but the government has refused because of a fear of drugs being sold on to addicts, which is a possibility. A woman who has worked tirelessly throughout her life to help provide the best possible future for her family will frequently count her suffering of little account compared to how the cost of her care will affect the family in the longer term.

There are lessons to learn for us all from the issues evident in this country: even Western economies cannot continue to support uncontrolled expenditure on healthcare; and it can be rational for a person to refuse treatment where she perceives that the costs may be greater than the benefits treatment could bring. And, of course, it is important to recognise the great disadvantage suffered by those with poor educational backgrounds and low socioeconomic status. In Australia one thinks particularly of Aboriginal and Torres Strait Islanders with their excessive burden of morbidity and mortality from all diseases, including gynaecological cancer. Overall, Lao women remind me of the amazing resilience of the human spirit in the face of adversity, and the need to strive to respond as positively as possible to the circumstances to provide the best possible care.

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2 Kanovis P. The rising burden of cancer in the developing world. *Annals of Oncology* 2006; 17: (Supplement 8) viii5-viii23.

3 Phongsavan K, Phengsavanh A, Wahlstrom R, Marions L. Women's perception of cervical cancer and its prevention in rural Laos. *Int J Gynecol Cancer* 2010; 20: 821-826.

Let's work together

Dr Andrew Pesce
FRANZCOG

Collaborative Care Agreements, privately practising midwives and obstetricians: where to from here in the Australian maternity system?

It is now some 18 months since the framework setting in place Commonwealth Government support for midwifery care was finalised. Eligible midwives have had access to Medicare Benefits Scheme (MBS) and Pharmaceutical Benefits Scheme funding and professional indemnity insurance cover for care they provide pregnant women since December 2010, potentially increasing access for women seeking care from privately practising midwives (PPMs). Continuity of care by a midwife throughout pregnancy and labour is highly valued by women who receive it, in the same way as women value continuity of care by an obstetrician. It follows that collaborative care from both a midwife and obstetrician known to the woman is likely to provide the 'gold standard' in care when that can be arranged. Such collaboration is evidenced by a Collaborative Care Agreement (CCA), which defines and documents the relationship between two otherwise independent practitioners.

I have been fortunate to have discussed and agreed CCAs with five midwives and have provided care collaboratively for pregnant women with three of them. In all cases, the continuity of both midwifery and obstetric care led to a high level of care and satisfaction for the women giving birth.

However, significant barriers remain, as evidenced by a relatively low level of endorsement of eligible midwives by the Nursing and Midwifery Board and occasions of service. As of April 2012, there were 185 endorsed eligible midwives in Australia, with 157 of those in Queensland. The process of endorsement is slow and cumbersome. A midwife I know with over 25 years of continuous experience across the spectrum of antenatal, intrapartum and postnatal care applied for endorsement and the process took more than ten months to complete. MBS statistics show that although claims were made for 1215 first antenatal attendances and 10 737 subsequent antenatal consultations with eligible midwives from December 2010 to June 2012, only 55 claims were made for management of labour and delivery in the same period. Furthermore, 46 of those claims were from Queensland and the remaining nine were from New South Wales.

PPMs, as yet, do not have any systematic arrangements for credentialing and appointment to maternity units. This is a significant barrier to provision of MBS-funded care for labour and birth, as without it midwives must formally handover care of women in labour to maternity unit staff, and can only subsequently remain present as a support person. Without such credentialing in place, it is difficult for obstetricians to consider collaborative agreements with PPMs. Indeed, the two midwives with whom I have provided care collaboratively for women in labour are employees at the public hospital where the women give birth, allowing them to provide care for those women. However, those women are formally admitted under my care; those midwives are remunerated as hospital employees and therefore cannot access MBS benefits for their care during labour and delivery.

So the lack of a national or even state-wide system of credentialing of PPMs remains an issue to be resolved and, though jurisdictions are working on this, little progress has been seen to date.

It is also disturbing to hear from some PPMs that they have experienced difficulty in discussing CCAs with obstetricians. Often the decline of a request to discuss a CCA is based on lack of credentialing of the PPM at a maternity unit. On occasions the request is declined because a lack of agreement on a model of care (for example, request for collaboration for homebirth where the obstetrician does not wish to incorporate such a model into his/her practice).

However, I am also told that on several occasions the request for discussion of CCA is flatly rejected without any attempt at discussion. Given the low number of eligible midwives who are in a position to discuss CCAs with obstetricians, it is impossible to predict whether such reported reluctance is likely to be a barrier to collaborative care in the future. A recent survey by the National Association of Specialist Obstetricians and Gynaecologists revealed 70 per cent of responding specialist obstetricians revealed they would consider CCAs with midwives if approached. This is a positive sign, but remains to be reflected in practice.

Conversely, I have am aware that some midwives have declared they will never consider discussing CCAs with obstetricians, claiming that this compromises their professional independence. Of course this is true, but no more so than the independence obstetricians and midwives relinquish when they agree to work within policies and procedures of the hospitals in which they provide care for women on a daily basis.

It would be a lost opportunity if the failure of PPMs to discuss and conclude CCAs with obstetricians led to a move away from collaborative care, regardless of which party's reluctance is responsible. Without a doubt, the expectation of many women and midwives that continuity of care from midwives should be facilitated will continue. Continuity of care models are developing in the public maternity system and it is logical that the private sector should provide such a model of care as well. Currently, regulations make provisions for CCAs between midwives and delegated medical practitioners in hospitals, and it is likely that such agreements with institutions will increasingly be used if the lack of individual CCAs remains a barrier to women accessing MBS-funded midwifery care.

CCAs are an opportunity to move away from the 'us versus them' mentality that unfortunately some obstetricians and midwives have held in the past toward a truly collaborative effort in maternity care. It requires goodwill, communication and a focus on the needs of the women for whom we care to convert this opportunity into reality.

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.

Day case hysterectomy

These two small studies report the experience of two centres with experience of same-day hysterectomy services. One study asserts that less than one per cent of hysterectomies performed in Norway result in the patient going home the same day. They report on 150 women who received a vaginal hysterectomy as an outpatient. Their unit provides a protocol including enhanced preoperative assessment and information provision and management of postoperative nausea and pain. Of the 150 women in the study 84 per cent were able to be discharged as planned on the same day as the surgery and 93 per cent of women reported a satisfaction score of above seven on a ten-point visual analogue scale.

The second study reports 21 women were discharged the same day as total laparoscopic hysterectomy bilateral salpingo-oophorectomy and pelvic lymphadenectomy for stage 1 endometrial cancer. They reported no readmissions or significant complications. While both of these studies are quite small, they show that hysterectomy – either vaginally or laparoscopically – is a viable day-surgery procedure for some women. It is notable that both studies were performed in units where outpatient hysterectomy was specifically planned, with the concomitant resources for pre- and post-surgical care.

Rettenmaier MA, Mendivil AA, Brown JV et al. Same-day discharge in clinical stage I endometrial cancer patients treated with total laparoscopic hysterectomy, bilateral salpingo-oophorectomy and bilateral pelvic lymphadenectomy. *Oncology.* 2012, 82: 321-6.
Engh ME, Hauso W. Vaginal hysterectomy, an outpatient procedure. *Acta Obstetricia et Gynecologica Scandinavica*, 2012, doi: 10.1111/j.1600-0412.2012.01502.x.

HRT for menopausal symptoms

At various times over recent decades HRT has been seen as a panacea for the health problems of older women, or a dangerous prescription likely to lead to an early death from cancer. Many doctors will have seen a reduction in demand for HRT following concerns raised by the Women's Health Initiative (WHI) trial, with women fearful of the increased risk of breast cancer. However, in the ensuing years further analysis and new research has enabled doctors to reassess the WHI results.

This brief statement issued by a number of North American societies, including the North American Menopause Society, the American Society for Reproductive Medicine and the Endocrine Society, succinctly concludes that HRT including oestrogen – plus a progestogen if the uterus is present – is recommended for control of menopausal symptoms in women under 59 years of age, or within ten years of menopause. The authors recommend that clinicians individualise their use of HRT, considering issues such as risk of thromboembolic events, cardiovascular disease and breast cancer. They further recommend that HRT is used for the minimum duration needed to relieve menopausal symptoms. They suggest topical vaginal oestrogen as sole treatment of vaginal dryness and dyspareunia are the only symptoms.

Stuenkel CA, Gass LS, Manson JE et al. A decade after the Women's Health Initiative–the experts do agree. *Fertility and Sterility* 2012, 98: 313-314.

Antidepressants in pregnancy

With training in psychology before medical school, I have often been involved in the treatment of depression in women of reproductive age. To make a broad generalisation, many doctors involved in antenatal care seem to place more weight in *primum non nocere* (first do no harm) research and give insufficient weight to the burden of depressive disease in what is, for many women, a vulnerable time. This can result in women not receiving antidepressant treatment during pregnancy.

A US study retrospectively examined the Medicaid records of over 200 000 singleton pregnancies between 1995 and 2007. At the beginning of the pregnancy 23 000 women had prescriptions for antidepressants, but only ten per cent filled prescriptions during the pregnancy. Seventy-five per cent of those women did not fill a prescription in the second or third trimester. The study is limited by the indirect nature of much of the data presented. For example, LMP and gestation at delivery were estimated for 15 per cent of study participants as they were not available from the records examined by the authors. Similarly, there are no data on congenital malformation, a common concern of pregnant women. The presence of depression was determined by either the recording of an ICD depression code in patient records in the 180 days before LMP, or the filling of a prescription for antidepressants before or during the pregnancy. The authors report that antidepressant use in the second trimester significantly increased the risk of shortened gestation by two to six days, while use in the third trimester increased the duration of pregnancy. Selective serotonin reuptake inhibitor (SSRI) use in the third trimester was associated with an increased rate of neonatal convulsions, although this was a rare complication, reported in 119 of over 32 000 women using antidepressants in the pregnancy. These results should be interpreted with caution, given the limitations of the study; in particular, the ascertainment of data regarding the diagnosis of depression from hospital and Medicaid records.

A recent Norwegian study used self-administered questionnaires at 17 and 30 weeks gestation to 63 000 pregnant women. Of these, 699 women reported using antidepressants in pregnancy. Exposure to SSRIs during the first trimester was not associated with increased risk of congenital malformation or cardiovascular malformations, which have previously been linked to SSRI use. They also concluded that antidepressant use during pregnancy did not increase the risk of preterm birth or low birthweight.

The results of these studies leave the obstetrician uncertain what advice to give women considering using antidepressants during pregnancy. The Norwegian study, while smaller, has the benefit of directly gathering information from women during their pregnancy, but this is limited by being a self-report. The US study is larger, but data on filling prescriptions does not necessarily reflect actual medication use. There is a large difference in the rate of reported antidepressant use in the Norwegian and US studies (about one and ten per cent, respectively). Obstetricians will have to be guided by individual cases; however, it may be that antidepressant use is a reasonably safe option for many women, with fewer adverse sequelae than previously reported.

Hayes RM, Wu P, Shelton RC et al. Maternal antidepressant use and adverse outcomes: a cohort study of 228,876 pregnancies. *AJOG* 2012, 207:49.e1-9.
Nordeng H, van Gelder MMHJ, Spigset O et al. Pregnancy outcome after exposure to antidepressants and the role of maternal depression, *Journal of Clinical Psychopharmacology,* 2012, 32: 186-194.

Friendships forged in Fiji

Dr Kenneth Clark
FRANZCOG
Chair, Asia Pacific Committee

Carmel Walker
Senior Coordinator, Asia Pacific Services

Celebrating the advances in professional links in maternal and child health in the Asia Pacific region during 2012.

In June 2012, the planets aligned to bring together, for the first time, representatives of the Asia and Oceania Federation of Obstetrics and Gynaecology (AOFOG), the Pacific Society for Reproductive Health (PSRH), the Fiji National University College of Medicine, Nursing and Health Sciences (FNU CMNHS) and RANZCOG, at a groundbreaking educational and professional forum hosted by RANZCOG, in conjunction with FNU. The Educational Forum and the 2012 AOFOG Council Meeting were held in Lautoka, bringing together more than 150 O and G specialists, midwives, radiologists, pathologists, ultrasonographers, surgeons, nurses and local reproductive healthcare workers from over 20 countries.

The initiative began with an offer from RANZCOG to AOFOG to host the Federation's 2012 Council meeting in Fiji, with a view to introducing AOFOG councillors to health issues facing Pacific women and facilitating links with Pacific colleagues. This allowed for discussions of pathways for interaction between Asian and Pacific colleagues, to complement the work that is currently being undertaken in the Pacific through RANZCOG and PSRH. Key support for expanding the initiative into a week-long educational forum came from a number of partners, including AusAID and UNFPA, the Australian Society for Colposcopy and Cervical Pathology (ASCCP) and Friends of Fiji Health (FOFH) who have undertaken significant support activities for Lautoka Hospital, in particular, in recent years.

The forum started with a one-day seminar titled Evidence-Based Updates in O and G Clinical Practice, on Sunday 24 June, attended by more than 100 participants. The program was developed by Prof Rajat Gyaneshwar from FNU CMNHS, member of the RANZCOG Asia Pacific Committee and chief local organiser. The target audience was O and G postgraduate trainees and current specialists in Fiji and those who had completed their MMed in Fiji to return to their own Pacific Island countries. The seminar and workshop program took advantage of the attendance of a number of high-profile AOFOG representatives with international standing.

Participants of the RANZCOG/PSRH Emergency Obstetrics and Neonatal Care workshop.

RANZCOG/FNU CMNHS Seminar participants.

Fijian O and G Specialists with the President of Fiji.

Secretary General of AOFOG, Prof Sumpaico; President of AOFOG, Prof Yang; President of RANZCOG, Dr Sherwood; and Prof Gyaneshwar.

The Forum continued with four workshops, with RANZCOG faculty, including representatives from AOFOG, PSRH, ASCCP and FOFH. Workshops were conducted in emergency obstetric and neonatal care, colposcopy, laparoscopic surgery and O and G ultrasound.

At the conclusion of the seminar, the inaugural meeting of the Fiji O and G Society (FOGS) was held. The FOGS Constitution was adopted, with Dr Swaran Naidu elected President, Dr James Fong elected Vice-President and Dr Amanda Noovao-Hill elected Secretary/Treasurer, as well as two other general members.

A highlight of the event was the seminar dinner, hosted by FNU CMNHS and RANZCOG. His Excellency the President of Fiji, Ratu Epeli Nailatikau, delivered an impressive address demonstrating his commitment to the wellbeing of the women of Fiji, but more particularly to the involvement of men in a family support role during pregnancy, childbirth and continuing education and the raising of children. President Nailatikau has agreed to be the Patron of FOGS and this level of support is encouraging and exciting for the Society's Board and members. In his address at the seminar dinner, RANZCOG President Dr Rupert Sherwood spoke of the College's commitment to Fiji and the Pacific, and its intention to build on current partnerships across the region and expand existing programs of educational support and opportunity in collaboration with these partners in a multidisciplinary approach to addressing Millennium Development Goals 4 and 5.

The AOFOG President, Prof Yu-Shih Yang, also addressed the dinner, making a number of significant announcements and demonstrating practical support from the AOFOG member societies in Asia to assist with professional development and educational opportunities for O and G specialists in the Pacific region, through both FOGS and the existing Papua New Guinea O and G Society.

A number of the developments and outcomes arising from the Educational Forum will continue to be discussed and progressed. It is expected that the results of these, as well as the evaluation of the workshops in due course, will demonstrate steps forward in developing further avenues for collaboration and support between reproductive health professionals in the region and pathways to effectively strengthen human resource capacity in the Pacific. As always, the ultimate aim is improved healthcare for women and their families in the island nations of the Pacific.

Participants at the RANZCOG/PRSH Emergency Obstetrics and Neonatal Care workshop.

Treating fistula in Uganda

Prof Judith Goh AO
FRANZCOG, CU, PhD

Recently, RANZCOG donated money to fund operations for women with utero-vaginal prolapse and fistulae in Uganda.

Over 90 per cent of maternal deaths worldwide occur in Africa and Asia. There are, unfortunately, many more women suffering from injuries sustained during pregnancy and childbirth. In many rural areas, only five per cent of deliveries are attended by skilled health professionals. For the rest, traditional birth attendants are usually present. Thus, there is no access to emergency obstetric care. Uterovaginal prolapse is also commonly seen in parts of Africa.

Obstetric fistula is the most common genital fistula worldwide. Prolonged, obstructed and neglected labour is the most frequent cause. On average, a woman with an obstetric fistula has laboured for four days and over 90 per cent of babies do not survive this prolonged ordeal.

For about 60 per cent of women with an obstetric fistula, the fistula occurred during the first delivery. These are, therefore, young women and there are significant social consequences with the development of an obstetric fistula. Women with obstetric fistulas are embarrassed by their leakage of urine/faeces. They often face social ostracism.

Successful surgical closure of an obstetric fistula may be achieved in up to 90 per cent of cases in specialised fistula units. The chance of successful surgical closure of the fistula is reduced by significant vaginal scarring and the type of fistula, in particular, the circumferential fistula. Post-operative complications such as ongoing urinary incontinence, pelvic organ/sexual/reproductive and mental health dysfunction may occur following fistula surgery.

Donation by RANZCOG

The SA/NT Regional Committee recommended that a portion of the RANZCOG 2010 ASM monies be allocated to the Health and Development Aid Abroad Australia Fund Inc. (HADA), which supports Medical Training in Africa. The money was used to purchase surgical equipment and support women while they received treatment. It costs about $200 for the total care of a woman, including transportation to and from hospital, all hospital costs and food during her stay. With minimal income, the woman and her family are unable to afford this. As volunteers, we pay for our own travel, accommodation and food and we provide our services free of charge.

Prof Judith Goh with some of the women who have received treatment, thanks to money donated by RANZCOG.

College Statements Update

July 2012

Louise Farrell
FRANZCOG
Chair, Women's Health Committee

The Women's Health Committee (WHC) reviewed the following statements in March and July 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 RANZCOG Council and Board in March and July 2012:

- Attributes of a RANZCOG Fellow (C-Gen 19)
- Credentialing in Obstetrics and Gynaecology (WPI 23)
- Testing of Serum TSH Level in Pregnant Women (C-Obs 46)
- Vasa Praevia (C-Obs 47)
- Semi-retired RANZCOG Fellow (WPI 24)

Revised College Statements

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

- Measurement of Cervical Length in Pregnancy for Prediction of Preterm Birth (C-Obs 27) REWRITE
- Depot Medroxyprogesterone Acetate (C-Gyn 4)
- Prevention Detection and Management of Subgaleal Haemorrhage in the Newborn (C-Obs 28)
- Emergency Contraception (C-Gyn 11)
- Hormone Replacement Therapy Advice (C-Gyn 16) REWRITE
- The Use of Mifepristone for Medical Termination of Pregnancy (C-Gyn 21)

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

- Guidelines for HPV Vaccine (C-Gyn 18)
- Categorisation of Urgency for Caesarean Section (C-Obs 14)
- Responsibility for Neonatal Resuscitation at Birth (C-Obs 32)

New College Statements under development

- Substance Use in Pregnancy
- Combined Hormonal Contraceptives
- Fatigue and the Obstetrician/Gynaecologist

Other news

College statements shall 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 Department

Should you have any queries for the Women's Health Committee or WHS, please use the following contact details:
Ms Shamila Kumar
(t) +61 3 9412 2920
(e) skumar@ranzcog.edu.au.

College website

College statements

Can be viewed at: http://www.ranzcog.edu.au/womens-health/statements-a-guidelines/college-statements.html . If you have any difficulties with the webpage, please contact Shamila Kumar at the College (t) +61 3 9412 2920 (e) skumar@ranzcog.edu.au .

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 Practive Guidelines, National Institute of Clinical Excellence (NICE) Clinical Guidelines and Department of Health and Ageing reports. Access at: http://www.ranzcog.edu.au/members-services/fellows/resources-for-fellows.html .

Have you changed your address or email account recently?

Have you notified the College of these changes?

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

Notice of Deceased Fellows

The College was saddened to learn of the death of the following Fellows:
Dr Kevin Strathmore Malcolm, NSW, December 2010
Prof David Lindsay Healy, Vic, on 24 May 2012
Dr Robert (Bob) Harvey Higham, NSW, on 27 May 2012*
Dr Dorothy (Billie) Grace Greening, NSW, on 10 June 2012
Dr James Sedman Gibson, NSW, on 25 June 2012
Dr David Henry Eizenberg, NSW, on 14 July 2012

An obituary appears on page 70 of this issue of *O&G Magazine*.

Queen's Birthday Honours

Honorary Fellow

AC **Prof Ian Hector Frazer, St Lucia, QLD**
For eminent service to medical research, particularly through leadership roles in the discovery of the human papilloma virus vaccine and its role in preventing cervical cancer, to higher education and as a supporter of charitable organisations.

Fellows

AO **Dr Judith Teng Wah Goh, Gold Coast, QLD**
For distinguished service to gynaecological medicine, particularly in the field of fistula surgery, and to the promotion of the rights of women and children in developing countries.

Dr Keith George Hartman, Mosman, NSW
For service to medicine in the field of obstetrics and gynaecology as a clinician, and through executive and fund-raising roles with the Friends of the Mater Foundation.

AM **Prof Neville Frederick Hacker, Rose Bay, NSW**
For service to medicine in the field of gynaecological oncology as a clinician, researcher and educator, and through contributions to professional organisations on a national and international level.

Prof Robert Siebrand Jansen, Killara, NSW
For service to medical research and education in Australia and internationally as an academic, particularly in the field of human reproductive genetics and in-vitro fertilisation, and as a clinician and author.

Dr Peter William Mourik, Baranduda, Vic
For service to medicine as an obstetrician and gynaecologist, to the promotion of medical services in rural and remote areas, and to education.

OAM **Dr Christopher St John James, Wollongong East, NSW**
For service to medicine in the field of obstetrics and gynaecology, and to medical education.

Staff news

New appointments

Agnes Wilson joined RANZCOG as Guideline Developer/Women's Health Coordinator at the end of June. She has a background in basic research, with a PhD in breast cancer research from Monash University. Following her PhD, Agnes worked for the Victorian State Department of Health, supporting translational cancer research. She has spent the last five years with the National Health and Medical Research Council's National Institute of Clinical Studies as a research scientist. In this role, she supported the translation of research and developed NHMRC clinical practice guidelines and associated clinician, patient and implementation resources.

Jane Cumming started with RANZCOG in May, as the New Zealand Executive Officer. She brings to the position many years of experience working in health-related organisations. Her career to date has included roles with the College of Physiotherapy, the New Zealand Home Health Association and, most recently, a local mental health provider.

Monique Maloney joined RANZCOG in July, as an administrative officer in corporate services. In this role, Monique supports RANZCOG's committees and Council. Previously, she worked in the superannuation industry as a SMSF administrator. She returned to study in 2009, to do a Bachelor of Creative Arts at La Trobe University in Bundoora, which she finished in June this year.

Michelle Holzman joined RANZCOG's Rural Services department as administrative officer for the Specialist Obstetrician Locum Scheme program in July, after temping in the Victorian regional office. Michelle is originally from South Africa, where she worked in advertising and marketing. She completed her postgraduate qualifications in marketing management and is currently studying her Honours in clinical psychology.

Departures

Judy Walker left her role in Rural Services in June to take up a position at the College of GPs. We wish her all the best with her new role.

Deborah James left RANZCOG in June for an opportunity to work in the aged-care sector. We wish her every success.

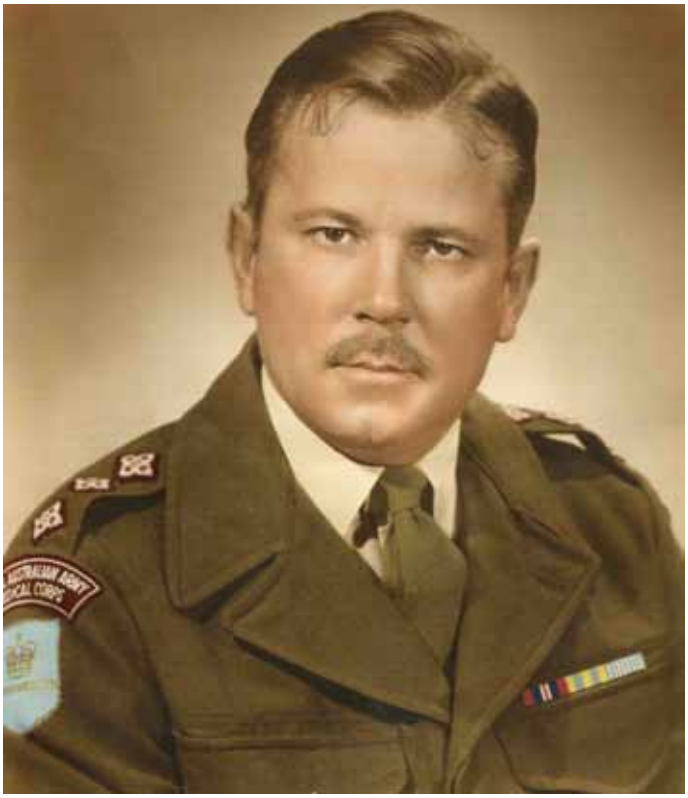
Do you have a RACOG Fellow's gown that you no longer need?

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

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

Any enquiries please contact:
Ros Winspear
Coordinator, Image & Regalia Working Party
ph: +61 3 9412 2934 fax: +61 3 9419 0672 email: rwinspear@ranzcog.edu.au

Obituaries



Dr Robert (Bob) Harvey Higham
1919 – 2012

Dr Robert (Bob) Harvey Higham, affectionately known as 'Digger' owing to his birth on 25 April 1919, died peacefully in Sydney on 27 May 2012.

Robert was born in Waverley Hospital in Sydney and educated at Sydney High School. He was awarded a scholarship to Wesley College at the University of Sydney where he studied medicine, excelling academically as well as in rugby and athletics. During World War II he served in the Sydney University Regiment from 1939–1942 and, in 1940, was attached to the Ninth Field Ambulance.

Two years of residency at Royal North Shore Hospital led to him being appointed Assistant Medical Superintendent at the Royal Hospital for Women, then at Paddington. In 1949, he was appointed Medical Superintendent at Manly Hospital and, in 1952, he became the first Honorary Obstetrician and Gynaecologist appointed at Manly. He held this position for 33 years.

In 1953–54, Bob saw active service as Regimental Medical Officer with the Second Infantry Battalion, Royal Australian Regiment, in Korea. Returning to Manly, he established a busy obstetric and gynaecological practice, and further enhanced his qualifications by travelling to England in 1957, where he gained the Diploma of the RCOG. When the Australian College was founded in 1980, Bob became a Fellow (FRACOG). He continued practising until 1995, delivering more than 10 000 babies, and remained Manly Hospital's Emeritus Consultant until his death. Despite this heavy schedule, he found time for academic pursuits, publishing papers on caesarean section and advanced abdominal pregnancy. He also continued a successful sporting career, excelling at golf, and was for 23 years Honorary Medical Officer to the Manly-Warringah Rugby League team.

Bob will be sadly missed by his two children, Trish Lloyd and Robert Higham, his second wife Trish and extended family.

Prof Caroline de Costa
FRANZCOG
Cairns

Mr Robert Fyfe Zacharin
1925 – 2012

Robert Zacharin was born in Melbourne on 11 April 1925, and educated at Wesley College. He graduated in medicine from the University of Melbourne in 1948, having achieved honours throughout his course, including the exhibitions in both O and G and in surgery in the final year. His postgraduate training included examination success (FRCOG and FRCS, London and FRACS) and extensive surgical training, leading to his appointment as senior consultant gynaecologist at the Alfred Hospital. His subsequent clinical research and scientific publications resulted in awards of postgraduate degrees, Doctor of Medicine and Master of O and G, by the University of Melbourne.

After years of careful dissections, Robert described the intricate anatomy of the female pubourethral ligament and an operation upon it to cure urinary stress incontinence in women. He was the founder of urogynaecological surgery in Australia. He was awarded many overseas lectureships and memberships of the societies of both pelvic surgeons and gynaecologic surgeons of the USA.

Robert made repeated visits to the Addis Ababa Fistula Hospital in Ethiopia, set up by the Hamlins, and became the local expert on surgical management of urinary tract and bowel fistula, the title of his book on this topic being Obstetric Fistula. In 1993, Robert was made an Officer of the Order of Australia for: 'service to obstetrics and gynaecology and to the health and welfare of women in developing countries.'

Robert variously served RANZCOG as a member of Council, Vice President and Chairman of the Board of Management of *ANZJOG*.

Robert was very alert and had an original mind. He was an avid traveller and expert photographer. He combined these talents to observe and marvel at the adaptation of the eucalypt to climate and

conditions in foreign lands. He also had the energy to capitalise on these observations and wrote Emigrant Eucalypts: gum trees as exotics, published by Melbourne University Press in 1978, which sold widely and received a CJ Dennis literary award.

In 1973, Robert purchased 75 acres on the Mornington Peninsula. Apart from cattle farming he created a four-acre plantation of proteas and banksias and he and his wife, Tricia, became commercial flower producers for the following 36 years.

Above all else, Robert was a family man. He died on 9 May 2012, and is survived by Tricia, their four children, 12 grandchildren and three great-grandchildren.

Prof Norman Beischer
FRANZCOG
Melbourne

Correction

O&G Magazine Vol 14 No 2 p78. The archive and editorial teams would like to apologise to the friends and family of Dr John Brian Greenwell. Owing to a transcription error on his Fellowship papers, the obituary that was published mistakenly referred to him as John Bryan Greenwell. Additionally, he was known by his initials 'JB' rather than by his middle name, as was stated.

Seeking O&G Magazine Peer Reviewers

Are you interested in being listed as a potential peer reviewer for *O&G Magazine*? We are planning to initiate a blinded peer-review process for selected clinical articles.

If you would like to be considered for the peer-review database, please send the following information to lwesthaven@ranzcog.edu.au:

- Qualifications
- Areas of special interest

We are looking for people who can review articles in a two-week timeframe.

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Membership of the RANZCOG Research Foundation is open to all members of the Royal Australian and New Zealand College of Obstetricians and Gynaecologists and to all others with an interest in the aims and objectives of the Foundation.

By joining the RANZCOG Research Foundation you are directly contributing to the internationally recognised research conducted in Australia and New Zealand.

Membership of the RANZCOG Research Foundation is free to all RANZCOG Fellows residing in Australia or New Zealand. Fellows wishing to accept membership of the Foundation should advise the RANZCOG Research Foundation Coordinator in writing.

RANZCOG trainees, clinical researchers and other interested individuals are warmly welcomed. Membership fees are tax deductible.

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The RANZCOG Research Foundation works closely with the RANZCOG Board, Council and College Committees to further the needs for research and research training in the broad fields of obstetrics, gynaecology, women's health and the reproductive sciences.

Our Scholars

The Foundation proudly supports promising young Fellows, clinical researchers and scientists undertaking high quality, innovative research and research training at an early stage in their career.

Scholars supported by the RANZCOG Research Foundation have a strong record of subsequent achievement in research and in academic careers in Australia and overseas.

Grants and Scholarships

Each year, approximately $120,000 is disbursed helping to support early career researchers in their work.

The Foundation continues to expand its program of grants, scholarships and other awards. Recent initiatives include Collaborative Bachelor of Medical Science Research Scholarships, Project Grants to assist RANZCOG trainees in undertaking their research project and the *Mary Elizabeth Courier Research Scholarship*, introduced following a bequest to the Foundation by her late husband, Australian lithographic artist, Jack Courier.

Further Information

Further information about the work of the Foundation is available on the website at: www.ranzcog.edu.au/research

Any questions should be directed to the RANZCOG Research Foundation Coordinator:

Ms Georgina Anderson:
t: +61 3 9417 1699
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