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THE PRESIDENT's MESSAGE

The battle of the real surgeons – a comic opera

Professor Hans Evers
WES President

You may have noticed a vicious war is raging. Between surgeons. About excellence. They are calling each other names. One of them, on his website, sets apart the endometriosis experts from the endometriosis "experts" (mind the quotation marks). It's about surgery, so coins count. Some of them seem to fear that if bowel resection is paid better than a selective resection of lesions, while being faster and easier, their fellow surgeons would go for an unnecessary bowel resection rather than take the trouble of removing just the invasive process. It has even been suggested that there should be a camera in every OR and mandatory video registration to prevent abuse and permit quality control (pity him who is going to watch all those tedious hours of deadly boring recordings).

And researchers stink. Some for considering endometriosis a 'monolithic' disease (which is either there or not) and correlating biological and genetic parameters to it; others for differentiating the disease into too many sub-groups *("Too many notes, Herr Mozart, too many notes"),* aggressive and dormant, occult and visible, superficial and deep, peritoneal and recto-vaginal, primary and secondary, waxing and waning, immunological and inflammatory, progressive and regressive, relapsing and recurring, cystic and solid, congenital and acquired.

Reports on series of patients are still the mainstay of surgical publications, which is why surgical research has been compared to comic opera: such studies are not scientifically serious, and as such a poor basis for surgical practice (Horton, 1996). On the other hand, performing a methodologically sound surgical trial is notoriously difficult and fraught with problems (even ethical ones). This should not deter us from doing them, however.

There are already too many examples of observational studies suggesting benefits from a treatment whereas subsequent randomised trials failed to provide evidence of such a benefit or even demonstrated harm. There is a lot to be gained by ending the hostilities and bringing researchers and surgeons together again.

If oncologists would fight their battles like this, who would take them serious? They are what they are now, well organised, with robust multicentre studies applying sound research methodologies, decent (inter)national research funding, appropriate reimbursement schemes, and considerable progress to show, both in cure-rates and survival, because they gathered in centres of excellence, with fundamental and clinical researchers, high quality patient care, and – among many medical and paramedical specialists – qualified surgeons.

John Sampson died in 1946. He was the last one-man centre of excellence. Now is the time to start gathering excellent research and multi-disciplinary patient care in polydimensional endometriosis centres. We need the knowledge *and* the skills. And we need the patient as a partner.

Horton R. Surgical research or comic opera: questions, but few answers. Lancet 1996;347:984-985.

In this issue of the WES e-Journal

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A WORD FROM THE EDITOR

Our chief wants the peace pipe

Dr Luk Rombauts
WES e-Journal Editor

The editorial team is pleased to bring you a new issue of the WES e-Journal. Still high on sugar following the Easter egg hunt, this issue is higher octane than usual. We have the usual contributions from the president, a guest editor and our book reviewer.

This month Hans Evers makes a rather passionate plea. I get the distinct feeling he is tired of some of the in-fighting in our discipline and he wants to see more cooperation between all stakeholders in the battle against endometriosis. He quotes many ongoing disputes and controversies, none of which have brought us much closer to a cure for endometriosis.

Although a healthy scientific debate is vital in scientific communities, Voltaire once said that "**a long dispute means both parties are wrong**". Or as Bertrand Russell put it: "**The most savage controversies are about matters as to which there is no good evidence either way**". So now seems the time to acknowledge that we can do better. Collaborating more cost-effectively should bring us closer to that elusive goal of understanding the endometriosis enigma. And as Alice (in Wonderland) said "it would be so nice if something would make sense for a change" (Lewis Carroll).

It is also a pleasure to have Paolo Vercellini as our guest editor for this issue. He brings us a provocative 'readers digest', in which he continues the debate on the link between endometriosis and cancer started in the Nov/Dec 2009 issue. The topic seemed controversial enough because we received not one but two letters from eminent members wanting to join into the debate. I regret that we have not had further responses to the letters by Ivo Brosens and Philippe Koninckx, published in our last issue, but Paolo addresses some of the previously raised issues in his contribution. We still encourage our readers to make their opinions known too.

Anusch Yazdani has managed to read, digest and critically review another book for us. In this issue he reveals what he liked and disliked about *Endometriosis: current management and future trends* by Juan Garcia-Velasco and Botros Rizk. Although nothing beats a personal perusal and appraisal, his insights may guide you with the purchase of your next medical library addition.

Enjoy!

UPCOMING MEETINGS

12th International Meeting on Gynaecological Surgery
5 - 8 May 2010
Avellino, Italy

58th Annual Clinical Meeting of the ACOG
15 - 19 May 2010
San Francisco, USA

66th Annual Clinical Meeting of the Canadian Society of Obstetrics & Gynaecology (SOGC)
2 - 6 June 2010
Montreal, Canada

ESHRE pre-congress course: Endometriosis - how new technologies may help
27 June 2010
Rome, Italy

ABSTRACT SUBMISSION DEADLINE
for this year's ASRM meeting is
3 May 2010 at www.asrm.org/presenters

Advancing the art and science of endometriosis: from stem cells to radical excision
20 May 2010
New York, USA

32nd British Congress of Obstetrics and Gynaecology
2 - 3 June 2010
Belfast, United Kingdom

World Congress of Minimally Invasive Gynecologic Surgery
26 - 29 June 2010
Dubrovnik, Croatia

26th Annual Meeting of ESHRE
28 - 30 June 2010
Rome, Italy

COMPLETE CONGRESS SCHEDULE

GUEST EDITOR's RESEARCH DIGEST

The endometriosis-ovarian cancer connection: challenging conventional wisdom

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Introduction

In the past few years the association between endometriosis and ovarian cancer has been the matter of intense research (Somigliana et al, 2006; Viganò et al, 2007). The WES e-Journal recently hosted a very interesting debate on this topic, with articles by Liselotte Mettler (2009), Luk Rombauts (2009), and Philippe Koninckx with Anastasia Ussia (2010). In the last issue our editor invited the readers to continue the discussion (Rombauts, 2010). Along this line, this Guest Editor's Digest is focused again on the endometriosis-ovarian cancer connection, with the aim of disentangling at least some of the many uncertainties surrounding the problem.

In light of a repeatedly observed increase in risk of gonadal malignancy, several experts fostered screening for endometriosis as well as early surgical treatment with the objective of timely eradicating what could reveal a pre-neoplastic condition (Nezhat et al, 2008; Mettler 2009).

However, much remains to be clarified with regard to the pathogenesis of ovarian cancer itself, the strength of the association and the potential causal relationship between endometriosis and ovarian malignancy, and, more importantly, the definition of endometriosis as a pre-malignant condition. Three recent publications on this issue may help the readers disentangle some doubts.

Traditional view flawed

In the March issue of the American Journal of Surgical Pathology, Robert Kurman and Ie-Ming Shih (2010) published a very interesting review and opinion paper on the origin and pathogenesis of epithelial ovarian cancer that all WES members are cordially invited to read.

Based on genetic, bio-molecular, and histopathological observation, the authors suggest that the traditional view of ovarian carcinogenesis may be flawed. According to current opinion, the various tumours are all derived from the ovarian surface epithelium (mesothelium), and subsequent metaplastic changes lead to the development of the different cell histotypes (serous, endometrioid, clear cell, mucinous, and transitional cell).

Contrary to this theory, the authors suggest that the vast majority of what seems to be primary ovarian cancers are derived from the fallopian tube and endometrium and not directly from the ovary. In particular, serous tumours would develop from the fimbriated portion of the fallopian tube, endometrioid and clear cell tumors from endometrial tissue passing through the fallopian tube resulting in endometriosis, and mucinous as well as Brenner tumours from transitional-type epithelium located at the tubal-mesothlial junction where the fimbria makes contact with the peritoneum.

With specific reference to endometriosis, Kurman and Shih maintain that, if retrograde menstruation accounts for most cases of the disease, it is logical to assume that endometrioid and clear cell tumours develop from endometrial tissue that implanted on the ovary. This hypothesis is further supported by epidemiologic evidence showing that a protective effect for tubal ligation was found only for these specific histotypes.

Defining neoplastic potential

This article by authoritative and recognised experts in the field, definitely clarify that endometrioid and clear cell ovarian carcinomas derives from endometriosis. Having said that, we are very far from demonstrating that endometriosis is a pre-malignant condition.

Endometrium has the potential to undergo neoplastic degeneration, be it within or outside the uterine cavity. Therefore, it is not surprising to observe neoplastic transformation of this mucosa even at ectopic sites. Moreover, uterine and ovarian endometrioid carcinomas share common risk factors. Defining a lesion as 'pre-malignant' implies a series of genetic, molecular, epidemiologic, and clinical requisites that do not seem to be fully met in the case of endometriosis.

The Origin and Pathogenesis of Epithelial Ovarian Cancer: A Proposed Unifying Theory
Am J Surg Pathol 2010;34:433–443

Kurman RJ and Shih IeM

ABSTRACT: Ovarian cancer is the most lethal gynecologic malignancy. Efforts at early detection and new therapeutic approaches to reduce mortality have been largely unsuccessful, because the origin and pathogenesis of epithelial ovarian cancer are poorly understood. Despite numerous studies that have carefully scrutinized the ovaries for precursor lesions, none have been found. This has led to the proposal that ovarian cancer develops de novo. Studies have shown that epithelial ovarian cancer is not a single disease but is composed of a diverse group of tumors that can be classified based on distinctive morphologic and molecular genetic features. One group of tumors, designated type I, is composed of low-grade serous, low-grade endo- metrioid, clear cell, mucinous and transitional (Brenner) carcinomas. These tumors generally behave in an indolent fashion, are confined to the ovary at presentation and, as a group, are relatively genetically stable. They lack mutations of TP53, but each histologic type exhibits a distinctive molecular genetic profile. Moreover, the carcinomas exhibit a shared lineage with the corresponding benign cystic neoplasm, often through an intermediate (borderline tumor) step, supporting the morpho- logic continuum of tumor progression. In contrast, another group of tumors, designated type II, is highly aggressive, evolves rapidly and almost always presents in advanced stage. Type II tumors include conventional high-grade serous carcinoma, undifferentiated carcinoma, and malignant mixed mesodermal tumors (carcinosarcoma). They displayTP53 mutations in over 80% of cases and rarely harbor the mutations that are found in the type I tumors. Recent studies have also provided cogent evidence that what have been traditionally thought to be primary ovarian tumors actually originate in other pelvic organs and involve the ovary secondarily. Thus, it has been proposed that serous tumors arise from the implantation of epithelium (benign or malignant) from the fallopian tube. Endometrioid and clear cell tumors have been associated with endometriosis that is regarded as the precursor of these tumors. As it is generally accepted that endometriosis develops from endome- trial tissue by retrograde menstruation, it is reasonable to assume that the endometrium is the source of these ovarian neoplasms. Finally, preliminary data suggest that mucinous and transitional (Brenner) tumors arise from transitional-type epithelial nests at the tubal-mesothelial junction by a process of metaplasia. Appreciation of these new concepts will allow for a more rational approach to screening, treatment, and prevention that potentially can have a significant impact on reducing the mortality of this devastating disease.

In fact, the risk of neoplastic degeneration of the endometrial mucosa appears similar whether at eutopic or ectopic site. Examples of pre-malignant conditions include actinic keratosis, Barrett's esophagus, atrophic gastritis, and cervical dysplasia.

The epithelium of the cervix itself does not constitute a pre-neoplastic condition, and nobody would suggest its systematic removal or destruction with the purpose of preventing squamous cell carcinoma. Only if and when cervical intraepithelial neoplasia (CIN) develops, is surgical treatment indicated. However, CIN is easily detected with a simple, non-invasive, and inexpensive test, whereas identification of cellular atypia within endometriotic implants is a very complex issue.

Atypia in endometriosis
The first question is then: how frequent are dysplastic lesions within endometriotic foci?

A recent paper by Mohamed Bedaiwy from the group of Tommaso Falcone gives us important information in this regard (2009). To gain more insight into the spectrum of histologic and cytologic abnormalities associated with endometriosis, the authors examined 2000 cases of endometriosis retrieved from the database of the Department of Pathology of the Cleveland Clinic Foundation in the period 2000-2003.

All available pathology slides were examined and reviewed by certified pathologists. Only six cases (6/2,000; 0.003%) had cytologic and histologic atypia. Most of the considered cases presented with cysts, therefore they could have been easily identified preoperatively with transvaginal ultrasonography.

Based on these figures from a very large series of consecutive cases collected in a tertiary care reference centre, the prevalence of histologic anomalies in women with undiagnosed endometriosis should be exceedingly low, rendering hardly justifiable embarking in screening programmes with the objective of preventing the development of endometrioid and clear cell ovarian cancer.

Screening in asymptomatic women
The problem here is also the appropriateness of extending the diagnostic process to asymptomatic women, and of performing surgery in subjects in whom there are no otherwise established indications. Moreover, whereas endometriomas may be recognised with ultrasonography, superficial implants escape detection. However, there is no proof that ovarian cancer arises only in cysts. Several experts foresee the development of more reliable markers than 'simple' CA 125, with the objective of identifying endometriosis more accurately (Nezhat et al, 2008).

Then the problem would arise on what to do next, as presence of superficial endometriosis in asymptomatic subjects may not be synonymous of disease or may be a transient phenomenon.

Screening for endometriosis is a very difficult and delicate issue. In many cases the disease may regress spontaneously or may need no treatment at all.

There are no data demonstrating that early surgical treatment in these conditions is associated with a reduced risk of progression or, ultimately, of ovarian cancer.

Moreover, women could be left with a generic diagnosis and without the knowledge for discriminating between different degrees of clinical severity.

This could generate excessive concerns and could potentially increase the risk for conflicts of interest involving companies producing the tests, pharmaceutical industries, and surgeons (Sheather, 2010). Many more women than today would be treated without robust evidence of effectiveness, and with possible misuse of health care resources.

Pelvic Endometriosis is Rarely Associated with Ovarian Borderline Tumours, Cytologic and Architectural Atypia: A Clinicopathologic Study
Pathol Oncol Res 2009;15:81-88

Bedaiwy MA, Abd-Elwahed Hussein MR, Biscotti C, and Falcone T

ABSTRACT: Endometriotic foci, especially ovarian ones, with epithelial cytologic atypia may be precursors of cancer. This study presents an overview of the atypical cytological and histopathological findings associated with endometriosis. Six cases of endometriosis, with atypical histological and cytological changes, were obtained from the archives of the Department of Pathology at Cleveland Clinic Foundation between year 2000 and 2003. The size of the base from which these cases were drawn was 2000 cases of endome- triosis. The age range of the patients was from 29 to 52 years. The clinical presentations included infertility (three cases), pelvic pain (three cases), adenexal and pelvic masses (four cases). Stage IV endometriosis with extensive pelvic involve- ment was found in two patients. Intraoperatively, the endometriotic lesions involved the ovaries (all cases); Cul de sac (four cases); urinary bladder (two cases); sigmoid colon, hemidiaphragms, and uterine vessels (one case each). The endometriotic lesions were associated with uterine leiomyomas (two patients) and adenocarcinoma of the vagina (one patient). Histologically, in addition to endometrial type glands and stroma, usually found in endometriosis, we observed both cytologic and pattern atypism involving the epithelium in all cases. The features of cytologic atypia included nuclear stratification, hyperchromatism, and pleo- morphism. The features of pattern atypia were complex glandular pattern, papillary formations and psammoma bodies. In two cases, these features were sufficient for diagnosis of borderline Mullerian seromucinous tumours. One patient had recurred with metastatic adenocarcinoma of the vault. She died later from disseminated metastatic disease. There is a rare association between pelvic endometriosis and borderline ovarian tumours (three cases), cytologic and pattern atypia (two cases); mesothelial hyperplasia, endo- salpingiosis (two cases), and metastasis (one case). Cytologic and pattern atypia can develop in the endometriotic foci and therefore, these lesions should be thoroughly scrutinized for presence of these changes. Our findings recommend surgical excision of these foci rather than their simple cauterization.

Increased ovarian cancer

The lifetime risk of developing endometrial cancer is 2/100, which does not appear to be lower than the probability of malignant degeneration of endometriosis.

The results of epidemiologic studies on the association between endometriosis and ovarian cancer are somewhat discordant, with a relative risk between 1.3 and 2 being the most frequent finding (Somigliana et al, 2006; Viganò et al, 2007; Nezhat et al, 2008). These figures have been recently confirmed by data from the latest study on the issue conducted in the region of Quebec, Canada. Aris (2010) retrieved information on women with ovarian cancer who were identified within the archive of the Centre Informatisé de Recherche Evaluative en Service et Soins de Santé, that manages clinical and pathological data obtained from the computerised patients' records of all residents in the Estrie region of Quebec (about 300,000 individuals).

The study, based on cases diagnosed during the period 1997-2006, included 292 patients with ovarian cancer and 41 with ovarian cancer and endometriosis. After adjusting for age, number of pregnancies, family history of ovarian cancer, race, oral contraceptive (OC) use, tubal ligation, hysterectomy, and breastfeeding, women with endometriosis were at

increased risk for developing ovarian cancer (Rate Ratio 1.6; 95% CI, 1.12 to 2.09). During the study period, a constant increase in the incidence of ovarian cancers was observed, but not in endometriosis-associated cases, which were detected at an earlier age with respect to the former group (48.3 versus 53.8 years).

Patient education and counselling

Based on this data, in the worst scenario, the lifetime probability of developing ovarian cancer increases from 1/100 to 2/100. In other words, a woman with untreated endometriosis has a 98% probability, instead of 99%, of not developing an ovarian malignancy.

One of the practical problems is translating these epidemiologic figures in simple terms in order to inform our patients correctly and to undertake sound clinical decisions. In fact, the same information might be delivered in terms of 30% to 100% increase in risk, thus appearing much more frightening to the patient.

Risk-benefit analysis

Radical excision of endometriotic lesions constitutes good surgical practice whenever operating women for pain, infertility or adnexal masses.

However, whether this might result in a reduction of the risk of ovarian cancer after conservative interventions is not demonstrated and, given the recurring nature of the disease, it does not seem to be supported by a robust rationale.

When ovarian cancer is the issue, bilateral salpingo-oophorectomy is the only "radical" procedure that would undoubtedly reduce the risk of malignant transformation in our endometriosis patients. However, whether the risks/costs/benefits balance of such an approach is favourable has yet to be determined.

Infertile subjects have a standardised incidence ratio for ovarian cancer of 2. In case of primary infertility the risk increases to 2.7 (Brinton et al, 2004). Women with a first-degree relative affected by ovarian cancer (except BRCA 1 and 2 subgroups) are at doubled risk of ovarian malignancy. Nevertheless, oncologic guidelines do not include preventive oophorectomy in any of the above subjects.

Kurman and Shih (2010), when addressing the issues of prevention of ovarian cancer, suggest long-term prescription of oral contraceptives (OCs) that is associated, in the general female population, with a 50% reduction in risk after five or more years of use.

Importantly, the use of OCs reduces the risk of ovarian cancer even more dramatically in patients with endometriosis. Modugno et al (2004) pooled data from four population-based, case-control studies that recruited women from four regions of the United States from 1993 to 2001, and found that women with endometriosis were slightly more likely to have ovarian cancer than controls.

Endometriosis-associated ovarian cancer: A ten-year cohort study of women living in the Estrie Region of Quebec, Canada

J Ovarian Res 2010 Jan 19;3:2.

Aris A

OBJECTIVES: Endometriosis has been believed to increase the risk of developing ovarian cancer, but recent data supporting this hypothesis are lacking. The aim of this study was to verify whether the incidence of endometriosis, ovarian cancer and the both increased during the last 10 years among women living in the Estrie region of Quebec.
Methods: We collected data of women diagnosed with endometriosis, ovarian cancer or both, between 1997 and 2006, from a population living in the Estrie region of Quebec. We performed this retrospective cross-sectional study from the CIRESSS (Centre Informatisé de Recherche Évaluative en Services et Soins de Santé) system, the database of the CHUS (Centre Hospitalier Universitaire of Sherbrooke), Sherbrooke, Canada.
RESULTS: Among the 2854 identified patients, 2521 had endometriosis, 292 patients had ovarian cancer and 41 patients had both ovarian cancer and endometriosis. We showed a constant increase in the number of ovarian cancer (OC) between 1997 and 2006 (r2 = 0.557, P = 0.013), which is not the case for endometriosis (ENDO) or endometriosis-associated ovarian cancer (EAOC). The mean age ± SD was 40.0 ± 9.9 and 53.9 ± 11.4 for patients having ENDO and OC, respectively. Mean age of women with EAOC was 48.3 ± 10.8, suggesting an early onset of ovarian cancer in women having endometriosis of about 5.5 years average, P = 0.003. Women with ENDO were at increased risk for developing OC (Rate Ratio [RR] = 1.6; 95% Confidence Interval [CI] = 1.12-2.09). Pathological analyses showed the predominance of endometrioid type (24.4%) and clear-cell type (21.9%) types in EAOC compared to OC, P = 0.0070 and 0.0029, respectively. However, the serous type is the most widespread in OC (44.5%) in comparison to EAOC (19.51%), P = 0.0023.
CONCLUSION: Our findings highlight that the number of cases of ovarian cancer is constantly increasing in the last ten years and that endometriosis represents a serious risk factor which accelerates its apparition by about 5.5 years

Oral contraceptive use decreased the risk of gonadal malignancy in the entire study population, but the effect was particularly evident in women with a history of endometriosis. Moreover, a dose-response relation was observed with lifetime duration of pill use.

In conclusion

A substantial modification in the standard management of patients with known endometriosis with the aim of preventing ovarian cancer does not appear justified based on the available, insufficient evidence.

In particular, lowering the level of surgical indications should be considered with caution, due to a probably unfavourable cost/benefit/harm ratio. Instead, patients must be informed fully and correctly, explaining the risk in terms of absolute and not relative increase, and including data on long-term oral contraceptive use.

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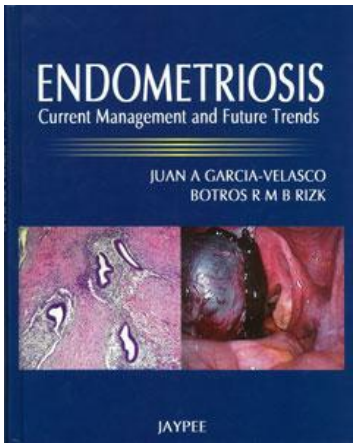

Endometriosis: current management and future trends

Editors: Juan A Garcia-Velasco and Botros RMB Rizk
Publisher: Jaypee
Target Audience: Healthcare professionals
Recommended retail price: USD 70.00 (www.jaypeebrothers.com); also available at Amazon and the RCOG Bookshop

Rating

Content:
Readability:
Interest:
Overall:

Reviewed by: Anusch Yazdani
Associate Professor, University of Queensland and Director of Research and Development, QFG Research Foundation, Australia

It's a familiar scenario: the book looked engaging in the store, but twenty pages in to the tome and you're falling asleep.

Undoubtedly, no genre is more guilty of this than scientific monographs on a single topic. In contrast, however, *Endometriosis: Current Management and Future Trends* manages to retain interest, though this is solely attributable to the strength of the individual contributing authors.

The editors, Juan Garcia-Velasco and Botros Rizk, have successfully assembled an exceptional collection of papers from eminent sources. The list of contributors is veritable a *who's who* of endometriosis publications.

Endometriosis: Current Management and Future Trends is divided into eight sections that cover over 37 papers in epidemiology, pathogenesis, diagnosis, medical and surgical therapies and future trends in the management of endometriosis. Particularly fascinating are the chapters on new and novel medical therapies and future trends. These chapters alone make this an essential purchase for any contemporary academic collection.

The detail is exquisite, though at times indigestible without significant background knowledge. The more mundane aspects of endometriosis are managed exceedingly well by each of the contributing authors: even the chapter on danazol was enjoyable.

Authoritative as such a collection may be, it lacks the integration of a single author and the overview of strong editors. The changes in writing style are distracting and the repetition is mildly annoying.

Almost without fail, each chapter in this book furnishes its own definition of endometriosis and quotes a new prevalence. Admittedly, this is only annoying if the reader attempts to engage this monograph as a cohesive entity, rather than a collection of individual papers.

More importantly, however, *Endometriosis: Current Management and Future Trends* lacks an integrated view. For example, the excellent chapters on novel treatment modalities lack any overview of where such treatment may fit in to the management of endometriosis.

While almost 150 pages have been dedicated to medical therapies, it is disappointing that little space (15 pages) is devoted to surgical management. However, this may reflect the paucity of evidence in the surgical domain, rather than an oversight of the authors.

Where *Endometriosis: Current Management and Future Trends* fails as a monograph, it is a resounding success as a collection of review papers, that accurately reflect our level of knowledge of this enigmatic disease in 2009.

DEADLINE for contributions to the May/June e-Journal is 25 May 2010