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The role of laparoscopy in managing infertility

The author details the use of laparoscopy as both a diagnostic and a treatment tool when managing patients who present with infertility due to such conditions as endometriosis, ovarian disorders, tubal damage, and uterine myomas.

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Laparoscopy is an indispensable tool for determining the cause of infertility, as it can provide a good indication of the overall condition of the patient's abdominopelvic cavity. Among the pathologies Ob/Gyns frequently identify during diagnostic laparoscopy are pelvic endometriosis, which may have invaded extrapelvic organs (*Figure 1*), leiomyoma, adhesions, polycystic ovaries, and neoplasms (*Figure 2*). Laparoscopy also makes it possible to evaluate the fimbriae for occlusion or other pathology and to carry out chromopertubation when indicated.

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In addition to its role as a diagnostic tool, the modality can increase a woman's chances of becoming pregnant, as well as improve her overall well-being—as long as appropriate treatment is performed at the time of laparoscopy. For example, the simultaneous evaluation and ablation or resection of endometriosis creates a more favorable environment for tubal and ovarian function, fertilization, and implantation, and minimizes the risk that the disease will spread to other structures such as the bladder, bowel, or ureter (*Figure 3*). Thus, the number of surgeries is reduced and, with it, the development of related adhesions. Nonetheless, laparoscopy should be reserved for the later stages of the diagnostic process—after ovulatory status has been assessed and a semen analysis and hysterosalpingography have been performed.

The causes of infertility

Infertility may be caused by conditions involving the uterus, fallopian tubes, or ovaries, or in 2 or more of these structures at the same time. It also may be linked to infection. The main etiologies, as well as the role of laparoscopy in evaluating and treating each particular condition, are discussed below.

By utilizing the multipuncture operative laparoscopy technique, the majority of female reproductive diseases can be diagnosed and treated successfully. After administering general endotracheal anesthesia, the physician places the patient in Allen stir-

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rups, which enables the assistant to perform a vaginal examination. The bladder is then catheterized. After introducing a 10-mm operative videolaparoscope infraumbilically, the surgeon inserts one to three 5-mm accessory cannulas suprapubically, depending on the severity of the case. The physician then inspects the intraperitoneal cavity to detect any pelvic abnormalities and to assess whether treatment is required, which, depending on the experience of the surgeon and the patient's condition, could be performed at the same time.

Endometriosis. One of the most common causes of female infertility is endometriosis; 30% to 60% of patients with infertility are diagnosed with the disease.[1] It has been suggested that endometriosis can hinder tubal and ovarian function, fertilization, and implantation due to abnormal peritoneal fluid, the adverse effects of prostaglandins and other toxic substances from endometriotic lesions, or immune system disturbances.[2]

Some researchers believe that women with mild endometriosis are no less likely to become pregnant than those without the disease, and that only patients with moderate to severe endometriosis have lower fertility rates than healthy women. However, recent studies have found that even treatment of minimal to mild endometriosis can increase fertility rates.[3,4] For example, in a randomized study of 341 women with minimal to mild endometriosis, the subjects underwent either diagnostic laparoscopy or operative laparoscopy with resection or ablation of visible endometriosis. All patients were followed for 36 weeks after the laparoscopy. Those who became pregnant were followed through the first 20 weeks of gestation. Of the 172 patients who underwent resection or ablation of endometriosis, 50 conceived and remained pregnant for more than 20 weeks compared to 29 women in the diagnostic laparoscopy group.[4]

This finding is comparable to that of a previous study in which researchers reported improved fertility in women with endometriosis who were treated by operative laparoscopy (*Table 1*). They determined that the overall success rate, i.e., the number of term pregnancies, in patients who underwent excision of their endometriosis at the time of laparoscopy was more than 60% for all stages of the disease.[5] This is significantly higher than the IVF success rate (27.2% per cycle, 31.3% per transfer).[6]

Key points

- Ablation or resection of endometriosis at laparoscopy, for all stages of the disease, yields an overall success rate (number of term pregnancies) of 60% compared to that of 27% for IVF.
- Laparoscopic excision of endometriomas is associated with a lower recurrence rate, a higher level of pain relief, and a higher pregnancy rate than both drainage-coagulation and fenestration.
- If the myomas are numerous, large, and deeply intramural, necessitating uterine reconstruction, laparoscopically assisted myomectomy is indicated.

Ovarian disorders. Ovarian cysts, polycystic ovaries, and endometriosis involving the adnexa can interfere with ovulation. Once cysts are identified during laparoscopy, they should be treated according to their type and size. When fertility is at issue, dermoid cysts are of particular concern. If their contents spill into the pelvic cavity, the result can be granulomatous peritonitis, which can cause adhesions and infertility. The use of retrieval bags to enclose such cysts prior to removal decreases the risk of spillage and reduces operative time.[7]

The surgical induction of ovulation through laparoscopic puncture is an option for women with polycystic ovary syndrome (PCOS). When compared with bilateral ovarian wedge resection at laparotomy, laparoscopic puncture requires less time, is more cost effective, and is associated with fewer adhesions. Gjonnaess concluded that ovulation occurred more frequently if 10 or more punctures, rather than only 6, were made in the ovary.[8-10] In 1998, Gjonnaess also reported that of those patients who had undergone laparoscopic puncture, two-thirds continued to ovulate 18 to 20 years after the procedure. The initial and long-term ovulation rates were higher in women

Figure 1: *Endometriosis can invade extrapelvic organs such as the diaphragm.*

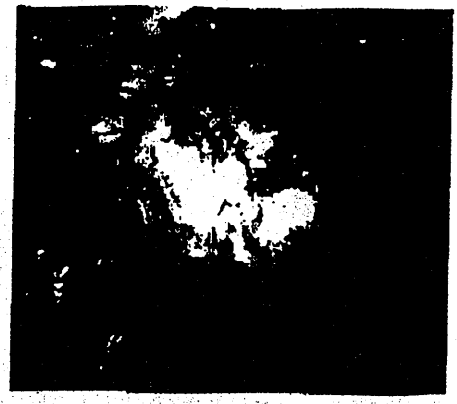

Figure 2: *Lesions, e.g., low-malignant-potential tumors, also can cause infertility.*

Figure 3: *Treatment of endometriosis at laparoscopy may prevent the spread of the disease to other structures such as the ureter.*

Figure 4: *Ovarian endometriomas, which occur secondary to ovarian endometriosis, affect 50% to 70% of women with the disease.*

of normal weight than in overweight women (those with a body mass index (BMI) greater than 25 kg/m^2).[8] Despite its effectiveness, laparoscopic puncture should be used with caution, as aggressive surgical treatment can destroy the ovary.[8,11]

Another condition affecting ovarian function is the formation of endometriomas (*Figure 4*). This pathology is secondary to ovarian endometriosis and occurs in 50% to 70% of women with the disease.[12] These masses often are bilateral and may adhere to the pelvic sidewalls, the posterior uterus, or the bowel.[13] In a randomized trial, cystectomy was found to be superior to drainage-coagulation in terms of pain relief, pregnancy rate, and recurrence.[14] Similarly, Saleh et al reported that laparoscopic excision of endometriomas is associated with a lower reoperation rate than is fenestration; while the recurrence rate after fenestration is independent of the age of the patient, it is higher in those with larger cysts.[15]

In cystectomy, the risk of endometrioma recurrence is significantly decreased compared to nonexcisional techniques. However, the method carries a risk of reduced follicular response in natural or clomiphene citrate-stimulated cycles. In a retrospective, controlled study, the mean follicular response was significantly lower in subjects younger than 35 years of age who had undergone cystectomy than it was in women with healthy ovaries and in those who did not undergo cystectomy but who had ovarian stimulation with clomiphene citrate. However, a patient's reduced follicular response after cystectomy can be offset if the physician employs aggressive gonadotropin stimulation.[16]

Tubal function. Another frequent cause of infertility is primary tubal disease, which affects up to 20% of infertile women. The most common etiologies are pelvic inflammatory disease (PID), adhesions from previous pelvic surgery, a ruptured appendix, and endometriosis.[17]

Salpingostomy can be performed easily at the time of laparoscopy, with restoration of patent fallopian tubes in the majority of cases. Success in opening blocked tubes depends on the degree of tubal damage, which, obviously, affects pregnancy rate and other fertility factors.

Research also supports the use of laparoscopic salpingectomy for hydrosalpinges in properly selected patients.[18-20] This condition adversely affects implantation, even in donor oocyte cycles. Cohen and colleagues found that patients with a hydrosalpinx had

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significantly lower implantation rates and higher miscarriage and ectopic pregnancy rates than did women with healthy fallopian tubes.[21] These rates also may reflect the toxicity of the hydrosalpinx fluid. Therefore, laparoscopic proximal tubal occlusion, performed to prevent the passage of hydrosalpinx fluid into the uterine cavity, may improve the developmental environment for embryos without requiring the removal of one or both tubes. However, when salpingectomy is indicated, removing only the severely damaged tube and leaving the other, relatively healthier tube intact may be sufficient to increase pregnancy rates and decrease miscarriage rates. Removing both fallopian tubes and proceeding to in-vitro fertilization (IVF) usually is advisable when both tubes are severely damaged.

Uterine myomas. Fibroids affect 20% to 25% of reproductive-age women.[22,23] Although they are seldom the sole cause of infertility, several studies show a link between fibroids, fetal wastage, and preterm birth.[22] Laparoscopic treatment of fibroids includes laparoscopic myomectomy (LM) and laparoscopically assisted myomectomy (LAM). LM is one of the most difficult laparoscopic procedures to perform, as it requires myoma morcellation and uterine reconstruction with laparoscopic suturing techniques to decrease the risk of uterine rupture during subsequent pregnancies. Therefore, LAM is a safe alternative to LM.

The decision to perform LAM usually is made in the operating room after the diagnostic laparoscopy and treatment of other pelvic abnormalities have been completed. The criteria for LAM are myomas larger than 8 cm; numerous myomas requiring extensive morcellation; and/or deep, large, intramural myomas that necessitate uterine repair in multiple layers and restoration of the myometrial integrity.

To perform LAM, the surgeon injects the myoma at its base with diluted vasopressin to reduce blood loss. He or she then makes a vertical incision in the uterine serosa and extends it to the surface of the tumor until the incision reaches the capsule of the leiomyoma. A corkscrew manipulator is then inserted into the myoma (*Figure 5*). With the trocar and manipulator attached to the myoma, the physician enlarges the midline 5-mm suprapubic puncture to a 4-cm transverse incision and raises the leiomyoma to the incision by using the corkscrew manipulator to elevate the uterus. The fibroid is then grasped, shelled, and morcellated sequentially (*Figure 6*). (When multiple leiomyomas are present, the surgeon should remove as many tumors as possible through 1 uterine incision.) After the myoma has been removed completely, the surgeon repairs the uterine wall defect; the conventional suturing technique, wherein 2 or 3 layers are closed, reduces the potential for uterine

TABLE 1 Pregnancy outcome in endometriosis patients treated laparoscopically

Stage of disease	No. of patients	No. of pregnancies	Success rate (%)	Spontaneous abortions	Abortion rate (%)
Mild	24	18	75.1	4	22.0
Moderate	51	32	62.7	4	12.5
Severe	19	8	42.1	2	25.0
Extensive	8	4	50.0	0	0.0
Total	102	62	60.8	10	9.8

Source: Nezhat C, Crowgey S, Garrison C. Surgical treatment of endometriosis via laparoscopy. Fertil Steril. 1986;45:778-

Figure 5: *In LAM, the corkscrew manipulator elevates the uterus to the abdominal incision to facilitate removal of the myoma.*

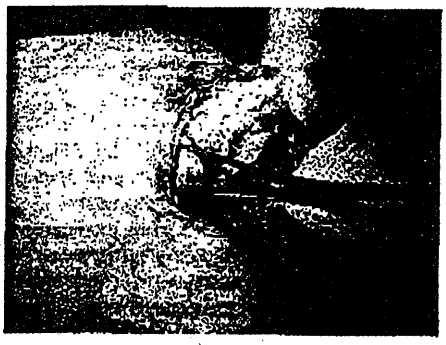

Figure 6: *After the uterus and myoma are elevated, the surgeon then grasps, shells, and morcellates the fibroid sequentially.*

dehiscence, fistulas, and adhesions. After closing the incision, the laparoscope is used to confirm hemostasis. Then, the physician assesses the pelvis again to identify and treat any endometriosis or adhesions that may have been previously obscured by myomas.

Patients should be counseled extensively about the risks associated with both LM and LAM and should be advised that, depending on the size and location of the myomas, future fertility may be compromised.

In summary

While laparoscopy should be included in an Ob/Gyn's armamentarium for assessing and managing infertility, it should be relegated to the later stages of the process. Ovulation assessment, a semen analysis, and hysterosalpingography should be performed prior to laparoscopy when an infertile woman presents for evaluation. However, the failure to perform diagnostic laparoscopy at some point could seriously jeopardize fertility—and the woman's overall health and well-being—when endometriosis, adhesions, or anatomic abnormalities are present.

Additionally, operative laparoscopy can increase a patient's chances of spontaneous conception and improve her response to ovarian stimulation, thus decreasing the need for IVF, risk of multiple gestation, associated maternal and fetal morbidity, and costs. It also can improve IVF outcome.

Laparoscopy, both diagnostic and operative, has its time and place in the management of infertile patients, and its effective use depends on an individual Ob/Gyn's level of comfort and experience. ■

REFERENCES

1. Pauerstein CJ. Clinical presentation and diagnosis in endometriosis. In: Schenken RS, ed. *Contemporary Concepts in Clinical Management*. Philadelphia, Pa: JB Lippincott; 1989:127-144.

2. Nezhat CR, Berger GS, Nezhat FR, et al. *Endometriosis: Advanced Management and Surgical Techniques*. New York: Springer-Verlag; 1995:62.

3. Panidis DK, Matalliotakis IM. Subfertility associated with minimal to mild endometriosis. *J Reprod Med*. 1998;43:1034-1042.

4. Marcoux S, Maheux R, Berube S. Laparoscopic surgery in infertile women with minimal or mild endometriosis. *N Engl J Med*. 1997;337(4):217-222.

5. Nezhat C, Crowgey S, Garrison C. Surgical treatment of endometriosis via laparoscopy. *Fertil Steril*. 1986;45:778-782.

6. Assisted reproductive technology in the United States: 1996 results generated from the American Society for Reproductive Medicine/Society for Assisted Reproductive Technology Registry. *Fertil Steril*. 1999; 71:798-807.

7. Campo S. Laparoscopic conservative excision of ovarian dermoid cysts with and without an endobag. *J Am Assoc Gynecol Laparosc*. 1998;5(2):165-170.

8. Gjonnaess H. Late endocrine effects of ovarian electrocautery in women with polycystic ovary syndrome. *Fertil Steril*. 1998;69(4):697-701.

9. Gjonnaess H, Norman N. Endocrine effects of ovarian electrocautery in patients with polycystic ovarian disease. *Br J Obstet Gynaecol*. 1987;94(8):779-783.

10. Gjonnaess H. Polycystic ovarian syndrome treated by ovarian electrocautery through the laparoscope. *Fertil Steril*. 1984;41(1):20-25.

11. Felemban A, Tan SL, Tulandi T. Laparoscopic treatment of polycystic ovaries with insulated needle cautery: a reappraisal. *Fertil Steril*. 2000;73(2):266-269.

12. Jenkins S, Olive DL, Haney AF. Endometriosis: pathogenic implications of the anatomic distribution. *Obstet Gynecol*. 1986;67:335.

13. Gomel V, Taylor P. *Diagnostic and Operative Gynecologic Laparoscopy*. St. Louis, Mo: Mosby; 1995:136.

14. Berera P, Franchi M, Ghezzi F, et al. Randomized clinical trial of two laparoscopic treatments of endometriomas: cystectomy versus drainage and coagulation. *Fertil Steril*. 1998;70:1176-1180.

15. Saleh A, Tulandi T. Reoperation after laparoscopic treatment of ovarian endometriomas by excision and fenestration. *Fertil Steril*. 1999;72:322-324.

16. Loh FH, Tan AT, Kumar J, Ng SC. Ovarian response after laparoscopic ovarian cystectomy for endometriotic cysts in 132 monitored cycles. *Fertil Steril*. 1999;72:316-321.

17. Nezhat CR, Nezhat FR, Luciano AA, et al. *Operative Gynecologic Laparoscopy: Principles and Techniques*. New York, NY: McGraw-Hill; 1992:205-213.

18. Mukherjee T, Copperman AB, McCaffrey C, et al. Hydrosalpinx fluid has embryotoxic effects on murine embryogenesis: a case for prophylactic salpingectomy. *Fertil Steril*. 1996;66(5):851-853.

19. Blazar AS, Hogan JW, Seifer DB, et al. The impact of hydrosalpinx on successful pregnancy in tubal factor infertility treated by in vitro fertilization. *Fertil Steril*. 1997;67(3):517-520.

20. Wainer R, Camus E, Camier B, et al. Does hydrosalpinx reduce the pregnancy rate after in vitro fertilization? *Fertil Steril*. 1997;68(6):1022-1026.

21. Cohen M, Lindheim S, Sauer M. Hydrosalpinges adversely affect implantation in donor oocyte cycles. *Human Reprod*. 1999;14(4):1087-1089.

22. Buttram VC Jr, Reiter RC. Uterine leiomyomata: etiology, symptomatology, and management. *Fertil Steril*. 1981;36(4):433-445.

23. Vollenhoven BJ, Lawrence AS, Healy DL. Uterine fibroids: a clinical review. *Br J Obstet Gynaecol*. 1990;97:285.

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DEBATE

Classification of endometriosis

Improving the classification of endometriotic ovarian cysts

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The need to modify the classification of endometriosis is both real and urgent, as has been clearly expressed in a recent expert review (Brosens *et al.*, 1993). We agree with the theoretical criteria suggested by these authors for an ideal classification. Such a system should be based on laparoscopic findings, have high descriptive value, though it should remain simple and practical to implement. Most importantly, the classification must be useful in selecting the proper treatment, while providing prognostic implications and allowing comparison of results. However, we find the proposed classification of endometriotic cysts to be an oversimplification. Scoring according to the ovarian cysts' colour and size (i.e. small <1 cm, medium $1-5$ cm and large >5 cm) seems completely arbitrary and of very limited practical value.

The ovary is involved in approximately half of all women with endometriosis (Jenkins *et al.*, 1986). The unique character of ovarian endometriosis is the development of large endometriomas. It is well recognized that endometriosis may also appear in the ovaries as superficial implants. Moreover, not all ovarian 'chocolate' cysts demonstrate histological evidence of endometriosis (Martin and Berry, 1990). We have, therefore, devised a new classification that takes into account these unique features of ovarian endometriosis, since they may have an important pathophysiological basis. In a series of 187 women with endometriosis, we performed a clinical and histological evaluation of haemorrhagic ovarian cysts (Nezhat *et al.*, 1992). It was observed that superficial ovarian endometriosis is similar to endometriosis in extra-ovarian sites, in that the formation of superficial cysts is limited in size by fibrosis and scarring. In contrast, large endometriomas may develop as a result of secondary involvement of functional (follicular or luteal) ovarian cysts by the endometriotic process.

Based on the clinical appearance and histological examination, we conceived the following classification.

Type I: primary endometrioma—true endometrioma origin, similar to that found on peritoneal surfaces. These 'pure' endometriomas are characterized as small superficial cysts containing dark 'chocolate' fluid. As these endometriomas develop on the surface of the ovary, they are typically found to be firmly adherent to the tissue and difficult to remove surgically. Histological analysis always reveals only endometrial glands and stroma.

Type II: secondary endometrioma—follicular or luteal ovarian cysts have been involved or invaded by cortical endometriotic implants or by primary endometrioma.

We further distinguish between three types of secondary endometriomas based on the relationship of cortical endometriosis with the cyst wall.

Type IIa endometriomas are usually large and with a capsule that is easily separated from the ovarian tissue. If endometrial implants are seen, they do not penetrate the cyst wall. Upon histological examination, the walls are found to be clear of any endometrial tissue. These haemorrhagic cysts are either follicular or luteal in origin.

Type IIb endometriomas have features of functional cysts but show deep involvement with surface endometriosis. The lining of these cysts is easily separated from the ovarian capsule and stroma, except adjacent to the area of endometriosis, where the ovarian capsule has adhered to the cyst wall. Histological findings reveal endometriosis implants in the cyst wall.

Type IIc endometriomas are similar to the former type, as these functional cysts show extensive surface endometrial implants. However, these ovarian cysts show deep penetration of the endometriosis into the cyst wall, spreading to at least one area of the ovarian capsule. The basis for differentiating between the Type IIb and Type IIc cysts is thus the degree of invasion of the endometriosis into the cyst wall. This characteristic can be distinguished clinically by the progressive difficulty in removing the cyst's capsule. A high degree of correlation was observed between the clinical findings regarding the presence of dense adhesions and the histological evidence of endometriotic extension into the wall of the ovarian cyst.

The challenge in distinguishing between endometriomas and functional cysts has long been recognized, and subsequently, the need for biopsy and histological confirmation has been well established. However, as Sampson (1921) has described over seven decades ago 'the histologic findings in these cysts vary in different portions of the same cyst'. Accordingly, we believe that our proposed classification has more value for laparoscopic or surgical diagnosis than the suggestion by Brosens *et al.* (1993). Furthermore, it facilitates in distinguishing between 'red' cysts with vascularized areas on a white surface and 'black' cysts with a dark, pigmented and fibrotic wall. Our classification has not only been validated histologically (Nezhat *et al.*, 1992) but may

also be of distinct clinical value in the management of endometriomas.

Medical therapy for ovarian suppression using either danazol or gonadotrophin-releasing hormone antagonists may be of greatest benefit in patients with Type II cysts. The latter are comprised of follicular and corpus luteum cysts which are likely to reduce in size or resolve. Pre-operative ovarian suppression may also reduce vascularity, resulting in decreased intra-operative haemorrhage, as well as minimizing manipulation and damage to normal ovarian tissue.

Surgical management may gain even more from the use of the proposed classification. In our experience, surgeons can successfully distinguish the type of the cyst prior to histological results, according to the following criteria: (i) size and content of cysts; (ii) ease of capsule removal; (iii) cyst adhesions to other structures; (iv) location of superficial endometriosis relative to cyst wall. We generally recommend definite treatment for all endometriomas, as aspiration alone has been shown to be associated with an unacceptably high recurrence rate (Hasson, 1990; Vercellini *et al.*, 1991). Surgical treatment should, however, be made on an individual basis according to cyst type. Although Type I endometriomas are difficult to remove intact because of associated fibrosis and adhesions, they can be easily biopsied and vaporized using laser or electrosurgery, or removed in pieces. The larger Type I cysts (2–3 cm) must be completely resected.

Type IIa cysts, which are essentially follicular or luteal cysts, are expected to resolve with pre-operative ovarian suppression. If the cysts persist, they should be evaluated laparoscopically, aspirated and deflated. Suspicious Type IIa lesions should undergo frozen section biopsy, and as they are characteristically easy to remove, complete excision with minimal trauma to the ovary can frequently be achieved.

Type IIb cysts are usually severely attached to the pelvic side wall and the back of the uterus, and tend to rupture during separation. For this type of endometrioma, the portion of the ovarian cortex which is involved with endometriosis is removed. Any spillage, which is common, is immediately suctioned and irrigated.

In Type IIc endometrioma, it may be difficult to resect the cyst due to lack of a plane between the cyst wall and the ovarian capsule, as they are severely attached. The portion of the ovary which is adherent to the cyst wall is removed until the ovarian plane is identified. The endometrioma is then completely resected, with approximation of the redundant ovarian capsule.

The aetiology and pathophysiology of endometriosis remain to be completely elucidated. Any attempt to classify endometriomas should, therefore, contribute to our understanding of these mechanisms. We believe that the proposed classification is logical and of clinical value as it is consistent with our current knowledge of endometriosis. Type I endometriomas represent superficial ovarian endometriosis, which is similar to endometriosis in extra-ovarian sites in that the formation of superficial cysts is limited in size by fibrosis and scarring. In contrast, large Type II endometriomas may develop as a result of secondary involvement of functional ovarian cysts by the endometriotic process.

References

Brosens,I., Donnez,J. and Benagiano,G. (1993) Improving the classification of endometriosis. *Hum. Reprod.*, **8**, 1792–1795.

Jenkins,S., Olive,D.L. and Haney,A.F. (1986) Endometriosis: pathogenic implications of the anatomic distribution. *Obstet. Gynecol.*, **67**, 335–338.

Hasson,H. (1990) Laparoscopic management of ovarian cysts. *J. Reprod. Med.*, **25**, 863–867.

Martin,D.C. and Berry,J.D. (1990) Histology of chocolate cysts. *J. Gynecol. Surg.*, **6**, 43–46.

Nezhat,F., Nezhat,C., Allan,C.J., Metzger,D.A. and Sears,D.L. (1992) Clinical and histologic classification of endometriosis: implications for a mechanism of pathogenesis. *J. Reprod. Med.*, **37**, 771–776.

Sampson,J.A. (1921) Perforating hemorrhagic (chocolate) cysts of the ovary. *Arch. Surg.*, **3**, 245–323.

Vercellini,P., Vendola,N., Bocciolone,L., Rognoni,M.T., Carinelli,S.G. and Candiani,G.B. (1991) Reliability of the visual diagnosis of ovarian endometriosis. *Fertil. Steril.*, **56**, 1198–1200.

Endoscopic exploration and classification of the chocolate cysts

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Endoscopy is the most powerful diagnostic tool in gynaecology. The visibility, magnification and minimal access technique all add to the detection and characterization of the pathology and the performance of atraumatic and accurate surgery. The ovary is an organ where the principles of atraumatic surgery are as important as in eye surgery because an intact cortex is as essential for the function of the ovary as the cornea for the eye. Unnecessary surgery inducing the risk of adhesion formation should be avoided. The discussion on the differentiation between an endometrial and other haemorrhagic cysts such as a functional and a neoplastic cyst is therefore an important issue in reproductive medicine.

Today the technique of minimal access in endoscopic surgery has made many pathologists dissatisfied because frequently they receive a morcellated specimen on which they are unable to perform properly the macroscopic examination of the specimen. It is therefore important that the endoscopist should perform the inspection carefully and identify the lesions for biopsy.

Hughesdon (1957) examined a series of 29 ovaries with in-situ endometriomas by serial sectioning and demonstrated that in 26 of them the inside wall was ovarian cortex. In no case was colonization of a functional cyst by endometrial tissue found. The theory of invagination and colonization of the invaginated ovarian cortex by endometrial tissue has been confirmed by our cystoscopic observations (Brosens et al., 1994). At the site of invagination which originally was described by Sampson (1921) as the site of perforation, free, active implants were found at biopsy with the characteristics of superficial endometrium.

Cytoscopy of an ovarian chocolate cyst is performed to examine the macroscopic aspects of the cysts and to differentiate between

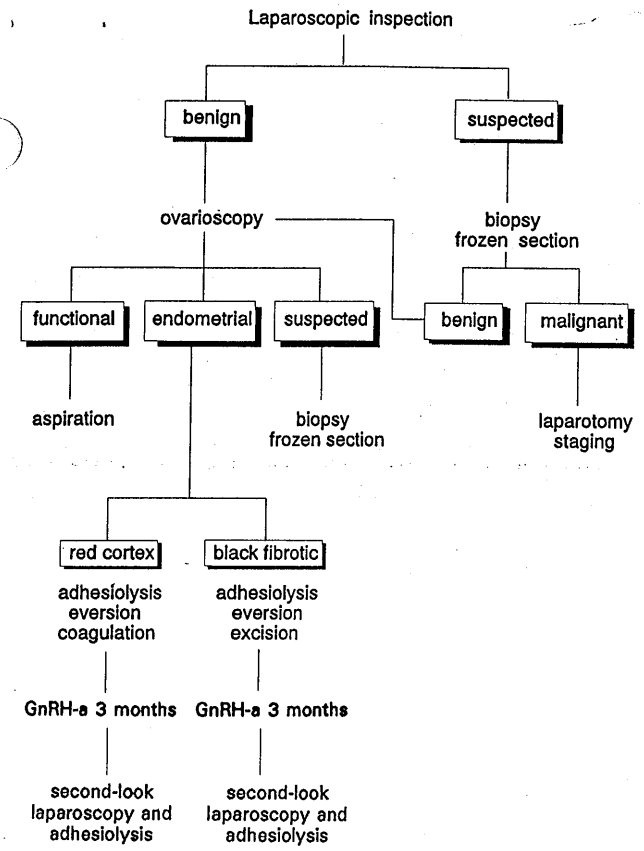

Fig. 1. Scheme of cystoscopic examination, identification and treatment of ovarian cysts.

an endometrioma and other cysts (Figure 1). The technique allows localization of the endometrial implants for biopsy. They can be identified as red areas supplied by an irregular network of vascularization overlying the wall, frequently loosely. They can be easily removed from the wall. Follicular and serous cysts have an undulating surface overlying a retinal network of vascularization and lutein cysts have a uniform but yellow, granular surface with a fine vascularization. These cysts are aspirated and biopsied but not resected.

The surgical treatment of the endometrioma is basically an adhesiolysis procedure with destruction of active implants. In endometriomas where the ovarian cortex can be identified by its white or yellowish appearance and red implants, the surgery is performed by adhesiolysis and opening the cyst at the point of inversion (similar to salpingostomy) and coagulation or vaporization of the red implants and their vascularization. In endometriomas with extensive fibrosis of the wall and a dark pigmented appearance at cystoscopy, the wall is excised. Large, multilocular or septate endometriomas can show a combination with one or more functional cysts such as a haemorrhagic corpus luteum or lutein cyst. It is no surprise to find this combination as the wall of the endometrioma is formed by ovarian cortex. After a three months gonadotrophin-releasing hormone agonist (GnRH-a) treatment large endometriotic cysts and associated functional cysts have regressed to normal sized ovaries and at this stage the second-look laparoscopy with adhesiolysis is easy to perform. The adhesions are more important than the size in determining the operability and the outcome (Canis, 1992). The classification of endometriomas should be based on the extent and type of adhesions and fibrosis because they are apparently the factors determining the operability and clinical outcome.

References

Brosens,I.A., Puttemans,P.J. and Deprest,J. (1994) The endoscopic localization of endometrial implants in the ovarian chocolate cyst. *Fertil. Steril.*, **61**, 1034–1038.

Canis,M., Pouly,J.L., Wattiez,A., Manhes,H., Mage,G. and Bruhat, M.A. (1992) Incidence of bilateral adnexal disease in severe endometriosis (revised American Fertility Society (AFS), stage IV): should a stage V be included in the AFS classification? *Fertil. Steril.*, **57**, 691–692.

Hughesdon,P.E. (1957) The structure of endometrial cysts of the ovary. *J. Obstet. Gynaecol. Br. Emp.*, **44**, 481–487.

Sampson,J.A. (1921) Perforating hemorrhagic (chocolate) cysts of the ovary. *Arch. Surg.*, **3**, 245–323.

The need for modification

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It was with great interest that we read the article by Nezhat *et al.* (1994) in which the authors propose a modification to the endometriosis classification for ovarian endometriomas. It is an extremely interesting piece of work but prompts us to make the following comments.

When dealing with endometriosis, it is indeed essential that there should be a classification that everyone is happy to use, so that everyone speaks the same language and it is possible to make comparisons between series and multi-centre studies. Since the first classification proposed in 1927 by Sampson, a great many others have been suggested [Wick and Larsen, 1949; Huffman, 1951; Acosta *et al.*, 1973; Kistner *et al.*, 1977; Buttram, 1978; American Fertility Society (AFS), 1979], with the 1985 revised version of the AFS classification being the reference classification today.

Like Nezhat *et al.* (1994) and Brosens *et al.* (1993), we feel that this classification is no longer satisfactory and that it should be modified. Among the shortcomings of this classification, the most important in our opinion are that the stages are defined solely on the basis of visual examination and that no histological elements are taken into account. The consequence is that the pathology is poorly assessed, and this can be due to the following: (i) There is underestimation of the severity and spread of the pathology because certain lesions can be misread. This can happen for two reasons. Firstly, the peritoneum which appears to be healthy upon visual examination may be the site of microscopic forms of endometriosis that can only be diagnosed

by making multiple, systematic biopsies (Vasquez *et al.*, 1984; Murphy *et al.*, 1987). Secondly, endometriosis can take on several aspects (Jansen and Russel, 1986; Stripling *et al.*, 1988; Martin *et al.*, 1989) and only histological examination of biopsies can determine whether endometriosis is present or not. (ii) The development of the lesions is not well understood. For example, Vernon *et al.* (1986) showed that red-coloured peritoneal lesions were more active than brownish ones, which in turn were more active than black ones. (iii) The depth of the lesions is not assessed, or lesions which penetrate further than 5 mm are more active (Cornillie *et al.*, 1990) and pain is associated with the degree of penetration (Koninckx *et al.*, 1991).

The AFS classification (1985) also has three other important defects. (i) It is very subjective because it depends to a large extent on visual laparoscopic exploration. (ii) It does not take certain lesions into account, such as lesions on the tubes or ureters, and does not include any extra-pelvic lesions on, for example, the bowel. (iii) It uses the same scoring for the prognosis for fertility as for pain. However, painful cases are very often associated with deep lesions but patients suffering from infertility are more likely to present with superficial implants (Cornillie *et al.*, 1990).

All these reasons mean that a modification to the AFS (1985) classification is definitely required within the next few years. Concerning ovarian endometriosis several suggestions have been made recently. Based on the histological findings of Hughesdon (1957), who reported that 90% of endometriomas present as a proliferation of endometrial tissue on the surface of an invaginated ovarian capsule, Brosens (1993) distinguishes between two types of endometriomas. Given that endometriotic implants do not penetrate the ovarian cortex as a rule, and depending on the quality of the responding fibrous tissue, two types of endometriomas exist: the red and black (Brosens, 1993). Bearing these observations in mind, Brosens *et al.* (1993) proposed that any future classification of endometriomas should take into account the type (red, black or mixed) and size of the cyst (small: <1 cm, medium: $1-5$ cm and large: >5 cm). Feeling that this classification based on colour and size is somewhat 'arbitrary' and of limited practical use, Nezhat *et al.* (1994) proposed another classification system for endometriomas, this time based on a clinical and histological study of 187 patients with endometriosis. They draw a distinction between primary and secondary endometriomas. Primary endometriomas are superficial, small and always contain endometriomal tissue. Secondary endometriomas are bigger and polymorphous. Using histological findings which seem to be different from those of Brosens (1993), who believes that the endometrioma is a colonization of an invagination of the ovarian cortex by endometrial tissue, without the ovary itself being involved, Nezhat *et al.* (1994) classify secondary endometriomas into three types according to the degree to which the endometriomal tissue penetrates the cyst wall.

These two propositions are the concrete result of the efforts made over the past few years to gain a better understanding of the physiopathology of endometriosis. The various suggestions (Brosens, 1993; Nezhat *et al.*, 1994) regarding the histology of the endometriomas are proof of the considerable uncertainty which still exists. These lines of work must be pursued further so that when, and only when, they reach conclusions which convince everyone, should they be integrated into the very necessary modification of the present-day classification for endometriosis (AFS, 1985).

Although this basic research is absolutely essential, conclusions should not be drawn prematurely with regard to how to treat endometriosis. The proposals of Brosens (1993) and Nezhat *et al.* (1994) are indispensable in the sense that they enable our knowledge of the physiopathology of endometriosis to progress. We do not agree, however, with the practical applications suggested, which say that on the basis of these histological studies, certain endometriotic cysts could be treated simply by coagulation or laser vaporization (Brosens *et al.*, 1994). As all cysts with chocolate contents are not necessarily endometriomas (Martin and Berry, 1990), it is an advantage to be able to pick out corpus luteum cysts which do indeed only require a simple biopsy. In our opinion, however, any suspicion of an endometriotic cyst requires not just a simple biopsy (Brosens *et al.*, 1994; Barbieri, 1992) but a systematic and complete histological study of the cyst, which consequently means we recommend cystectomy in every case. This is because histological analysis of endometriotic lesions of the ovary shows that 4% of cases are atypical (Czernobiliski and Morris, 1979) and represent a risk factor for the development of cancer (LaGranadie and Silverberg, 1988; Chalas *et al.*, 1991). Although laparoscopic exploration is reliable when diagnosing whether ovarian cysts are malignant or not, it is not totally infallible, and neoplastic lesions can go unnoticed if in an endometrioma (Nezhat *et al.*, 1992). This is reason enough to justify systematic analysis of the whole cyst pouch. Finally, the subdivision of secondary endometriotic cysts into the three types proposed by Nezhat *et al.* (1994) would not seem to present any major advantage at present, from the point of view of treatment. The type of treatment depends on the degree to which the cyst wall has been invaded by the endometriotic lesions, which can only be established histologically and therefore requires prior surgical removal of the cyst wall, just as we suggest.

One thing is for certain, and that is that the considerable progress made during the past few years regarding the physiopathology of endometriosis renders the present classification (AFS, 1985) inadequate, based as it is on purely visual observation of the lesions. The classification definitely needs modifying. This modification should take recent, confirmed findings into account, including the histology, state of development and depth of the lesions. The proposals made by Brosens *et al.* (1993) and Nezhat *et al.* (1994) are quite definitely a step forward in our knowledge of the physiopathology of endometriomas but, from the treatment point of view, they need to be interpreted with considerable caution before drawing any practical conclusions. For the present, endometriomas should continue to be treated surgically, with complete cystectomy and histological analysis of the entire tissue sample removed.

References

Acosta,A.A., Buttram,V.C., Besh,P.K., Malinak,L.R., Franklin,R.R. and Vanderheyden,J.D. (1973) A proposed classification of pelvic endometriosis. *Obstet. Gynecol.*, 42, 19–23.

American Fertility Society (1979) Classification for endometriosis. *Fertil. Steril.*, **32**, 633–634.

American Fertility Society (1985) Revised American Fertility Society Classification of Endometriosis. *Fertil. Steril.*, **43,** 351–352.

Barbieri,R.M. (1992) Visual diagnosis of endometriosis—Reliability? *Fertil. Steril.*, **58,** 221–222.

Brosens,I. (1993) Classification of endometriosis revisited. *Lancet*, **341,** 630.

Brosens,I., Donnez,J. and Benagiano,G. (1993) Improving the classification of endometriosis. *Hum. Reprod.*, **8,** 1792–1795.

Brosens,I., Puttemans,P.J. and Desprest,J. (1994) The endoscopic localization of endometrial implants in the ovarian chocolate cyst. *Fertil. Steril.*, **61,** 1034–1038.

Buttram,V.C. (1978) An expanded classification of endometriosis. *Fertil. Steril.*, **30,** 240.

Chalas,E., Chumas,J., Barbieri,R.L. and Mann,W.J. (1991) *Gynecol. Oncol.*, **40,** 260–263.

Cornillie,F., Oosterlynck,D., Lauwereyns,J.M. and Koninckx,P.R. (1990) Deeply infiltrating endometriosis: histological and clinical significance. *Fertil. Steril.*, **53,** 978–983.

Czernobiliski,B. and Morris,W.J. (1979) A histologic study of ovarian endometriosis with emphasis on hyperplastic and atypical changes. *Obstet. Gynecol.*, **53,** 318–323.

Huffman,J.W. (1951) External endometriosis. *Am. J. Obstet. Gynecol.*, **62,** 1243.

Hughesdon,P.E. (1957) The structure of the endometrial cyst of the ova. *Br. J. Obstet. Gynaecol.*, **44,** 481–487.

Jansen,R.P.S. and Russel,P. (1986) Nonpigmented endometriosis: clinical, laparoscopic and pathologic definition. *Am. J. Obstet. Gynecol.*, **155,** 1154–1158.

Kistner,R.W., Siegler,A.M. and Behrman,S.J. (1977) Suggested classification for endometriosis: relationship to infertility. *Fertil. Steril.*, **28,** 1008.

Koninckx,P.R., Meuleman,C., Demeyere,S., Lesaffre,E. and Cornillie,F.J. (1991) Suggestive evidence that pelvic endometriosis is a progressive disease, whereas deeply infiltrating endometriosis is associated with pelvic pain. *Fertil. Steril.*, **55,** 759–765.

LaGranadie,A. and Silverberg,G. (1988) Ovarian tumors associated with atypical endometriosis. *Hum. Pathol.*, **19,** 1080–1084.

Martin,D.C. and Berry,J.D. (1990) Histology of chocolate cysts. *J. Gynecol. Surg.*, **6,** 43–46.

Martin,D.C., Hubert,G.D., Vander Zwaag,R. and El-Zeky,F. (1989) Laparoscopic appearances of peritoneal endometriosis. *Fertil. Steril.*, **51,** 63–67.

Murphy,A.A., Green,W.R., Bobbie,D., de la Cruz,Z.C. and Rock,J.A. (1987) Unsuspected endometriosis documented by scanning electron microscopy in visually normal peritoneum. *Fertil. Steril.*, **46,** 522–524.

Nezhat,F., Nezhat,C., Welanger,C.E. and Benigno,B. (1992) Four ovarian cancers diagnosed during laparoscopic management of 1011 women with adnexal masses. *Am. J. Obstet. Gynecol.*, **167,** 790–796.

Nezhat,C., Nezhat,F., Nezhat,C. and Seidman,D.S. (1994) Improving the classification of endometriotic ovarian cysts. *Hum. Reprod.*, **9,** 2212–2213.

Sampson,J.A. (1927) Peritoneal endometriosis due to the premenstrual dissemination of endometrial tissue into the peritoneal cavity. *Am. J. Obstet. Gynecol.*, **14,** 422–469.

Stripling,M.C., Martin,D.C., Chatman,D.L., Vander Zwaag,R. and Poston,W.M. (1988) Subtle appearances of endometriosis. *Fertil. Steril.*, **49,** 427–431.

Vasquez,G., Cornillie,F. and Brosens,I. (1984) Peritoneal endometriosis: scanning electron microscopy and histology of minimal pelvic endometriotic lesions. *Fertil. Steril.*, **42,** 696–703.

Wilson,M.W., Beard,J.S., Graves,K. and Wilson,E.A. (1986) Classification of endometriotic implants by morphologic appearance and capacity to synthesise prostaglandin F. *Fertil. Steril.*, **46,** 801–806.

Wick,M.J. and Larson,C.P. (1949) Histologic criteria for evaluating endometriosis. *Northwest Med. J.*, **48,** 611–613.