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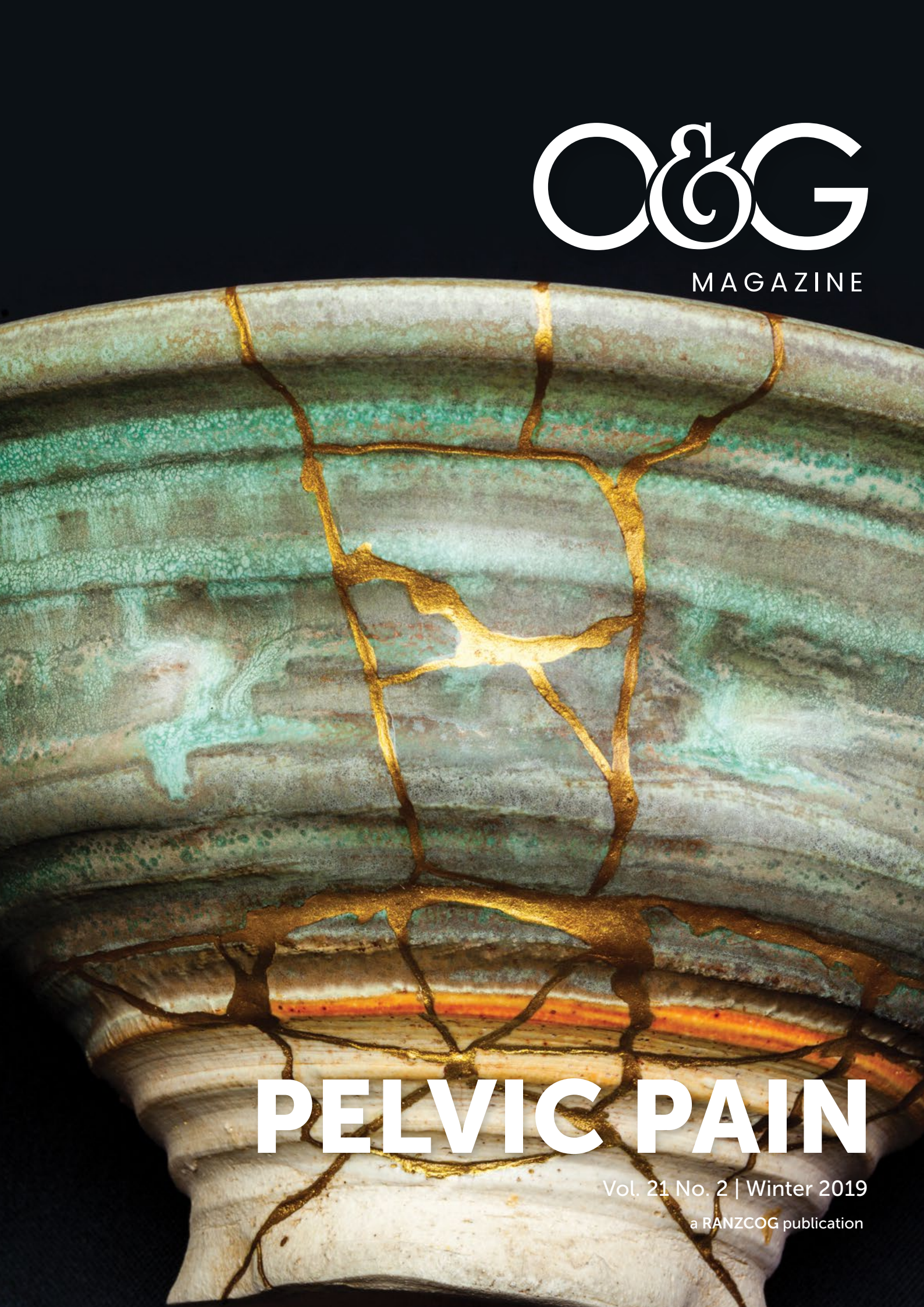

PELVIC PAIN

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The College

Pelvic Pain

Women's health

The College

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From the President

Dr Vijay Roach
President

This issue of *O&G Magazine* discusses the clinical enigma that is endometriosis. In the 13th century, pain during menses was described as 'strangulation or suffocation of the womb'.[1] Like many perceptions about women's illnesses, the pain and suffering was attributed to a woman's failure to fulfil her biological destiny, that is, become pregnant and bear children. It's interesting, and important, to reflect on the impact of social mores and expectations and how they influence doctors and scientists and our assessment and management of patients. In far more modern parlance, our authors in *O&G Magazine* discuss the pathophysiology, clinical assessment, investigation and management of this complex disease. RANZCOG is taking a leading role in Australia, having received a Commonwealth grant to develop country-specific guidelines for endometriosis management. New Zealand has recently released their guidelines, and this will provide a useful reference point. I am pleased that internationally recognised endometriosis expert, Prof Jason Abbott, has agreed to Chair the committee.

Your RANZCOG Board will be attending the New Zealand ASM in Hamilton in May. We're looking forward to spending time with our colleagues in New Zealand, reflecting on the events in Christchurch and learning from an interesting and varied scientific program. We will be joined by newly appointed RANZCOG Board observer, Julie Hamblin. Julie is a lawyer with considerable expertise in the corporate sector and was the consumer representative on the Obstetrics and Gynaecology MBS Review Committees. I am sure that she will bring a fresh and insightful perspective to our discussions.

Your RANZCOG CEO, Vase Jovanoska, has already made a very positive impact at the College. She's hit the ground running and the consistent feedback from staff and RANZCOG members is that Vase is willing to listen. Vase has made a huge effort to spend time with each Board member and has sought advice and feedback from staff and members. I feel very confident that, in Vase, we have strong, competent, respectful and reflective leadership.

One of the special duties of the President is to attend international scientific meetings and I am writing this on the long flight home from Nashville, Tennessee, having attended the 2019 ACOG ASM. The conference dinner included line dancing at the Wild Horse Saloon, but I also concentrated on meeting former ACOG Presidents, Mark De Francesca (Honorary FRANZCOG) and Tom Gelhaus (to receive Honorary FRANZCOG at the Melbourne ASM), outgoing President Lisa Hollier, incoming President Ted L Anderson and President-elect Lisa Chalas. The Americans were, as always, warm and welcoming and there were many insights to be learned discussing their governance and processes. I attended the Fellowship Ceremony and was proud to hear the citations for RANZCOG Immediate Past-President Prof Steve Robson, who received the prestigious ACOG Distinguished Service Award, and Prof Suzanne Garland, from the Royal Women's Hospital Melbourne, who received an ACOG Honorary Fellowship for her pioneering work in human papilloma virus research.

After too many years of inertia, the College announced our Gender Equity and Diversity Working Group, led by Chair, Dr Gillian Gibson. Recognising the need for broader representation at a leadership level at RANZCOG, we have set ourselves some ambitious targets. At the time of writing, we have sought feedback from the RANZCOG membership that will be collated and discussed. With awareness of our lack of experience in this area, we have sought advice, and RANZCOG is the first medical college to engage with the Workplace Gender Equality Agency (WGEA). I am particularly grateful to Libby Lyons and Kate Lee from WGEA for their support and sage advice.

In honour of International Day of the Midwife, 5 May, a message from RANZCOG to the Australian College of Midwives and New Zealand College of Midwives was warmly received. The College is progressing a Joint Committee to bring together the many entities that contribute to maternity care in Australia and New Zealand. Collaboration and our common purpose will underpin the work of this multidisciplinary group.

Finally, in line with the Board's determination to proactively address issues relevant to the College, we want your feedback and suggestions. We want to know what you think is important and we invite constructive criticism. The world is changing and expectations of communication from the College are higher than ever before. We recognise this and are endeavouring to do more to keep you informed through various media. RANZCOG is *your* College.

Reference

1. C Nezhat, F Nezhat, C Nezhat. Endometriosis: ancient disease, ancient treatments. Fertil Steril. 2012;98(6 Suppl):S1-62. https://doi.org/10.1016/j.fertnstert.2012.08.001.

From the CEO

Vase Jovanoska
Chief Executive Officer

I am delighted to contribute to what is my first CEO report for *O&G Magazine* and I am equally excited about what the future holds for RANZCOG. I would like to take this opportunity to thank the President, the Board, our members and College staff who have given me a warm welcome. It is an honour and a privilege to be part of the College and to be working together in delivery of excellence and equity in women's health.

Strategic Plan

The College's Strategic Plan 2019–2022 was launched in March this year, defining our strategic priorities. I believe that through stronger engagement with members via better support, advocacy and education provisions; the development of an agile, sustainable and innovative organisation, and the development and maintenance of productive and mutually beneficial relationships, together we will create meaningful outcomes for the benefit of women's health across Australia, New Zealand and the Pacific region.

Respectful Workplaces and Thrive Workshops

The College's Training Support Unit has been instrumental in setting up the Supporting Respectful Workplaces workshops, which aim to tackle the issues of workplace bullying and harassment faced by many consultants and trainees. There has been an overwhelming response to the workshops, with positive feedback from participants about the interactivity, openness of conversation and insight from both trainees and senior colleagues that has been enlightening.

Thrive workshops that were created to promote the wellbeing and support of doctors, especially those in their first years of medicine, are designed using research evidence, including cognitive and positive psychology, and in consultation with our trainee cohort to provide support and skills to trainees, provide practical techniques and strategies to improve their personal wellbeing and workplace performance.

The feedback received on the workshops has been of high praise as being practical and relevant and I am pleased that we have created a positive learning environment with both workshops for trainees and Fellows alike.

Endometriosis Project

As reported in previous issues of *O&G Magazine*, the College was awarded a contract for services by the Department of Health (DoH) to develop an Australian Clinical Practice Guideline for the Diagnosis and Management Of Endometriosis, in line with the National Action Plan for Endometriosis 2018.

Endometriosis affects one in every 10 Australian women, with the average diagnosis taking between seven to ten years.[1] Currently, there are no national evidence-based clinical practice guidelines for the diagnosis and management of endometriosis for use in Australia.

RANZCOG's first Endometriosis Expert Working Group meeting is scheduled to be held in July and we will be required to submit periodic progress reports to DoH. A Project Plan had been devised by RANZCOG in consultation with the guideline methodologists and had been approved by the DoH. The first progress report is due on 30 August and the final version of the Guideline is to be delivered to the Department by February 2020.

International Women's Day

In March, RANZCOG hosted an event at College House in celebration of International Women's Day. Stephanie Woollard of Seven Women was our guest speaker and told a fascinating story of how her organisation started and the hurdles she faced along the way. We also had the opportunity to ask questions of three very different and intriguing female doctors at different stages in their RANZCOG journey: Dr Bernadette White, Dr Amber Moore and Dr Manarangi De Silva.

Staff were very appreciative and inspired by this event as it gave them the chance to pause and connect with co-workers and our members on a different level from day-to-day work interactions. It was an opportunity to celebrate the accomplishments of successful women and recognise the opportunities that are available to us all.

DRANZCOG Advanced Oral Examination April 2019

The Advanced Diploma of RANZCOG is a hospital-based training program intended for medical practitioners who have gained skills in obstetrics through the Diploma and who wish to develop them to a level that will enable them to safely undertake complex deliveries and perform more advanced gynaecological procedures. Following a successful pilot conducted during 2018, Adelaide Health Simulation Centre at the University of Adelaide has been adopted as the venue for delivery of this exam.

Adelaide Health Simulation is a world-class, purpose-built, clinical education, training and assessment centre. Some of the benefits of the centre include integrated IT systems that allow examination results to be recorded electronically in real time, video and audio recording, access to modern medical and simulation equipment and high-fidelity medical suites and observation rooms.

The Advanced RANZCOG Oral Examination was held on Sunday 7 April 2019. The examination followed a structured oral examination format, including 10 cases from the areas of obstetrics and gynaecology.

The feedback received from examiners and candidates on the conduct of this exam has been positive. Both examiners and candidates felt the examination was delivered professionally, was very well organised and they felt supported by RANZCOG and Adelaide Health Simulation staff.

Finally, on behalf of the College, I would like to extend our deepest sympathies to our members in New Zealand, and all of those affected by the tragic events of March 15 in Christchurch. We are united in our grief and stand with you in solidarity.

Reference

1. The Department of Health. National Action Plan for Endometriosis. Commonwealth of Australia. Available from: www.health.gov.au/internet/main/publishing.nsf/Content/endometriosis.

LEADERS IN FOCUS

Dr Kirsten Connan
MBBS(Hons), FRANZCOG, DDU
MMedEd (Gender and Leadership)

This feature sees Dr Kirsten Connan in conversation with RANZCOG members in a broad range of leadership positions. We hope you find this an interesting and inspiring read.

Join the conversation on Twitter
#CelebratingLeadership @RANZCOG @connankf

Dr Gillian Gibson FRANZCOG

Dr Gillian Gibson is a RANZCOG Fellow in New Zealand. She holds the Clinical Director role of the Auckland District Health Board, is a RANZCOG federal Board member (currently the only female elected Board member), is the past president of the NZ Society of Gynaecology, holds a board position on two women's health related trusts, and is the director and clinician of a private practice.

Dr Gibson has held a VMO position at National Women's Health since 1997, is the clinical lead for the Pregnancy Termination Service, and she holds an honorary lecturer post at the University of Auckland.

Why did you choose O&G as your career pathway?
I developed an interest in O&G as a clinical undergraduate. Our clinical teachers at Wellington Women's Hospital were passionate and took a keen interest in students. I was inspired by a senior trainee who had returned from training in the UK. She was heavily pregnant in her final six months of training and I admired her capability. She was the first woman O&G role model I clearly identified with.

What are the highlights of your professional career?
Being part of the team that pioneered the first group O&G practice in New Zealand.

Holding the Chair position for the RANZCOG Organising Committee ASM in Auckland 2017. This ASM was a resounding success and was able to attract the largest attendance at a RANZCOG-associated conference in New Zealand.

Holding a professional career while raising our daughter who recently graduated with a MBChB (all credit to my husband who maintained our solid home base).

Would you describe yourself as a leader and why?
I would not say I am a natural leader, but I am privileged to hold positions that influence people and policy.

How did your leadership journey occur?
I have not sought leadership roles; they seem to have found me!

I have not been mentored into leadership roles, but have been 'shoulder tapped' for many. These 'shoulder tap' nominations have provided me with both a sense of obligation as well as the confidence to take on roles. I have not had any formal leadership training, although acknowledge this would be valuable. Media training has been provided for my leadership roles with the NZ regional Committee, my Service Clinical Director role, and as a RANZCOG Board member.

What do you feel are essential characteristics as a leader?
Be available, responsive and fair. Good relationships and networks are key.

What do you feel have been the biggest challenges/barriers to your leadership journey?
Time and resources! Clinical work has much more clearly defined boundaries in comparison.

What future leadership goals do you have?
I hope to have a further term on RANZCOG Board. I am fortunate that most of the incumbent Board members have been re-elected, so I have a wonderful opportunity to 'learn the ropes' from their institutional knowledge and experience.

What have been the biggest challenges/ vulnerabilities during your career?
Combining parenting with a career. Lost opportunities due to work commitments, mainly long hours – less time to spend with wider family/whānau, ageing parents, friendships.

Strategies for coping include outstanding support, advice and wisdom of my spouse. My exercise routine that must not be compromised. Delegating is not an easy skill, but a good one to acquire, and I rarely have my computer on after 8pm for sleep hygiene reasons!

What does being a RANZCOG board member entail?
RANZCOG is governed by a seven-member Board who are the directors of the College and we meet face to face six times during the year.

The Board manages the financial, legal and business operations of the organisation and is supported in its activities by the Council and a range of committees. The timetable during Council week (held three times a year) is quite busy, but does achieve results. Support from College staff is outstanding, with a significant amount of work undertaken by teleconference and email.

'For me a highlight is to attend the RANZCOG Annual Scientific Meeting and to be part of the new Fellows ceremony.'

My responsibilities include ex-officio membership of RANZCOG NZ Committee Executive, with quarterly meetings in Wellington, weekly teleconference, and contributing responses to submissions and policy development at a national level.

You are the only woman on the RANZCOG Board this term, how does this influence your role?
I am Chair of the Gender Equity Working Group. My responsibility is to represent all members, but I am motivated to develop policy that will maximise opportunities within the College for women who are well qualified to contribute, but for whom there may be barriers.

There are many men and women who have contributed enormously to College work at the Council and Board level. My time as Chair of the New Zealand Regional Committee in 2006–2008 overlapped with Dr Christine Tippett's tenure as RANZCOG President, an inspirational woman with huge institutional knowledge.

What role has the College played in your career?
Networks, friendships, collegiality, career development, job satisfaction and multiple trips to Melbourne! As a generalist O&G, College work has provided the opportunity for professional development.

What role do you feel RANZCOG should play with regards to leadership training?
There is no formal leadership program, but this is an area ripe for career development.

Dr Gillian Gibson.

What role do you feel RANZCOG should play with regards to Indigenous health?
We have a responsibility to address health inequities by contributing to policy development, interaction with government and other agencies. The New Zealand Committee has created a Māori women's health committee, He Hone Wāhine, that has the aim of addressing inequities in women's healthcare.

Do you see yourself as a feminist?
I'm proud that New Zealand was the first nation to give women the vote! I believe women can achieve all the same goals as men, if given the opportunity. The women in our communities need to be in control of their sexual and reproductive health. The College has a responsibility to make this a priority to achieve excellence in women's healthcare.

How do you feel about the gender imbalance within O&G leadership and O&G trainee posts?
I believe it is more likely trainees and younger Fellows will pursue a role at the College if women in leadership positions becomes the norm. The pipeline effect of sheer numbers coming through cannot be relied upon entirely, and specific policy for gender equity is needed as well. There are examples of medical colleges creating gender-based targets for leadership positions to change the imbalance. In future, this includes ensuring there are sufficient men becoming members and having leadership roles.

How do you balance your personal and professional life?
I belong to a book club and manage to read at least 20 books a year! I try to enforce boundaries with respect to exercise, taking holidays, getting out of town and leaving all work responsibilities behind. Good cover is needed if you are in private practice. I recommend group obstetric practice to achieve a balance.

What advice would you give to a trainee starting out at the beginning of their career?
Get through training as quickly and efficiently as possible, then if you plan to have a family, get on with it. Be reassured the career opportunities and earning potential will be there for many years to come.

If you could do things differently in your career, what would it be?
Completing a BSc(hons) added three years to achieving my goal of entry to medical school. It caused, in part, the delay for starting a family and ultimately secondary infertility.

Do you have any regrets?
I have a deep regret that I put my career ahead of having more children.

Have you seen workplace culture change during your career?
Our Otago University medical school class in 1983 was the first with 50 per cent women intake. As a medical student, the concept of informed consent was not very evident. After Cartwright (cervical cancer enquiry 1987–88) significant changes were introduced to protect patient rights.

What role do you see for the College in the future?
We are the peak body and authority for women's healthcare in Australia and New Zealand. I envisage a more outward-facing College that makes it easy for women and families to get information that affects their health. This role will be easier if our Council and Board membership reflects the diversity of the communities we care for.

What three words best describe your life?
Demanding, varied, rewarding.

What would you tell your younger self if you had the chance to go back in time?
Sooner or later an adverse event will occur during your career despite your best efforts. Seek out help and support early.

Are you willing to be contacted by trainees for career advice/mentoring, and what would you bring to that relationship?
I would welcome contact from trainees. I have over 20 years of perspective and experience looking after women in private and public practice, as well as familiarity with College people and processes.

Editorial

Dr Marilla Druitt
MBBS, BMedSc, FRANZCOG
University Hospital Geelong

In 2019 the world of pain is full of innovative researchers, shifting paradigms of understanding and great hope. We have gathered together authors from around Australia and New Zealand to make you think, make you want to know more and look up all the references!

Momentum is building for pain management and endometriosis research in the political arena, multidisciplinary pain clinics are being created and a new language is filtering out.

We have articles from gynaecologists, anaesthetists, psychologists, physiotherapists, scientists, educators and lobbyists. We have controversies, and nothing makes authors happier than people who write letters to discuss and engage in the conversation.

Explain pain

Who would have thought that explaining pain to patients can treat it? Language matters – I challenge you all to convey the new neuroscience knowledge and be pain educators in your interactions with patients. Women need to be listened to and understood; as does any patient seeking care. Talking with them is treatment. Explain that pain is multifactorial and we have no way of knowing what proportion of their pain is due to endometriosis – a muscle response, contributions from visceral hypersensitivity, other pain states (pain begets pain) or sleep and mood disorders – so everything matters; you can't have pain if you don't have a brain. No fancy business required; the best tool is an A4 piece of paper to draw the brain, spinal cord, organs and body and use arrows to describe options to treat each part of the complex human.

Controversies

Do we know if it matters that someone has endometriosis if their pain is resolved with medical treatment? Does it matter that it took seven years to diagnose the histology? Injustice is the new catastrophising – and a sense of injustice, especially in the compensation arena is a poor prognostic sign for pain chronicity. We need to treat pain, but if they are all better, what does histology add?

Gather your team and shift the curve

As Geoffrey Rose suggested in the 1960s, targeting the extreme of the spectrum will not have as much impact on a population as shifting the curve to the left (classically seen with smoking and heart disease). If we are all better at treating pain, a little could go a long way. Gather your team – if not in a co-located fashion, in a virtual one – and endeavour to be on message as a group. Know your resources for patients and know how they learn best (watching, reading, listening). Further your learning – here are some suggestions below.

Enough evangelising – on with the show!

Further reading

Pelvic Pain Foundation Australia: www.pelvicpain.org.au

Endometriosis Australia: www.endometriosisaustralia.org/

Endometriosis New Zealand: https://nzendo.org.nz/

New Zealand Pain Society: www.nzps.org.nz

West Australian Pain Stories: https://painhealth.csse.uwa.edu.au/

International Pelvic Pain Society: www.pelvicpain.org/

Brainman on YouTube: www.youtube.com/user/HunterBrainman

TARPS (The Auckland Regional Pain Service): www.healthpoint.co.nz/public/pain-management/tarps-the-auckland-regional-pain-service/

Counties Manukau Health Chronic Pain Service: www.healthpoint.co.nz/public/pain-management/counties-manukau-health-chronic-pain-service/

Explain Pain resources:

https://noijam.com/

https://bodyinmind.org

https://painrevolution.org

Unlocking the aetiology of endometriosis

Prof Grant W Montgomery
Professorial Research Fellow
Institute for Molecular Bioscience,
The University of Queensland, Brisbane

The underlying causes for endometriosis remain enigmatic. The disease is defined by the presence of lesions containing tissue resembling endometrium at sites outside the uterus.[1,2] Lesions mainly occur in the peritoneal cavity, but can also occur in other sites within the body. Definitive diagnosis requires the observation of lesions at the time of surgery with histological confirmation of ectopic endometrial glands and stroma.[1,2] To complicate matters, there are different types of lesions and different combinations of lesion types in individual patients. Women present with symptoms of pelvic pain and infertility that overlap several other conditions and there is a poor correlation between disease symptoms, lesion type and disease severity.

Explanations for the aetiology of endometriosis must account for the origin(s) of cells responsible for initiation of lesions, the range in type and location of lesions and variable presentation of the disease between patients. Several theories have been proposed including (i) deposition of viable endometrial cells into the peritoneum through retrograde menstruation followed by adhesion and initiation of lesions (ii) activation of misplaced endometrial cells left behind from unusual differentiation and migration of Müllerian ducts during development and (iii) metaplasia or transformation of one differentiated cell type into another, for example, transformation of the coelomic epithelium covering the ovary.[3,4] No one theory provides an adequate explanation for all presentations of the disease. For example, some cases of endometriosis are seen in young women before the onset of puberty. The theory of retrograde menstruation could be extended to include the retrograde transport of endometrial stem or progenitor cells increasing the chance of lesion formation, and/or activation of stem or progenitor cells shed at the time of neonatal retrograde bleeding in a proportion of babies.[3,4] However, these explanations do not explain the rare occurrence of endometriosis in males. The theories are not mutually exclusive and it is likely some cases of endometriosis arise from different causes.

The disease is complex – influenced by both genetic and environmental factors – with an estimated heritability of approximated 50 per cent.[5,6] In the last 10 years, genome-wide association studies (GWAS) have transformed our understanding of genetic contributions to complex diseases.[7] Several GWAS for endometriosis have been published describing genetic risk factors and providing insight into the genetic architecture of the disease. The most recent published study[8] included data from 11 case-controlled data sets (17 045 endometriosis cases and 191 596 controls). Results replicated nine previously reported genomic regions and identified five novel regions significantly associated with endometriosis risk. Follow-up studies have identified likely target genes in four of the 14 genomic regions associated with endometriosis.[9-12] Initial results suggest genetic effects mostly influence genes associated with cell proliferation, cell adhesion and angiogenesis. However, extensive functional studies will be required to confirm the specific genes and functional changes increasing endometriosis risk.

The results show genetic risk is most likely due to a large number of common variants, each with small effects.[8] There is little evidence for protein-modifying variants with moderate or large effect sizes associated with endometriosis.[13] The GWAS results include women with surgically diagnosed and self-report of disease and include women with a range of different lesions types. Overall, GWAS results show good consistency between and within studies suggesting genetic risk factors are similar for women with different lesion types. However, confirmation of this conclusion must wait a formal analysis of genetic risk factors in women with different combinations of lesions types.

Environmental factors contributing to endometriosis risk include an inverse association between body mass index and endometriosis, specific dietary factors and possibly exposure to endocrine disrupting chemicals.[2] Specific factors have been hard to define since environmental risk factors may operate at any time during development. Measurements at the time of diagnosis are unlikely to reflect exposures during early stages of development and questionnaire studies are subject to recall bias.

Environmental risk estimated from twin studies is the variation remaining after estimating the contribution of genetic risk factors. This represents approximately 50 per cent of risk for endometriosis.[6] This can include exposures to specific environmental insults or other non-genetic risk factors. Recent studies in endometriosis have highlighted the high burden of somatic mutations in endometriosis lesions.[14-16] Somatic mutations were identified in 79 per cent

of deep infiltrating endometriosis lesions, with 21 per cent of lesions harbouring known somatic cancer driver mutations.[14] The somatic mutations are DNA changes in a small number of specific cells, distinct from genetic risk factors and part of the 'environmental' or non-genetic risk. They may result from specific environmental exposures (like the role of sun exposure causing DNA damage in skin and increasing the risk for melanoma and non-melanoma skin cancers) or from errors in DNA replication with constant tissue regeneration in the endometrium at each menstrual cycle and high proliferation rates in specific cell types.

We have not yet solved the riddle of endometriosis, but recent discoveries from genetics and genomics provide important clues about the origins of the disease and new avenues for study. The discovery that the same somatic mutations seen in lesions are already found in cells in endometrial glands within the endometrium gives strong support to the view that endometrial tissue transported to the peritoneum by retrograde menstruation is an important source of cells initiating endometriosis lesions.[16] Genetic risk factors and somatic mutations may both contribute to survival of endometrial cells during this process and subsequent initiation and progression of lesions. Future work should define the target genes and functional consequences of known genetic risk factors and overlap with the spectrum of somatic mutations in both lesions and eutopic endometrium. Armed with knowledge of fundamental genomic changes responsible for disease initiation and progression, we will be able to develop better methods of diagnosis and novel and more personalised treatments.

References

1. LC Giudice. Clinical practice. Endometriosis. *The New England Journal of Medicine*. 2010;362(25):2389-98.
2. KT Zondervan, CM Becker, K Koga, et al. Endometriosis. *Nat Rev Dis Primers*. 2018;4(1):9.
3. S Gordts, P Koninckx, I Brosens. Pathogenesis of deep endometriosis. *Fertil Steril*. 2017;108(6):872-85.
4. P Vercellini, P Vigano, E Somigliana, L Fedele. Endometriosis: pathogenesis and treatment. *Nat Rev Endocrinol*. 2014;10(5):261-75.
5. R Saha, HJ Pettersson, P Svedberg, et al. Heritability of endometriosis. *Fertil Steril*. 2015;104(4):947-52.
6. SA Treloar, DT O'Connor, VM O'Connor, NG Martin. Genetic influences on endometriosis in an Australian twin sample. *Fertil Steril*. 1999;71(4):701-10.
7. PM Visscher, NR Wray, Q Zhang, et al. 10 Years of GWAS Discovery: Biology, Function, and Translation. *American Journal of Human Genetics*. 2017;101(1):5-22.
8. Y Sapkota, V Steinthorsdottir, AP Morris, et al. Meta-analysis identifies five novel loci associated with endometriosis highlighting key genes involved in hormone metabolism. *Nat Commun*. 2017;8:15539.
9. JN Fung, SJ Holdsworth-Carson, Y Sapkota, et al. Functional evaluation of genetic variants associated with endometriosis near GREB1. *Hum Reprod*. 2015;30(5):1263-75.
10. SJ Holdsworth-Carson, JN Fung, HT Luong, et al. Endometrial vezatin and its association with endometriosis risk. *Hum Reprod*. 2016;31(5):999-1013.
11. H Nakaoka, A Gurumurthy, T Hayano, et al. Allelic Imbalance in Regulation of ANRIL through Chromatin Interaction at 9p21 Endometriosis Risk Locus. *PLoS Genet*. 2016;12(4):e1005893.
12. JE Powell, JN Fung, K Shakhbazov, et al. Endometriosis risk alleles at 1p36.12 act through inverse regulation of CDC42 and LINC00339. *Hum Mol Genet*. 2016;25(22):5046-58.
13. Y Sapkota, I Vivo, V Steinthorsdottir, et al. Analysis of potential protein-modifying variants in 9000 endometriosis patients and 150000 controls of European ancestry. *Sci Rep*. 2017;7(1):11380.
14. MS Anglesio, N Papadopoulos, A Ayhan, et al. Cancer-Associated Mutations in Endometriosis without Cancer. *The New England Journal of Medicine*. 2017;376(19):1835-48.
15. M Noe, A Ayhan, TL Wang, IM Shih. Independent development of endometrial epithelium and stroma within the same endometriosis. *J Pathol*. 2018;245(3):265-9.
16. K Suda, H Nakaoka, K Yoshihara, et al. Clonal Expansion and Diversification of Cancer-Associated Mutations in Endometriosis and Normal Endometrium. *Cell Rep*. 2018;24(7):1777-89.

What else could it be? Causes of pelvic pain

Dr Sumi Saha
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Persistent pelvic pain (PPP) has been defined as noncyclical pain of six or more months duration that localises to the anatomic pelvis, anterior abdominal wall at or below the umbilicus, the lumbosacral back or the buttocks, and is of sufficient severity to cause functional disability or lead to medical care.[1] The prevalence of PPP is estimated to be 15–27 per cent.[2]

PPP is a well-recognised, frustrating symptom for both the patient and physician. Due to the variable nature of presentation involving various organ systems, patients often undergo several expensive and unnecessary investigations and surgical procedures. Women with PPP use three times more medications, have four times more gynaecological surgeries, and are five times more likely to undergo hysterectomy than women without PPP.[3] Despite these interventions, about 30 per cent of patients continue to experience persistent pain for more than five years.[4]

PPP has been viewed as a complex neuromuscular-psychosocial disorder sharing the characteristics of chronic regional pain syndrome (such as reflex sympathetic dystrophy) or functional somatic pain syndrome (such as irritable bowel syndrome and nonspecific chronic fatigue).[5] Up to 50 per cent of patients with PPP have history of physical, sexual and emotional abuse or trauma and about one third have positive screening results for posttraumatic stress disorder.[5]

To induce reflection of this multidimensional complexity, Shoskes et al were first to propose a clinical phenotype-based classification system with six domains: urinary, psychological, organ specific, infection, neurological/systemic and tenderness (UPOINT).[6] See Figure 1. Several researchers have demonstrated the clinical applicability of UPOINT to prostate and bladder pain syndrome.[7]

Urological domain in PPP

The lower urinary tract consists of the bladder and urethra. The coexistence of symptoms and the common embryologic origin of the structures involved have led to the suggestion of a 'urogenital pain syndrome,' including some or all of interstitial cystitis and bladder pain syndrome, vulvodynia, urethral syndrome, coccyodynia, and perineal pain.[9] For the bladder pain, patients may complain of increased urinary frequency (day and night), urgency, hypersensitivity, pressure, discomfort, hesitancy, pain with filling, and sensation of incomplete emptying. If the pain is secondary to urethral syndrome, it is perceived usually with voiding and associated dull pressure sensation, radiating towards the perineum and groin.[10] Because of the varying nature of presentation and the impact of the condition on women's lives, treatment needs to be individualised. A multidisciplinary treatment approach including dietary modification, pharmacological agents (such as pentosan polysalphate sodium) and physiotherapy has been recommended. Patients with urethral syndrome secondary to paraurethral gland infection may require treatment with a prolonged course of antibiotics.

Table 1. Other causes of PPP (adapted from ISPOG European Consensus Statement 2015[8]).

Diseases	Causes and findings
Urological	Interstitial cystitis Urethral syndrome Malignant urological disease Bladder function disorders Chronic inflammatory urinary tract Urolithiasis
Gastrointestinal	Irritable bowel syndrome Chronic constipation Chronic-inflammatory intestinal diseases Malignant intestinal diseases Stenosis of the small or large intestine Chronic intestinal pseudo-obstruction
Musculoskeletal and connective tissue	Fibromyalgia Myofascial pain, trigger points Chronic back pain Neuralgia/neuropathic pain syndrome Dysfunction of the pelvic floor Scar pain Malignant disease of the musculoskeletal system and of connective tissue Nerve compression syndromes Hernia
Psychiatric/ psychological disorder	Somatoform disorders Adaptation disorder Schizophrenic, schizotypal and delusional disorders

Gastrointestinal domain in PPP

The gastrointestinal tract (GI) can be the primary pain generator or may play a critical role in inducing pain symptoms by visceral hypersensitivity syndrome. Patients affected in the GI domain frequently report constipation, diarrhoea and obstructive defecation, abdominal bloating and cramping, pain with defecation, bleeding, discharge, recurrent rectal pain/pressure/burning or aching

episodes.[10] Anorectal problems may result from haemorrhoids, abscess, fissures, ulcers, levator ani syndrome or chronic proctalgia. Functional bowel disorders, including irritable bowel syndrome, inflammatory bowel disease and malignancy, may present with similar 'red flag' symptoms and should be excluded by thorough examination and investigation.

Musculoskeletal domain in PPP

Dysregulation in the musculoskeletal system can be the primary cause of PPP or may be secondary to the patient's physical adaptation to the primary pathology involving other domains. Musculoskeletal problems may also coexist with other pelvic pathology such as endometriosis.[11] Features indicative of a musculoskeletal problem include: point tenderness, abnormal movement and alterations in the muscle (tone, stiffness, tension, spasms, cramping, fasciculation and trigger points). PPP related to musculoskeletal disorders includes myofascial pain syndrome, fibromyalgia, hernia, osteitis pubis, trigger point and sacroiliac joint pain. Researchers have also discovered an association of soft tissue disease, such as fibromyalgia, with other comorbid conditions, such as irritable bowel syndrome, primary dysmenorrhoea and chronic functional headache.[5]

Physiotherapy addressing pelvic floor and hip muscle is the mainstay of the treatment for this group of patients. Some other measures proven to be helpful include self-directed pelvic floor contraction and relaxation exercises, muscle relaxants and Botox injections.

Neurological domain in PPP

Patients with predominantly neurological domain related pain commonly describe burning, throbbing, electric shock-like sensation, tingling, stinging and/or painful paraesthesia in the pelvis and/or perineal area.[12] In persistent pelvic pain, secondary sensitisation of the peripheral or central nervous system can occur, perpetuating the overall pain experience with extending referral areas along with the involvement of another organ system.[13] The relevant nerves are sacral, pudendal, thoracolumbar, ilioinguinal, illiohypogastric, genitofemoral or obturator. Local anaesthetic nerve blocks may offer successful treatment for a subgroup of patients, but require repeated injections. In some rare circumstances, surgical decompression of the affected nerve may be an option.

Psychosocial domain in PPP

Psychological factors including depression, anxiety, catastrophising and somatisation are more common among women with PPP compared to control groups.[14] Reports of sleep disturbances, helplessness, hopelessness, difficulty concentrating and pain impairing daily enjoyment are frequently encountered. This vulnerable group of women reports more health-related disability, incur higher health costs and have associated sexual dysfunction, particularly dyspareunia, invariably affecting quality of life. Many women also fear to be stigmatised by health professionals who may consider their pain is not real, but rather 'in her head'.

A formal psychological assessment by valid questionnaires has been recommended to evaluate the extent of psychological comorbidities prior embarking on any specific treatment. Multimodal, personalised approaches involving active communication with primary care providers and a chronic pain team provide the basic framework for comprehensive bio-psychosocial model of holistic and thoughtful care. Most of the patients require extensive psychotherapy (such as cognitive-behavioural therapy), physiotherapy, individual and couples counselling along with pharmacological treatment and social support.

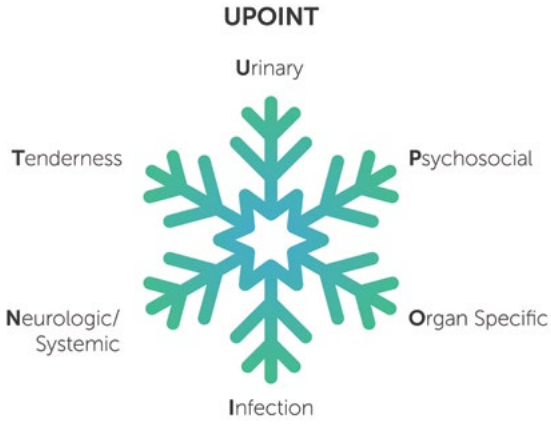

Figure 1. UPOINT is a clinical phenotyping classification system based on the 'snowflake hypothesis' for chronic pelvic pain syndrome. Each of the six domains remains open to subcategorisation as new mechanisms and biomarkers are discovered.[6]

In a nutshell, PPP management requires in-depth understanding of the multidimensional pain mechanism by expert interdisciplinary team members. Following exclusion of any treatable cause, the goal should focus on empowering the patient to resume her normal activity and function by a shared pathway along with the treating team.

References

1. J Steege, M Seidhoff. Chronic pelvic pain. *Obstet Gynecol.* 2014;124:616-29.
2. A Ahangari. Prevalence of chronic pelvic pain among women: an updated review. *Pain Physician*. 2014;17(2):e141-7.
3. SR Till, S As-Sanie, A Schrepf. Psychology of Chronic Pelvic Pain: Prevalence, Neurobiological Vulnerabilities, and Treatment. *Clin Obs and Gyn.* 2019;62(1):22-36.
4. KT Zondervan, PL Yudkin, et al. Prevalence and incidence of chronic pelvic pain in primary care: evidence from a national general practice database. *BJOG.* 1999;106(11):1149-55.
5. D Engeler, AP Baranowski, J. Borovicka, et al. European Association of Urology. Guidelines on Chronic Pelvic Pain. Available from: http://uroweb.org/wp-content/uploads/EAU-Guidelines-Chronic-Pelvic-Pain-2015.pdf.
6. JC Nikel, D Shoskes. Phenotypic approach to the management of chronic prostatitis/chronic pelvic pain syndrome. *Curr Urol Rep*. 2009;10:307-12.
7. JC Nikel, D Shoskes, et al. Clinical phenotyping of women with interstitial cystitis/painful bladder syndrome: a key to classification and potentially improved management. *J Urol.* 2009;182:155-60.
8. F Siedentopf, P Weijenborg, M Engman, et al. ISPOG European Consensus Statement - chronic pelvic pain in women (short version). *J Psychosom Obstet Gynaecol*. 2015;36(4):161-70.
9. U Wesselmann. Pain of urogenital origin. *Pain Clin Updates.* 2000;8(5):1-4.
10. N Rana, MJ Drake, et al. The fundamentals of chronic pelvic pain assessment, based on international continence society recommendations. *Neurology and Urodynamics*. 2018;37:S32-8.
11. D Raimondo, A Youssef, et al. Pelvic floor muscle dysfunction on 3D/4D transperineal ultrasound in patients with deep infiltrating endometriosis: a pilot study. *Ultrasound Obstet Gynaecol*. 2017;50:527-32.
12. M Haanpää, R Treede. Diagnosis and classification of neuropathic Pain. *Pain: Clinical Updates*. 2010;18(7):1-6.
13. LH Whitaker, J Reid, et al. An exploratory study into objective and reported characteristics of neuropathic pain in women with chronic pelvic pain. *PLoS One*. 2016;11(4):e0151950.
14. CE Martin, E Johnson, et al. Catastrophizing: a predictor of persistent pain among women with endometriosis at 1 year. *Hum Reprod.* 2011;26:3078-84.

Core outcome sets for endometriosis

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Variability in reporting outcomes of clinical trials is a significant issue in obstetrics and gynaecology. In a systematic review of published randomised controlled trials (RCTs) addressing therapeutic interventions for pre-eclampsia, 79 RCTs reported 119 different maternal and infant outcomes, with significant variability with regards to what was reported.[1] Most importantly, fewer than half of the studies reported any information regarding harms of the therapeutic interventions.[1] Similar results have been found when reviewing clinical trials in other areas of obstetric and gynaecological research, including preterm birth,[2] management of women with epilepsy in pregnancy[3] and heavy menstrual bleeding trials,[4] among others. This variability limits the ability to perform meaningful systematic reviews and meta-analyses and impacts on the conclusions that may be made.

What are the core outcome sets?

Core outcome sets are an agreed standardised minimum set of outcomes for clinical trials, in an attempt to reduce this variability in clinical trial reporting. They are created by consensus of multiple stakeholders, including clinicians, researchers and affected women. The development and dissemination of core outcome sets aims to standardise outcome collection and reporting in clinical trials, and allow for meaningful systematic review and meta-analysis.

> 'The core outcome sets represent a minimum reporting standard for clinical trials.'

The CROWN Initiative (www.crown-initiative.org) represents a collective of obstetrics and gynaecology journals and journal editors who are committed to the robust development and promotion of core outcome sets. Over 80 journals are involved, including *ANZJOG*.[5] Researchers planning trials and submitting papers to these journals are encouraged to find out if there are published core outcome sets in their areas of research and to incorporate them into trial design and reporting.

What are the core outcome sets for endometriosis?

Similar variability in reporting of outcomes in endometriosis trials is present, with a systematic review of outcome reporting revealing 164 outcomes and 113 outcome measures.[6] This significantly limits the ability to draw meaningful conclusions and provide clinicians and women with evidence-based recommendations for care. The development of a core outcome set for endometriosis trials involved input from healthcare professionals, researchers and women with endometriosis. A Delphi survey was used to assess the relative importance of potential core outcomes in endometriosis research. The Delphi survey method uses multiple rounds of surveys to determine a consensus.

Table 1. Endometriosis core outcome sets.

Pain	Infertility
• Overall pain • Improvement in most troublesome symptom • Quality of life • Adverse events • Patient satisfaction with treatment	• Viable pregnancy confirmed by ultrasound • Pregnancy loss (ectopic, miscarriage, stillbirth) • Livebirth • Gestational age at delivery • Time to pregnancy leading to livebirth • Birthweight • Neonatal mortality • Congenital abnormality

The final core outcome set included five core outcomes under the heading of pain, and eight core outcomes under the heading of infertility (detailed in the table below; from presentation and personal communication). While the goal is that these outcomes are addressed by all future clinical trials in endometriosis, the intention is not to limit outcome reporting, and investigation and reporting of additional outcomes of interest is encouraged.

These core outcomes were presented at the FIGO World Congress 2018, in Rio de Janeiro, Brazil, by Dr James MN Duffy (@JamesMNDuffy), and are in the process of being submitted for publication.

References

1. J Duffy, M Hirsch, A Kawsar, et al. Outcome reporting across randomised controlled trials evaluating therapeutic interventions for pre-eclampsia. *BJOG*. 2017;124(12):1829-39.
2. S Meher, Z Alfirevic. Choice of primary outcomes in randomised trials and systematic reviews evaluating interventions for preterm birth prevention: a systematic review. *BJOG*. 2014;121(10):1188-94; discussion 95-6.
3. BH Al Wattar, A Placzek, J Troko, et al. Variation in the reporting of outcomes among pregnant women with epilepsy: a systematic review. *Eur J Obstet Gynecol Reprod Biol*. 2015;195:193-9.
4. MC Herman, J Penninx, PM Geomini, et al. Choice of primary outcomes evaluating treatment for heavy menstrual bleeding. *BJOG*. 2016;123(10):1593-8.
5. J Oliver Daly. Harmonisation of research outcomes for meaningful translation to practice: The role of Core Outcome Sets and the CROWN Initiative. *ANZJOG*. 2018;58(1):15-6.
6. M Hirsch, JMN Duffy, JO Kusznir, et al. Variation in outcome reporting in endometriosis trials: a systematic review. *Am J Obstet Gynecol*. 2016;214(4):452-64.

Scope of medical imaging for pelvic pain

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'I have had lots of ultrasounds, but no one can ever tell me why I have pain.' Sadly, this is an all-too-common refrain from women suffering pelvic pain. Ultrasound can be a very valuable imaging tool for the assessment of pelvic pain, but it has its limitations. Understanding what it 'can and cannot see' is very important for maximising its clinical efficacy. When pelvic ultrasound is limited to the assessment of the uterus, endometrium and ovaries, many causes of pelvic pain will be missed or misdiagnosed. A more complete assessment, which incorporates the Pouch of Douglas and the uterosacral ligaments, has been described and widely published in recent years.[1,2]

Pelvic pain is a complex and diverse medical problem. Delayed diagnosis and misdiagnosis are common problems, with the median time interval from first experience of symptoms to a diagnosis of seven years.[3] This has been reported for endometriosis, but is also relatively common for other gynaecological conditions causing, or contributing to, pelvic pain. There is considerable overlap between the causes of acute and chronic pelvic pain. The initial consultation with a health provider is of crucial importance. Establishing trust and arriving at a timely diagnosis can prevent many causes of pelvic pain progressing to a chronic pain condition.[4,5]

Medical imaging has often been inadequate in providing the answers that patients and gynaecologists need to manage pain. The investigations for pelvic pain must consider the broad range of possible aetiologies and comorbidities. Correct use and interpretation of medical imaging needs to take account of whether the pain is acute or chronic, as well as individual patient considerations such as age, prior surgery and use of hormonal medications. Providing this information to your medical imaging team is very important to allow a thorough and complete investigation. As clinicians, you have an important role in providing this information. An appropriately detailed referral or request form will allow your radiologist or COGU to provide a more complete and clinically pertinent report.

Table 1. Things your imaging specialist wants to know.

Factor	What to include
Nature of the pain	Duration, cyclical/non-cyclical, location of pain
Comorbidities	Other medical conditions that may be relevant
Age	Premenarchal, reproductive age, peri- or post-menopausal
Use of hormonal medications	Mirena, Implanon, OCP, HRT
Prior surgery	
Gynaecology and obstetric history	

While there is a role for MRI and CT scanning in the investigation of some forms of pelvic pain, ultrasound remains the investigation of first choice. It is readily available, relatively inexpensive and free from radiation. It provides high-resolution imaging of pelvic organs and, importantly, is a dynamic real-time assessment, allowing the localisation of pain and assessment of tissue mobility.

A disadvantage of ultrasound is that it is operator-dependant, and the ability to review or reassess images can be limited. The preferred imaging modality may relate to the local expertise in this area.

Acute pelvic pain

Acute abdominal and pelvic pain often presents as a diagnostic dilemma between gynaecological and general surgical teams. Imaging is central to establishing the diagnosis. Once pregnancy-related complications have been excluded with a current negative beta human chorionic gonadotropin test, ultrasound remains the first line in medical imaging. This article will not cover pregnancy-related causes of pelvic pain.

Ultrasound is useful in the diagnosis of many of the conditions listed in Table 2. Transvaginal (TV) sonography performs well in the diagnosis of pelvic inflammatory disease[6], ovarian cysts and some forms of torsion. The difficulty with the diagnosis of ovarian cysts as causes of acute pelvic pain is that they are common, often transient, and may be an incidental, rather than causative, finding. Approximately 7 per cent of asymptomatic pre-menopausal women will have a cyst greater than 25 mm, and 7 per cent of postmenopausal women will have a cyst greater than or equal to 15 mm.[7,8] Complications of a cyst, such as internal haemorrhage or rupture, are readily identified with TV ultrasound.

Table 2. Types of acute pelvic pain.

Adnexal	Uterine	Non-gynaecological
• Torsion - Ovarian - Tubal - Adnexa • Cyst complication - Torsion - Haemorrhage - Rupture • Endometriosis • Pelvic inflammatory disease	• Fibroids - Degeneration - Torsion • Mal-positioned IUD • Congenital uterine anomaly - Haematometra - Haematocolpos	• Appendicitis • Renal uropathy • Diverticulitis • Inflammatory bowel disease (IBD)

Torsion is a partial or complete rotation of the adnexal structures around their vascular pedicle and can involve the ovary, tube or entire adnexa. Torsion is relatively common, representing approximately 3 per cent of presentations to emergency departments.[9] Given the risk of adnexal necrosis, a timely diagnosis is important. Torsion creates a diffuse enlargement of the adnexal structure secondary to the oedema. It must be remembered that torsion remains a clinical diagnosis that cannot be completely excluded or confirmed with imaging. The most predictive imaging findings where torsion has been confirmed surgically are adnexal enlargement (90 percent greater than 5 cm in size) and displacement of the ovary to a position superior to uterus or to the contralateral pelvis. Doppler can be misleading, with up to 50 per cent of cases of torsion demonstrating normal blood flow patterns.[9] This is likely secondary to the varied degrees of arterial, venous and lymphatic occlusion. Absence of blood flow to the adnexa has a high positive predictive value for torsion, but is a late sign and indicates a degree of necrosis.[9] In the premenarchal paediatric group, torsion often occurs in the setting of a normal ovary and tube.

Congenital anomalies of the uterus and vagina in the adolescent population may present acutely secondary to the haematocolpos or haematometra. These conditions can be mislabelled as ovarian masses, typically endometriomas.

Non-gynaecological causes of pelvic pain should be assessed with every scan. Appendicitis is a common cause of acute right lower quadrant pain and imaging is often used in the less obvious clinical presentations. The sensitivity of transabdominal/TV ultrasound for acute appendicitis ranges from 75–90 per cent.[6] Diverticulitis presents as lower left quadrant pain in adults. Ultrasound offers similar sensitivity and specificity to CT scanning.

Chronic pelvic pain

Chronic pelvic pain is a condition defined as pelvic pain present for more than six months that is severe enough to cause functional disability or require treatment.[10] Chronic pelvic pain presents as frequently to the medical system as migraine or lower back pain[5] and is associated with significant impairment in quality of life and significant economic implications. Endometriosis alone is estimated to affect 176 million women worldwide,[11] with pain accounting for approximately 20 per cent of hysterectomies for benign conditions and at least 40 per cent of gynaecological laparoscopies.[10] The aetiologies of chronic pain are more complex and diverse, and it is more frequently associated with significant comorbidities, such as, irritable bowel syndrome 50 per cent and interstitial cystitis 38–84 per cent.[5] Musculoskeletal pain arising from the pelvic floor is common.

The efficacy of ultrasound in the investigation of chronic pelvic pain has not been well evaluated, but it has been reported that up to 20 per cent of patients undergoing ultrasound for chronic pelvic pain will have abnormalities identified.[12] TV ultrasound performs well in identifying structural anomalies such as adnexal/ovarian pathology, adenomyosis and fibroids, but these may be incidental findings and not the cause of the pain. MRI and TV ultrasound perform well and similarly in the identification of deep endometriosis. There have been recent publications outlining a systematic approach to the ultrasound evaluation of deep endometriosis. It is an important preoperative investigation to locate lesions of deep infiltrating endometriosis and to adequately consent patients and plan surgery. MRI remains the second line investigation, owing to cost and availability. MRI is particularly useful to use in conjunction with TV ultrasound imaging or in patients where TV ultrasound is not acceptable, where the origin or nature of pathology is uncertain, or where immobility of the pelvis has limited assessment of the upper rectal wall. This is often encountered with severe endometriosis/adhesive disease or when large fibroids or ovarian masses fill the pelvis.

The use of medical imaging to provide a non-invasive diagnosis for peritoneal or superficial endometriosis remains the next frontier. A Cochrane

Table 3. Types of chronic pelvic pain.

Adnexal	Uterine	Non-gynaecological
• Ovarian pathology • Hydrosalpinges • Endometriosis	• Adenomyosis • Fibroids • Malignant myometrial • Congenital uterine anomalies • Prolapse	• IBD • Diverticular disease • Uropathy • Interstitial cystitis • Pelvic congestion • Adhesive tissue

review concluded that no imaging modality could detect pelvic endometriosis with enough accuracy to replace surgery.[13] A study assessing the role of 'soft markers' in ultrasound found a LR of 1.9 for the presence of these markers and positive findings at laparoscopy.[12] A more recent study identified a correlation of 79 per cent of ultrasound findings of inflammatory change (thickened uterosacral ligaments and pericolic fat) and positive findings of endometriosis at laparoscopy.[14]

The ability of ultrasound to diagnose peritoneal/superficial forms of endometriosis is the current challenge. This would reduce the reliance on diagnostic laparoscopy and allow patients and their doctors greater insight into the cause of their pain.

References

1. S Guerrero, et al. Systematic approach to sonographic evaluation of the pelvis in women with suspected endometriosis, including terms, definitions and measurements: a consensus opinion from the international deep endometriosis analysis (IDEA) group. *Ultrasound Obstet Gynecol*. 2016;48:318-32.
2. M Goncalves, et al. Transvaginal ultrasound for diagnosis of deep infiltrating endometriosis. *International J Obst and Gynecol*. 2009;104:156-60.
3. H Taylor, et al. An evidence-based approach to assessing surgical versus clinical diagnosis of symptomatic endometriosis. *International J Obst and Gynecol*. 2018;142:131-42.
4. G Harkins G. Female pelvic pain. *Semin Reprod Med*. 2018;36:97-8.
5. The initial management of chronic pelvic pain. RCOG: Green Top Guidelines, No. 41, May 2012.
6. E Okaro, L Valentin. Best Practice and Research. *Clin Obst and Gynaecol*. 2004;18:105-23.
7. C Borgfeldt. Transvaginal sonographic ovarian findings in random sample women, 25 – 40 years. *Ultrasound in Obst and Gynecol*. 1999;13:345-50.
8. C Conway. Simple cyst in the post-menopausal patient: detection and management. *Journal of Ultrasound in Medicine*. 1998;17:369-372.
9. P Callen. Ultrasonography in Obstetrics and Gynaecology, 6th Edition, Elsevier, 2016.
10. J Chandler. Evaluation of female pelvic pain. *Seminars in Reproductive Medicine*. 2018;36:99-106.
11. D Bush, S Evans. The $6 Billion Woman and the $600 Million Girl. 2011: The Pelvic Pain report. Available from: http://fpm.anzca.edu.au/documents/pelvic_pain_report_rfs.pdf.
12. E Okaro, G Condous, et al. The use of ultrasound-based 'soft markers' for the prediction of pelvic pathology in women with chronic pelvic pain – can we reduce the need for laparoscopy? *BJOG*. 2006,113:251-56.
13. V Nienblat, PMM Bossuyt, C Farquhar, et al. Imaging modalities for the non-invasive diagnosis of endometriosis (Review). *Cochrane Database of Syst Rev*. 2016, Issue 2.
14. P Chowdary, K Stone. Multicentre retrospective study to assess the diagnostic accuracy of ultrasound for superficial endometriosis – are we any closer? *ANZJOG*. 2019;59:279-84.

Managing pelvic pain in adolescents

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Adolescents presenting with pelvic pain deserve a considered approach for several reasons. Firstly, by recognising and treating pain at an early stage, we can reduce the likelihood of a lifelong battle with chronic pain. We know that repeated or prolonged episodes of pain lead to central sensitisation and predispose to chronic/persistent pelvic pain.[1] Most importantly, how pain is explained and the language that is used will significantly shape how the adolescent sees and copes with the pain that she is experiencing. Secondly, adolescence itself represents a time of self-discovery and growth, the crucial time in which a young woman is developing her sense of self and paving the way for the adult she will become. There is Australian evidence that 20–30 per cent of women with dysmenorrhea are missing school and activities, making it an important issue to address,[2] not just for her current health and wellbeing, but for her future. Additionally, there are a few other specific causes of pelvic pain that typically present in adolescence, or even during childhood, that we will cover in this article.

Causes of pelvic pain in adolescents:

- dysmenorrhoea
- ovulation pain
- abdominal wall/pelvic floor
- ovarian cysts
- vulvodynia
- imperforate hymen
- Müllerian tract anomalies
- pelvic inflammatory disease (PID)
- acute pain, such as ovarian torsion, ectopic pregnancy, appendicitis, PID
- other causes, such as bladder, inflammatory bowel disease, constipation

A considered approach to pelvic pain in adolescents

A careful history should be taken with any consultation, including questions exploring HEADS (home/housing, education/employment, activities, drugs/depression, sexuality/suicide). Much can be gained by understanding the context and life of the young woman and her pain. Questions also need to cover the development of secondary sexual characteristics, if there has been a growth spurt, if and when menarche was achieved. Getting an idea of cycle length, duration, menstrual flow and when in the cycle she has pain will often help identify a cause. For example, pain two days before the period starts and for the first few days, but not at any other time of the month, associated with nausea, vomiting and diarrhoea is consistent with primary dysmenorrhoea.[3]

On the other hand, pain throughout the month with a palpable one-sided pelvic mass worse with a period might represent an obstructed uterine horn in an adolescent with a Müllerian tract abnormality. Pain may also be ongoing post an acute event, such as cyst rupture or haemorrhagic corpus luteum. Patients may also transition from experiencing intermittent pain to experiencing ongoing abdominal pelvic symptoms as a result of central sensitisation. Careful examination of the abdomen and, in particular, the abdominal wall to ascertain whether there are any trigger points will help identify a musculoskeletal cause. This can be done by asking the patient to lift their head off the bed (that is, tensing the abdominal wall) while examining the abdomen, known as Carnett's sign. Bowel and bladder symptoms should also be discussed to identify any contributing factors. External genital examination should be performed with consent, especially in girls who have not yet achieved menarche but may have cyclical abdominal pain or an abdominal mass, to identify an imperforate hymen or a transverse vaginal septum. A sexual history should be taken without parental/carer presence, if possible, to assess other risk factors that may contribute to pelvic pain, such as previous history of sexual abuse/assault, risk factors for PID/pregnancy.

If dysmenorrhoea is not well controlled with simple analgesia, including nonsteroidal anti-inflammatory drugs (NSAIDs), or if associated with atypical features, such as pain persisting beyond the end of the menses and pain between menses, a pelvic ultrasound should be performed. Ultrasound can be useful to identify

Table 1. Management of causes.

General management	• Identify likely cause • Education • Exercise, stretching, heat, mindfulness • Multidisciplinary care should be considered for all complex patients • Emotional support for parent and child including school supports and involvement of a psychologist • Involve patient in management plan based on maturity level
Dysmenorrhoea	• NSAIDs: regularly three times a day (TDS) at the onset of symptoms • Tranexamic acid 1 g TDS to reduce blood loss • Suppression of menses with hormonal therapies e.g. progesterone only pill, COCP, progesterone injection, or Levonorgestrel IUS • If ongoing pain despite an adequate period of menstrual suppression and a specialist women's imaging ultrasound ruling out other causes of pain, a laparoscopy should be considered
Ovulation pain	• Track expected ovulation; use regular tranexamic acid and NSAIDs • Ovulation suppression e.g. COCP, medroxyprogesterone injection, continuous progestogens
Abdominal wall/ pelvic pain	• Physiotherapy • Injection with steroid and local anaesthetic – partly diagnostic, partly treatment • Consider role of neuromodulators • Involvement of paediatric pain team
Ovarian cysts	• Identify type of cyst simple vs complex • Monitoring: need repeat scan in 6–8 weeks • Ovulation suppression if recurrent simple cysts • If complex bloods, surgery as required
Vulvodynia	• Avoid irritants • Education/emotional support • Neuromodulators that control symptoms >6 months before consideration of weaning e.g. Amitriptyline
Imperforate hymen	• Surgical management
Müllerian anomalies	• Specialist pelvic ultrasound +/- MRI • Supress periods to control symptoms until ready for surgery • Seek advice for optimal management from experienced gynaecologist • Surgery to correct if/when appropriate
Pelvic Inflammatory disease	• Antibiotics • Education about safe sexual practice, including contact tracing • Counsel long-term complications
Acute pain	• Gynaecology review • Pelvic ultrasound to rule out acute causes of pelvic pain, eg. torsion, ectopic pregnancy, cyst rupture
Other causes	• Surgical, gastroenterology, adolescent medicine, urology as appropriate

ovarian cysts or obstructive Müllerian anomalies. In addition, ultrasound can be better than laparoscopy for identifying some unusual adolescent problems, such as obstructed uterine horn/s that at laparoscopy might simply appear as a bicornuate uterus.

Blood tests are often of little value to investigate causes of pain in adolescence; however, if a patient has recurrent haemorrhagic cysts and/or heavy menstrual bleeding, history of easy bleeding/ bruising or a strong family history, investigations for a bleeding disorder should be considered. This would include screening for Von Willebrand's disease, full blood count and platelet function assay (PFA-100).

Dysmenorrhoea

Primary dysmenorrhoea in teenagers is common and can be associated with a range of associated symptoms that commonly include nausea, vomiting, diarrhoea, myalgia, headaches, dizziness and fainting.

Some girls present with cyclical exacerbations of chronic fatigue, pain in other body parts, epilepsy, asthma and even anaphylaxis related to their periods. This tells us that the prostaglandins and pro-inflammatory cytokines produced to provoke endometrial shedding (which is known to be an inflammatory process)[4] are potent and cause systemic effects. We know that by involving the patient in their management and explaining the physiology behind why they may feel pain, we can empower them to understand their condition better. An age-appropriate explanation of the process the body goes through during menstruation can be therapeutic in itself and is reassurance that the pain is not necessarily reflective of underlying pathology. Endometriosis (and even adenomyosis) should be considered as a differential diagnosis, but, in adolescents, first-line management should be education, pain management strategies and reduction of heavy menstrual bleeding/suppression of menses.

Vulval and genital pain

The occurrence of distressing nocturnal pain, waking the pre-pubertal child from sleep, raises the possibility of pinworms and should be treated with weekly mebendazole for three weeks.[5] Vulvodynia commonly presents in childhood, but can be ongoing into adolescence. Presentations can include urinary symptoms (80 per cent), burning sensation, or a bubbly/ticklish feeling on the perineum/vulval area. It is also seen after a triggering event, such as urinary tract infection or vulvovaginitis. In the adolescent, the presentation may be inability to use tampons or dyspareunia. Careful history and subsequent examination using cotton swab test can aid diagnosis. External genitalia usually appear normal, but 94 per cent will have altered sensation at the vestibule on cotton swab test examination.[6] Diagnostic delay is common and can be very distressing for the patient and family.

Special considerations for the use of hormonal therapies in adolescents

Medical treatment options for dysmenorrhoea are essentially the same in adolescents as they are in adults. First-line options include heat packs/stretching, simple analgesia and regular NSAIDs. For girls with pain uncontrolled by simple measures or seeking contraception, hormonal treatments such as combined oral contraceptive pill (COCP) and progesterone-only medications are appropriate. It is common to see adolescents referred to our tertiary paediatric centre by doctors who are hesitant to use hormonal treatments in adolescents. This is not surprising, given the amount of conflicting information on this topic in both the medical literature and the media, and it is important that we address these concerns. There is no evidence that using COCP in postmenarchal girls, even those shortly after menarche, results in any significant effect on height. By the time a girl achieves menarche, the majority of her growth has been completed and she will already have high circulating oestrogen. A clinically significant effect of the pill on bone mineral density has not been demonstrated and concerns about skeletal health should not affect COCP use in healthy women of any age.[7] It is also safe to recommend continuous use of the pill for symptom control with the aim of skipping periods.[8]

Medroxyprogesterone injection can be safely uses in adolescence, if other options are not suitable to control symptoms, with consideration for monitoring of bone density. It is sensible to ensure adequate dietary calcium intake, optimal sunshine exposure/vitamin D, and encourage weight-bearing exercise. If bone density becomes an issue, continuous use of oral or patch oestrogens could be added.

Etonogestrel implant can be effective in suppressing menses and reducing ovarian activity in some adolescents but has the risk of unpredictable bleeding in others and therefore wouldn't be considered first-line management. It is, however, a very effective and reliable contraceptive and very acceptable to many young women.

Levonorgestrel-IUS have been used for 20 years in the adolescent population in Melbourne and can be considered in nulliparous and non-sexually active teenagers when inserted under general anaesthetic. Keeping in mind that ovulation pain or symptoms related to cyclical hormonal changes are unlikely to be well controlled by this alone.

References

1. OR Zekavat, MY Karimi, A Amanat, F Alipour. A randomised controlled trial of oral zinc sulphate for primary dysmenorrhoea in adolescent females. *ANZJOG*. 2015;55(4):369-7.
2. S Evans. Chronic pelvic pain in Australia and New Zealand. *ANZJOG*. 2012;52:499-501.
3. A Subasinghe, L Happo, Y Jayasinghe, et al. Prevalence and severity of dysmenorrhoea, and management options reported by young Australian women. *Australian Family Physician*. 2016;45(11):829-34
4. J Evans, L Salamonsen. Inflammation, leucocytes and menstruation. *Rev Endocr Metab Disord*. 2012;13:277.
5. J Dennie, S Grover. Distressing perineal and vaginal pain in pre-pubescent girls: an aetiology. *J Paediatr Child Health*. 2013;49:138-40.
6. A Dunford, D Rampal, M Kielly, S Grover. Vulval pain in paediatric and adolescent patients. *J Pediatr Adolesc Gynecol*. 2019. 10.1016/j.jpag.2019.03.005.
7. MM Isley, AM Kaunitz. Update on hormonal contraception and bone density. *Rev Endocr Metab Disord*. 2011;12(2):93-106.
8. Royal Children's Hospital. Oral contraceptives – skipping periods when taking the Pill. 2018. Available from: www.rch.org.au/kidsinfo/fact_sheets/Oral_contraceptives_skipping_periods_when_taking_the_Pill/.

Linking subfertility with endometriosis

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Endometriosis is a chronic inflammatory disorder defined by the presence of endometrial glands and stroma outside the uterus and is characterised by pelvic adhesions and fibrotic tissue formation.[1] Three main subtypes of endometriosis exist; namely, superficial peritoneal, ovarian endometrioma and deeply infiltrative disease (DIE). There exists an association between all these forms of endometriosis and subfertility, although the extent of the link with DIE is less clear.[2] While the exact prevalence is unknown, endometriosis is estimated to affect up to 10 per cent of the general female population; however, is thought to be present in up to 50 per cent of women presenting with subfertility.[3,4] In normal fertile couples, the fecundity rate, potential to reproduce, is estimated to be in the range of 15–20 per cent per month, while the rate of fecundity in women with untreated endometriosis may be as low as 2–10 per cent per month.[5,6] Women with endometriosis have, overall, been shown to have a significantly lower probability of pregnancy over three years than women with unexplained fertility (36 per cent versus 55 per cent, respectively).[7]

Present treatment options for endometriosis-associated subfertility include medical treatment, which may be given alone or as an adjunct to surgery, and artificial reproductive techniques (ART), including controlled ovarian stimulation (COS) with intrauterine insemination (IUI) and in vitro fertilisation (IVF).

Medical management of endometriosis and natural conception

Given the demonstrated oestrogen dependency of endometriosis, significant work has been done to investigate the potential for the use of hormonal suppressive treatment of endometriosis to improve fertility, despite the fact that all treatments inhibit ovulation. A Cochrane review published in 2007 found no evidence of benefit in the use of suppression (GnRH agonists, danazol, gestrinone, medroxyprogesterone and the combined oral contraceptive pill [COCP]) in subfertile women with endometriosis, in terms of clinical pregnancy and live birth.[13] There is also a lack of evidence for adjunctive medical treatment before or after surgery for improvement in spontaneous pregnancy rate.[4,14]

Medical management as an adjunct to ART

There is high-quality evidence to suggest that three to six months of pre-treatment with GnRH agonists prior to ART will increase the clinical pregnancy rate by a factor of up to four.[4] The creation of a hypo-oestrogenic environment is thought to reduce the inflammatory state.[15] There is no evidence available to suggest benefit of pre-treatment with any other form of medical suppression, such as the COCP.

Surgical treatment of stage I/II endometriosis and natural conception

Direct visualisation and biopsy at the time of laparoscopy is the gold standard diagnostic test for endometriosis and enables identification of the location, extent and severity of the disease.[14] Surgical excision of all visible disease should be performed simultaneously. In patients with minimal

Box 1. List of biological mechanisms identified to explain the link between endometriosis and subfertility.

Mechanism of subfertility

Several biological mechanisms have been identified explaining the link between endometriosis and subfertility. These include:

- Distortion of pelvic anatomy by adhesions, impairing oocyte release, capture and transport[6]
- Altered peritoneal fluid volume and increased concentrations of prostaglandins, proteases and inflammatory cytokines[8,9]
- Altered hormonal and cell-mediated function, resulting in altered endometrial receptivity due to increased levels of endometrial IgG and IgA antibodies and lymphocytes[9]
- Endocrine and ovulatory abnormalities, including luteal phase defect, abnormal follicular phase and abnormal progesterone secretion[10]
- Disordered endometrial function, resulting in impaired implantation
- Reduced oocyte and embryo quality. Altered progesterone and cytokine concentrations have been found in follicular fluid from women with endometriosis[11]
- Abnormal utero-tubal transport when compared to healthy controls[12]
- Painful intercourse leading to decreased frequency
- Inflammatory hydrosalpinges in 30% of cases

to mild endometriosis (revised American Society for Reproductive Medicine (rASRM) classification stage 1–2),[16] laparoscopic excision or ablation of lesions adhesiolysis will increase the ongoing pregnancy and live birth rate, when compared to diagnostic laparoscopy alone.[4,17] There is a lack of prospective data comparing effectiveness of modalities such as CO2 laser vaporisation, monopolar electrocoagulation and excision.[4] There is no evidence to support the use of repeat laparoscopic surgery to improve fertility, although it may be considered for treatment of pain.[19]

Surgical treatment of stage I/II endometriosis prior to ART

There is moderate quality evidence to suggest that in women with rASRM stage I/II endometriosis, laparoscopic treatment of disease prior to ART may improve live birth rate;[4,20] however, other indications for surgery may be taken into consideration. Complete surgical excision of minimal to mild endometriosis may improve the rate of embryo implantation, pregnancy rate and live birth rate as well as reduce time to first pregnancy.[20] There are no randomised controlled trials comparing ablative with excisional treatment prior to ART. There is also a lack of data directly comparing laparoscopic surgery to IVF for patients with minimal endometriosis and infertility.[15]

Surgical treatment of stage III/IV endometriosis and natural conception

In patients with moderate to severe endometriosis (rASRM stage 3–4) the data demonstrates that laparoscopic surgery is better than expectant management.[4,18] One controlled prospective study, published in 2018, demonstrated a higher rate of natural conception in women following surgical treatment of rectal DIE.[21,22] Prospective cohort studies have also shown that in women with infertility and stage III/IV endometriosis, operative laparoscopy may increase spontaneous pregnancy rates.[4] There is no benefit in repeat surgery.[6] There is a paucity of data examining the effect of bowel resection on both spontaneous pregnancy rate and success of ART, although one observational study found that in women with DIE affecting the bowel, complete excision and bowel segmental resection resulted in higher monthly fecundity rates than removal of endometriosis without bowel resection.[23] Should resection of DIE be performed, there is, of course, benefit for fertility of laparoscopy over laparotomy.[24] Multidisciplinary review, case-by-case decision making and careful consideration of risks of surgery are essential pre-requisites for a good outcome.

Surgical treatment of stage III/IV endometriosis prior to ART

The impact of surgical treatment of DIE as an adjunct to IVF is controversial. The available published studies are observational and are not large enough to allow for any definitive conclusions. Thus, it remains impossible to define the absolute effect of surgery in this group beyond the benefit of facilitation of ovarian access.

Management of ovarian endometrioma

In patients with ovarian endometrioma undergoing surgery, excision of the endometrioma capsule increases the postoperative spontaneous pregnancy rate, compared to drainage and electrocoagulation.[25] All techniques carry potential risks for ovarian reserve, either by removal of normal ovarian tissue during excision or by thermal damage to the ovarian cortex during ablation. Therefore, women should be counselled regarding the risks of reduced ovarian function after surgery.[25] Prior to commencement of ART, there is no evidence that surgical treatment of endometrioma will improve pregnancy rate and should only be considered for management of pain or to improve ovarian accessibility.[25-27] Other benefits include potential avoidance of contamination of follicular fluid with endometrioma content and other potential complications of endometrioma, such as rupture or malignant transformation.[6]

ART for endometriosis-associated infertility

IUI, COS and IVF are the most established techniques available to women with endometriosis. There is no consensus on the optimal approach. In stage I/II endometriosis-associated infertility, IUI with COS has been shown to increase the live birth rate by a factor of almost six when compared to no treatment.[28] This may be more effective if done within six months of surgery and may be a reasonable first line option for younger women.[6,21] Older women (over 35 years) or those who have already undergone surgery to improve fertility and failed to conceive, may be better served by earlier recourse to IVF.[6] However, while IVF is an effective treatment, the pregnancy rate in women with endometriosis is almost half that of women with other indications for IVF. This may be because endometriosis affects oocyte and embryo quality and development, as well as endometrial receptivity.[29] In the most recent ANZARD report, however, IVF cycles reporting male factor infertility as the only cause of infertility had the highest live delivery rate (19.7 per cent), followed by cycles reported with female tubal disease and endometriosis as the only causes of infertility (19.2 per cent and 19.2 per cent).[30]

Pregnancy outcomes

Although the exact mechanism for the relationship remains unclear, there is evidence to suggest an increase in adverse obstetric outcomes in women who achieve live birth with endometriosis, including endometriosis-associated spontaneous haemoperitoneum, placenta praevia, preterm birth, pre-eclampsia, antepartum haemorrhage and caesarean section.[31,32] There is no evidence for any association with intrauterine growth restriction or stillbirth.[6] Women with untreated deeply infiltrative rectovaginal endometriosis have also been found to be at higher risk of caesarean delivery and surgical complications, namely hysterectomy, haemoperitoneum and bladder injury, than controls.[33]

Conclusion

Endometriosis is a common condition that may have significant impact on fertility via a variety of mechanisms. In terms of fertility, it would appear that a laparoscopy plus excision of endometriosis and restoration of normal anatomy offers the most benefit. Surgical treatment increases the spontaneous conception rate and improves the success rate of ART in stage I/II disease, and may assist in stage III/IV. There is need for more good-quality research to guide management of such patients.

References

1. C Bulletti, ME Coccia, S Battistoni, et al. Endometriosis and infertility. *J Assist Reprod Genet*. 2010;27(8):441-7.
2. D de Ziegler, B Borghese, C Chapron. Endometriosis and infertility: pathophysiology and management. *Lancet*. 2010;376(9742):730-8.
3. C Meuleman, B Vandenabeele, S Fieuws, et al. High prevalence of endometriosis in infertile women with normal ovulation and normospermic partners. *Fertil Steril*. 2009;92(1):68-74.
4. D Bush, S Evans, T Vancaillie. The $6 Billion Woman and the $600 Million Girl: The Pelvic Pain Report, 2011. Available from: fpm.anzca.edu.au/documents/pelvic_pain_report_rfs.

5. ML Macer, HS Taylor. Endometriosis and infertility: a review of the pathogenesis and treatment of endometriosis-associated infertility. *Obstetrics and Gynecology Clinics of North America*. 2012;39(4):535-49.
6. Practice Committee of the American Society for Reproductive Medicine. Endometriosis and infertility: a committee opinion. *Fertil Steril*. 2012;98(3):591-8.
7. VA Akande, LP Hunt, DJ Cahill, et al. Differences in time to natural conception between women with unexplained infertility and infertile women with minor endometriosis. *Hum Reprod*. 2004;19(1):96-103.
8. MA Bedaiwy, T Falcone, RK Sharma, et al. Prediction of endometriosis with serum and peritoneal fluid markers: a prospective controlled trial. *Hum Reprod*. 2002;17(2):426-31.
9. DI Lebovic, MD Mueller, RN Taylor. Immunobiology of endometriosis. *Fertil Steril*. 2001;75(1):1-10.
10. JS Cunha-Filho, JL Gross, CA Bastos de Souza, et al. Physiopathological aspects of corpus luteum defect in infertile patients with mild/minimal endometriosis. *J Assist Reprod Genet*. 2003;20(3):117-21.
11. N Garrido, J Navarro, J Remohi, et al. Follicular hormonal environment and embryo quality in women with endometriosis. *Hum Reprod Update*. 2000;6(1):67-74.
12. S Kissler, N Hamscho, S Zangos, et al. Diminished pregnancy rates in endometriosis due to impaired uterotubal transport assessed by hysterosalpingoscintigraphy. *BJOG*. 2005;112(10):1391-6.
13. E Hughes, J Brown, JJ Collins, et al. Ovulation suppression for endometriosis. *Cochrane Database Syst Rev*. 2007(3):Cd000155.
14. C Yap, S Furness, C Farquhar. Pre and post operative medical therapy for endometriosis surgery. *Cochrane Database Syst Rev*. 2004(3):Cd003678.
15. J Koch, K Rowan, L Rombauts, et al. Endometriosis and Infertility – a consensus statement from ACCEPT (Australasian CREI Consensus Expert Panel on Trial evidence). *ANZJOG*. 2012;52(6):513-22.
16. Revised American Society for Reproductive Medicine classification of endometriosis: 1996. *Fertil Steril*. 1997;67(5):817-21.
17. JM Duffy, K Arambage, FJ Correa, et al. Laparoscopic surgery for endometriosis. *Cochrane Database Syst Rev*. 2014(4):Cd011031.
18. P Vercellini, L Fedele, G Aimi, et al. Reproductive performance, pain recurrence and disease relapse after conservative surgical treatment for endometriosis: the predictive value of the current classification system. *Human Reprod*. 2006;21(10):2679-85.
19. P Vercellini, E Somigliana, R Daguati, et al. The second time around: reproductive performance after repetitive versus primary surgery for endometriosis. *Fertil Steril*. 2009;92(4):1253-5.
20. HK Opoien, P Fedorcsak, T Byholm, et al. Complete surgical removal of minimal and mild endometriosis improves outcome of subsequent IVF/ICSI treatment. *Reproductive Biomedicine Online*. 2011;23(3):389-95.
21. H Roman, et al. Conservative surgery versus colorectal resection in deep endometriosis infiltrating the rectum: a randomized trial. *Hum Reprod*. 2017;33(3):47-57.
22. H Roman H, I Chanavaz-Lacheray, M Ballester, et al. High postoperative fertility rate following surgical management of colorectal endometriosis. *Hum Reprod*. 2018;33(9):1669-76.
23. A Stepniewska, P Pomini, F Bruni, et al. Laparoscopic treatment of bowel endometriosis in infertile women. *Hum Reprod*. 2009;24(7):1619-25.
24. E Darai, B Lesieur, G Dubernard, et al. Fertility after colorectal resection for endometriosis: results of a prospective study comparing laparoscopy with open surgery. *Fertil Steril*. 2011;95(6):1903-8.
25. RJ Hart, M Hickey, P Maouris, et al. Excisional surgery versus ablative surgery for ovarian endometriomata. *Cochrane Database Syst Rev*. 2008(2):Cd004992.
26. L Benschop, C Farquhar, N van der Poel, et al. Interventions for women with endometrioma prior to assisted reproductive technology. *Cochrane Database Syst Rev*. 2010(11):Cd008571.
27. JA Garcia-Velasco, NG Mahutte, J Corona, et al. Removal of endometriomas before in vitro fertilization does not improve fertility outcomes: a matched, case-control study. *Fertil Steril*. 2004;81(5):1194-7.
28. IS Tummon, LJ Asher, JS Martin, et al. Randomized controlled trial of superovulation and insemination for infertility associated with minimal or mild endometriosis. *Fertil Steril*. 1997;68(1):8-12.
29. K Barnhart, R Dunsmoor-Su, C Coutifaris. Effect of endometriosis on in vitro fertilization. *Fertil Steril*. 2002;77(6):1148-55.
30. O Fitzgerald, RC Paul, K Harris, et al. Assisted reproductive technology in Australia and New Zealand 2016. Sydney: National Perinatal Epidemiology and Statistics Unit, the University of New South Wales Sydney. 2018.
31. P Vigano, L Corti, N Berlanda. Beyond infertility: obstetrical and postpartum complications associated with endometriosis and adenomyosis. *Fertil Steril*. 2015;104(4):802-12.
32. U Leone Roberti Maggiore, S Ferrero, et al. A systematic review on endometriosis during pregnancy: diagnosis, misdiagnosis, complications and outcomes. *Hum Reprod Update*. 2016;22(1):70-103.
33. C Exacoustos, I Lauriola, L Lazzeri, et al. Complications during pregnancy and delivery in women with untreated rectovaginal deep infiltrating endometriosis. *Fertil Steril*. 2016;106(5):1129-35.

A pragmatic approach to surgical management of endometriosis

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With three placebo-controlled trials for the surgical treatment of endometriosis-associated pain, there is good evidence that surgery works to relieve pain symptoms.[1] What those trials demonstrate, however, is both a placebo response of 30 per cent – common to all medical and surgical treatments when they are studied in this manner – and a nonresponse rate of approximately 20 per cent. These figures must be kept in mind when undertaking surgical treatments for endometriosis.

For any women presenting with pelvic pain symptoms, differential diagnoses must exclude life-threatening conditions often associated with pregnancy, with chronic pain symptoms more likely to be endometriosis – the commonest single diagnosis of chronic pelvic pain in women. There are increasing data to recommend clinical diagnosis based on history taking, thorough pelvic examination (where appropriate) and limited investigations, such as a pelvic ultrasound, with recognition that high-quality sonography has increasing sensitivity at discriminating small-volume disease on the uterosacral ligaments and bowel lesions, in addition to the traditional recognition of ovarian disease.[2] It is early-stage disease that remains difficult to diagnose by sonography and serological studies are not helpful in making the diagnosis.

Medical options with simple analgesics and site-specific symptoms should be considered first line, with hormonal treatments demonstrated to be beneficial, although discontinuance of the oral contraceptive pill and progestogens may be as high as 50 per cent, owing to side effects.[3] For women who do not tolerate these medications, have side effects associated with their use or have ongoing pain symptoms despite using them, surgery may be an option for management. The first question when considering surgery is 'what is the expected outcome?' Diagnostic procedures should not be considered in modern management of endometriosis as they increase the risk to the woman with no real benefit. The plan should always be a see-and-treat approach and it should be rare that the gynaecological surgeon is surprised by the findings at laparoscopy.

A surgeon's insight into their own skillset is important. Investigation following a high index of suspicion of endometriosis based on history taking and clinical examination may reveal pelvic masses and a fixed, and often retroverted, uterus. This is an indication of high-stage disease and the surgeon must be comfortable undertaking sidewall and cul-de-sac dissection. Based on evidence, for any endometrioma there is a 99 per cent chance that there is sidewall disease,[4] and, again the treatment of both the endometrioma and the sidewall disease must be the preoperative aim and the surgeon must have this in their skillset (minimum RANZCOG-AGES level 4 procedure, with possibility of level 5 or 6 skills being required[5]) before they undertake the procedure. Even without the much-discussed deep invasive endometriosis ultrasound, a good 2D scan and clinical examination will be able to guide direction for the degree of difficulty in most surgical situations. Where a deep infiltrating endometriosis (DIE) scan is available (and affordable), this offers outstanding mapping of the pelvic disease, often informs when additional staff, such as colorectal colleagues or urologists, may be needed to contribute to the procedure and allows more tailored counselling and consenting of women preoperatively.

Many cases of endometriosis are not in the moderate–severe stage, which presents with deeply infiltrating lesions and the involvement of ovary, bowel, bladder or ureter. Peritoneal disease or deep disease affecting the uterosacral ligaments, cul-de-sac or sidewalls is common and an approach to treatment is required. There has been considerable debate over ablation versus excision and the simple fact is that it is likely to make no difference which technique you use, as long as the lesions are removed in toto. Ablation (better named vapourisation, since this is actually the effect that is desirable) is perfectly reasonable and can be carried out using standard electrosurgical electrode (such as scissors or needlepoint) and an electrosurgical generator. Vapourising the lesion with a low-power continuous waveform current will remove the

lesion with minimal thermal spread and damage to surrounding tissue that may induce cicatrisation and neuropathic sensitisation. Vapourisation of deep lesions is also perfectly reasonable but requires an understanding of the potential anatomical structures that may be injured in proximity to the lesion, and carbonisation of the tissues must be avoided since this leads to superficial insulation and an increased risk of unintended thermal injury. The technique of vapourisation does not allow for histological assessment of the abnormal tissue and it is well documented that not all lesions that are considered to be endometriosis are this disease. This is important, since while there is a definite recurrence rate with endometriosis, which may respond to a second surgical procedure; where there is no endometriosis, recurrence of symptoms and subsequent surgery may not improve the situation and, in fact, may exacerbate the problem.

Excisional surgery does allow for both histological assessment of the tissues and to gauge the depth of the lesion. The surgical principles of traction-countertraction may be readily employed with dissection of non-involved anatomical structures away from the lesion to preserve their function and prevent unintended injury. The instruments required for excision are exactly the same as for ablation, with an electrosurgical generator and an electrode (such as laparoscopic scissors) readily and inexpensively available wherever surgery is undertaken. For disease that involves the ovaries, it is imperative that not only is the ovarian disease treated, but so too is the sidewall disease removed since persistence does not allow for an adequate surgical response to be evaluated. It is also easiest to do this at the primary surgery, rather than subsequent surgery when ovarian adhesions necessitate further ovarian dissection to allow access to sidewall disease that can deplete ovarian follicular reserve and volume reduce the ovary. As a general principle, it should be considered that 'the first go is the best go' with respect of removing disease and all subsequent surgeries for endometriosis are higher risk, since the fibrosis that may be associated with previous surgical excision is difficult to distinguish from fibrosis associated with recurrent (or persistent) disease and surgical risk is increased in these cases.

Even with the very best surgical treatments and removal of all lesions in the pelvis, symptom recurrence for pain is approximately 50 per cent at five years (although actual disease recurrence is not always present) and re-assessment and evaluation is mandatory in this situation. Consideration of hormonal treatments, analgesic and neuroleptic medications, evaluation and treatment of muscular involvement by a skilled physiotherapist when appropriate, alongside simple self-management strategies for the woman including local heat, meditation, yoga and exercise may all offer benefit. Any subsequent surgery must be considered higher risk for complication and histology in this setting should be considered mandatory. Scheduled surgery in the asymptomatic woman to 'check up' on disease offers no benefit, carries risk and should not be undertaken. Evaluation as per first surgery with history, examination and sonography is good practice, and referral to centres or surgeons skilled in both the surgical and medical management of endometriosis and pain is appropriate where the primary surgeon is unfamiliar with the range of options or not confident in the increasingly complex surgical pelvis in this situation.

Women often ask how long their disease has been present, but there is no way of knowing this based on surgery. Progression is usually quite slow, with studies suggesting that no progression–minimal progression in two years is apparent for 80 per cent of women. For a small number of women, progression may be very rapid (as may recurrence). It is unclear as to the triggers for this and we have no way of currently identifying this group of women, although that would be helpful since it may improve counselling and consent. The endometriosis fertility index (EFI) is a surgically based and validated assessment tool that has prognostic implications for subsequent pregnancy and may also guide whether expectant management or IVF should be the recommendation post-operatively for women desiring pregnancy.[6] Photodocumentation should be a mandatory part of the procedure, since this may also aid in fertility counselling and EFI evaluation. Furthermore, given the range of disease presentations, the potential for further surgery and the risks associated with any subsequent procedure, pre-operative planning will be more readily facilitated by photographic evidence to determine recurrent versus new disease and for prognostic discussions with the woman.

With some 16,000 primary surgeries for endometriosis and the same number of secondary surgeries, this is a common procedure commensurate with the prevalence of the disease. The National Action Plan for endometriosis[7] has clinical care as one of its three foundation pillars and includes the recommendation of clinical care pathways for women with suspected endometriosis. Large-scale national research studies via the MRFF-funded National Endometriosis Clinical and Scientific (NECST) network[8] will further improve our knowledge in the area of the surgical management of women with endometriosis and contribute to improving the quality of life of some 10 per cent of women living with this disease globally.

References

1. Abbott J. Surgical Therapies: Randomized Controlled Trials in Endometriosis. Endometriosis: Science and Practice. Blackwell Publishing Ltd. 2012. p.410-18. DOI:10.1002/9781444398519.
2. Taylor HS, Adamson GD, Diamond MP, et al. An evidence-based approach to assessing clinical diagnosis of symptomatic endometriosis. *International Journal of Gynaecology & Obstetrics.* 2018;142(2):131-142.
3. Brown J, Farquhar C. Endometriosis: an overview of Cochrane Reviews. *Cochrane Database Syst Rev*. 2014(3):CD009590.
4. Redwine DB. Ovarian endometriosis: a marker for more extensive pelvic and intestinal disease. *Fertility & Sterility.* 1999;72(2):310-5.
5. RANZCOG. Guidelines for performing gynaecological endoscopic procedures. 2018. Available from: www.ranzcog.edu.au/RANZCOG_SITE/media/RANZCOG-MEDIA/Women%27s%20Health/Statement%20and%20guidelines/Clinical%20-%20Training/Guidelines-for-performing-gynaecological-endoscopic-procedures-(C-Trg-2)-Amended-November-2018.pdf?ext=.pdf.
6. GD Adamson, DJ Pasta. Endometriosis fertility index: the new, validated endometriosis staging system. *Fertil Steril.* 2010;94:1609-15.
7. Department of Health. National Action Plan for Endometriosis. 2018. Available from: www.health.gov.au/internet/main/publishing.nsf/Content/endometriosis.
8. Jean Hailes. The National Endometriosis Clinical and Scientific Trials (NECST) Network. 2019. Available from: https://jeanhailes.org.au/necst.

Interventional management options

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Interventions are a small, but important, component of pain management. Recognition of interdisciplinary management and the role of intervention as part of a sociopsychobiomedical paradigm of care is necessary before undertaking procedures. The management of pelvic pain should not be confined to a Cartesian model of practice, where pain is viewed as a symptom and treatment is directed to an identified cause. Pain management targets the mechanisms of pain and the goals of treatment are to improve patient quality of life and function in concordance with patient-defined goals.[1]

Gynaecologists and obstetricians have a unique role in managing pelvic pain because of their understanding of women's broader quality of life outcomes and goals that come with experiencing pelvic pain. These include sexual health, fertility and pregnancy, and bladder and bowel function.

Pain management in our specialty is not restricted to caring for women with endometriosis (Table 1). A pelvic pain service should offer support for women with any pain, collaborate with our anaesthetic and palliative care colleagues, and has a unique opportunity to address pain as it intersects with changes in a woman's life.

Accordingly, there is a breadth of indications for interventions in the management of pelvic pain. Some of the more common interventions are outlined in this article.

Botulinum toxin to the pelvic floor

Women with pelvic floor myalgia may benefit from botulinum toxin.[2] Pelvic floor myalgia arises from dysfunction in muscle and the surrounding connective tissue. It presents commonly in women with persistent pelvic pain and can be associated with endometriosis, bladder pain syndromes, vulvodynia and irritable bowel syndrome. Management is interdisciplinary and includes patient education, physical therapy, psychology and pharmacotherapy for neuropathic pain.

Women with pain refractory to these measures may be offered botulinum toxin to pelvic muscles. Botulinum toxin engenders antidystonic effects by inhibiting acetylcholine release at the neuromuscular junction. It may also have actions on afferent signalling and nociception via blockade of substance P and glutamate. Onset of action begins to occur in two to five days and reaches its nadir after two weeks. The duration of action reduces after three months.

The clinical potential of botulinum toxin has long been recognised from the 19th century, when Justinus Kerner studied the toxin from sausage poisoning on animals and, ultimately, himself.[3] Botulinum toxin is utilised widely for spasticity, dystonia, migraine and overactive bladder. Botulinum toxin to the pelvic floor can reduce dyspareunia, non-menstrual pelvic pain and vaginal pelvic floor pressures. Lower doses applied to select muscles, such as bulbospongiosus, were not found to be effective.

It is prudent to note that the application of botulinum toxin is directed to the muscle and is distinct from trigger point injection. It is not clear that the use of botulinum toxin, local anaesthetic, saline or physical therapy are superior to each other in trigger point treatment. The concept of trigger points in clinical practice has also been challenged, as trigger points may be a manifestation of altered central nociceptive mechanisms, rather than a generator of pain of itself.[4]

Pudendal block

Pudendal blocks are part of the diagnostic criteria for pudendal nerve entrapment syndrome and may be therapeutic in a subset of women with pudendal neuralgia. Women with pudendal neuralgia present with pain in the distribution of the nerve, which may include the clitoris, vagina and anus. Pudendal nerve entrapment syndrome, one type of presentation of pudendal neuralgia, is defined by Nantes criteria (Box 1).[5] When pain is relieved at pudendal block, even temporarily, the block is considered positive. Pudendal blocks are best undertaken from a transgluteal route, with the needle directed under radiological guidance to the space between the sacrotuberous and sacrospinous ligaments. A transgluteal approach is more likely to target the anatomical site of entrapment between these ligaments and block early branching of the pudendal nerve, which can occur above the ischial spine. This approach is more proximal than the vaginal approach, which blocks the nerve in Alcock's canal.

The addition of steroids is not helpful to the therapeutic effect of the block.[6] A minority of patients derive therapeutic benefit from a local anaesthetic block alone. The addition of other agents or the use of pulsed radiofrequency are being investigated.

Table 1. Typical consultations of a pelvic pain service.

Gynaecology	Persistent pelvic pain Dyspareunia Levator myalgia Peripheral neuralgias (including pudendal, iliohypogastric) Chronic post-surgical pain (including mesh related pain) Bladder pain syndrome Visceral hyperalgesia Migraine
Obstetrics	Chronic pain conditions in pregnancy including rheumatoid arthritis, SLE, Ehlers-Danlos, CRPS Opioid use in pregnancy Pelvic girdle pain Coccydynia Obstetric trauma and pudendal neuralgia Chronic pain after LSCS
Acute pain	Preventive analgesia Management of opioid-dependent patients
Cancer pain	Sexual dysfunction and pain Interventional management (including nerve blocks, intrathecal therapy)
Education	Patient – individual and group Primary health College

Older women and women who have subacute pudendal neuralgia after an event such as birth or surgery are most likely to derive therapeutic benefit.

Sympathetic blocks

Sympathetic blocks include superior hypogastric blocks and blocks to the impar ganglion. These blocks are typically undertaken in the prone position under radiological guidance. For superior hypogastric blocks, a long spinal needle is directed in a paravertebral direction to lie anterolateral to L5/S1. The impar ganglion can be accessed through a coccygeal junction, or anterior and cephalad from the tip of the coccyx, or through a paravertebral approach.

Superior hypogastric blocks have been described in cohort studies and case series, particularly in the setting of persistent pain in malignancy. Their benefit in chronic non-cancer pain is select given that visceral nociception is not defined by a single neuroanatomical pathway and that central pain mechanisms tend to be involved in visceral hyperalgesia. Patients with cancer pain undergo a diagnostic block with local anaesthetic, and if they derive improvement, then an ablation with phenol, alcohol or radiofrequency is undertaken.

The impar ganglion is the coalescence of the paravertebral sympathetic chains at the anterior surface of the coccyx. Visceral afferent fibres from the rectum travel with sympathetic fibres. Some women with coccydynia may benefit from radiofrequency treatment to the impar ganglion. Coccyx pain has mixed mechanisms, however, including from coccyx trauma, ligaments, levators and somatic innervation from the sacral plexus and women should be offered non-interventional management first.

Sacroiliac blocks

Pain derived from the sacroiliac joint may present with low back pain or posterior pelvic pain. It can be associated with joint inflammation in rheumatological diseases or with joint and ligamentous dysfunction, such as during and after pregnancy, or in connective tissue disorders. In the setting of pregnancy, exercise and physical therapy are most commonly used.

A minority of women with persistent pain may benefit from sacroiliac joint injections. Bipolar and monopolar radiofrequency ablation of the lateral sacral branches has been described, but most presentations are for back pain and to a pain clinic. Platelet-rich plasma applied to the sacroiliac ligaments is being investigated and may be an option for women with continued dysfunction or laxity despite conservative measures.

Sacral and pudendal neuromodulation

Neuromodulation is a promising avenue of management for well selected patients. Sacral neurostimulation is familiar to gynaecologists and is a well-established intervention for bladder dysfunction with leads directed to the S3 nerve root. The Sydney protocol described by Thierry Vancaillie treats pain and organ dysfunction using multiple leads, multiple waveforms and a combination of waveforms.[7]

Leads are typically placed via the sacral hiatus over the sacral dorsal root ganglia, through the S3 or S4 foramina or may include peripheral stimulation of the pudendal nerve. Mechanisms of action include altering the excitability of cells in the dorsal root ganglia involved in nociception, inhibition of wide dynamic range neurons through GABAergic or other mechanisms and possibly through effects on the brainstem and limbic system. This addresses several mechanisms of pain, including central mechanisms, with the dorsal root ganglia an important target.

Box 1: Nantes criteria

Pain in the distribution of the pudendal nerve
Pain worse on sitting
Pain does not wake at night
No objective sensory deficit features
Positive response to a pudendal block

Other techniques and targets have been described. Pudendal neuromodulation alone may be a treatment option for persistent genital arousal disorder. Conus medullaris stimulation for pudendal neuralgia has been described for patients who have failed pudendal decompression surgery. A retrograde approach to lead insertion has also been described, but is technically difficult and not widely applied.

Conclusion

The outlined interventions are helpful for well-selected patients and may be offered as part of interdisciplinary management for the woman with persistent pelvic pain. Patients may disengage or be caused psychological distress if an intervention first approach is applied, or if surgery or intervention is used as the triage to interdisciplinary management. An evaluation of the patient's functional goals and mechanisms for pain in a sociopsychobiomedical context is essential.

References

1. PJ Siddall, MJ Cousins. Persistent pain as a disease entity: implications for clinical management. *Anesth Analg*. 2004;99:510-20.
2. JA Abbott, SK Jarvis, SD Lyons, et al. Botulinum toxin type A for chronic pain and pelvic floor spasm in women. *Obstetrics and Gynecology*. 2006;108(4):915-23.
3. FJ Ergbuth, M Naumann. Historical aspects of botulinum toxin. *Neurology.* 1999; 53(8):1850-3.
4. JL Quintner, GM Bove, ML Cohen. A critical evaluation of the trigger point phenomenon. *Rheumatology.* 2015;54(3):392-9.
5. J-J Labat, T Riant, R Robert, et al. Diagnostic criteria for pudendal neuralgia by pudendal nerve entrapment (Nantes critera). *Neurourol Urodyn.* 2008;27(4):306-10.
6. J-J Labat, T Riant, A Lassaux, et al. Adding corticosteroids to the pudendal nerve block for pudendal neuralgia. *BJOG.* 2017;124(2):251-60.
7. T Vancaillie, L Kite, E Howard, et al. Sacral neuromodulation for pelvic pain and pelvic organ dysfunction. *ANZJOG.* 2018;58(1):102-7.

Hormonal management of endometriosis

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Endometriosis is a modern epidemic; it affects one in 10 women. Women present to health practitioners with a spectrum of symptoms that can affect them physically, sexually and psychosocially. Endometriosis can lead to subfertility, infertility and chronic pelvic pain. More than 73 per cent of adolescents and young adults with a history of severe and primary dysmenorrhea have endometriosis. It is estimated that 700 000 Australian women have endometriosis.

Endometriosis is characterised by endometrial-like tissue growing outside of the uterine cavity. Our current understanding of the pathophysiology of endometriosis describes dysfunction of molecular and immunological responses.[1] These alterations result in increased oestrogen synthesis and progesterone resistance, both of which drive the proliferation of endometriosis. Hormonal management aims to suppress these pathophysiological processes.

Overview of management

As diverse as the disease, women have complex and individual needs and their treatment and management should be personalised. A stepwise approach to management is well documented and involves:[2]

- a short trial of analgesics (paracetamol and/or NSAIDs) for first-line management of endometriosis-related pain
- hormonal treatment for women with suspected, confirmed or recurrent endometriosis. Table 1 summarises an approach to hormonal management in endometriosis
- combination of medical and surgical treatment
- neuromodulators and neuropathic pain treatments

Women need to be educated about pharmacological management in the setting of their disease. As a chronic disease, the balance of symptom management, side-effect profiles, fertility treatment and decreasing disease recurrence following surgery are all important.

Hormonal therapy

The overarching role of hormonal therapy in endometriosis is chronic suppression of the disease process. Hormonal treatments can be used to avoid surgical intervention, as an adjunct to surgical intervention, or post-surgical intervention.[3] By suppressing follicular growth and ovulation, a hypo-oestrogenic environment is created and ectopic endometrial growth is thus inhibited and when withdrawal bleeds are avoided so is the risk of recurrence following retrograde menstruation. Hormonal treatment is effective and tolerated in about 70 per cent of women. Available hormonal treatments are outlined below.

Combined oral contraceptive pill

The oral contraceptive creates a relative hypo-oestrogenic environment by inhibiting the mid-cycle peak of gonadal oestrogen production. The evidence suggests that low dose combined oral contraceptive pill (COCP) prescribed as continuous therapy is most effective as management of cyclical pelvic pain associated with endometriosis. The COCP is cheap and generally well tolerated with mild side effects. It may be contraindicated in women who smoke, are older than 35, have high blood pressure, breast cancer, high risk of cardiovascular disease or venous thromboembolism. As a hormonal therapy for endometriosis, the COCP can be used in in three ways. Firstly, for symptom management of primary dysmenorrhea that is not responsive to NSAIDs. Secondly, for the prevention of disease recurrence post-surgery. Thirdly, as 'add back' therapy (described below).[3,4]

Prescribe:

- **Levonorgestrel** 150 µg/**Ethinylestradiol** 30 µg daily oral dose (4x28, PBS: $17.44)

Progestins

Progestins are synthetic progesterone-like hormones, which can be administered via multiple routes (orally, implant or IUD). Progestins can act on multiple pathways, including stopping proliferation of endometrium, altering oestrogen receptors and inhibiting growth of endometriosis lesions. Furthermore, progestins have been shown to have an anti-inflammatory and anti-angiogenic activity.

Table 1. Summary of approach to hormonal management of endometriosis.

	Class	Drug
First line	Oestrogen and progestin combination	Monophasic oestrogen/progestin
Second line	Progestins	Norethisterone Medroxyprogesterone Levonorgestrel-releasing intrauterine system Dienogest
Third line	GnRH agonists	Goserelin Nafarelin
Consider	Androgenic steroids Aromatase inhibitors GnRH antagonists	Danazol Letrozole

Side effects of progestins include irregular bleeding, mood changes, breast tenderness and weight gain. Importantly, they have an intrinsic bone-sparing effect and can be used as 'add-back' therapy (see below). Progestins cannot be used in pregnancy, but can be used while breastfeeding.[3,4]

Prescribe:
- **Norethisterone acetate** 5 mg daily oral dose, for up to 6 months (1x30, PBS: $33.54)
- **Medroxyprogesterone acetate** (depot) 150 mg intramuscular injection every 12 weeks (PBS: $25.46)
- **Levonorgestrel-releasing intrauterine system** 52 mg (PBS: $208)
- **Dienogest** 2 mg/day (available on private script as Visanne)

Gonadotropin-releasing hormones agonists

Gonadotropin-releasing hormone agonists (GnRHa) induce a hypo-oestrogenic state by blocking GnRH receptors in the pituitary. Initially, this results in a transient surge of FSH and LH, but ultimately leads to a down regulation of GnRH receptors. FSH and LH levels are subsequently reduced, leading to a 'medical menopause' with many undesirable side effects. The hormonal surge may briefly cause increased endometriosis symptoms while the subsequent menopausal state may lead to hot flushes, decreased libido, mood swings and decreased bone mineral density (BMD). The current recommendations are that GnRHa therapy should be combined with 'add back' therapy to counter the side-effect profile and to protect against osteoporosis if given for longer than six months. 'Add back' therapy is the concurrent prescription of a synthetic progestin as well as the consideration of bisphosphonates and/or oestrogen to combat the side effects. As hormonal therapy for endometriosis, the GnRHa can be used in a variety of ways, including as an option to avoid surgery, adjunct to surgery or to prevent recurrence post-surgery.[3,4]

Prescribe:
- **Goserelin** subcutaneous implant 3.6 mg every four weeks for up to six months (PBS: $264)
- **Nafarelin** 200 µg intranasal twice daily (1*60 doses, PBS: $121.65)

Androgenic steroids

Androgenic steroids are synthetic hormones that inhibit ovulation by directly suppressing folliculogenesis. While androgens induce a hypo-oestrogenic state, these are less favoured due to significant and poorly tolerated androgenic side effects (hirsutism, acne) with some risk of irreversible virilisation (voice change) as well as increased metabolic risks (liver toxicity, lipid derangement).[3,4]

Prescribe:
- **Danazol** 100–400 mg orally, twice daily for three–nine months (100x100mg, PBS: $49)

Newer hormonal agents

Aromatase inhibitors

While inhibition of ovulation can lead to a hypo-oestrogenic state, research has shown that aromatase is abnormally expressed in endometriotic lesions. The enzyme aromatase converts androgens into oestrogen and local production of oestrogen is thus likely even when ovulation is suppressed. Side effects include vaginal dryness, hot flushes, headache and decreased BMD. As hormonal therapy for endometriosis, aromatase inhibitors are potent new agents with a poor side-effect profile and therefore are used when other hormonal therapies have been ineffective. It is recommended that 'add back' therapy should be initiated when commencing aromatase inhibitors.[3,4]

Prescribe
- **Letrozole** 2.5 mg daily oral dose (1x30, PBS: $31.12)

GnRH antagonists

GnRH antagonists induce a hypo-oestrogenic state by competitively blocking GnRH receptors in the pituitary. This results in immediate suppression of LH and FSH and does not cause an initial flare. GnRH antagonists have been showed to decrease symptoms and suppress disease progression. There is a lower degree of hypo-oestrogenism, which leads to a better side-effect profile than GnRHa. They have the potential to be better tolerated and an oral form is now clinically available but, as yet, not in Australia.[3,4]

Other

The use of newer and novel agents have aimed to prevent the action of oestrogens on endometriosis lesions, block receptors and inhibit enzymes. The majority of these medications have only been investigated experimentally. They include selective oestrogen receptor modulators (SERMs), selective progesterone receptor modulators (SPRMs), anti-angiogenesis factors and tumour necrosis factor alpha (TNF-a) blockers.

Clinical considerations

There are a couple clinical considerations when prescribing hormonal management.

Firstly, there is limited research into the cost effectiveness of hormonal management of endometriosis.[5] Preliminary models into the COCP showed increased quality adjusted life years (QALYs) and lower cost to no hormonal treatment. This is assumed to be a result of decreased visits to the GP and days away from work. Furthermore, GnRHa and dienogest have been demonstrated to be equally effective for long-term treatment of pain symptoms associated with endometriosis; however, have very different costs (in favour of GnRHa, which is available on PBS) and side-effect profile (in favour of dienogest).[4]

Secondly, since endometriosis is a disease most prominent during childbearing years, the main prescribing conundrum is balancing a woman's desire for fertility with the safety and side-effect profiles of medications. The outlined hormonal treatments have no fertility benefit and are generally counterproductive as they suppress ovulation or are contraceptive in other ways. GnRHa (with add back therapy) may be used in preparation for IVF in those women with endometriosis and adenomyosis seeking fertility.

Conclusion

Overall, there are three main issues in the long-term medical management of endometriosis.[3] Firstly, women experience a recurrence of symptoms following cessation of medication. Secondly, the medications have significant and sometimes intolerable side effects. Lastly, many women desire fertility. Appropriate pharmacological management has the capacity to synergistically contribute to a reduction in the impact and burden of disease at individual and population levels. New pharmacological research should focus on finding medical treatment that does not prevent or preclude pregnancy and has a favourable side-effect profile as well as being affordable.

References

1. T Falcone, R Flyckt. Clinical Management of Endometriosis. *Obstet Gynecol*. 2018;131(3):557-71.
2. GA Dunselman, Vermeulen N, Becker C, et al. ESHRE guideline: management of women with endometriosis. *Hum Reprod*. 2014;29(3):400-12.
3. S Rafique, AH DeCherney. Medical Management of Endometriosis. *Clin Obstet Gynecol*. 2017;60(3):485-96.
4. F Barra, G Grandi, G Tantari, et al. A comprehensive review of hormonal and biological therapies for endometriosis: latest developments. *Expert Opinion of Biological Therapy*. 2019;19(4):343-60.
5. TS Grand, H Basarir, LJ Jackson. The Cost Effectiveness of Oral Contraceptives compared to 'no hormonal treatment' for endometriosis-related pain: an economic evaluation. *PLoS ONE*. 2019;14(1):e0210089.

All in their mind? The stigma of pain

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Persistent pelvic pain (PPP) is considered one of the most complex and difficult pain conditions to treat.[1] Like most forms of persistent pain, PPP can have a profound impact on many aspects of life and functioning, affecting a woman's identity, ability to be intimate and the capacity to reproduce and care for children. Specific underlying causes of pelvic pain can be difficult to detect, and even where lesions, scarring, or inflammation are found, effective treatment is difficult. In a qualitative study with women experiencing medically unexplained disorders, Werner and Malterud[2] showed that women often feel a sense of shame and disempowerment as they struggle to search for a satisfactory diagnosis. Moreover, as with other conditions with unclear medical aetiology, there is a high prevalence of prior trauma, including childhood sexual abuse, and co-morbid psychiatric disorder, which add to the consequences of PPP for quality of life.[3]

In recognition of the complexity of most pain conditions, including PPP, it is not surprising that a range of psychological approaches to their treatment have been suggested. The best evidence exists for cognitive behaviour therapy (CBT). The fundamental principle of CBT is that the interpretation of events (the cognition) shapes emotional reactions and subsequent behaviour. Applied to persistent pain, this can help to understand the cycles of avoidance and distress that can arise; a woman may experience an exacerbation of pain, giving rise to the thought that 'the pain is unbearable'. This kind of thinking is sometimes referred to as 'pain catastrophising' and is known to predict a poor response to treatment.[4] As a consequence, she decides to stay home from work, which reinforces the belief that the pain is indeed unbearable. Meanwhile, she feels guilty that she has stayed away from work and misses out on the social interaction that this could have provided, which in turn, lowers her mood. Lower mood leads to lower pain tolerance, and a reduced sense of self-worth. CBT seeks to intervene at a number of points in this cycle, using interventions such as pain education, relaxation, coping skills, imagery techniques, goal setting, pacing, and challenging unhelpful thoughts. Increasingly, approaches such as mindfulness-based stress reduction or acceptance and commitment therapy (ACT) are also being used, either to supplement more traditional CBT or as approaches in their own right. With all these approaches, the goal is that the patient develops a 'toolkit' of skills and strategies that enable them to manage their pain, rather than fight against it, and carry out valued activities more effectively.

Although there is a wealth of evidence supporting the efficacy of CBT in the treatment of musculoskeletal pain, the evidence for what works with PPP is more limited. A 2014 Cochrane review of non-surgical treatments for chronic pelvic pain[5] found some evidence (albeit in low- or very low-quality studies) for reduced reporting of pain in women who had undergone reassurance ultrasound scans and received counselling, and in women who had taken part in writing therapy as a form of emotional disclosure. Two very small recent studies have reported some benefits for mindfulness interventions, and one of the better quality studies in the literature found that interpersonal therapy (IPT) was beneficial in reducing depression, but not pain severity.[6] This was a randomised controlled trial (RCT) with 61 participants, most of whom were African American, a rare example in this field of an RCT and a study conducted with non-Caucasian participants. IPT is a time-limited psychotherapy that focuses on interpersonal issues associated with both the onset and maintenance of depression, and for this study was modified to incorporate pain-related content.

Why are these outcomes so disappointing?

Firstly, it is likely that a multidisciplinary approach is more effective than psychological interventions alone.[4] Secondly, as Panisch and Tam[3] noted, very

few interventions for PPP address the history of trauma, which, arguably, will reduce their efficacy by neglecting to address underlying causes of distress. Thirdly, almost all the studies we reviewed have very small numbers of participants and are weak methodologically. This area of research may attract unusual approaches to treatment. For example, Meissner and colleagues[7] reported on the efficacy of an intervention combining the following disparate elements: mindfulness-based psychotherapy, hypnotherapy, problem-solving therapy and CBT, together with 'techniques of somatosensory stimulation from traditional Chinese medicine such as acupuncture, moxibustion (heat), and cupping'.[7]

What's the way forward for psychological interventions for PPP?

One striking feature of the studies reviewed is the absence of the voices of the women for whom these treatments have ostensibly been designed. We know very little about what women themselves find useful. We do know that some women react negatively when doctors recommend psychological therapies to them, because they feel that this means that treating clinicians don't believe their pain is real.[2] Others are reluctant to attend psychological therapy because of the stigma attached to undergoing therapy and potentially being labelled with a mental illness. In addition, many PPP patients suffer reduced mobility and find it difficult to consistently attend treatment sessions.

As part of a study in which we designed an app to help women manage their pelvic pain, we sought to overcome our lack of knowledge about what women themselves want by running a series of focus groups. We wanted to understand how they felt about receiving psychological therapy, and if they were open to receiving it via technological means. We found that the patients responded positively to the CBT and mindfulness exercises we presented and appeared open to using psychological strategies. Some reported helpful experiences with psychologists in the past, and some were already aware of how their mental states affected their experience of pain. Consistent with the literature, some women reported that they were reluctant to engage in psychotherapy, citing stigma, cost and lack of time as reasons.[8] Others were concerned about being labelled with a psychological condition that may limit medical treatments, while others feared the distress of being told that their pain was 'all in their mind'.

Based on the information gathered, we built a mobile phone app that we called appEASE. Pilot data indicate that more than 50 per cent of participants experienced clinically significant reductions in pain catastrophising and clinically significant increases in pain self-efficacy. Participants reported that by learning to cope more adaptively with pain, they were more confident doing the things they needed to do despite experiencing pain, and they attributed gaining new ways of thinking and coping from their use of the app. Other features of the app highlighted by women as particularly useful were the session on self-compassion and the sense of connection with the narrator of the sessions, suggesting that even in technology-based interventions a therapeutic relationship is important.

What can the clinician take away from our current understanding of the psychological contributions to the treatment of PPP?

Firstly, our evidence base is poor, and we have, by and large, neglected to ask women themselves what they need. Our own data suggest that women experiencing PPP sometimes feel misunderstood, but are open to psychological interventions, and should be involved in their design. Standalone psychological interventions may have a place but are likely to be more effective when integrated with interdisciplinary care that includes a gynaecologist and physiotherapist. Secondly, our study found that women experiencing PPP can feel a sense of failure, exacerbated by inconclusive diagnostic processes, so an orientation towards self-compassion may be particularly relevant for this population and their treating clinicians. Finally, much remains to be learned about how best to help this diverse, but often troubled, group of women.

References

1. P Latthe, M Latthe, L Say, et al. WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity. *BMC Pub Health.* 2006;6(1):177.
2. A Werner, K Malterud. It is hard work behaving as a credible patient: encounters between women with chronic pain and their doctors. *Soc Sci Medicine*. 2003;57:1409-19.
3. LS Panisch, LM Tam. The role of trauma and mental health in the treatment of chronic pelvic pain: A systematic review of the intervention literature. *Trauma, Violence, & Abuse*. 2019. https://doi.org/10.1177/1524838018821950.
4. C Allaire, C Williams, S Bodmer-Roy, et al. 2018. Chronic pelvic pain in an interdisciplinary setting: 1-year prospective cohort. *Am J Obstet Gynecol*. 2018;218:114.e1-12.
5. Y Cheong, G Smotra, AC Williams. Non-surgical interventions for the management of chronic pelvic pain. *Cochrane Database of Syst Rev*. 2014;(3):CD0087972014.
6. E Poleshuck, S Gamble, K Bellenger K. Randomized controlled trial of interpersonal psychotherapy versus enhanced treatment as usual for women with co-occurring depression and pelvic pain. *J Psychosom Res*. 2014;77:264–72.
7. K Meissner, A Schweizer-Arau, A Limmer, et al. Psychotherapy with Somatosensory Stimulation for Endometriosis-Associated Pain: A Randomized Controlled Trial. *Obstet Gynecol.* 2016;128:1134–42.
8. C Rini D Williams, J Broderick, FJ Keefe. Meeting them where they are: Using the internet to deliver behavioral medicine interventions for pain: Using the internet to deliver behavioral medicine interventions for pain. *Translational Behavior Med.* 2012;2:82-92.

Physio? But I've got endometriosis!

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Chronic pelvic pain (CPP) is a common clinical manifestation of endometriosis[1] and the most common reason for referral to women's health services.[2] Unfortunately, many women continue to experience pain, bladder, bowel and sexual dysfunction despite optimal gynaecological treatment. Pelvic health physiotherapy often has a part to play in a collaborative approach to treatment.

Comorbid symptoms are common and associated with dysmenorrhoea (and its severity) regardless of endometriosis presence.[3] Most people with CPP or endometriosis have negative sexual effects[4] including severe deep dyspareunia, which is associated with bladder/pelvic floor muscle (PFM) myalgia regardless of stage/location of endometriosis.[5] Poor localisation of tactile input may suggest a degree of neglect or poor motor sensory awareness.[6]

Women with endometriosis have increased prevalence of PFM and obturator internus spasm[7] and hypertonicity (non-neurogenic). Reduced flexibility, dyssynergia, altered motor control with a habitual holding pattern and reduced capacity to relax their PFM may also be present both in voluntary and functional scenarios.[7-9]

Musculoskeletal symptoms also include extrapelvic and abdominal musculature.[4] Apical breathing with a braced abdominal wall is common.[8] CPP severity is associated with abdominal wall and PFM myalgia[10] and observed in women with endometriosis.[11] Positive Carnett's sign may suggest abdominal wall or nerve irritation components to pelvic pain.[8] Much of the literature refers to myofascial trigger points, the existence of which are currently debated.[12] PFMs may contract involuntarily in response to threat[13] and occur without conscious awareness.

Trauma-informed care, mindful of possible trauma history, is important. A history of distressing sexual events is associated with an increasing number of pain symptoms in women with dysmenorrhoea.[3]

Many women experience pain, fear and anxiety related to pelvic examinations.[14] Pelvic health physios do not perform intravaginal examinations to assess sinister pathology. Management can, therefore, involve a staged and graduated approach not only to therapy but also assessment itself. Subsequent assessments by medical colleagues may then be more comfortable.

There is a growing appreciation that CPP in women with endometriosis may be a manifestation of central sensitisation, regardless of endometriosis-specific factors.[3,15,16] Amplification, viscerovisceral and viscerosomatic convergence, peripherally or at the spinal cord, explain the link between gynaecological pain and other visceral (such as bladder) or somatic (such as PFM) structures. One may initiate or exacerbate another.[15,17-19,20]

Endometriosis may be considered an initiating event that continues to trigger CPP.[8] Thoughts alone may provoke inflammatory responses.[21] For example, women often reveal that merely contemplating physical intimacy triggers their pain. Nervous system and myofascial contributions to deep dyspareunia show interindividual variation and may guide treatment, including in women requiring surgery for endometriosis.[5]

PFM myalgia,[22] hypertonicity and bladder and bowel dysfunction help identify women who may benefit from referral to pelvic health physio. Physiotherapy management is individualised and widely advocated. Addressing the complexity of pain experiences physiotherapeutically means assessing and managing local tissue issues within a broader context of sensitised protective mechanisms resulting from central nervous system (CNS) sensitivity.[6,23] This takes time. Allowing a woman to tell her story may be therapeutic in itself, permitting development of woman-centred management plans.[8,24] Fortunately, pelvic health physio appointments often provide additional time for women with complex pain presentations. Treatment seeks to build self-efficacy and reduce catastrophising, which has been associated with pelvic pain severity.[8,10,25] A focus on self-management and home-based components of management are essential.

Physiotherapy modalities may include education, PFM relaxation, manual therapy and stretches (within and around the pelvis), graded imagery and motor activity, neural dynamics, optimising bladder and bowel health, breathing and body scanning techniques, exercise, pacing and goal setting. This approach sits nicely within our current understanding of the neuroscience of pain.[6,8,26] Somatocognitive therapy of CPP, in addition to standardised gynaecological treatment, improves pain, function and psychological distress, which continue to improve beyond the completion of treatment.[27] This therapy overtly recognises the mind/body relationship and the importance of a positive working alliance,[27] shown to predict behavioural change and outcomes.[28]

Education may form the mainstay of treatment. Neuroscience-based pain education is an effective treatment in itself[29,30] and can be readily understood by patients.[31] Education and reassurance around normal laparoscopy findings can be important regarding the resolution of pain.[32] Many women have never seen images of genitalia or looked at their own vulva. Demystifying a woman's genitalia is powerful and potentially achieved via education about anatomy, function and vulval care. We need to normalise varied presentations as media images are very homogenised. Mirrors, images and visualisation may be helpful for some.[6]

Advice and education around sexual activity and the female sexual response cycle[33] with a focus on intimacy and pleasure-based, rather than fear-avoidant, activity can be helpful for women who experience dyspareunia. Fear and anticipation of pain negatively impact on the sexual response cycle.[34,35] We can explain a cycle of pain and protective muscle guarding in response to perceived or real threat.

Physiotherapy aims to rehabilitate and retrain the full range of functional capacity of PFMs, including contraction, holding, relaxing and stretching. Historically, much attention has been placed on PFM contractile activity. The ability of muscles to relax and stretch is also essential for normal function.

Ultimately, women can be assisted to identify and change habitual holding patterns, voluntarily relax PFMs and integrate this into functional activities (bladder and bowel evacuation, tampons, pelvic examinations, penetrative sexual activity). Intrapelvic manual therapy is useful for some.

Manual therapy has the potential to reduce medication requirements in women with CPP;[36] however, it may need delaying until some downregulation of a sensitised CNS has occurred.[6] There is much we still don't know about the efficacy of specific techniques and monitoring individual responses allows treatments to be individualised.

Commonly, pelvic health physios utilise digital techniques, with or without biofeedback and intravaginal devices, to improve awareness of contraction/relaxation, proprioception, confidence, knowledge and self-awareness of the vagina and PFMs, reduce hypersensitivity and stretch soft tissues. Positive physical experiences can start to modify fear-based reactions.

Adverse neural tension in pudendal, ilioinguinal, iliohypogastric, femoral or obturator nerves may contribute to pelvic pain and gentle neural mobilisation techniques can be helpful.[26] Posture and movement should be dynamic and varied, rather than static and bracing. Exercise has benefits beyond physical fitness, including regulating sleep, improving energy, mood and inflammation.

Kinesiophobia is frequently observed and fear of a pain flare may lead to reduced activity. Mechanisms by which physical activity may be protective for women with endometriosis include decreasing bioavailable oestrogens and insulin resistance, while increasing anti-inflammatory mediators.[37] Activity pacing is crucial to achieve patient-centred goals.

Many women with pelvic pain describe their experience not only in terms of physical pain but also detail the profound impact it has on their perception of being able to live a meaningful life. They talk of distress, reduced desire and arousal, avoidance of intimacy, social withdrawal, loneliness, limited employment opportunities, inability to care for those who rely on them, and intimate relationships, both with others and themselves, as crumbling. They often describe intense frustration with multiple visits to consecutive health professionals. They struggle to understand why they continue to attend countless appointments at immense personal and financial cost without any improvement, a meaningful explanation or cohesive management plan.

An interdisciplinary approach is widely recommended in the literature and international guidelines and requires all components of the program.[38-40] Unfortunately, interdisciplinary care remains very difficult for many women to access. We need to examine barriers both within ourselves and our health systems to providing this care. For those who have endometriosis, an interprofessional setting improves pelvic pain severity, quality of life and healthcare utilisation.[8] Paying equal attention to multifactorial contributors initially and concurrently with an interprofessional approach (rather than only after exclusion of organic pathology) may be more likely to achieve positive results.[8,41,42]

Pain is complex and working with this population presents many challenges and joys for clinicians. A team can support and inspire each other, provide women with consistent messages, multifactorial evidence-based explanations and management plans that help ensure comorbidities are managed optimally in a timely fashion.

Providing women with the highest possible standard of care is, of course, at the forefront of our minds. Equally, we need to value working with one another in a supportive manner in order that we may therefore continue to serve the women we hope to help. We should therefore seek to develop meaningful interprofessional communication that engenders open, respectful, curious relationships with one another and our patients.

References

1. J Aredo, K Heyrana, B Karp, et al. Relating Chronic Pelvic Pain and Endometriosis to Signs of Sensitization and Myofascial Pain and Dysfunction. *Seminars in Reproductive Medicine*. 2017;35(1):88-97.
2. P Latthe, M Latthe, L Say, et al. WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity. *BMC Public Health*. 2006;6:177.
3. SF Evans, TA Brooks, AJ Esterman, et al. The comorbidities of dysmenorrhea: a clinical survey comparing symptom profile in women with and without endometriosis. *J Pain Res*. 2018;11:3181-94.
4. R Doggweiler, KE Whitmore, JM Meijlink, et al. A standard for terminology in chronic pelvic pain syndromes: A report from the chronic pelvic pain working group of the international continence society. *Neurourol Urodyn*. 2017;36(4):984-1008.
5. NL Orr, H Noga, C Williams, et al. Deep Dyspareunia in Endometriosis: Role of the Bladder and Pelvic Floor. *J Sex Med*. 2018;15(8):1158-66.
6. S Hilton, C Vandyken. The Puzzle of Pelvic Pain—A Rehabilitation Framework for Balancing Tissue Dysfunction and Central Sensitization, I: Pain Physiology and Evaluation for the Physical Therapist. *Journal of Women's Health Physical Therapy*. 2011;35(3):103-13.
7. AP Dos Bispo, C Ploger, AF Loureiro, et al. Assessment of pelvic floor muscles in women with deep endometriosis. *Arch Gynecol Obstet*. 2016;294(3):519-23.
8. C Allaire, T Aksoy, M Bedaiwy, et al. An interdisciplinary approach to endometriosis-associated persistent pelvic pain. J*ournal of Endometriosis and Pelvic Pain Disorders*. 2017;9(2):77-86.
9. K Bo, HC Frawley, BT Haylen, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for the conservative and nonpharmacological management of female pelvic oor dysfunction. *Neurourol Urodynam*. 2016;9999:1-24.

10. A Yosef, C Allaire, C Williams, et al. Multifactorial contributors to the severity of chronic pelvic pain in women. *Am J Obstet Gynecol*. 2016;215(6):760.e1-.e14.
11. J Jarrell. Endometriosis and abdominal myofascial pain in adults and adolescents. *Current Pain and Headache Reports*. 2011;15(5):368-76.
12. JL Quintner, GM Bove, ML Cohen. A critical evaluation of the trigger point phenomenon. *Rheumatology (Oxford)*. 2015;54(3):392-9.
13. J van der Velde, W Everaerd. The relationship between involuntary pelvic floor muscle activity, muscle awareness and experienced threat in women with and without vaginismus. *Behav Res Ther*. 2001;39(4):395-408.
14. HE Bloomfield, A Olson, N Greer, et al. Screening pelvic examinations in asymptomatic, average-risk adult women: an evidence report for a clinical practice guideline from the American College of Physicians. *Ann Intern Med*. 2014;161(1):46-53.
15. J Brawn, M Morotti, KT Zondervan, et al. Central changes associated with chronic pelvic pain and endometriosis. *Hum Reprod Update*. 2014;20(5):737-47.
16. P Stratton, I Khachikyan, N Sinaii, et al. Association of chronic pelvic pain and endometriosis with signs of sensitization and myofascial pain. *Obstet Gynecol*. 2015;125(3):719-28.
17. E Ustinova, M Fraser, M Pezzone. Cross-talk and sensitization of bladder afferent nerves. *Neurourology and Urodynamics*. 2010;29(1):77-81.
18. MA Pezzone, R Liang, MO Fraser. A model of neural cross-talk and irritation in the pelvis: implications for the overlap of chronic pelvic pain disorders. *Gastroenterology*. 2005;128(7):1953-64.
19. MA Giamberardino, R Costantini, G Affaitati, et al. Viscero-visceral hyperalgesia: characterization in different clinical models. *Pain*. 2010;151(2):307-22.
20. A Latremoliere, CJ Woolf. Central sensitization: a generator of pain hypersensitivity by central neural plasticity. *J Pain*. 2009;10(9):895-926.
21. GL Moseley, N Zalucki, F Birklein, et al. Thinking about movement hurts: the effect of motor imagery on pain and swelling in people with chronic arm pain. *Arthritis Rheum*. 2008;59(5):623-31.
22. CE Neville, CM Fitzgerald, T Mallinson, et al. A preliminary report of musculoskeletal dysfunction in female chronic pelvic pain: a blinded study of examination findings. *J Bodyw Mov Ther*. 2012;16(1):50-6.
23. J Nijs, B Van Houdenhove, RA Oostendorp. Recognition of central sensitization in patients with musculoskeletal pain: Application of pain neurophysiology in manual therapy practice. *Man Ther*. 2010;15(2):135-41.
24. SJ Moore, SH Kennedy. K. The initial management of chronic pelvic pain. Green-top Guideline No. 41. Royal College of Obstetricians and Gynaecologists. 2012. Available from: www.rcog.org.uk/globalassets/documents/guidelines/gtg_41.pdf.
25. G Desrochers, S Bergeron, S Khalife, et al. Provoked vestibulodynia: psychological predictors of topical and cognitive-behavioral treatment outcome. *Behav Res Ther*. 2010;48(2):106-15.
26. C Vandyken, S Hilton. The Puzzle of Pelvic Pain. J*ournal of Women's Health Physical Therapy*. 2012;36(1):44-54.
27. GK Haugstad, U Kirste, S Leganger, et al. Somatocognitive therapy in the management of chronic gynaecological pain. A review of the historical background and results of a current approach. *Scand J Pain*. 2011;2(3):124-9.
28. AM Hall, PH Ferreira, CG Maher, et al. The influence of the therapist-patient relationship on treatment outcome in physical rehabilitation: systematic review. *Phys Ther*. 2010;90(8):1099-110.
29. A Louw, EL Puentedura, P Mintken. Use of an abbreviated neuroscience education approach in the treatment of chronic low back pain: a case report. *Physiother Theory Pract*. 2012;28(1):50-62.
30. A Louw, I Diener, DS Butler, EJ Puentedura. The effect of neuroscience education on pain, disability, anxiety, and stress in chronic musculoskeletal pain. *Arch Phys Med Rehabil*. 2011;92(12):2041-56.
31. L Moseley. Unraveling the barriers to reconceptualization of the problem in chronic pain: the actual and perceived ability of patients and health professionals to understand the neurophysiology. *J Pain*. 2003;4(4):184-9.
32. AFF Ghaly. The psychological and physical benefits of pelvic ultrasonography in patients with chronic pelvic pain and negative laparoscopy. A random allocation trial. *Journal of Obstetrics and Gynaecology*. 1994;14(4):269-71.
33. R Basson. Women's sexual dysfunction: revised and expanded definitions. *CMAJ: Canadian Medical Association Journal*. 2005;172(10):1327-33.
34. N Di Donato, G Montanari, A Benfenati, et al. Do women with endometriosis have to worry about sex? *Eur J Obstet Gynecol Reprod Biol*. 2014;179:69-74.
35. N Fritzer, D Haas, P Oppelt, et al. More than just bad sex: sexual dysfunction and distress in patients with endometriosis. *Eur J Obstet Gynecol Reprod Biol*. 2013;169(2):392-6.
36. RUAnderson, RH Harvey, D Wise, et al. Chronic pelvic pain syndrome: reduction of medication use after pelvic floor physical therapy with an internal myofascial trigger point wand. *Appl Psychophysiol Biofeedback*. 2015;40(1):45-52.
37. L Buggio, G Barbara, F Facchin, et al. Self-Management and psychological-sexological interventions in patients with endometriosis: strategies, outcomes, and integration into clinical care. *International Journal of Womens Health*. 2017;9:281-93.
38. LD Kames, AJ Rapkin, BD Naliboff, et al. Effectiveness of an interdisciplinary pain management program for the treatment of chronic pelvic pain. *Pain*. 1990;41(1):41-6.
39. JF Jarrell, GA Vilos, C Allaire, et al. No. 164-Consensus Guidelines for the Management of Chronic Pelvic Pain. *Journal of Obstetrics and Gynaecology Canada*. 2018;40(11):e747-e87.
40. S Loving, J Nordling, P Jaszczak, T Thomsen. Does evidence support physiotherapy management of adult female chronic pelvic pain? A systematic review. *Scand J Pain*. 2012;3(2):70-81.
41. AA Peters, E van Dorst, B Jellis, et al. A randomized clinical trial to compare two different approaches in women with chronic pelvic pain. *Obstet Gynecol*. 1991;77(5):740-4.
42. SPK Friggi, M Garcia, C Petta, et al. Physical therapy and psychological intervention normalize cortisol levels and improve vitality in women with endometriosis. *Journal of Psychosomatic Obstetrics and Gynecology*. 2012;33(4):191-8.

Multidisciplinary team management of chronic pelvic pain

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Chronic pelvic pain (CPP) is a common presentation to GPs and gynaecologists. Effective management of CPP, however, is often found to be challenging. A 2018 survey[1] of UK gynaecologists found that 45 per cent felt they were unable to adequately manage CPP, and a 2016 audit of gynaecologists at a tertiary women's hospital in NZ found that only 7 per cent rated the quality of their CPP assessment as 'good' and none as 'excellent'.[2]

The ICD-11 defines chronic pain as 'Pain that persists or recurs for more than three months' and, unlike acute pain, represents a distinct disease state in itself. An accumulating body of evidence demonstrates that CPP shares the same mechanisms and comorbidities of other chronic pain syndromes.[3-5]

Lesion-focused management

In practice, persistent pelvic pain is often not recognised as a distinct diagnosis; with clinical focus remaining entirely on attempting to locate and remediate an assumed end-organ pathology. This lesion-focused approach is at odds with the well-recognised multidisciplinary approaches recommended for other persistent pain conditions. This means that the mainstay of management of pelvic pain remains heavily biomedical and end-organ focused. This approach neglects the influence of the surrounding structures and wider nervous system inputs on pain, as well as the wider pain-related symptoms and distress that women living with pelvic pain experience.[5,6]

There is also often a mistaken belief that persisting pelvic pain is always a sign of, or directly synonymous with, endometriosis; thus, surgical investigation and intervention are imperative. This is a view that is unfortunately strongly advocated for within many media forums. It is consistently reported in the literature, however, that there is no good correlation between stage or laterality of lesions and symptoms experienced, and over a third of laparoscopies performed for this indication find no pathology.[7,8]

Women with persisting pelvic pain report a wide range of extra-pelvic pain syndromes, such as migraine, irritable bowel syndrome and widespread musculoskeletal or myofascial pain syndromes.[5,9] A focus centred on the presence of visible lesions risks ignoring these, and other, comorbid difficulties that are frequently experienced alongside chronic pain, such as anxiety and depression, chronic fatigue, neurocognitive changes, and the associated impacts on social relationships, work and productivity.

This lesion-focused approach can also creates a false dichotomy; implying that normal investigations suggest pain is somehow the fault of the patient, or imagined; when it has been shown that those with pelvic pain and no demonstrable lesions share the same characteristics[9,10] and central changes as those with them.[3,4] This risks leaving the patient feeling there are no treatment options for their pain and unnecessarily exacerbates distress.

Current management of pelvic pain usually comprises of hormonal manipulation or surgical procedures. Neither of these approaches has been shown to be superior to the other,[6-8] nor to bring long-term improvement. Medical therapy has been shown to improve quality of life in up to two thirds of women; however, it requires long-term use and can bring significant side effects. Surgery often results in only partial or temporary pain relief, with one-in-five experiencing no improvement and about half of patients who do benefit experiencing symptom recurrence by two-year follow up.[7-8]

It is increasingly recognised that repeated laparoscopic procedures are neither successful nor appropriate management for pelvic pain.[7,8] However,

data from health insurer Southern Cross NZ places surgery for endometriosis within the top five across all surgeries for women between the ages of 21–50. A 2014 patient poll by Endometriosis NZ also demonstrated the large number of these procedures being undertaken, reporting a mean number of four surgeries per respondent, with a range from 1–25. Despite this, 70 per cent of respondents who had undergone surgery reported no improvement in pain.

The benefits of multidisciplinary team managment

Recommended treatments for persistent pain, in comparison, include biomedical approaches as just one component in management. This allows for all inputs driving ongoing pain to be addressed; a multifactorial approach that optimises outcomes for women living with pelvic pain.

The benefits of multidisciplinary team (MDT) management for people living with persisting pain has been recognised for over four decades,[11,12] with the supporting evidence base continuing to grow as knowledge about pain neurobiology increases.

The adoption of this model to pelvic pain has, however, been lagging; despite mounting evidence that CPP shares the same mechanisms and comorbidities of other chronic pain syndromes,[3,9,10] and over 30 years of evidence that multimodal MDT approaches to CPP demonstrate superiority over solely biomedical ones in quality-of-life outcomes, cost-effectiveness, iatrogenic risk and reducing future surgeries and emergency department visits.[13-16]

Symptom-focused approaches: treating the whole person

Unlike unimodal approaches for CPP, pain specialist MDTs have expertise both in scope of practice and specialist pain knowledge in order to address the multifactorial contributors of pain and associated comorbidities.

MDT approaches are multi-pronged, to address both bottom-up and top-down contributors to ongoing pain. While the scopes of team members may vary depending on the needs of an individual patient, professions involved typically include: pain specialist doctors, physiotherapists, psychologists, occupational therapists, specialist nurses and social workers; all with specialist understanding of pain management. For pelvic pain, in particular, pelvic floor physiotherapists and therapists with training in sexual disorders are also included.

For all professionals working in pain, it is not sufficient to simply hold a qualification in their scope. Just as different areas of medicine require some specialism, appropriate education, experience and supervision pertaining to pain is essential to ensure that unhelpful messages about pain are not being inadvertently reinforced by practitioners who are not adequately schooled in the complexities and subtleties of this disease.

All in their heads?

Pain is by IASP definition 'a sensory and emotional experience'. The significant contribution of psychosocial elements to the development and maintenance of persistent pelvic pain is well recognised in the literature as an important target for treatment to improve outcomes, though largely ignored within clinical practice. The Australasian Faculty of Pain Medicine recognises this key element to managing pain and has reconceptualised the required approach to a sociopsychobiomedical one.

Figure 1. MDT management can be likened to a door with many locks; unlocking one is insufficient to allow patients to open the door. Instead, multiple locks need to be targeted in order to free women from the constraints of ongoing pain.

Many patients are initially wary of the involvement of psychology or psychosocial elements of pain management, as this suggests to them that practitioners believe the pain that they experience is in some way imaginary or psychological. Unfortunately, this also seems to be the belief of many other health practitioners who see the role of psychologists within pain management as one of simply support or distress tolerance, or there to intervene with 'histrionic' or 'psychosomatic' patients. Education about the place of psychosocial elements within pain management is therefore a vital part of engaging patients in treatment, as well as helping them to use all the specialities that can improve their experience where these are available to them.

Common themes of MDT approaches include education about pain and how pain works and what happens in the pain system when pain becomes persistent. Pain management promotes active and adaptive approaches that provide experiential exposure to feared and painful activity in a safe and supported way. These programs aim to reduce fear-avoidance, increase activity tolerances, redress functional losses, increase pain-self efficacy, improve quality of life, and, for some patients, decrease or even eliminate pain.

Role of the doctor within an MDT

The medical specialist working within a chronic pain MDT has many roles, as identified in the CanMEDS framework. Specialist pain medicine physicians work to understand the current and previous pain history, review and collate patient history, balancing the need to rule out 'red flag conditions' with iatrogenic harms from over investigation, and act as a health educator and advocate for both patients and other MDT members.

Unfortunately, there is a paucity of specialised MDT pelvic pain centres, so all gynaecologists who work with women with pelvic pain need to develop the above skills, rather than acting solely as a 'surgical technician', and then engaging in endless 'speciality ping-pong' referrals to other end-organ specialists when a laparoscopy is normal or fails to resolve symptoms. A 2016 audit found that the majority of the surveyed gynaecologists felt their ability to explore underlying non-gynaecological and psychosocial contributors to pain was poor,[2] indicating a need for greater attention to this during medical training.

There is often some reluctance to accept both self-management and MDT approaches from both the general public and some clinicians, so even when MDT pain clinics are available, there is a need for the MDT approach to be supported and validated within gynaecology clinics and by the wider gynaecological speciality so that patients receive a consistent message and are supported to see that there are alternatives or adjuncts to biomedical therapies that have fewer side effects and may have greater long-term benefits for wellbeing and quality of life.[5,8,16] This also prevents dependence on passive service provision and helplessness to manage pain and pain-related distress during times of symptom flare ups.

Shifting focus from the pelvis to the person who lives with pain

Current management of pain perceived within the pelvis focuses on locating and removing lesions in the hopes of eliminating pain, even though the relationship between lesions and symptoms is poorly correlated and this approach often unsuccessful.

CPP needs to be recognised as a distinct disease state that has a specific treatment framework that focuses on all contributors and symptoms.

As growing evidence base attests, an MDT approach for persistent pain conditions offers the greatest chance for best outcomes, and as with other conditions, clinical outcomes are better where evidence-based guidelines are followed. Further education, investment and systemic change will be required before women living with pelvic pain are able to access the services they deserve.

References

1. HW Leow, W Szubert, AW Horne. 45% of UK gynaecologists think chronic pelvic pain is managed badly. *European Journal of Obstetrics and Gynecology and Reproductive Biology*. 2018;224:200-2.
2. K Joseph. Unmet healthcare-needs audit of attendees with chronic pelvic pain at the Christchurch Women's Hospital Outpatient Clinic. 2016. Unpublished data.
3. J Brawn, M Morotti, KT Zondervan, et al. Central changes associated with chronic pelvic pain and endometriosis. *Hum Reprod Update*. 2014;20(5):737-47.
4. S As-Sanie, RE Harris, V Napadow, et al. Changes in regional gray matter volume in women with chronic pelvic pain: a voxel-based morphometry study. *Pain*. 2012;153(5):1006-14.
5. S As-Sanie, R Black, LC Giudice, et al. Assessing research gaps and unmet needs in endometriosis. *American Journal of Obstetrics and Gynecology*. pii: S0002-9378(19)30385-0.
6. A Yunker, NA Sathe, WS Reynolds, et al. Systematic review of therapies for noncyclic chronic pelvic pain in women. *Obstetrical & Gynecological Survey*. 2012;67(7):417-25.
7. P Vercellini, PG Crosignani, A Abbiati, et al. The effect of surgery for symptomatic endometriosis: the other side of the story. *Hum Reprod Update*. 2009;15(2):177-88.
8. P Vercellini, F Facchin, L Buggio, et al. Management of endometriosis: toward value-based, cost-effective, affordable care. *Journal of Obstetrics and Gynaecology Canada*. 2018;40(6):726-49.
9. SF Evans, TA Brooks, AJ Esterman, et al. The comorbidities of dysmenorrhea: a clinical survey comparing symptom profile in women with and without endometriosis. *Journal of Pain Research*. 2018;11:3181.
10. H Grundström, B Gerdle, S Alehagen, et al. Reduced pain thresholds and signs of sensitization in women with persistent pelvic pain and suspected endometriosis. *Acta Obstetricia et Gynecologica Scandinavica*. 2019;98(3):327-36.
11. WE Fordyce, R Fowler, JF Lehmann, et al. Operant conditioning in the treatment of chronic pain. *Archives of Physical Medicine and Rehabilitation*. 1973;54(9):399-408.
12. H Flor, T Fydrich, DC Turk. Efficacy of multidisciplinary pain treatment centers: a meta-analytic review. *Pain*. 1992;49(2):221-30.
13. AA Peters, B Jellis, J Hermans, JB Trimbos. A randomized clinical trial to compare two different approaches in women with chronic pelvic pain. *Obstetrics and Gynecology*. 1991;77(5):740-4.
14. RC Reiter, JC Gambone, SR Johnson. Availability of a multidisciplinary pelvic pain clinic and frequency of hysterectomy for pelvic pain. *Journal of Psychosomatic Obstetrics & Gynecology*. 1991;12(sup1):109-16.
15. JC Gambone, RC Reiter. Nonsurgical management of chronic pelvic pain: a multidisciplinary approach. *Clinical Obstetrics and Gynecology*. 1990;33(1):205-11.
16. C Allaire, C Williams, Bodmer-Roy, et al. Chronic pelvic pain in an interdisciplinary setting: 1-year prospective cohort. *American Journal of Obstetrics and Gynecology*. 2018;218(1):114-e1.

Mater Mother's Hospital: acute management of persistent pelvic pain

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Persistent pelvic pain (PPP) affects 15–26 per cent,[1,2] of women. It is defined by pelvic pain for more than 3–6 months that is not solely related to menstruation, sexual activity or bowel movements.[3] PPP is caused by a complex combination of visceral and musculoskeletal pain, central sensitisation and pelvic floor hypertonicity, often accompanied by evolving psychological dysfunction.[3-6] Acute exacerbations (flares) of pelvic pain are common and often triggered by menstruation, constipation, UTI/bladder pain or pelvic muscle spasm.[7] Flares can last days to months and the patient will often have significant fear and anxiety relating to escalation of their pain. The result is frequent presentation to primary care providers and the emergency department (ED), where previously normal investigations are often repeated and admission (with repeat diagnostic laparoscopy) undertaken. This scenario is frustrating to both patient and clinician and results in excess hospital expenditure. We developed a guideline for acute management of PPP flares with the aim of preventing unnecessary investigation, admission and surgery. Management focuses on identification and treatment of specific triggers while providing validation, reassurance and education to the patient.

Persistent pelvic pain: the burden

PPP poses a significant societal and healthcare burden. In Australia, the cost of medical and surgical treatment for endometriosis alone amounts to over six billion dollars per year. Globally, this results in 11 hours and $200–250 lost per woman, per week, due to absenteeism and presenteeism, where an employee is working with reduced productivity.[8] Given that many ED presentations result in admission and diagnostic laparoscopy, the cost of which is up to $4289 USD,[9] there is clearly benefit to be gained from avoiding such intervention in the previously investigated PPP patient, when clinically appropriate. Additionally, if adequate education and reassurance can be provided in the acute setting, the patient may be more likely to accept ongoing outpatient management.

It is well established that a multidisciplinary approach to the management of PPP results in improved patient outcomes.[10-12] In June 2017, the Mater Mothers' Hospital Persistent Pelvic Pain Clinic (PPPC) was opened with the dual aims of improving patient outcomes and reducing hospital costs associated with unnecessary admissions or procedures. The changes made include the development of guidelines for the acute management of pelvic pain in the ED, and an effective multidisciplinary team approach to management in the clinic.

Management in the emergency department

The following guidelines provide a consistent approach to management following exclusion of acute intra-abdominal pathology. They educate women on strategies to improve their pain over the longer term, and support them to manage pain flares, avoiding the need for unnecessary presentation to ED. The principles of management are:

- Exclude acute intra-abdominal pathology
- Confirm symptoms consistent with an exacerbation of long-term pain
- Recognise and manage the likely trigger for the recent flare of pain
- Appropriate analgesia with avoidance of opioids
- Avoidance of repeat laparoscopy
- Assessment of psychological stressors and risk of self-harm
- Reassurance and acknowledgement of the patient's pain despite normal investigations
- Education and emphasis on self-management
- Appropriate follow up

Ongoing management in the PPPC

As of November 2018, 58 patients have commenced in the service. The core team includes advanced

History

A thorough, but timely, patient history should be obtained that is not biased by previous admissions or diagnosis

- Symptoms suggestive of pelvic muscle spasm include pain of sudden onset, unilateral or bilateral location, pain worse with movement, referring to anterior thigh, tender lower back/gluteal region, often described as 'stabbing' and may be related to a period of overactivity or stress
- Surgical history: prior laparoscopic surgery and findings
- Psychiatric history with assessment of risk of self-harm and current psychological symptoms of depression and/or anxiety, recent significant life events
- Medication history: opioids and doses, chronic pain medications (eg. amitriptyline/gabapentin/pregabalin/duloxetine), psychiatric medications
- Current engagement with and names of gynaecologist, pain services, psychiatrist, psychologist, pelvic floor physio

Examination

The patients examination should ideally be performed in a safe, and comfortable environment by an experienced practitioner

- Basic observations and abdominal palpation to elicit signs of peritonism that may indicate an anatomical cause
- Speculum examination may be warranted if history of bleeding or vaginal discharge, otherwise this may be avoided
- Vaginal examination: bimanual evaluation of the uterus and adnexa for localised tenderness or masses
- Pelvic floor examination: pelvic floor hypertonicity will be evident in most women with PPP. It is acute spasm of these muscles that contributes significantly to pain flares. Directed examination of the vulva, pubococcygeus, puborectalis, piriformis and obturator internus can localise the causative muscle group and replicate the patient's pain. This should only be undertaken by a clinician experienced in pelvic floor examination.

Investigations

The patients investigations should be performed to support clinical findings and not used in a 'scattergun', cover all possibilities approach

- Urine for exclusion of pregnancy and assessment of infection if indicated
- Consider blood tests and imaging only if clinical evidence of alternate pathology
- If indicated, USS is the best imaging modality to define acute pelvic pathology

Management

- Treat acute pathology as indicated
- Reduce fear and enhance management with an explanation of exacerbation of their long-term pain and the likely trigger for the flare where this is known, such as acute pelvic muscle spasm where this has been demonstrated
- Address reversible causes, such as constipation, UTI, dysmenorrhoea, hypertonic pelvic floor
- Non-pharmacological management: heat pack, mindfulness/deep breathing
  - Encourage patient to breathe to RR of 6
- Stepwise analgesia: IV/PO Paracetamol, PR Voltaren/IV Parecoxib if vomiting or PO Ibuprofen
  - Pregabalin 25–75 mg PO may also be helpful if there is a component of anxiety and pain related to central sensitisation
- For pelvic floor muscle spasm: PV or PR diazepam 5 mg[13] (in a fatty base, made by a compounding pharmacist), refer to gentle pelvic stretches available at: www.pelvicpain.org.au/for-women/easy-stretches-to-relax-the-pelvis-women
- For painful bladder symptoms: Ural or 500 mL water with 1 tbsp bicarb soda, increase PO fluid intake.
- For constipation:
  - Mild: movicol 2 sachets daily
  - Moderate: movicol 2–4 sachets daily, 2 dulcolax tabs mane until bowel movement
  - Severe: 2 microlax enemas, 3 dulcolax tablets and 8 sachets of movicol in 1L of liquid to drink over 12 hours
- For dysmenorrhea:
  - Diclofenac suppository 100 mg PR
- Opioids increase central sensitisation when used regularly and should be avoided where possible when acute pathology is excluded. Options if required:
  - Tramadol 50–100 mg PO or slow IV, beware of risk of serotonin syndrome with concomitant use of SSRI/SNRIs
  - Tapentadol IR 50 mg PO
  - Temgesic (Buprenorphine) 0.2–0.4 mg SL (this is equivalent to 10–20 mg PO morphine, therefore care with dosing in opioid naive)
  - If patients are on SR or IR opioids already, their treating GP must remain the sole prescriber
  - Opioids (SR or IR) generally should not be given for discharge. If opioids are administered in the ED, consideration should be given to co-administration of aperients such as movicol, rather than stimulants such as coloxyl/senna
- Encourage gentle mobilisation rather than bed rest to reduce muscle spasm
- Provide the patient with written information on pelvic pain, eg. Pelvic Pain Foundation of Australia (www.pelvicpain.org.au)
- Early follow up with primary healthcare provider, gynaecologist or persistent pelvic pain clinic

laparoscopic gynaecologist, pain specialist, psychologist and physiotherapist, with referral pathways to psychiatry, functional gastroenterology and colorectal surgeons. The structure comprises a six-month program with an initial, three- and six-month visit, as well as regular physiotherapy and psychology intervention. A general summary of long-term management in the PPPC includes:

- Emphasis on self-management and expectation setting: aim for pain reduction to a level that facilitates improved functioning and a return to usual activities with ongoing management planned as outpatient
- Provision of written and verbal information regarding long-term treatment and management of pain flares
- Complete laparoscopic excision of endometriosis by experienced endometriosis surgeon, as clinically indicated
- Achieving amenorrhoea with hormonal suppression
- Weaning off opioid medication
- Modulation of pain pathways using antidepressants (amitriptyline/duloxetine) or membrane stabilisers (gabapentin/pregabalin)
- Physiotherapy for pelvic floor downtraining with experienced women's health physiotherapist
- Psychology/psychiatric input
- Persistent Pain Education module
- Administration of pelvic floor botulinum toxin as clinically indicated

The acute exacerbation of PPP can be a frustrating encounter for both patient and clinician, frequently resulting in unnecessary intervention that yields little information or clinical improvement. By directing acute management at diagnosing and treating triggers, providing education and instituting multidisciplinary follow up, the patient can potentially be adequately managed as an outpatient, avoiding admission and its associated problems. As part of an auditing cycle, the Mater Mothers' PPPC is collecting prospective outcome data on all clinic attendances and is in the process of integrating with the Electronic Persistent Pain Outcomes Collaboration system.

References

1. Latthe P, Latthe M, Say L, et al. WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity. *BMC Public Health*. 2006;6:177.
2. Grace V, Zondervan K. Chronic pelvic pain in New Zealand: prevalence, pain severity, diagnoses and use of the health services. *ANZ J Pub Health*. 2004;28:369-75.
3. Jarrell JF, Vilos GA, Allaire C, et al. Consensus guidelines for the management of chronic pelvic pain. *J Obstet Gynaecol Can*. 2005;27:781-826.
4. Alappattu MJ, Bishop MD. Psychological factors in chronic pelvic pain in women: relevance and application of the fear-avoidance model of pain. *Phys Ther*. 2011;91:1542-50.
5. Kwon JK, Chang IH. Pain, catastrophizing, and depression in chronic prostatitis/chronic pelvic pain syndrome. *Int Neurourol J*. 2013;17:48-58.
6. Meltzer-Brody S, Leserman J. Psychiatric comorbidity in women with chronic pelvic pain. *CNS Spectr*. 2011;16:29-35.
7. Evans S. Management of Persistent Pelvic Pain in Girls and Women. *Aust Fam Phys*. 2015;44:454-9.
8. Bush D, Evans S, Vancaillie T. The Pelvic Pain Report: The $6 billion woman and the $600 million dollar girl. 2011, Australia.
9. Fuldeore M, Chwalisz K, Marx S, et al. Surgical procedures and their cost estimates among women with newly diagnosed endometriosis: a US database study. *J Med Econ*. 2011;14(1):115-23.
10. Allaire C, Williams C, Bodmer-Roy S, et al. Chronic pelvic pain in an interdisciplinary setting: 1 year prospective cohort, *Am J Obstet Gynecol*. 2018;218(1):114.e1-12.
11. Allaire C, Aksoy T, Bedaiwy M, et al. An interdisciplinary approach to endometriosis-associated persistent pelvic pain. *J Endo Pelvic Pain Disord*. 2017. doi:10.5301/jeppd.5000284.
12. Stanos S, Houle TT. Multidisciplinary and interdisciplinary management of chronic pain. *Phys Med Rehabil Clin N Am*. 2006;17:435-50.
13. Rogalski MJ, Kellogg-Spadt S, Hoffmann AR, et al. Retrospective chart review of vaginal diazepam suppository use in high-tone pelvic floor dysfunction. *Int Urogynecol J*. 2010;21(7):895-9.

Does endometriosis really cause pain?

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It's an outrageous question, and it's meant to be outrageous; however, it sets the state of mind when approaching pelvic pain. Too many colleagues will 'look for endometriosis' rather than 'look for pain'. That's fundamentally wrong in the context of our current understanding of pain. The answer to the question 'Does endometriosis really cause pain?' is 'yes, it can, but…' Pain is very much like haemostasis, it involves a cascade of events, with the pain cascade the more complex one. One can argue that endometriosis is merely a variation of normal. The tissue is 'normal', it belongs to the patient and in most cases behaves in a predictable physiological manner. Why the endometrial tissue ends up in the wrong place is still a mystery. How the tissue in the wrong place, or not, can lead to pain is far better understood now than a few decades ago.

Endometrial tissue can trigger inflammatory and immune responses through its activity, such as release of prostaglandins. Regardless of location, this can trigger pain. Peripheral receptors are triggered, a signal in the sympathetic nervous system is generated, multiple connections occur in the pre-vertebral chain, the spinal cord and finally the brain. The list of factors influencing this cascade of neurotransmission is long and unfinished.

One important element, which influences the individual's response to a trigger, is the hormonal environment.[1] Oestrogen is particularly active, not only in the direct trophic stimulation of the endometrial tissue, but also in the interpretation of the signal at a central level. Understanding the impact of oestrogens completely alters the approach to treating the patient with pelvic pain. When a patient presents with pelvic pain, it is as important to take a full pain history – to find out if she also suffers headaches, for instance – as it is to perform a thorough pelvic examination. Headaches can indeed also be significantly modulated by the hormonal environment. Manipulation of the hormonal environment, therefore, is and will remain an important element in the treatment of pain.

Pain is normal. In days gone by, this would seem like another outrageous statement, but not in the context of our current understanding of pain. The function of pain is to protect us from danger – it is one of our homeostatic mechanisms. Pain is the interpretation of a sensory signal by our brain and is designed to receive our full attention. And now the difficult part begins: how does the brain interpret these signals and determine how much pain to provide as the output? How significant is that signal? What does it mean for me now, in context, according to my past, and what does it mean for my future? The brain will do this assessment amazingly quickly. These questions come up every day and this question is also one of the main issues a patient brings to the consultation: why am I hurting? Often, finding and understanding the answer to that question is enough to 'treat' the pain. Therefore, it is exceedingly important to answer the question 'does endometriosis really cause pain?' correctly. Telling the patient that endometriosis always equals pain is wrong and only reinforces the quest for more surgery – and poorer results.

We need to change our treatment algorithm. That's not so outrageous a statement. Great progress has been made over the past decades in approaching resection of endometriosis using laparoscopic techniques. However, we need to move laparoscopy for resection of endometriosis off its pedestal and place it in the context of pain management of the whole person and stop promoting surgery as a cure for pelvic pain.[2]

The main impediment to a change of attitude toward treating pelvic pain is the stubborn belief among physicians, as well as the public at large, that surgery can remove pain. In many cases, surgery will indeed improve the patient's symptoms, but rarely does it actually 'cure' the pain. As a matter of fact, laparoscopic surgery itself can turn into an important trigger for pain, as any surgery can trigger post-surgical neuropathy. At the first laparoscopy for resection of endometriosis, the surgeon removes one element in the cascade of pain genesis, but introduces a new one – the surgery itself. With each subsequent surgery, the weight of the introduced trigger threatens to tip the balance in favour of greater, rather than reduced, pain triggers and leads to an even more sensitised nervous system. A better understanding of the pathophysiology of pain requires education. Improved pain education is needed within the curriculum of medical schools. Improved pain education is needed among surgical specialists. Improved pain education should reach the general public.[3]

Once it is understood that it's not the location of the tissue, but the interaction of the tissue with its environment regardless of location, that is at the origin of the pain cascade, then it opens up a whole new world of possibilities for treatment. Rather than feeling threatened, the physician-surgeon should embrace this opportunity of providing a greater positive impact on the patient's health and wellbeing. The lack of understanding of pain pathophysiology is reflected in the attitude of physicians toward allied health. How could osteopathy or physiotherapy or psychotherapy possibly remove the pain from endometriosis? Posited that way, it does seem impossible: ectopic endometrium will not vanish after a session of pelvic floor muscle manipulation. Within the context of pain pathophysiology, where pain triggers do correlate with aberrant muscle contractions, it becomes quite understandable, however. The pervasive false belief that surgery cures pain is reflected in the medical insurance and government reimbursement systems. The rebate for surgery still dominates, whereas reimbursement for pain education is essentially non-existent. Yet, education is at least equally important in the management of chronic pain. Change is on its way. A Medicare Benefits Schedule Review Task force was convened in 2018 and its recommendations will be published this year. It is anticipated that emphasis will be shifted away from surgical interventions towards a more holistic approach.

References

1. K Vincent, J Moore, S Kennedy, J Tracey. Steroid hormones and pain-related brain activity and functional connectivity in healthy women. *Lancet*. 2014;383(1):S104.
2. ET Carey, CE Martin, MT Siedhoff, et al. Biopsychosocial correlates of persistent postsurgical pain in women with endometriosis. *Int J Gynaecol Obstet*. 2014;124:169-73.
3. NE Morone, DK Weiner. Pain as the fifth vital sign: exposing the vital need for pain education. *Clinical Therapeutics*. 2013;35(11):1728-32.

To 'scope or not to 'scope, that is the question

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The standard teaching and gynaecological approach to period and pelvic pain is to undertake a laparoscopy as the 'gold standard' for the identification of endometriosis,[1] to identify the cause of the pain and, ideally, to surgically correct any pathological findings in an effort to try and resolve the pain problem. In the setting of period and pelvic pain, studies suggest that 30–50 per cent of women will have endometriosis found at laparoscopy. A further number will have some adhesions found. So why not laparoscope everyone with significant period and pelvic pain who has not responded to NSAIDs and the oral contraceptive pill (OCP)?

There are a number of reasons, which include my experiences of working at Royal Children's Hospital (RCH) in Melbourne where there have been many unusual, but very instructive, clinical problems, but also the lack of correlation between pain and endometriosis, the lack of RCTs demonstrating any long-term benefit of laparoscopy for pain and the increasing knowledge regarding the mechanisms associated with pain, pain sensitisation and the development of persistent or chronic pain.

Starting with my experience at RCH; remember that it has often been an unusual presentation of a problem that teaches us something or leads to improved understanding of a condition.

By listening to adolescents with dysmenorrhoea, we recognised that 40 per cent experience nausea, 30 per cent have vomiting and/or diarrhoea, 20 per cent have headaches, 10 per cent feel faint or are dizzy. This is classic primary dysmenorrhoea, which begins a few days prior to menses and lasts usually until day 2 or 3, caused by inflammatory cytokines and prostaglandins.

Extending from this, adolescent girls are seen at RCH with anaphylaxis as well as ICU admissions for severe asthma – occurring only with onset of their menses. Adolescent girls have also been admitted for severe vomiting occurring four-five weekly, prior to their first period, and then with their periods, when they begin. Exacerbations of Crohn's disease and irritable bowel syndrome occur at times of menses, as do migraines and the symptoms associated with chronic fatigue and fibromyalgia. Most of these events can be attributed to inflammatory cytokines and prostaglandins, which are known to be critically involved in the process of endometrial shedding[2] and are clearly pretty potent substances.

Again listening to the adolescents with dysmenorrhoea, the pain patterns of those with dysmenorrhoea and heavy menses, but not the prostaglandin-related symptoms, is actually different – with their pain occurring on days of heavy bleeding. We know that 95 per cent of women get retrograde bleeding and that those that bleed heavily are doing more of this.[3] Thus, the pain pattern is different, either because they are cramping to expel a blood clot or they have more retrograde bleeding with the peritoneal free fluid causing irritation provoking bladder and bowel symptoms and their different pain pattern.

Through my work at the RCH, I have been involved in the care of more than 100 women with uterovaginal agenesis. Given that one in 10 women are supposed to develop endometriosis, why aren't there a similar number of women with Mayer-Rokitansky-Küster-Hauser with endometriosis? I have yet to see a case (when there is no endometrial tissue present), and neither are they found in the medical literature. In contrast, I have seen numerous young women with obstructed Müllerian anomalies and most of these have significant endometriosis due to substantial retrograde menses. In young women, it is often not appropriate or possible to operate immediately, so periods are suppressed and the corrective surgery is undertaken some months to years later. In these young women, the endometriosis has invariably resolved by the time of their surgery. This spontaneous resolution, of even moderate to severe endometriosis, has been reported by others.

Bleeding disorders in adolescents with heavy menstrual bleeding (HMB) have long been recognised. On taking a history, including family history, I rapidly realised that the mothers of the adolescents with HMB also had heavy periods (and endometriosis), and we subsequently often identify bleeding disorders in both the teenager and the mother. Kadir has shown that more than 10 per cent of women with HMB in adult gynaecology clinics have a mild bleeding disorder,[4] but this rate also applies to women with histologically proven endometriosis. Various epidemiological studies have already shown that women who bleed more often, for longer and more heavily, are more likely to have endometriosis.[5] So, at a clinical level – if you don't bleed, you do not seem to make endometriosis, and if you bleed lots you are more likely to do so.

Now add to this background the science of pain and central sensitisation. It is the presence of pain that is associated with lower thresholds of pain sensitivity and thus central sensitisation, not the presence or absence of endometriosis.[6] Additionally, repeated cyclic pain is thought to predispose to persistent or chronic pain.[7] But further to this, the poor correlation between symptoms, location and pain severity with location of endometriosis raises concerns regarding endometriosis and its failure to fulfil the Bradford-Hill criteria for causality. The relationship between pain and endometriosis is not helped by the trials relating to pain and laparoscopy. There have been three RCTs that have involved women who had presented with pain, and then when endometriosis was identified at the time of laparoscopy, had either sham (diagnostic laparoscopy only) or excision/laser undertaken.[8-10] The initial outcome measures for two of these studies was endometriosis at six-month follow up[8] or at their repeat surgery.[9] The study by Abbott demonstrated no difference in pain scores at six or 12 months between those who had delayed or immediate excisional surgery.[9] Long-term follow up (mean six years) of the former study demonstrated pain recurrence in both groups.[11] Likewise, the study by Jarrell with follow up to 12–14 years, demonstrated no difference between those with sham surgery and those with excision of endometriosis in pain score outcomes, an equal likelihood of repeat surgery and with the only predictor of repeat surgery being pain score prior to first laparoscopy.[12]

What about the risk of progression of endometriosis? Several studies have reported on delays of 4–11 years to the diagnosis of endometriosis.[1] Adolescents who have been seen at RCH are likely to be in the 10 per cent of teenagers with significant dysmenorrhoea[13] who should be at high risk, in theory, of having endometriosis. Their management involves reducing menstrual loss and often suppressing menses altogether to manage their symptoms. In a retrospective cohort, we had performed a laparoscopy in only 8 per cent (12/150) and only one had endometriosis. A long-term follow up study of these adolescents located 50 per cent of the young women 5–15 years later, with a 95 per cent participation rate (n=70). 25 per cent had no pain, 25 per cent had some pain, and 50 per cent had ongoing pain. If we follow the argument that endometriosis will progress without excision and specific treatment, then of this adolescent cohort, most, if not all, should have had significant endometriosis by the time they had a laparoscopy as adults. This is particularly the case as others have reported that of adolescents who fail cyclic OCP and NSAIDs, 38–100 per cent[14-16] will have endometriosis at the time of laparoscopy. Yet, in the long-term follow up cohort, only 13 of the 26 who had had a laparoscopy under adult care, had endometriosis – all minimal/mild disease. So despite a long history of dysmenorrhoea and never having had excision of any theoretical/potential spots of endometriosis, there was no moderate or severe endometriosis. The fertility rate among the cohort was as good, if not better, than the state age matched figures. Maybe the outcomes were better than expected due to the reduction in menstrual loss during adolescence. I won't argue that the ongoing pain in 50 per cent is a problem; but, there were no predictive features of this cohort and I suspect the lack of psychological input may have contributed.

You may argue that adolescents are different, and you may be correct. Nevertheless, in adult women, with normal, careful ultrasounds (and ignoring comments regarding immobile and tender ovaries, due to lack of evidence that this is relevant), I mostly avoid laparoscopies for pain. I admit I do not know who will benefit from a negative laparoscopy, and I am sure there is a cohort, but there is also a significant cohort who will be angry about a negative laparoscopy. There will be 20 per cent who will develop a new pain – often a trigger point on their abdominal wall.

Women report that their period and pelvic pain symptoms are ignored and trivialised, not managed. What I am advocating, and what others are also saying, is that dysmenorrhoea and pelvic pain should not be trivialised and ignored. It needs to be actively managed to endeavour to avoid the development of persistent pelvic pain and central sensitisation, with the incumbent risk for many other chronic pain syndromes. The aim is to manage pain symptoms, rather than excising some endometriosis, which may well return with a subsequent heavy period. The aim is to reduce menstrual loss (tranexamic acid or hormones), plus or minus suppression of menses and ovulation (a potential source of pain), provide and link patients to period and pelvic pain education and seek the assistance of physiotherapists. I know I need to do better at finding psychologists with an interest in pain, as I am sure they will have an impact on pain rumination, magnification and helplessness[17] and offer patients the skills to better manage the stressors and contributors to pain.

References

1. SK Agarwal, C Chapron, LC Giudice, et al. Clinical diagnosis of endometriosis: a call to action. *Am J Obstet Gynecol*. 2019;220(4):354.e1-e12.
2. J Evans, LA Salamonsen. Inflammation, leukocytes and menstruation. *Rev Endocr Metab Disord*. 2012;13(4):277-88.
3. TM D'Hooghe, CS Bambra, BM Raeymaekers, PR Koninckx. Increased prevalence and recurrence of retrograde menstruation in baboons with spontaneous endometriosis. *Hum Reprod*. 1996;11(9):2022-5.
4. R Kadir, D Economides, C Sabin, et al. Frequency of inherited bleeding disorders in women with menorrhagia. *Lancet*. 1998;351(9101):485-9.
5. BPaW Eskenazi, L Marcella. Epidemiology of Endometriosis. *Obstetrics and Gynecology Clinics*. 1997;24(2):235-57.
6. S As-Sanie, RE Harris, SE Harte, et al. Increased pressure pain sensitivity in women with chronic pelvic pain. *Obstet Gynecol*. 2013;122(5):1047-55.
7. G Hardi, S Evans, M Craigie. A possible link between dysmenorrhoea and the development of chronic pelvic pain. *ANZJOG*. 2014;54(6):593-6.
8. CJ Sutton, SP Ewen, N Whitelaw, P Haines. Prospective, randomized, double-blind, controlled trial of laser laparoscopy in the treatment of pelvic pain associated with minimal, mild, and moderate endometriosis. *Fertil Steril*. 1994;62(4):696-700.
9. J Abbott, J Hawe, D Hunter, et al. Laparoscopic excision of endometriosis: a randomized, placebo-controlled trial. *Fertil Steril*. 2004;82(4):878-84.
10. J Jarrell, R Mohindra, S Ross, et al. Laparoscopy and reported pain among patients with endometriosis. *J Obstet Gynaecol Can*. 2005;27(5):477-85.
11. KD Jones, P Haines, CJ Sutton. Long-term follow-up of a controlled trial of laser laparoscopy for pelvic pain. *JSLS*. 2001;5(2):111-5.
12. J Jarrell, R Brant, W Leung, P Taenzer. Women's Pain Experience Predicts Future Surgery for Pain Associated With Endometriosis. *J Obstet Gynaecol Can*. 2007;29(12):988-91.
13. M Parker, A Sneddon, P Arbon. The menstrual disorder of teenagers (MDOT) study: determining typical menstrual patterns and menstrual disturbance in a large population-based study of Australian teenagers. *BJOG*. 2010;117(2):185-92.
14. P Vercellini, L Fedele, L Arcaini, et al. Laparoscopy in the diagnosis of chronic pelvic pain in adolescent women. *J Reprod Med*. 1989;34(10):827-30.
15. MR Laufer, J Sanfilippo, G Rose. Adolescent endometriosis: diagnosis and treatment approaches. *J Pediatr Adolesc Gynecol*. 2003;16(3 Suppl):S3-11.
16. EC Dun, KA Kho, VV Morozov, et al. Endometriosis in adolescents. *JSLS*. 2015;19(2).
17. M Sullivan, S Bishop, J Pivik. The Pain Catastrophizing Scale: development and validation. *Psychological Assessment*. 1995;7(4):524-32.

The Pelvic Pain Foundation of Australia

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The Pelvic Pain Foundation of Australia (PPFA) is a not-for-profit organisation that promotes and supports education, advocacy and research for girls, women and men with pelvic pain from any cause. While dysmenorrhea-related pain is a significant proportion of our work, we also recognise the difficulties that Australians, both male and female, with other pelvic pain conditions have in accessing reliable, medically accurate and practical information. A key part of our work is in the support of clinicians, including gynaecologists, when caring for the complex needs of women with pain. Solutions for pelvic pain need to work for both sides of the consulting desk.

The origins of PPFA

PPFA was co-founded in January 2014 by Dr Susan Evans (gynaecologist, pain physician), Ms Kathy Allen (accountant), Ms Donna Benge (solicitor) and Dr Meredith Craigie (pain physician).

The need for representing those with pelvic pain was first recognised at the National Pain Summit held at Parliament House, Canberra in 2010. The Pain Summit was a turning point for Australians with chronic pain, and led to the development of Australia's first National Pain Strategy; however, the needs of women with pelvic pain had not been addressed. No organisation was advocating on behalf of girls and women with pelvic pain.

In 2011, Ms Deborah Bush (Endometriosis New Zealand), Prof Thierry Vancaillie and Dr Susan Evans, in collaboration with PainAustralia and the Faculty of Pain Medicine, looked at ways of remedying this problem. For their report entitled 'The $6 Billion Woman and the $600 Million Girl: The Pelvic Pain Report' they consulted widely with health professionals and those affected by pelvic pain to outline the problems with current pelvic pain services and policies. Since then, the extent of pelvic pain in men has also been recognised. PPFA was formed to represent the interests of those with pelvic pain regardless of age, gender or pelvic pain condition.

PPFA's achievements so far

Online education

The pelvicpain.org.au website provides information and education to all Australians, including those from rural, remote and international locations. The website focuses on practical information that patients can action themselves, or in consultation with their GP. Self-care and a better understanding of pain is emphasised. Patients can begin learning pelvic stretches, pelvic muscle relaxation, better use of medications and self-care from the first day of consultation.

Seminars

In May 2019, PPFA held its fourth Health Practitioner Training Seminar. These inexpensive and practical seminars welcome all AHPRA registered health professionals with an interest in broadening their pelvic pain management skills and building contacts across different professions. The speakers and topics are chosen to provide maximal practical application to everyday clinical care. The seminar allows professionals to upskill outside their traditional area of expertise, in a wide range of areas such as Understanding Pelvic Pain, The Management of Bowel Pain in Primary Care, Avoiding Burnout, Male Pelvic Pain, Laparoscopy for Pelvic Pain, Recognising Musculoskeletal Pelvic Pain, Opioids in Pelvic Pain, and Motivational interviewing.

Schools program

In 2019, PPFA is contracted to provide an innovative, neuroscience-based schools education program to 80 schools in South Australia (SA). In 2017, PPFA piloted the Endometriosis New Zealand, ME Program in 10 SA schools with great success, and presented an evaluation of the program to the Australian Pain Society. The PPFA-developed Periods, Pain and Endometriosis (PPEP-Talk) Program was created to accommodate the requirements of the Australian education system and incorporate new aspects of neuroscience for the benefit of all students. It explains pain, periods and endometriosis with a focus on positive health options to reduce pain, early effective management of dysmenorrhea in a primary care setting, and early referral for consideration of endometriosis where required. PPEP-Talk is part of the National Action Plan for Endometriosis (NAPE) and is funded as a collaboration between the federal government and the SA State Government. A pilot PPEP-Talk program will be provided in Queensland in 2019, and PPFA welcomes enquiries from other states.

Subscriber program

Health professionals who choose to become PPFA subscribers are supported in several ways. Subscribers are entitled to have their practice details

available for the public on the website (optional) with a description of the specific services each practitioner provides, for example, laparoscopic surgery for endometriosis, physiotherapy for male pelvic pain, pain specialist and so on. They are entitled to use PPFA patient and teaching resources including the PPFA Pelvic Pain Questionnaire to facilitate history taking, the Introduction to Pelvic Pain booklet for patient pre-reading before consultations, and view specific pelvic pain training videos to improve their management skills. Subscribers are also supported where they encounter difficulties when caring for their patients, where possible.

Patient resources

PPFA provides products for girls, women and men through an online shop accessed via the pelvicpain.org.au website. Products include Dr Patricia Neumann's pelvic muscle relaxation audio (male and female), Ms Meike Wise's 'This Endo Life' book, Dr Susan Evans's 'Endometriosis and Pelvic Pain' book (paperback and ebook), TENS machines for teens, Olive and Bee lube for sensitive vulval skin, vaginal trainers, Peter Dornan's 'Musculoskeletal approach to pelvic pain' book, the Explain Pain 'Protectometer workbook', and health practitioner training videos for individual sale.

Collaboration with other groups

PPFA collaborates with a wide range of clinician organisations (Queensland Persistent Pelvic Pain Network, Pelvic Pain Victoria, Tasmanian Pelvic Pain group), advocacy groups (QENDO, Endometriosis Australia, EndoActive, EndoNet, EndoSupportSA, EndoHelp, the Endometriosis Perth Sisterhood), Womens Health Organisations (WHRIA, TRUE, Jean Hailes), research organisations (Robinson Research Institute) and several universities.

MBS Review advocacy

PPFA has actively engaged with the MBS Review Committee, seeking a new Medicare Item Number for prolonged consultations by gynaecologists when caring for women with pelvic pain. PPFA has advocated that a timed consultation remunerated at the same rate as physicians for patients with complex conditions would allow gynaecologists to provide more comprehensive care and incentivise upskilling in this area. PPFA's submission to the MBS Review Board requesting this new item number was followed by the opportunity to meet for a long and comprehensive interview with Prof Bruce Robinson (Chair, MBS Review), and senior members of the Department of Health. We continue to advocate for this as an essential part of health reform.

NAPE

PPFA has been an active participant in the advocacy for, the preparation for, and the implementation of, the NAPE. The NAPE is an ambitious document, which, if fully implemented, has enormous potential to change the lives of girls and women with pelvic pain. It is a credit to the collaboration of Federal and State Governments with the five peak advocacy bodies that make up the Australian Coalition for Endometriosis (QENDO, Endometriosis Australia, EndoActive, Canberra Endometriosis Network and PPFA).

Research

PPFA has supported a wide range of research projects, either financially or with in-kind support. These include the development of a Pelvic Pain Impact Questionnaire, basic science research into the relationship between endometriosis lesions and pain, the continuing development of a home use tool for male pelvic muscle pain, and a large survey investigating the Language of Pelvic Pain.

Facebook

PPFA provides items of news to the public that are perceived as contributing to readers' lives in a positive and collaborative way.

What PPFA doesn't do

PPFA does not provide consumer support, pelvic pain advice or services to individuals with pain.

How PPFA is funded

PPFA is led by a diverse and highly qualified volunteer board. The skill mix includes legal, accounting, journalism, marketing, education, gynaecology and pain medicine, supported by a part-time office administrator. Income is derived from the sale of online goods, training seminar registration fees, PPFA subscriber subscriptions and private donations. A 2019 grant from the Federal Department of Health and the SA State Government will support the PPEP-Talk Schools Program.

How you can support PPFA

PPFA's foremost aim is to support clinicians wishing to improve the care they provide to girls, women and men with pelvic pain. We believe that the lack of a trained workforce willing to work in this area is the major impediment to improved services. You support the aims of PPFA most effectively by choosing to take up the challenge of improving your own pelvic pain management skills, and PPFA is ready to help you. If you wish to support PPFA financially, donations to PPFA are tax-deductable. We are a highly cost-efficient organisation that supports an area close to your heart: women's health. PPFA subscribers who wish to have their practice included on the PPFA website and access patient resources and training materials pay a subscriber fee of $200 per year or $450 for three years.

Summary

While all areas of healthcare have patients with complex pain needs, we suggest that in no area of medicine is this need more apparent than in pelvic pain. Much of the knowledge required to assist women with pain is already available. It crosses a range of traditional skillsets, requiring learning outside traditional comfort zones. Gynaecologists are already experts in endometriosis, menstrual and hormonal issues. The upskilling required to manage the majority of pelvic pain needs is relatively straightforward, and well within the ability of gynaecologists. PPFA looks forward to helping you increase the number of women that leave your care with less pain.

Pelvic Pain Foundation OF AUSTRALIA

A new paradigm, science and learning opportunities

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Persistent pelvic pain (PPP) presents challenges above and beyond those suffered by others living with chronic pain. In the past, patients have been reluctant to present to healthcare providers as their pain has embarrassing gender, fertility and sexual health overtones. Their reluctance has been reinforced by their experiences of frequently being dismissed, downplayed or normalised as 'just period pain', blamed on their mental health or straight disbelief by healthcare providers based on unhelpful stereotypes and inadequate training to provide the comprehensive assessment and care these patients need. Patients have often had myriad unsuccessful treatments or no treatment at all so that they eventually avoid and even distrust healthcare practitioners. This situation has been exacerbated by poor understanding of the mechanisms contributing to chronic pain in general and PPP in particular.

However, there are glimmers of light for PPP sufferers in 2019. Pain medicine is a rapidly evolving discipline with exciting new frontiers now being explored in the science of neuro-immune interactions and the social and psychological disciplines. Pain medicine was recognised as a medical specialty by the Australian Medical Council in 2005, and endorsed as a scope of practice in New Zealand in 2012. This recognised the importance of the problem of unrelieved pain in the community and the need for a comprehensive medical response through education, training and practice. The Faculty of Pain Medicine (FPM) of the Australian and New Zealand College of Anaesthetists (ANZCA) was established in 1998 to address these needs.

Sources of PPP include disorders of the gynaecologic, urologic, gastro-intestinal and neurological systems, either as the sole or, more commonly, multi-factorial aetiology. Genetic and environmental influences, gender-specific mechanisms, including the role of sex hormones, as well as changes related to pregnancy may also contribute. The unique neuroanatomical features of the lumbo-sacral nerve plexus, a predilection for somatic referral and an ability to provoke strong emotional responses add to the complexity of the PPP phenotype.

Known mechanisms of PPP include pelvic organ and somatic causes of nociception (including inflammation) and neuropathic pain. Persistent or cyclical nociceptive inputs from the periphery to the spinal cord can lead to increased membrane excitability, synaptic facilitation and disinhibition resulting in reduced thresholds for activation, spontaneous activity and enlargement of receptive fields whereby spinal cord neurons now respond to noxious and innocuous stimuli. In addition, recent research in the contribution of immune mechanisms to persistent pain show that the neuronal processes involved in conducting heightened nociception are triggered by anatomically distributed immune signals.[1] These signals arise from glia, immune-like cells, distributed throughout the nervous system exhibiting bidirectional communication between peripheral immune, spinal immune and brain immune systems. Glia, particularly microglia, engulf the synapses and control neurotransmitter release both pre- and post-synaptically. Normally glia maintain neuronal reactivity; however, they can amplify neuronal signalling sufficiently to create persistent pain states through fundamental reorganisation with recruitment of maladaptive microcytic and glial processes as well as T-cell involvement, changing trophic support and releasing pro-inflammatory cytokines that modify the pre- and post-synaptic terminals permanently. Together this suggests that the pathology of persistent pain resides in the glial component, but is expressed by adaptations in the neuronal system, namely peripheral and central sensitisation and cross sensitisation between organs.[2]

These neuro-immune changes can be triggered by end-organ dysfunction anywhere in the body. In PPP, multiple organ systems appear to be involved either as the primary pathology generates a secondary response to pain in other tissues or as a result of viscero-somatic or viscero-visceral convergence with expansion of the receptive fields of spinal cord neurons. So, it is not surprising that PPP is often described as multiple pains in several different sites, commonly associated with a range of other somatic and autonomic symptoms.

Changes extend to the brain where characteristic alterations in brain morphology, commonly decreases in regional grey matter, can be seen on functional MRI scans suggesting altered neuronal activity at the brain

level as well as affecting the 'top-down regulatory pathways' (Tor Wager). Multiple areas, including the ventro-medial thalamus, parabrachial amygdala, somatosensory pathways and the periaqueductal grey, are involved. The ventro-medial prefrontal cortex is particularly important in pain appraisal, driving aversive behaviours. Neuroplastic changes in these areas lead to changes in the appraisal of pain, altering descending modulation and avoidance learning, underpinning the emotional and behavioural responses to pain. The extent of these changes is modulated by genetic factors, brain development throughout childhood, particularly adverse childhood events, and past pain experiences. Much depends on the language used to communicate pain experiences. The language of pain and the influence of cultural contexts are relatively new, but very important, areas of pain research. Pain is a subjective and very personal experience that is communicated to others through language and behaviours. Women with PPP frequently report unsatisfactory conversations with healthcare practitioners and not surprisingly feel misunderstood.

Treating patients with PPP therefore requires a sound understanding of the language of pain, the social context in which patients experience pain and the psychological impact of the pain in addition to knowledge of pelvic organ pathology and neuroanatomy in order to develop a comprehensive management plan. The most complex patients are best served by a multidisciplinary team working in an interdisciplinary environment using a sociopsychobiomedical paradigm.[3] Discipline-specific training is required to address the wide range of biomedical contributions to PPP. Additional training in pain medicine addresses the sociopsychological dimensions, changing attitudes and behaviours while enhancing the knowledge and skills required to manage the more challenging PPP conditions. However, not every gynaecologist will wish to or be able to undertake an additional training program in pain medicine. Simple steps such as raised awareness of the potential complexity of the patient's presentation of PPP and altering lines of inquiry when taking the history, considering different approaches to investigations, preoperative preparation and postoperative pain relief can make a real difference. Engaging early with a pain medicine specialist and allied health practitioners with pain training can be useful.

Gynaecologists are in a prime position to set expectations for the patient and their other healthcare providers regarding recovery times and opioid analgesic tapering. The language used in history taking is critically important. Not infrequently, the language used has inadvertent high threat value for patients. Reframing information into less threatening language can help. Judicious use of adjuvant analgesics, such as low-dose amitriptyline, from the first night postoperatively through to the follow up visit can be a useful strategy for reducing opioid use. Learning about pain has been shown to empower patients to manage pain more effectively. Gynaecologists can assist by directing patients to reputable online resources such as the Pelvic Pain Foundation of Australia website[4] or the New South Wales Agency for Clinical Innovation website, the Pain Management Network.[5]

For gynaecologists wishing to learn more about persistent pain, the FPM offers two options; a series of online learning modules and a specialist training program. There are 12 one-hour modules in the Better Pain Management[6] program. Participants may choose to do one, a few or all 12 modules. For gynaecologists wanting more immersive learning, the faculty's specialist training pathway leads to the qualification of specialist pain medicine physician. The FPM was the first multidisciplinary medical academy in pain medicine in the world. The two-year fellowship is an additional fellowship to a primary specialist or general practice qualification. The faculty's 2015 curriculum and training program reflects the sociopsychobiomedical paradigm and is based on the CanMEDs structure. It consists of workplace-based clinical supervision with in-training progressive feedback and targeted summative assessments. A range of resources are available on the faculty's e-learning platform to support learning. They include modules introducing nine essential topic areas for the core training stage (first year) of which visceral pain is one. The practice development stage (second year) provides an opportunity for trainees to develop expertise in topic areas of their choice. PPP is one option for which learning outcomes have been defined.[7]

Gynaecologists face many challenges in looking after women with PPP. It takes a lot of time and emotional resilience. Currently, the remuneration structures in private practice are grossly inadequate and indeed there are perverse incentives for gynaecologists providing long consultations. In the public sector, there are high demands on outpatient clinics and pressures on surgical waiting lists. Limited access to pelvic physiotherapists, pain trained psychologists and specialist pain clinics across both sectors also contributes to less-than-optimal care. As individual physicians, we may feel we cannot do much about these problems; however, persistent pain is finally being recognised as a major public health issue and federal government funding is now targeting specific pain conditions, especially endometriosis, and by association PPP. We can lobby our respective professional organisations, RANZCOG and FPM ANZCA, to work together and strongly advocate for change. Our patients are depending on us.

References

1. KN Dodds, EAH Beckett, SF Evans, MR Hutchinson. Spinal Glial Adaptations Occur in a Minimally Invasive Mouse Model of Endometriosis: Potential Implications for Lesion Etiology and Persistent Pelvic Pain. *Reprod Sci*. 2019;26(3):357-69.
2. CA Von Hehn, R Baron, CJ Woolf. Deconstructing the neuropathic pain phenotype to reveal neural mechanisms. *Neuron*. 2012;73(4):638-52.
3. DB Carr, YS Bradshaw. Time to flip the pain curriculum? *Anesthesiology*. 2014;120(1):12-14.
4. Pelvic Pain Foundation of Australia. www.pelvicpain.org.au.
5. Pain Management Network. www.aci.health.nsw.gov.au/chronic-pain.
6. Better Pain Management program. www.fpm.anzca.edu.au/resources/better-pain-management.
7. Faculty of Pain Medicine 2015 training program www.fpm.anzca.edu.au/training/2015-training-program.

Notice of Deceased Fellows

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

- Dr John McNeil Campbell, Qld, 25 December 2018
- Dr Graeme Dickinson Desmond Cable, NZ, 13 February 2019
- Dr Eva Irene Popper, Qld, 2 March 2019
- Dr Philip Vincent Moon, Qld, 8 April 2019
- Dr Sarwat Fouad Shenouda, Vic, 10 May 2019

Q&A

For the broader *O&G Magazine* readership, balanced answers to those curly-yet-common questions in obstetrics and gynaecology.

Q What should I tell my patients about monitoring their baby's movements?

Dr Jenny Dowd
FRANZCOG

A Historically, maternal perception of fetal movements, called 'quickening', was likely the first clinical sign of a live pregnancy. These movements are usually noticed between 18 and 20 weeks, but may be well after 20 weeks in a first pregnancy or as early as 16 weeks in subsequent ones. The definition of 'normal' fetal movements is difficult as women perceive the sensation differently and one woman's description of fetal activity may not apply to another. The focus on noting a reduction in fetal movements (RFM) has become a topic of interest as a way of identifying fetuses at risk of stillbirth in time to act and prevent a tragic outcome. Stillbirth affects 2500 families in Australia and New Zealand annually[1,2] and the majority of women experiencing a stillbirth noted such a reduction prior to diagnosis.[3]

Various bodies (RCOG, RANZCOG, PSANZ) have guidelines about RFM. Currently, these guidelines assume that acting on RFM will avoid stillbirths and, to that end, all mention women should be educated about the need to monitor fetal movements by being given verbal and written information about such. Fetal movements should be felt by 24 weeks and note taken of RFM from 28 weeks. Actual counting of the number of fetal movement (as in the previously used Cardiff Kick Charts) is not advised as this has been shown to raise maternal anxiety levels. More emphasis is now placed on an individual woman's perception of the pattern of her own baby's movements.

Once the problem has been acknowledged, the recommended urgency of action varies, with PSANZ recommending a woman immediately contact her caregiver after noticing RFM and the RCOG Green Top guideline recommending she lies down on her side for two hours and then act if the baby is still quiet. Community-based organisations, such as Still Aware (stillaware.org), have an online presence and distribute written material urging women to 'bond with your bump' daily and 'if anything seems different or irregular contact your healthcare provider immediately, no matter what time of the day.'

Recommendations for management after presentation beyond 28 weeks gestation all begin with auscultation of the fetal heart and performance of a CTG. Assessment is then made of other causes of RFM, such as medication (opioids, sedatives, methadone) or fetal malformations (check that a morphology ultrasound has been performed). Stillbirth risk factors, such as maternal medical conditions, previous intrauterine growth restriction, smoking, advanced maternal age, IVF, BMI greater than 25, should be assessed.

In the presence of continued RFM or other clinical problems, an ultrasound scan is recommended for fetal biometry, amniotic fluid assessment, and if possible, middle cerebral artery dopplers. The timing of such a scan must obviously depend on the availability of appropriate resources. PSANZ alone seems to recommend a Kleihauer to exclude fetomaternal haemorrhage.

The management aspects of these guidelines may need to be reviewed in light of the evidence from the AFFIRM Trial published in the *Lancet* in November 2018.[4] This involved 400 000 pregnancies from 33 hospitals, in which women were told to monitor changes in fetal movements from 24 weeks and should refer themselves immediately if they detected altered movements after 28 weeks. The control group were given usual care and the primary outcome was stillbirth. The results show a slight decrease in the stillbirth rate from 44 per 10 000 births after standard care to 41 per 10 000 births with the intervention, but this drop was too small to prove that the intervention was beneficial. Other outcomes noted were an increase in induction of labour from 35.8–40.7 per cent, caesarean section from 25.5–28.3 per cent and preterm birth from 8.1–8.6 per cent, with the flow on of more prolonged admissions to the neonatal nursery. This led to a published comment about these data in the same journal from Walker and Thornton[5] who assert that 'encouraging awareness of fetal movements is harmful'. They suggest that, on the basis of the current evidence, we should not encourage programs that promote awareness of preterm RFM and tighten guidelines that lead to delivery based on RFM alone.

Two other cluster trials are currently in progress: 'Mindfetal' involving 39 000 women in Sweden and 'My Baby's Movements' in Australia. The latter began in August 2016. It involves 27 hospitals, 256 700 births

over three years, and is a stepped wedge cluster-randomised trial. The package of intervention involves a smart phone app, which women receive after 28 weeks, that asks them to log their fetal movements daily. It also provides them with information and prompts about fetal activity and is intended to support them in making decisions about seeking care if RFM occurs. This has been going for several months at the Royal Women's Hospital, Melbourne, and so far, only 30 per cent of those patients logging an interest have started using the App when sent a link at 28 weeks (personal communication).

So where does that leave us with respect to advising our patients? Anyone touched by stillbirth, either personally or professionally, hopes there will be a way to reduce the incidence of this event and many are vocal in producing resources that encourage women to act. Clinicians all have anecdotal stories about women who present out of the blue with RFM and a critical CTG leads to urgent delivery and, we presume, a saved life. The flipside is women who lose a baby feeling guilty that they didn't present in time. Many pregnancy blogs and websites have advice along these lines.

In light of the AFFIRM trial, current guidelines still stand, but maybe we should pause and await better evidence regarding promoting urgent action on the basis of RFM alone, especially preterm, in order to avoid unnecessary interventions and inductions.

References

1. Perinatal and Maternal Mortality Review Committee (PMMRC). Tenth Annual Report of the Perinatal and Maternal Mortality Review Committee: Reporting Mortality 2014. Wellington: Health Quality and Safety Commission, 2016 Contract No.: ISBN 978-0-908345-29-8 .
2. Australian Institute of Health and Welfare. Australia's mothers and babies 2014—in brief. Canberra: AIHW, 2016.
3. T Stacey, JM Thompson, EA Mitchell, et al. Maternal perception of fetal activity and late stillbirth risk: Findings from the Auckland Stillbirth Study. *Birth*. 2011;38:311-16.
4. JE Norman, EPA Heazell, A Ridriguez, et al. Awareness of fetal movements and care package to reduce fetal mortality (AFFIRM): a stepped wedge, cluster-randomised trial. *Lancet*. 2018:392;1629-38.
5. KF Walker, JG Thornton. Encouraging awareness of fetal movements is harmful. *Lancet*. 2018;392;1601-2.

Case report

The local effects of fertility tourism

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Ms N, 35 years old primigravida, underwent IVF overseas. Five embryos were transferred and Ms N became pregnant with septuplets. She had an early dating scan that noted presence of six viable fetuses and one fetal demise. Upon return to Australia, she and her partner were counselled regarding risks of higher order pregnancy and they opted for fetal reduction at 12 weeks of pregnancy. After a detailed scan in a tertiary centre, she underwent reduction of four fetuses to continue with twin (DCDA) gestation.

In view of twin pregnancy, she was managed in a high-risk clinic. Ms N developed diabetes at 28 weeks and discordant growth of twins was noted at 31 weeks, further increasing the fetal surveillance. Her pregnancy was further complicated by onset of preeclampsia at 33–34 weeks.

Ms N was admitted for management of hypertension and, during her inpatient stay, her membranes spontaneously ruptured at 34 weeks. Two days later, she went into spontaneous labour and proceeded to have a vaginal birth of twin boys, weighing 2230 g and 1730 g.

The delivery was complicated by a postpartum haemorrhage of 800 mL that was managed medically; however, she continued to have ongoing moderate vaginal bleed over the next 12 hours, losing a further 400 mL. In view of the ongoing vaginal bleed, it was decided to take her for an examination under anaesthesia.

Initially, multiple fragments of adherent placenta were removed manually. Surprisingly, a fetus of 50 mm crown-rump length was also retrieved. This unusual find gobsmacked not just the mother, but also the obstetric team, despite their years of experience.

Post the evacuation, the bleeding settled. The total blood loss was 1200 mL. Ms N recovered well and was discharged home on day 4 while the babies remained in the nursery.

Discussion

A direct consequence of increase in the popularity of assisted reproductive technology (ART) is the increase in the incidence of multiple birth rates.

Our patient, unfortunately, ended up having all the major complications associated with multiple pregnancies, which included gestational diabetes, preeclampsia, preterm rupture of membranes, preterm labour, discordant growth of twins and postpartum haemorrhage. She also brought to our attention the risks of cross-border reproductive care (CBRC).

Women are increasingly accessing ART overseas, known as CBRC, for various reasons that include gender selection, avoiding long waiting lists, cheaper treatments, multiple embryo transfer and perceived increased success rates.[1,2]

Australia has strict laws for gamete selection, commercial surrogacy, multiple embryo transfer and restriction for IVF, in case of disabilities and diseases. Implementation of polices such as single embryo transfer has resulted in significantly reducing the incidence of higher order pregnancies, secondary to ART, thereby greatly mitigating the complications associated with these pregnancies. However, adherence to these policies has also resulted in countries like Australia facing problems associated with CBRC

The explosion of CBRC has raised not only legal and ethical questions, but has also raised concerns in terms of 'commodification' of ART.

Growth of CBRC has been exponential and resulted in development of commercial hubs for CBRC in countries such as India and Thailand. It is estimated that surrogacy in India is a $2.5bn industry.[3] There are CBRC 'brokers' that facilitate the CBRC interaction, ranging from hotels to maternity waiting homes for the reproductive travellers.

When access to fertility treatment at home is restricted legally or ethically, couples look at accessing services in countries that do not have the same restrictions. When compared with Australia, countries such as India have highly permissive laws governing reproductive treatments.[4] There are concerns about the nature of informed consent for gestational surrogates and commercial exploitation of surrogates and donors.

The wide variation in law between countries that leads to fertility tourism has resulted in children being stateless and questioning their parentage. There have been cases where it has taken almost two years for children to be united with their IVF parents.[3]

CBRC also has economic implications. In Western Australia, a study of the effects of CBRC on the local healthcare system found that one third of multiple pregnancies was a result of fertility treatment overseas. The study estimated that the healthcare cost exceeded $1m AUD and added significantly to the economic burden of the local healthcare system.[5]

RANZCOG has a guideline in place for management of CBRC.[1] Clinicians in Australia are obligated to optimise the woman's health prior to her accessing ART services. When the patients make use of fertility services overseas, they are not necessarily given the same education and counselling in order to minimise the morbidity associated with any coexisting medical conditions. So, women who seek ART overseas may be further exposed to risks secondary to their pre-existing diabetes, obesity, hypertension or other medical conditions, further adding to the disease burden.[5]

Lessons learned

Fertility treatment effects a woman's health both physically and psychologically. Our case highlights complications of CBRC, multi embryo transfer, feticide and multiple pregnancies. Several strategies have been suggested to reduce the complications of multiple pregnancies associated with multi-follicular development seen in ART cycles. These include selection of high-quality embryos, single embryo transfer, patient selection, preimplantation genetic screening and patient education.

With CBRC, there are the added burdens of cost, travel and language that the woman and her family may have to face. Fertility societies like ESHRE have stressed that the ideal scenario is fair access to fertility treatment at home for all patients.[6]

It is an ideal that healthcare providers all over the world should aim for. The onus of responsibility rests with the fertility physicians and obstetric care providers to keep the risk associated with CBRC and multiple pregnancies to a minimum by educating and counselling the woman appropriately.

References

1. RANZCOG. Cross-border reproductive care. Available from: https://www.ranzcog.edu.au/RANZCOG_SITE/media/RANZCOG-MEDIA/Women's%20Health/Statement%20and%20guidelines/Clinical%20-%20Gynaecology/Cross-Border-Reproductive-Care-(C-Gyn-36)-New-March-2016.pdf?ext=.pdf.
2. MC Inhorn, P Patrizio. The global landscape of cross-border reproductive care: twenty key findings for the new millennium. *Current Opinion in Obstetrics & Gynecology*. 2012;24(3):158-63.
3. SL Crockin. Growing families in a shrinking world: legal and ethical challenges in cross-border surrogacy. *Reproductive BioMedicine Online*. 2013;27(6):733-741.
4. A Whittaker. Cross-border assisted reproduction care in Asia: implications for access, equity and regulations. *Reproductive Health Matters*. 2011;19(37):107-16.
5. KA Waller, JE Dickinson, RJ Hart. The contribution of multiple pregnancies from overseas fertility treatment to obstetric services in a Western Australian tertiary obstetric hospital. *ANZJOG*. 2017;57(4):400-4.
6. Cross-border reproductive care: a committee opinion. *Fertil Steril*. 2013;100(3):645-50.

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An underdiagnosed cause of infertility: isthmocele

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Caesarean section is one of the most commonly performed surgeries and rates are continuing to increase all over the world. Well-known complications of a caesarean section scar include placenta acreta, placenta praevia, scar ectopic, and scar dehiscence (either antenatally or intrapartum). However, we are less familiar with the implications from a caesarean scar defect, such as dysfunctional uterine bleeding, pelvic pain and secondary infertility.

History

Isthmocele, also known as caesarean scar defect, uterine scar deficiency, uterine niche or pouch, was first described by the obstetrician gynaecologist Morris in 1995.[1] This entity is only just being identified and described in the literature, and is becoming a subject of attention, especially in those who present with secondary infertility. The reported incidence of isthmocele ranges from 24–84 per cent.[2,3] With the incidence of caesarean section increasing, it is important that we are aware that isthmocele could be a cause of these symptoms.

Presentation

Isthmocele may present with secondary infertility, menstrual disturbance (typically postmenstrual spotting), continuous brown discharge, dysmenorrhoea, pelvic pain, and/or dyspareunia.[1,2,4] Isthmocele can also be asymptomatic. The intensity of symptoms is thought to be related to the size of the isthmocele.[1] As other pathology may present with these symptoms, there is often a delay in diagnosis.[5]

Definition and diagnosis

There is no international standardised definition or diagnosis for isthmocele. It can be identified on imaging with transvaginal ultrasound scan (TVUS), magnetic resonance imaging (MRI), or at the time of hysteroscopy. Isthmocele or caesarean scar defect has been defined as 'any visible filling defect in the anterior isthmus of the uterus on transvaginal ultrasound scan'.[6] A 'larger' isthmocele is thought to be when the remaining myometrial thickness is less than 2.5–3.0 mm.[7]

Prevalence

Studies have found wide prevalence ranges. Bij de Vaate et al estimated the incidence to between 24–84 per cent, Wang et al reported 6.6–69 per cent.[8] While Florio, Tower and Frishman reported and incidence of 30–52 per cent and 19.4–88 per cent respectively.[8]

Although isthmocele is quite prevalent, it is not always associated with secondary infertility.

One of the largest systematic reviews on caesarean section scar-associated secondary infertility showed a correlation between caesarean delivery and increased odds of subfertility when compared to vaginal delivery (1.6 95% CI 1.45-1.76, p<0.00001).[9]

There is growing evidence that IVF success rates are lower in patients who deliver via caesarean section when using embryos from the same cohort,[10] suggesting a possible impact of embryo implantation.

Risk factors

A number of risk factors are thought to be implicated in the development of isthmocele. The list includes more than one caesarean section, retroflexed uterus, pre-eclampsia, maternal age less than 30 years, duration of labour more than five hours, cervical dilation more than 5 cm at time of delivery, lower station at delivery (below pelvic inlet), incision in the cervical area, use of oxytocin, exclusion of endometrial layer during repair, one-layer closure of myometrium, delayed absorbable sutures, and more ischemic closure.[1,6]

Investigations

TVUS and MRI are considered to be the gold standard when investigating for isthmocele,[7] although ultrasound is usually considered as the first diagnostic tool.[1] The depth and length of the defect are measured along with the remaining myometrial wall thickness. Other key sonographic findings to consider are inward and outward protruding of the scar, scar retraction and haematoma.[6] On TVUS, the defect is seen as a triangular hypoechogenic zone in the uterine scar area; the term for this is 'niche'.[1] Saline infusion sonohysterography and hysterosalpingography have also been used to detect isthmocele.

Pathophysiology

The exact mechanism of how isthmocele or caesarean scar defect occurs is not completely understood.[8] It is possible that two mechanisms are at play; inadequate healing of the caesarean section scar and the resulting thickness of the myometrium.[11] Reduced scar contractility and abnormal myocontraction, along

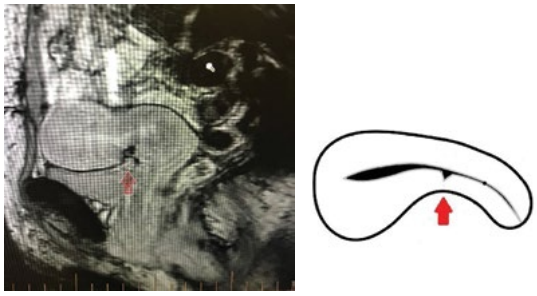


Figure 1. MRI showing isthmocele and pictorial diagram.

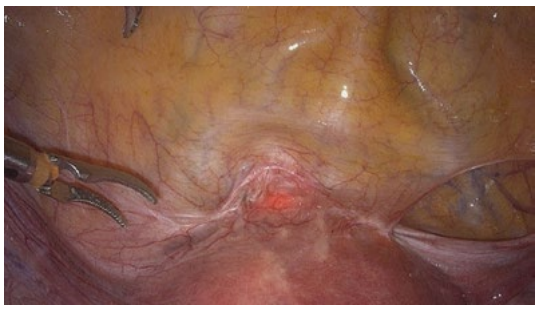


Figure 2. Isthmocele seen at laparoscopy.

with the presence of functional endometrium leads to the accumulation and impaired drainage of menstrual blood in the cavity.[2,12] This results in intermenstrual bleeding and pelvic pain.[11]

The retained menstrual fluid is likely to impair the quality of cervical mucous, sperm viability and disrupt sperm swim-up. Bleeding may cause the blastocyst to be washed out of the uterus, interfering with embryo implantation.[1,2]

In addition, endometrial abnormalities around the scar defect, such as overhanging endometrium and/or inclusion of endometrium within the scar, could lead to abnormal bleeding. Careful re-approximation of the endometrial layer during uterine closure could reduce endometrial abnormalities and dysfunctional bleeding.[2]

Treatment

Treatment should be offered to symptomatic women. There is no 'gold standard' or treatment protocol for isthmocele repair, and no technique that has shown a statistically superior outcome.[7]

Although some report medical therapy can help to improve symptoms by suppressing menstruation,[11] others would argue that medical treatment with the combined oral contraceptive pill or Mirena shows no benefit.[13] Hormonal treatment is also not suitable for patients who are planning a pregnancy.

Both hysteroscopic and laparoscopic resection and repair of isthmocele have high success rates of treating both infertility and bleeding disturbance, and a variety of different surgical approaches have been described.[1]

Isthmocele repair was first performed laparoscopically by Jacobson in 2003.[7] The laparoscopic method has the advantage of increasing the thickness of the myometrial layer after repair.[14] It is the preferred surgical approach, particularly if the residual myometrium is less than 3 mm thick.[7,9] Restoring normal myometrial thickness is important in those patients planning a pregnancy to help reduce the risk of scar rupture.[7] Furthermore, the laparoscopic approach has a reduced risk of incomplete removal of scar tissue, uterine perforation and bladder injury, which is associated with hysteroscopic resection.[1,7]

The crucial step of laparoscopic repair is to correctly identify the uterine scar defect.[7] A popular technique described in the literature is the 'Rendezvous technique'.[1] This is a combined technique where the isthmocele is identified at laparoscopy with the simultaneous use of hysteroscopy to outline the defect with its light source. The defect can be seen under the light of the hysteroscope and is known as the 'Halloween sign'[1] (See Figure 3). A Foley catheter or TVUS can also be used during laparoscopy to help identify the location of the isthmocele.[11,15] It is unclear which type of energy source or suture material is best to repair the scar defect.[1]

Symptoms are generally improved after repair of isthmocele, with one study reporting a 97 per cent improvement in pain.[5]

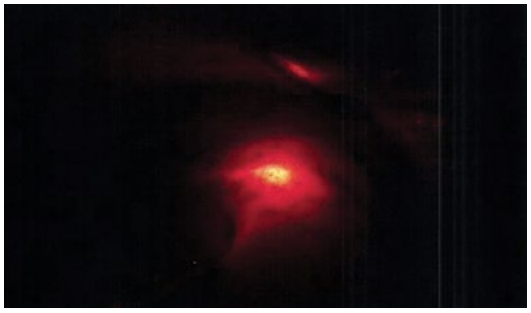


Figure 3. The defect can be seen under the light of the hysteroscope, 'Halloween sign'.

Future pregnancy

Secondary infertility is an indication for treatment, and the reported pregnancy rates after endoscopic repair of isthmocele are 44–92 per cent.[1,16] As with caesarean section, the risk following treatment of isthmocele is scar dehiscence in subsequent pregnancies. The current recommendation is to wait at least three months after repair to get pregnant. It is also recommended that delivery is by caesarean section at term, although successful vaginal deliveries after surgical repair of isthmocele have been reported in the literature.[1]

There is only limited data regarding isthmocele and pregnancy complications, including uterine rupture in subsequent pregnancies. However, what has been observed is the relationship between the size of the isthmocele and the risk of scar rupture, where the larger the defect the greater the risk of scar rupture. Lower uterine thickness near term could also be predictive of uterine rupture.[17]

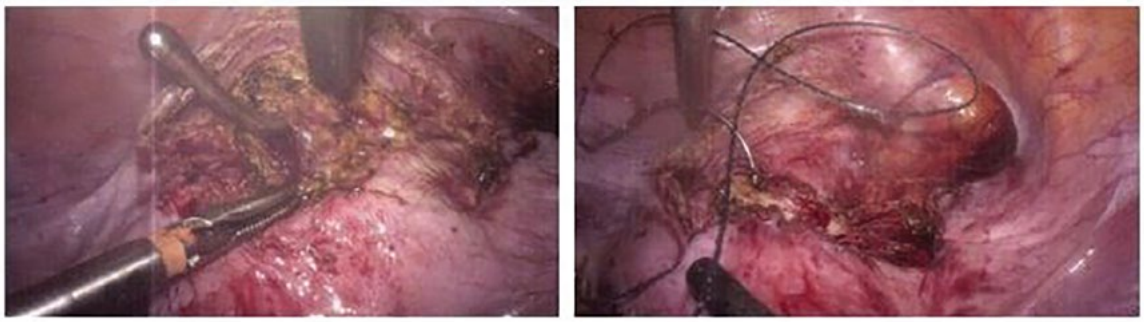


Figure 4. Intra-operative laparoscopic excision and repair of isthmocele.

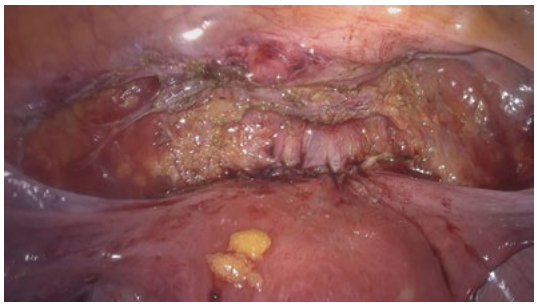


Figure 5. After laparoscopic excision and repair of isthmocele.

Future trends

Isthmocele is common following caesarean section and is associated with infertility. We need a prospective multicentre database to develop an understanding of this condition with regards to diagnosis, surgical techniques and outcomes of surgery so that we can give robust information to our patients.

Conclusions

Isthmocele must be considered in patients with a history of caesarean section that present with secondary infertility, abnormal bleeding or pelvic pain. With caesarean section rates rising globally, it is predicted that we will encounter this issue with increasing frequency. By increasing awareness of this common complication, we can help to prevent a delay in diagnosis.

There are few modifiable risk factors in preventing the development of isthmocele following caesarean section. Attention to surgical technique, in particular, avoiding incision into the cervical area, not performing a one-layer closure and not using delayed absorbable sutures could all be taken into consideration at the time of caesarean section.

One of the first steps in diagnostic workup is TVUS; however, often other imaging modalities are employed. Surgical treatment needs to be considered for symptomatic patients, including those who present with secondary infertility. The laparoscopic approach appears to be gaining popularity as the preferred method for treatment primarily because it aims to restore normal myometrial thickness. However, no technique has actually shown a statically superior outcome.

Patients need to be counselled to wait at least three months after treatment before they try for pregnancy, that it does not eliminate the risk of uterine rupture, and that the recommended mode of delivery is caesarean section at term.

References

1. G Bakavičiūtė, et al. Laparoscopic repair of the uterine scar defect – successful treatment of secondary infertility: a case report and literature review. *Acta Medica Lituanica*. 2016;23(4):227-31.
2. H Masuda, et al. Successful treatment of atypical cesarean scar defect using endoscopic surgery. *BMC Pregnancy and Childbirth*. 2015;15:342.
3. AJ Bij de Vaate, HA Brolmann, LF van der Voet, et al. Ultrasound evaluation of the cesarean scar: relation between a niche and postmenstrual spotting. *Ultrasound Obstet Gynecol*. 2011;37(1):93-9.
4. B Urman, et al. Laparoscopic Repair of Cesarean Scar Defect "Isthmocele". *J Minim Invasive Gynecol*. 2016;23:857-58.
5. AJ Bij de Vaate, LF van der Voet, O Naji, et al. Prevalence, potential risk factors for development and symptoms related to the presence of uterine niches following Cesarean section: systematic review. *Ultrasound Obstet Gynecol*. 2014;43:372-82.
6. AM Tower, et al. Cesarean Scar Defects: An Underrecognized Cause of Abnormal Uterine Bleeding and Other Gynecologic Complications. *J Minim Invasive Gynecol*. 2013;20:562-72.
7. A Setubal, et al. Treatment for Uterine Isthmocele, A Pouchlike Defect at the Site of a Cesarean Section Scar. *J Minim Invasive Gynecol*. 2018;25:38-46.
8. F Istvan, et al. Isthmocele: Successful Surgical Management of an Under-Recognized Iatrogenic Cause of Secondary Infertility. *Women's Health & Gynecology*. 2017;3(4):1-5.
9. OE Keag, JE Norman, SJ Stock. Long-term risks and benefits associated with cesarean delivery for mother, baby, and subsequent pregnancies: Systematic review and meta-analysis. *PLoS Med*. 2018;15(1):e1002494.
10. MK Hayes, et al. Repeat in vitro fertilisation success rates are lower in patients who deliver via caesarean section when using embryos from the same cohort. *Fertil Steril*. 2016;106(3):e48.
11. H Nazik, E Nazik. A new problem arising after the cesarean, cesarean scar defect (isthmocele) a case report. *Obstet Gyncol Int J*. 2017;7(5):00264.
12. A Murji, K Glass, N Leyland. Isthmocele. *Journal of Obstet Gynaecol Can*. 2013;35(9):779.
13. X Zhang, et al. Prospective evaluation of five methods used to treat cesarean scar defects. *Int J Gynaecol Obstet*. 2016;134:336-9.
14. G Aimi, et al. Laparoscopic repair of a symptomatic post–cesarean section isthmocele: a video case report. *Fertil Steril*. 2017;107:17-8.
15. A Akdemir, et al. Determination of Isthmocele Using a Foley Catheter During Laparoscopic Repair of Cesarean Scar Defect. *J Minim Invasive Gynecol*. 2018;25:21-2.
16. O Donnez, P Jadoul, J Squifflet, J Donnez. Laparoscopic repair of wide and deep uterine scar dehiscence after cesarean section. *Fertil Steril*. 2008;89:974-80.
17. D Bolla, et al. Laparoscopic Ultrasound-Guided Repair of Uterine Scar Isthmocele Connected With the Extra-Amniotic Space in Early Pregnancy. *J Minim Invasive Gynecol*. 2016;23:261-4.

Summary

- Isthmocele, also known as caesarean scar defect, is only just being identified and described in the literature and is becoming a subject of attention especially in those who present with secondary infertility.
- Caesarean section is one of the most commonly performed surgeries, and rates are continuing to increase all over the world.
- Isthmocele is an underdiagnosed cause of infertility.
- Isthmocele may present with secondary infertility, menstrual disturbance, dysmenorrhoea, pelvic pain or dyspareunia. Isthmocele must be considered in patients with a history of caesarean section that present with these symptoms.
- One of the first steps in diagnostic workup is TVUS, although other imaging modalities, such as MRI, are often employed.
- Surgical treatment should be offered to patients who present with secondary infertility.
- Reported pregnancy rates after endoscopic repair of isthmocele are high.
- The laparoscopic approach appears to be gaining popularity as the preferred method for treatment, primarily because it aims to restore normal myometrial thickness. However, no technique has actually shown a statically superior outcome.
- With caesarean section rates rising globally, it is predicted that we will encounter this issue with increasing frequency. Raising awareness of this common complication can help to prevent a delay in diagnosis.

ANZJOG

From the editor's desk

Prof Caroline de Costa
FRANZCOG
Editor-in-Chief
ANZJOG

Welcome to the report for readers of this issue of *O&G Magazine*.

First up, I am happy to report some additions to our Editorial Board. Gynaecological oncologists Dr Bryony Simcock and Dr Rhonda Farrell have joined us in place of A/Prof Penny Blomfield, who will retire from the Board at the end of May. The number of high-quality oncology submissions likely to be of interest to the clinicians reading *ANZJOG* has increased significantly over the past two years, hence the decision to appoint two new Associate Editors in Penny's place. I thank Penny for her major contribution to *ANZJOG* during her time with us.

Dr Jason Waugh has also joined the Board and brings with him enormous experience, both as a clinician and as the former editor of the RCOG publication, *The Obstetrician and Gynaecologist*. He will be a valuable addition to current Board members assessing the many submissions we are now receiving. In 2018, we saw a 10 per cent increase in numbers of submissions over the previous year and the majority of these are on obstetric topics.

I am also pleased to tell you that Sarah Ortenzio has returned as Senior Coordinator of Periodical Publications and has been joined by Lindsey Mathews and Foong-Ee Wyatt at Wiley who are assisting with submissions and managing many day-to-day queries from authors.

The April issue is now available and will, I hope, provide stimulating reading. Within it are two excellent reviews. Firstly, McCarthy et al review pregnancy outcomes for women with pre-pregnancy diabetes (PPDM) in Australia, in both urban and rural areas,[1] concluding that women with PPDM 'continue to experience excess adverse pregnancy outcomes, including maternal morbidity, complicated birth, perinatal loss, congenital anomalies and mother-infant separation' and calling for more and better research around outcomes, especially in rural areas. Secondly, Tanaka et al present a systematic review of the incidence of adverse events, their predictability and their associated mortality in gynaecogical hospital admissions in Australia.[2] These authors conclude that approximately one in ten gynaecological inpatients suffer at least one adverse event, and around 50 per cent of adverse events are considered preventable. Again, further research is needed on how adverse events may be prevented. Both reviews should be of considerable interest to clinician readers.

Among the many original articles on obstetric topics in this issue is a contribution from Yoong and associates recommending revisiting the techniques of abdomino-pelvic packing for intractable venous obstetric haemorrhage.[3] This paper has provoked some spirited discussion in the Letters to the Editor, which can be found in the e-pages at the end of the April issue.[4,5] As if to emphasise the importance of this topic, Flood et al present the recent figures for primary postpartum haemorrhage for the state of Victoria in 2009–2013;[6] disturbingly, these show a significant increase. There are also a number of articles around the topics of diabetes and obesity in pregnancy, topics also linked to increased PPH rates. Meloncelli et al discuss the role of multidisciplinary teams,[7] McGuane and others look at the role of early induction of labour for diabetes, obesity and/or macrosomia in their hospital[8] and Little et al discuss the association of maternal obesity with failed induction of labour.[9]

Two original articles in gynaecology look at the role of ultrasound, both in assisting the diagnosis of superficial endometriosis,[10] and in the post-operative assessment of treatment of endometriosis.[11]

In the area of sexual and reproductive health an interesting study from New Zealand finds that Māori women are willing to participate in self-taken collection of vaginal samples for HPV testing;[12] these findings have relevance to Australian practice as well.

The Current Controversies series has been held over until the June issue, but under Opinion in the April issue you will find a very thoughtful and informative discussion by Hayden Homer on preimplantation genetic testing for aneuploidy;[13] he concludes that the use of this 'very expensive IVF add-on' has so far only been proven to be of benefit for small groups of patients likely to have a good prognosis, and thus 'has not yet been shown to be effective, or indeed without harm' for other patient populations. Definitely good reading.

I have left the April editorials until last; there are two of these and both deal with the hot topic of gender equity in obstetrics and gynaecology. Carcel et al[14] (including two RANZCOG Fellows) make very important points, not only on education and training in O&G but also around research, stating that 'when sex differences are neglected (in obstetric and gynaecological research) we risk making the same mistakes we have seen in cardiac or other diseases where one sex or gender is disadvantaged due to poor treatment or care.' These authors also spell out the differences between sex and gender and acknowledge the issue of non-binary gender, which is receiving increasing recognition in our larger society. If we are truly to address the large issue of gender equity in O&G practice and as a College, we need first to acknowledge that gender is not binary.

The other editorial comes from Angstmann et al[15] and I am an author of this. We have looked at the gender composition of the College – Fellows and trainees over the years 1978–2018 – and at the concurrent composition of College committees. We have used data from College reports across nearly 40 years to show that the percentage of female Fellows has risen from around 5 per cent at the beginning of the period to 50 per cent today. Figures for gender of trainees are not available for the earlier years but, as one of the few Fellows who was active in College affairs in 1981 and still active, I can assure readers that the percentage of women admitted to train in Australia in 1981 was less than 5 per cent; in 2018 it was 83 per cent. Across state committees now women are in the majority; only Queensland has less than 50 per cent female representation and most others are well above 50 per cent, with Tasmania being 100 per cent female. At Council level, 50 per cent of members are women. Only at Board level is there a major discrepancy – there has only been one woman on the Board for the past three Boards, with five or six men.

I understand the Board is addressing the latter issues by co-opting some members; lawyer Julie Hamblin has a new observational and consumer role, the Chair of the Diplomates' group Dr Judith Gardiner and the RANZCOG CEO Vase Jovanoska are also present at meetings, so with Board member Dr Gill Gibson the makeup around the table is 36 per cent female. Board members almost always have been elected by the Council, the Councillors having themselves gained experience at state committee level before nominating for Council.

Council membership requires attendance at three sets of meetings each year in Melbourne, each lasting several days, plus much work in preparation. Board membership requires considerably greater commitment in time and unpaid work. This of course can be much more difficult for women juggling O&G practice with family and home commitments, and I believe that innovative ways of running these various bodies, which are essential to the functioning of our College and hence our professional independence, need to be found if more women are to put their hands up to serve the College in these roles. Shared roles and more use of tele-and video-conferencing are among the sensible suggestions in this direction.

For the past three Council elections, the overall percentages of women among candidates nominating have been 25 per cent, 42 per cent and 39 per cent. In the first two of these elections, the resulting Council membership corresponded exactly to these percentages, but in the current Council the percentage of women is higher (50 per cent); women are being elected to Council by their peers both female and male.

I had hoped that these two editorials would provoke thoughtful Letters to the Editor of *ANZJOG*, but so far there have been none. There has, however, been much comment on social media and this has taken two directions: that the College should address what is seen as a 'leadership crisis' in committee membership, and that there is no barrier to male applicants for specialist FRANZCOG training.

In fact, figures show that we already have gender equity across almost all College committees; the 'pipeline' is not leaking, except at the Board level. This does need to be addressed and it seems that will happen.

I also dispute the claim that 'there is no barrier to males entering specialist O&G training' at this point in the 21st century. In the 1970s and early 1980s there was 'no barrier' to women entering O&G training (application then was to individual hospitals, there were no College training schemes), but in 1974, even though I had my MRCOG Part 1, I could not find a job, while I saw many men accepted who had not passed that exam. Now applicants for training must have completed at least two years doing O&G at house officer level, have followed the pre-vocational pathway, have at least one relevant publication as first author, possibly be part-way or fully through a relevant post-grad degree, and generally have spent four or five years preparing themselves for the RANZCOG application process. When they see that the gender composition of the trainee intake is 83 per cent female, and likely to reach 100 per cent if left unchecked, I believe interested junior docs who are male may decide upon another specialty. I believe this is genuinely a barrier. I also believe that having a completely female RANZCOG would be as dystopian as I found the virtually all-male College when I returned from training overseas in the early 1980s. We live in a gender- and ethnically-diverse society and this needs to be reflected in our membership.

I welcome Letters to the Editor for *ANZJOG* on these (and on other) important topics, and I look forward to the next three years of participating in a College that has enthusiastic teams both gender- and ethnically-diverse, working together in the area of women's reproductive healthcare.

References

1. EA McCarthy, R Williamson, A Shub. Pregnancy outcomes for women with pre-pregnancy diabetes mellitus in Australian populations, rural and metropolitan: A review. *ANZJOG*. 2019;59(2):183-94.
2. K Tanaka, L Eriksson, R Asher, A Obermair. Incidence of adverse events, preventability and mortality in gynaecological hospital admissions: A systematic review and meta-analysis. *ANZJOG*. 2019;59(2):195-200.
3. W Yoong, A Lavina, A Ali, et al. Abdomino-pelvic packing revisited: An often forgotten technique for managing intractable venous obstetric haemorrhage. *ANZJOG*. 2019;59(2):201-7.
4. S Matsubara, R Usui, Y Sakuma. Additional procedures for pelvic gauze packing for obstetric haemorrhage. *ANZJOG*. 2019;59(2):E5.
5. A Govind, W Yoong. Response to Letter to Editor 'Additional procedures for pelvic gauze packing for obstetric haemorrhage'. *ANZJOG*. 2019;59(2):E6.
6. M Flood, SJ McDonald, W Pollock, et al. Incidence, trends and severity of primary postpartum haemorrhage in Australia: A population-based study using Victorian Perinatal Data Collection data for 764 244 births. *ANZJOG*. 2019;59(2):228-34.
7. N Meloncelli, A Barnett, F Pelly, S de Jersey. Diagnosis and management practices for gestational diabetes mellitus in Australia: Cross-sectional survey of the multidisciplinary team. *ANZJOG*. 2019;59(2):208-14.
8. JT McGuane, L, Grlj, MJ Peek. Obesity, gestational diabetes and macrosomia are associated with increasing rates of early-term induction of labour at The Canberra Hospital. *ANZJOG*. 2019;59(2):215-20.
9. J Little, R Nugent, V Vangaveti. Influence of maternal obesity on Bishop Score and failed induction of labour: A retrospective cohort study in a regional tertiary centre. *ANZJOG*. 2019;59(2):243-50.
10. P Chowdary, K Stone, T Ma, et al. Multicentre retrospective study to assess diagnostic accuracy of ultrasound for superficial endometriosis—Are we any closer? *ANZJOG*. 2019;59(2):279-84
11. R Seracchioli, D Raimondo, S Del Forno. Transvaginal and transperineal ultrasound follow-up after laparoscopic correction of uterine retrodisplacement in women with posterior deep infiltrating endometriosis. *ANZJOG*. 2019;59(2):288-93.
12. A Adcock, F Cram, B Lawton, et al. Acceptability of self-taken vaginal HPV sample for cervical screening among an under-screened Indigenous population. *ANZJOG*. 2019;59(2):301-7.
13. HA Homer. Preimplantation genetic testing for aneuploidy (PGT-A): The biology, the technology and the clinical outcomes. *ANZJOG*. 2019;59(2):317-24.
14. C Carcel, Z Wainer, A Henry, M Hickey. Why should the obstetrics and gynaecology community care about sex and gender issues in health? *ANZJOG*. 2019;59(2):181-2.
15. M Angstmann, C Woods, CM de Costa. Gender equity in obstetrics and gynaecology – where are we heading? *ANZJOG*. 2019;59(2):177-80.

Dr Andrew Browning: a man with a mission

RANZCOG

Andrew Browning was six years old when he first heard about Africa's medical missionaries. He was at Church with his family (an ordinary Sunday), but the stories of countless women dying in labour stayed in his mind. In that moment, he knew what he wanted to do with his life.

Today, with more than seventeen years' experience in Tanzania and Ethiopia, Dr Andrew Browning (AM) is widely regarded as the finest fistula surgeon in the world. He was a lead author of the WHO Training Manual for prospective fistula surgeons and is highly respected in countries where this problem is prevalent.

Early beginnings

'My biggest challenge? Trying to run a hospital without water, with staff that haven't been paid by the government for months and patients dying because of the lack of equipment.' – Dr Andrew Browning

As a medical student Dr Browning spent time in a remote mission hospital in Tanzania, on the border with Rwanda, for his medical elective. It was 1993, at the beginning of the genocide in Rwanda, and literally tens of thousands of refugees flooded into the area overnight. It was here that, for the first time, he was faced with the stark reality of practicing medicine without adequate resources.

Wishing to do more to help, he then visited his aunt Valerie Browning who has been working in the desert area of Ethiopia for 40 years. He also visited the Hamlin Fistula Hospital in Addis Ababa, Ethiopia, where he was invited to work.

After five years operating, researching and teaching with Dr Catherine Hamlin, Dr Browning moved to Barhidar, where he started and directed the first regional Hamlin Fistula hospital. He and two Ethiopian nurses would spend the next years operating on up to 600–700 fistula patients per year. He then started his own charity, the Barbara May Foundation, to build and run maternity hospitals to prevent fistula, train midwives and to establish fistula centres around Africa where the Hamlin's didn't work.

Thinking big

'All in all, I spent thirteen years in Ethiopia coordinating training programs and establishing health centres, but I wanted to take this to work outside Ethiopia. There is a huge fistula need in the rest of Africa; to build more hospitals' says Browning.

In 2011, Dr Browning moved to Tanzania, funded the refurbishment of another hospital and built a dedicated fistula clinic near Arusha. It was in this same year that he met Dr Geoff Kelsey.

'I was trying to find an organisation that had experience with fistulas. Dr Browning had written one of the best books on fistula I had laid my hands on, so I decided to write him an email. He replied straight away: he was moving to Tanzania and asked me if I wanted to join him' says Dr Kelsey.

Dr Kelsey went to Tanzania, taking with him donated sutures, catheters and other supplies from Australia. He was eager to learn more about obstetric fistulas. However, despite his interest, Dr Kelsey had only identified two fistula surgeries in over 35 years, so Browning tasked him with general gynaecology practice and allowed him to accompany him on several fistula surgeries for him to learn more.

'I enjoyed it so much that I have gone back every year since. Dr Browning is an amazing teacher that leaves his ego at the door. He maintains a huge workload himself on top of travelling around Africa teaching local doctors working on the frontline on the best way to manage fistulas as well as building maternity clinics to teach midwives and arranging support of under-resourced clinics with the belief that prevention is better than cure.'

Dr Browning's preventative approach has resulted in a marked increase in hospital usage by Tanzania's local communities. Dr Kelsey states that deliveries increased from 40 a month in 2010 to near 100 a month in 2012 at the hospital where he volunteered and later to 200 per month. 'This is great, but it also means that the local medical and nursing staff are stretched to the limit.'

Frustrated by a continuous lack of resources and wishing to adopt a prevention-focused approach, Dr Browning decided to establish the Barbara May Foundation in 2009. The Foundation's funds allow him to continue running projects in Tanzania, South Sudan, Uganda, Nepal and Ethiopia, at no cost to patients, that aim to reduce the high incidence of maternal death and extreme injury in pregnancy and childbirth.

'Treating obstetric fistula is one of the few operations that you can do that transforms completely someone else's life. It brings women back from being ostracised to having a normal life' says Browning. 'I'd love to treat more patients; especially because it only costs $200 AUD to ensure a woman has a safe delivery at a hospital in Africa.'

On the horizon

Dr Browning relocated to Australia last year while his sons finish high hchool.

'After 17 years working in Ethiopia and Tanzania, I now feel like Africa is my home. It's been really hard for me to adjust to Australia. In Africa, everything is unexpected. Nothing goes as planned. Australia is facing a very different set of barriers that requires me to think another way.'

Andrew remains dedicated to helping the more than two million women estimated to be suffering with existing obstetric fistula injuries throughout Africa by teaching surgeons via 'fistula camps' in Malawi, Sierra Leone, Kenya, Chad, Uganda, Congo, South Sudan, Somaliland, Nepal and Togo. He also consults on global maternal healthcare issues to United Nations Fund for Population Activities and the International Federation of Gynaecology and Obstetrics. He is very keen to talk to midwives and doctors, like Dr Kelsey, who wish to volunteer overseas.

'Go,' advises Kelsey. 'I found Africa to be exceptional. The people are friendly, welcoming and grateful. But be prepared; you don't want to have an agenda. You'll see that things are terrible but make sure to look at the local situation before you start wanting to make changes. You've got to be aware of context; not put people offside by telling them they should be doing things they cannot do. Also, go for long enough! You can't do or learn much in a fortnight.'

If you want to help financially or by volunteering, visit www.barbaramayfoundation.com.au.

A new standard: developing O&G care in the Solomon Islands

Dr Rebecca Mitchell
MBBS, MPH&TM, FRANZCOG

Some things are familiar – morning handover, ward rounds, teaching sessions – but there are aspects of O&G practice here that are vastly different.

I'm sitting on a bench inside the outpatients clinic at Honiara's National Referral Hospital (NRH), flanked by the department's four consultants and three registrars. A handful of junior doctors have also gathered, most recently returned to Solomon Islands having completed their undergraduate studies in Cuba.

This is the only O&G unit in the country. It's a precious resource in a county of 640 000 people, dispersed over a large swathe of the Pacific Ocean. Solomon Islands are an archipelago of approximately 900 islands – it's an incredibly beautiful country, but incredibly remote.

The handover from the night shift is telling. We hear about several neonatal complications, a stillbirth and, unfortunately, a maternal death. The latter is uncommon, but frequent enough that this feeling – a nauseating mix of hopelessness, regret and sadness – is familiar.

As a group, we discuss the issues affecting these women and the factors that contributed to their poor outcomes. Frequently, they have had limited antenatal care due to poor healthcare access. Occasionally, NRH has been short on necessary equipment and staff.

The Head of Department, Dr Leeanne Panisi, is all too familiar with these issues. From November 2011 until 2014, she was the sole local O&G in the country. She is an inspirational Solomon Islander who is doing an outstanding job as a clinical leader.

'Many women have a lot of difficulty getting to clinics and health centres, and sometimes people can take a long time to seek help, often because of the lack of transport, and sometimes because of the cost of the journey,' she told the World Bank in a recent profile piece.[1] 'And we don't have many specialist doctors in provincial areas; most are general practitioners. Specialists who deal with obstetrics and gynecology – there are only a few of us, and we are all based in Honiara.'

Despite the incredible efforts of Dr Panisi and colleagues, it's easy to see why neonatal and maternal mortality rates are high. The challenges are complex and resources are few. But there is a sense of optimism here at NRH, and it's an absolute pleasure to visit Dr Panisi and her team as they continue their pursuit of improved women's health across the country.

Obstetric care at NRH

The night team hand over the care of the women in birth suite, which consists of three sections – the first-stage area, the second-stage area and the immediate postnatal area.

With approximately 15 births per day (5600 births per year), the women in the first-stage area are not individually discussed at handover. Nor are they allocated a private area; they congregate in the room, swaying in a trance-like state, until they feel the urge to push. At this point, they are examined and taken to one of the second-stage rooms.

Today, three out of the four beds in the second-stage area are occupied. In bed one, Barbara* is pushing to deliver the second twin, learning only five minutes ago that she was expecting two babies. This is often the case for multiple pregnancies as women do not routinely undergo ultrasound scans antenatally.

In bed three, Cathy* is in obstructed labour with her first baby. A caesarean section has been arranged; however, before getting into the operating theatre, required blood needs to be cross-matched. Her relatives are currently at the blood bank donating blood in case she requires a transfusion; unfortunately, this is often a cause of delay for delivery. At NRH, as with many developing settings, the caesarean section rate is much lower than Australia and New Zealand, at approximately 10 per cent.

The mood is lighter in bed four. Janet* is cradling her newborn while a midwife delivers her placenta. It's a beautiful moment in an otherwise chaotic environment.

I meet these women on the morning ward round following handover. As with many low-income countries, they are alone in birth suite, supported only by the midwives and doctors.

Husbands and relatives mill around outside the doors awaiting news and updates. I smile and say good

morning to them as we walk into birth suite. 'Morning iufela,' they reply, in a calm and affectionate manner that sharply contrasts the struggles beyond the door.

Improving outcomes and consistency of care

So what am I doing here? In 2017, I volunteered as a senior O&G registrar at NRH. My role was part of the Solomon Islands Graduate Internship Supervision and Support Project (SIGISSP), and my primary responsibility was to support and mentor Cuban-trained junior doctors transitioning to practice in the Solomon Islands.

I'm back in Honiara for a project that's become a labour of love (pardon the pun) over the last 18 months. I have been working with Dr Panisi and her team to develop a national Standard Treatment Manual (STM) for O&G. It's an exciting initiative and has just reached completion.

When I first arrived in Honiara, I asked many questions of the local consultants, registrars and midwives. I wanted to be sure that the treatment I provided was relevant, appropriate and consistent with local practice. I also spoke with patients about their journey to NRH and their healthcare experiences.

After many conversations, it became apparent that there was significant variation in quality of care across the country. Dr Panisi and colleagues explained that many healthcare workers, especially those in the provinces, are uncertain about best practice women's healthcare and lack access to clinical resources.

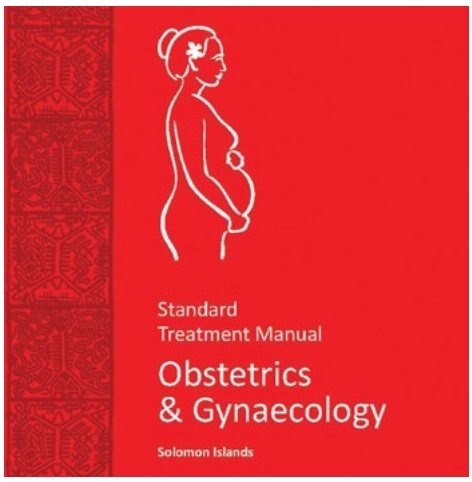
Together, we identified that a standardised O&G treatment manual could address some of these challenges. While the World Health Organization reference text 'Managing Complications in Pregnancy and Childbirth' is theoretically available to Solomon Islands clinicians, it is not locally specific and many of the treatment options are not available.

With much gusto, we formed a working group to develop a STM in O&G for the country. This was chaired by Dr Panisi and included the three other Solomon Islands O&G specialists, two local registrars, a senior midwife and me. The Papua New Guinean STM (a document with similar objectives, but relevant to the PNG context) was used as a template (with permission), and each chapter was reviewed and updated by a member of the working group. My role was to compile the chapters, edit the content and arrange external review by provincial health workers and two international O&G specialists.

The end product is a 200-page, A6 manual – designed to fit in the pocket of Solomon Islands healthcare workers. It has been endorsed by the Solomon Islands Ministry of Health & Medical Services, and professionally designed thanks to generous sponsorship from RANZCOG.

The World Health Organization has kindly funded printing of the manual, which means that every Solomon Islands healthcare worker dealing with women will have a personal copy. They will be disseminated using the pharmacy distribution network and delivered during O&G outreach visits. The content will even be available via an app sponsored by the Therapeutic Guidelines Foundation.

On 21 May, the Manual was officially launched at NRH. This included a celebratory lunch and, in true Solomon Islands fashion, lots of music and dancing.



Funding for this event, and associated activities, came from the AVI Go Back Give Back competition.

In the week of the launch, the STM Working Group also facilitated a series of education sessions on the Manual and its content. Workshops were delivered to senior clinicians (using a train-the-trainer model) as well as end users. Emergency management flowcharts, based on content in the Manual, have been printed and placed on the walls of birth suite as a quick reference guide.

The process of developing and publishing the STM took close to 18 months of work. It required a huge amount of time and dedication on the part of local clinicians, who pursued the project with vigour from the outset.

The end product is excellent (well, at least the working group thinks so!), but the development process also extremely positive. The STM is an example of Australian and Pacific colleagues collaborating towards improved women's health, and I am proud to have played a small part. The clinical and educational aspects of my volunteer assignment were rewarding, but it is capacity development initiatives such as the STM that will have lasting effects.

A new standard?

At the end of the day, the O&G team gather briefly in birth suite to discuss patient management plans for the night. There's a new flowchart on the wall and a copy of the STM on the work desk. Neither of these things guarantee good clinical outcomes, but my optimistic ambition is that the Manual will positively impact on the care of women in this birth suite and beyond.

This morning's handover, and the morbidity and mortality attached to it, is a sad reality for now. The hard work of the O&G team at NRH is changing that however, and it's been inspiring to watch the consultants, registrars and midwives galvanise around the STM project.

For Dr Panisi's part, her vision is clear. 'I'm hopeful that we will improve maternal health in Solomon Islands. Preventing maternal deaths and helping women with gynaecology problems is our goal.'

* all patient names have been changed.

Reference

1. The World Bank. Inspiring women in the Pacific: Leeane's story. 2018. Available from: www.worldbank.org/en/news/feature/2018/03/05/inspiring-women-in-the-pacific-leeanne-story.

The Royal Australian and New Zealand College of Obstetricians and Gynaecologists

Excellence in Women's Health

Introducing the **New Fellow Resource Guide**

This handy guide contains everything New Fellows need to know as they take the next step in their journey. The Membership team will provide a copy to all New Fellows upon elevation.

For further information contact: **membership@ranzcog.edu.au**

www.ranzcog.edu.au/members

Letters to the Editor

Dr Polly Weston
MBChB (Hons), MRCOG, FRANZCOG

I very much enjoyed the article 'Threatened preterm labour out bush' by Drs Jared Watts and Han-Shin Lee, *O&G Magazine* Vol. 21 No. 1 Autumn 2019.

I read with interest that patients 'in active labour, at high risk of bleeding or with fetal distress, should not be considered for transfer except in extreme circumstances.' This obviously makes sense. Unfortunately, it contrasts sharply with my experience working in rural New Zealand. I live in Queenstown, which despite huge population growth and a minimum of 600 births a year, has only a primary birthing unit. We are almost three hours by road from the nearest secondary unit – only there is analgesia, continuous CTG monitoring, theatre and obstetric assistance available. In winter the road is often deep in snow and a large volume of tourist traffic significantly hampers travel.

Helicopter evacuation is the usual route of transfer, and this is weather dependent. There is at least one helicopter evacuation per day – sometimes for labouring VBACs or women requesting epidural, but more often for obstructed or preterm labour, fetal distress, prolonged second stage over three hours or postpartum haemorrhage. As the patient departs in the helicopter, their partner faces a terrifying journey alone by road, often arriving hours after delivery or surgery has occurred. In fact, it appears that most transfers here occur for all the reasons you would prefer to avoid it.

This isn't a question, just demonstrates the enormous range of available resources to an obstetrician in rural practice and difference in management planning according to location.

Dr Rosemary Anne Jones
MBChB, FRACOG, FRANZCOG

I was tickled pink to read the article 'PMT, PMS and PMDD: is there a difference?' *O&G Magazine* Vol. 20 No. 3 Spring 2018, by three psychiatrists about these questions of ovarian dysfunction causing hormonal upsets. Not that I am about to suggest that those of us who trade in hormones should seek a monopoly on these overworked elements, but a balance of expertise might have been more convincing.

In my reading of their review article, there seemed little reference to hormonal matters apart from some alarming comment such as 'Thus, hormone levels and flux alone appear to be irrelevant, however, the relevant neurobiological and physiological changes may represent an underlying sensitivity to such changes'. In fact, there seemed to be no discussion of the actual levels of oestrogen and testosterone.

Full marks to them then for attempting to ram the contents of this elusive condition into a categorisation box, but sadly then to consider, in the main, psychotropic medication as their holy grail. In fact there was another reference to oestrogen in that '...there are no demonstrable differences in reproductive hormonal levels...', by which I assume they are referring to oestrogen and possibly progesterone, but surely not testosterone! And if they were thrown by the absence of measurable hormonal fluctuations, then they have failed to understand what a slippery beast they are dealing with.

Recognising that PMS is not an easy matter, I have taken an alternative approach and evolved a 'paradigm of understanding' that I am happy to report yields well to treatment with hormones. I have presented this concept on three occasions and have succeeded only in boring the proverbial pants off my audience. Perhaps I should be so bold as to submit a copy of the paradigm not for publication as such but for the amusement of you, the Editor, and see how many paragraphs you can consume before your eyelids droop.

At least I could identify with the criteria set out in the DSM-V for PMDD. I would have changed the emphases perhaps. As for aetiology, good luck with that. And, as a bottom line, have the researchers made any attempt to relate the incidence of breast cancer with this condition; and what might the rates of hysterectomy (for any reason) be in this population of women?

Provincial Fellows Practice Visits in Australia

Why you should sign up for a visit in 2020

A/Prof Ian Pettigrew
FRANZCOG

Practice visits are an excellent form of peer review and audit. By the end of 2019, 72 Provincial Fellows Practice Visits will have been held in Australia over eight years.

Background and funding

RANZCOG Practice Visits commenced in New Zealand many years ago and have become commonplace there. They are looked upon favourably by the Medical Board of New Zealand.

Based upon the very successful New Zealand model, Practice Visits began in Australia as a pilot project in 2005. They were championed by Dr Philip Hall and supported by the Provincial Fellows Committee and the Continuing Professional Development Committee.

The visits were initially funded via two Commonwealth Government funding programs; firstly the Support Scheme for Rural Specialists (SSRS), then by the Rural Health Continuing Education (RHCE) program. The cost of a visit to an individual is $3000. The visits now continue to operate on a partially self-funded model, with some additional support funding generated through profits from the Provincial Fellows Annual Scientific Meetings.

Currently, individuals may apply for funding support via the Support for Rural Specialists in Australia (SRSA) program. https://ruralspecialist.org.au

What is a Practice Visit?

A Practice Visit is a collegial peer review of a specialist within their work environment intended to provide detailed, in-depth professional development relevant to the context and setting of the specialist receiving the visit. It provides doctors with an opportunity to gain an understanding of their performance/competencies and allows them to compare themselves to RANZCOG standards, with the aim of improving safety and quality of care. Visits are conducted with the support of Fellow colleagues.

The visit aims to provide constructive feedback about positive aspects of practice and areas of vulnerability that could be addressed to improve patient care.

What is involved?

Before the visit takes place, the Fellow is required to complete:

- a memorandum of understanding in relation to the visit
- a practice profile questionnaire
- a self-assessment survey
- the dispersion and collection of 50 patient satisfaction questionnaires
- a logbook of the last three months of procedures
- compilation of the program for the day of the visit, including the arrangement of interview times with colleagues and obtaining patient consent for the theatre observation

During the visit the Fellow will:

- receive a one-day site visit by two visiting Fellows
- be interviewed by the two Fellows
- undergo review of clinical workload, case mix and record keeping
- be observed carrying out one major and one minor procedure in theatre
- have their practice surroundings reviewed, including equipment and processes such as OHS, record systems in public and private (both practice settings are reviewed where possible)
- have multi-source feedback involving interviews with relevant personnel such as unit managers, CEO, Director of Nursing, Director of Medical Services, RANZCOG trainees and other junior staff about the context of care provided within the organisation and systems issues
- receive feedback about the visit and initial findings

After the visit:

- the results of the visit will be reviewed confidentially by a Steering Committee
- the visited Fellow will be provided with a written report outlining positive aspects of their practice and areas of vulnerability with suggestions on practice improvement

What are the benefits?

Practice Visits are beneficial for both the Fellow who is being visited as well as the two visiting Fellows.

Fellows visited:

- gain valuable feedback on how their practice is progressing
- identify areas of vulnerability and reduce risk
- receive confidential reporting on performance
- a reduction in professional isolation
- earn Practice Audit & Reflection (PAR) points

Visiting Fellows:

- view how other practices operate
- gain training and development in how to conduct peer review
- earn Practice Audit & Reflection (PAR) points

What do participants think?

'It was a very useful exercise to look at all these areas of my practice critically before the actual visit and to get organised for the actual day of the visit.'

'The best part of receiving a visit was having an objective appraisal of my solo practice and interacting with other regional specialists.'

'The visit forced a review of every aspect of practice – things on the 'to do' list for years got done.'

'Input from visitors was very constructive and delivered with a supportive attitude.'

'Excellent exercise – my practice and I are stronger for the experience.'

Commonwealth Qualified Privilege Scheme

Practice Visits held in Australia are currently covered under the Commonwealth Qualified Privilege Scheme allowing for free communication within this activity.

How to be involved

To be eligible for a visit, the visited Fellow must be a RANZCOG Provincial Fellow currently working in a regional, rural and/or remote area (classification RA2-5) for the past year. See www.doctorconnect.gov.au/locator for classification details.

To enquire regarding 2020 visits, please contact RANZCOG Provincial Fellows Coordinator, Ms Angie Spry, for further details:
Email: aspry@ranzcog.edu.au
Phone: (03) 9412 2971

The future of Practice Visits

It is hoped that Practice Visits will continue to expand and be offered to the greater Fellowship.

College Statements update March 2019

Revised College Statements

The following statements were approved by RANZCOG Council and Board in March 2019:

Use of prostaglandins for induction of labour (C-Obs 22)
Revisions include:
- Updated references

Birth after previous Caesarean Section (C-Obs 38)
Revisions include:
- Updated references
- Inter-delivery interval was changed to inter-pregnancy interval (delivery to conception) and the RCOG period of 12 months applied.
- It was agreed that the advice on oral intake should be in line with recommendations on fluids and oral intake in the College's Routine Intrapartum Care (C-Obs 31) statement.
- Reference to 'Off-label' drug use (5.10.3 on page 14) was changed to indicate the specific use of prostaglandins for induction of labour is against manufacturer's advice
- A table outlining risks of uterine rupture associated with different methods of induction and success rates was added.
- Recommendation 14 regarding the use of prostaglandins for IOL was softened.

Alcohol in Pregnancy (C-Obs 54)
For retirement
- Rationale: this statement has been amalgamated with the statement Substance use in pregnancy (C-Obs 55).

Genetic Carrier Screening (C-Obs 63) – new
This statement was developed to address the gap in guidance to health professionals regarding advice on the counselling of women and couples prior to, and in the early stages of, pregnancy in relation to genetic carrier screening in Australia and New Zealand. This document aims to assist clinicians to provide women and couples with information regarding their chance of having a child with an inherited genetic condition, so that all carriers are given the option of genetic counselling to discuss the implications of their results with an expert.

Prevention of Congenital Cytomegalovirus (CMV) Infection (C-Obs 64) – new
As CMV is the most common congenital infection, the WHC felt it is appropriate that a statement should be developed to raise awareness among College Fellows. The committee discussed a stand-alone statement on CMV as opposed to a general statement on infections in pregnancy. Members felt that generally, people look up a statement by a specific name or procedure and due to the relatively high incidence of CMV a separate statement is preferred.

Termination of Pregnancy (C-Gyn 17)
Revisions include:
- Title change to Abortion
- Aligned to new legislation
- Updated references

Guidelines for HPV Vaccine (C-Gyn18)
Revisions include:
- New evidence re: rates of decline since introduction of vaccine
- Updated references

The use of mifepristone for medical abortion (C-Gyn 21)
Revisions include:
- New paragraph regarding oral contraceptives (specifically Diane-35) and risk of VTE in light of correspondence to the College and recent publicity surrounding the daughter of MP Julian Hill
- Updated references

Vaginal Rejuvenation and cosmetic vaginal procedures (C-Gyn 24)
Revisions include:
- Updated references

Combined Hormonal Contraceptives (C-Gyn 28)
Revisions include:
- Updated references

Interview and selection of Obstetricians and Gynaecologists for visiting medical officers, salaried medical officers and academic staff in Australia (WPI 4)
For Retirement
Rationale: It was felt that most hospitals would have their own policies and procedures around recruitment of staff.

The personally controlled electronic health record (PCEHR) (WPI 22)
For Retirement
Rationale: This statement is very out-of-date due to many new developments in this area, particularly the introduction of My Health Record.

A full list of College statements can be viewed at www.ranzcog.edu.au/Statements-Guidelines.

RANZCOG Patient Information

There are 37 RANZCOG Patient Information Pamphlets, including the Pregnancy and Childbirth pack of 18 pamphlets, now available. All of these products can be viewed and ordered at www.ranzcog.edu.au/patient-information-pamphlets.
The following titles were approved for publication and are now available:
- Pregnancy Loss
- Gestational Trophoblastic Disease
- Pelvic Organ Prolapse
- Stress Urinary Incontinence

Prof Yee Leung
Chair
RANZCOG Women's Health Committee