Andrea Tinelli
Luis Alonso Pacheco
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Editors

Atlas of Hysteroscopy

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Contents

Part I

Vaginoscopy

1

Osama Shawki and Yehia Shawki

The history of vaginal examination dates back to ancient times and has, hitherto, been a rite of passage into womanhood. Revolutions in this gateway field have been few and far between, with the most notable of which coming from J. Marion Sims in 1845 when he fashioned the first crude speculum from a pewter spoon [1]. Cusco's duck bill vaginal speculum was introduced around 1859 which proved to be more refined and practical than most [2] and remains to this day a staple in most gynaecological offices and clinics, being a routine portion of an obgyn's examination. This aged format has not seen developments in the last century and can even go so far as to be labelled a misnomer, as the blades of the speculum hide most of the vagina, and thus is more useful in viewing the cervix. On the opposite end of the spectrum, the patients are subjected to an unpleasant and painful procedure which leaves stinging memories from one visit to the gynaecologist to the next. With the dawn of optics and endoscopy came exceptional developments in the medical field, most important of which for the gynaecologist are laparoscopy and hysteroscopy. Hysteroscopy is available as an office procedure with minimal to no sedation needed and provides exemplary diagnostic capabilities as well as the potential for operative intervention. In 1997, a new means of performing the procedure was introduced which was intended to decrease pain by avoiding the need for tenaculum and speculum [3]. This method, however, still did not allow full examination of the vagina due to the inability to maintain distension of the potential space (vagina). The proposal of a new modification to inspect the vagina was proposed by Osama Shawki dubbed the Shawki technique which includes approximation of the vulva to avoid leakage and ballooning of the vagina exposing all the vaginal walls, fornices as well as the portio vaginalis of the cervix. This technique bears all the advantages of the aforementioned technique in terms of pain relief and gains a more effective view of the vaginal canal and ectocervix. It has also been revolutionary for the treatment of vaginal pathology such as OHVIRA syndrome for the obstructed hemi-vagina to be re-connected to the menstruating vagina.

These patients tend to present around the age of puberty when the menstrual cycle begins. Patients complain of severe cyclic lower abdominal pain during menstruation often requiring hospitalisation. As one hemi-vagina is communicating with the vulval orifice, there is no suspicion of Mullerian anomalies until further investigations are performed which include ultrasound examination and MRI.

These advanced studies would show a fluid collection in the vagina. The condition is most commonly associated with a bicornuate bicollis uterus, otherwise known as didelphys.

Another common association is ipsilateral renal agenesis on the same side as the obstructed hemi-vagina; thus a urological examination would prove prudent.

These cases were previously misdiagnosed and subjected to exploratory laparotomies which may have ended in a hemi-hysterectomy, removing the obstructed side's uterus. The fact that a lot of the patients are virgins proved a difficult point to navigate any vaginal approach in certain cultures where the hymen is of great importance.

With the new vaginoscopy technique implemented, exposure of the vagina is possible and the obstructed vagina can be accessed by utilising a resectoscope with a Collin's knife inserted trans-hymenal and incising through the obstructing vaginal septum. This unification is a minimally invasive approach which respects the hymen and avoids the abdominal route whilst providing an instant cure for the condition.

Similarly, a longitudinal non-obstructing vaginal septum can be tackled by the vaginoscopic route as well.

This pathology, which is usually associated with a cervico-uterine septum, was previously treated surgically by clamping and excising the vaginal septum. This was then followed by hysteroscopic release of the uterine septum with or without incision of the cervical septum [4].

In the vaginoscopic approach, the vaginal septum is tackled in a similar way to a uterine septum, again utilising the resectoscope with Collin's knife inserted and incising the

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A. Tinelli et al. (eds.), *Atlas of Hysteroscopy*, https://doi.org/10.1007/978-3-030-29466-3_1

septum from caudal to cephalad until reaching the external cervical os.

In this technique the tissues retract and there is no need for any further excision. Progression to resecting the cervical and uterine septa is then possible hysteroscopically.

Vaginoscopy has also proved to be advantageous in cases where vaginal examination of virgins is required. In cases of endometrial polyps, submucous fibroids and such. Here, the vaginoscopic approach saves any damage to the hymen, in cultures where integrity of the hymen is considered important. The diameter of the hysteroscope ranges from 3.4 to 9 mm depending on the modality of the sheath used and the different brands available, all of which can be inserted through the hymen without causing any damage.

Furthermore, cases of vaginal endometriosis can now be easily diagnosed vaginoscopically, through proper visualisation and examination of the vagina. Bluish or brownish endometriotic spots which would have been missed by a conventional vaginal examination can now be seen. This pathology can be implicated in cases of dyspareunia and can now be easily diagnosed and therefore treated by cauterising the endometrial tissue.

In conclusion, vaginoscopy is a new vision for an age-old practice, shifting gynaecological vaginal examination to the modern era (Figs. 1.1, 1.2, 1.3, 1.4, 1.5, 1.6, 1.7, 1.8, 1.9, 1.10, 1.11, 1.12 and 1.13).

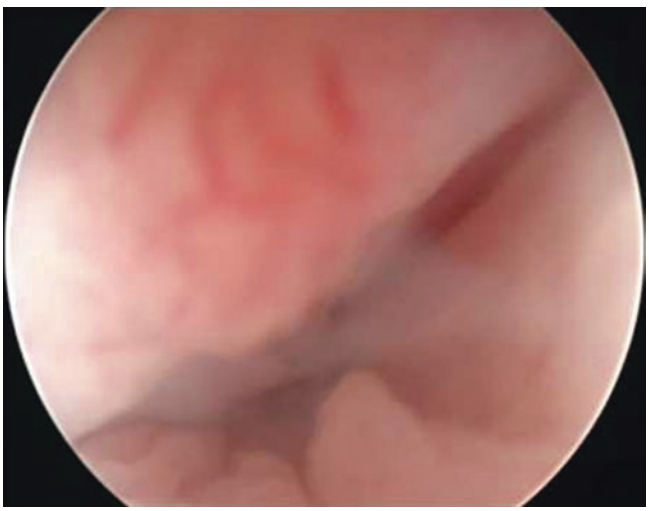

Fig. 1.1 Standard vaginoscopy without approximation of the vulva yielding a collapsed view of the vagina has limited capacity to inspect the vagina and is mainly used as a means of accessing the cervix

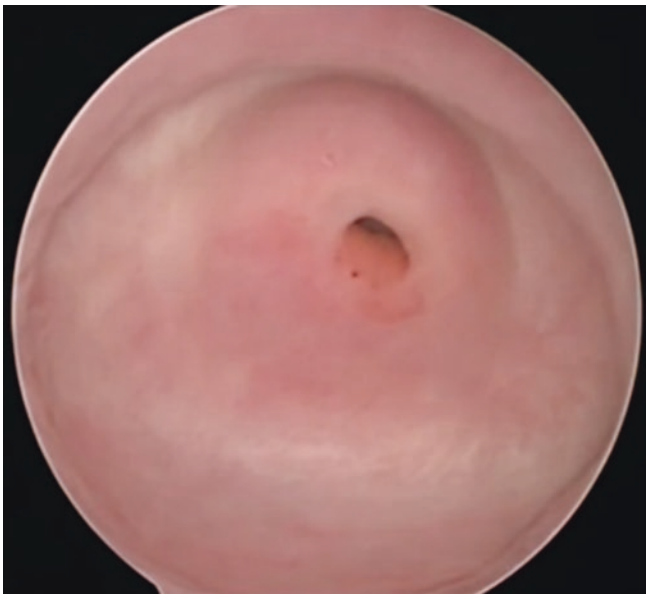

Fig. 1.2 Shawki technique to visualise the vagina, exposing the vaginal walls, fornices and portio vaginalis of the cervix

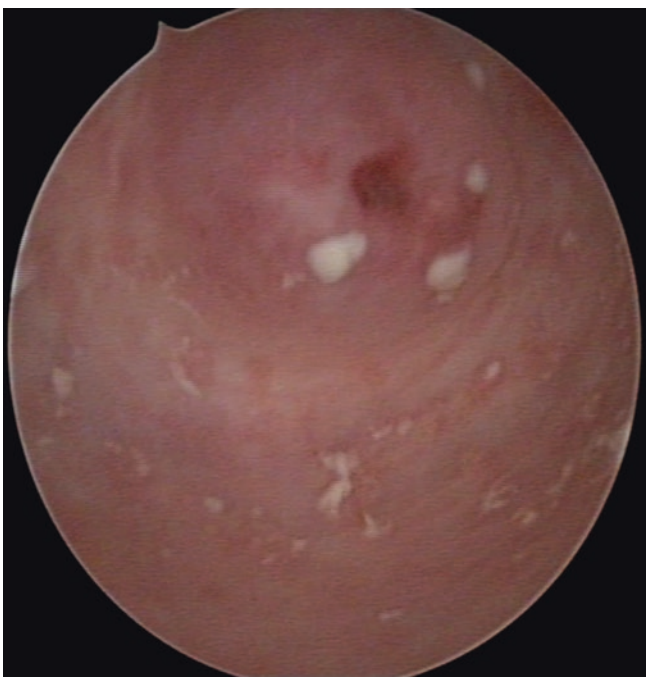

Fig. 1.3 A case of vaginal candidiasis depicting an example of infections of the vagina as seen by modern vaginoscopy. Creamy white congregations resulting from the yeast infection can be seen on the vaginal walls and on the cervix

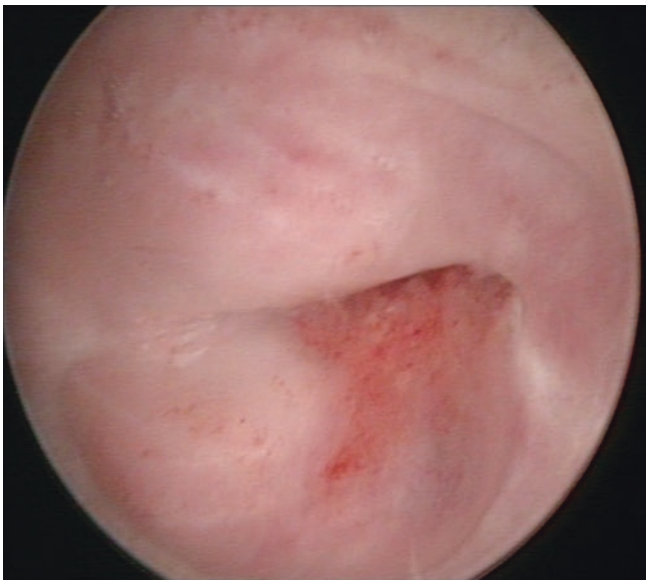

Fig. 1.4 Picture of bilateral cervical tears most likely as an obstetric complication. The anterior lip of the cervix is seen almost flush with the vaginal wall in compilation with the cervical tears

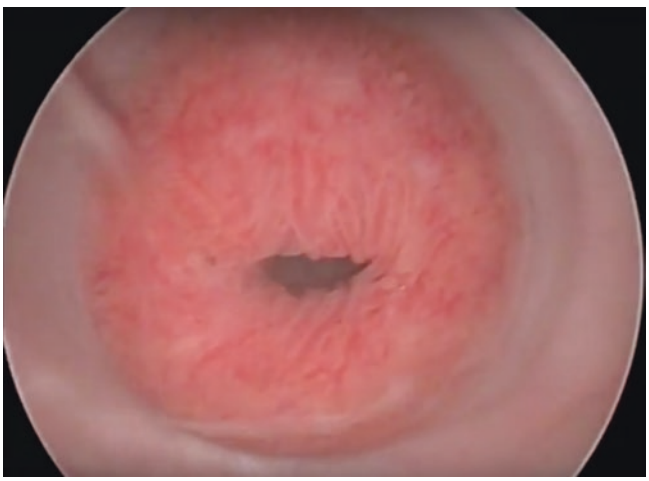

Fig. 1.5 Picture of cervical ectopy. Complete cervical erosion where the transformation zone has shifted from the external os to cover the portio vaginalis of the cervix by columnar epithelium

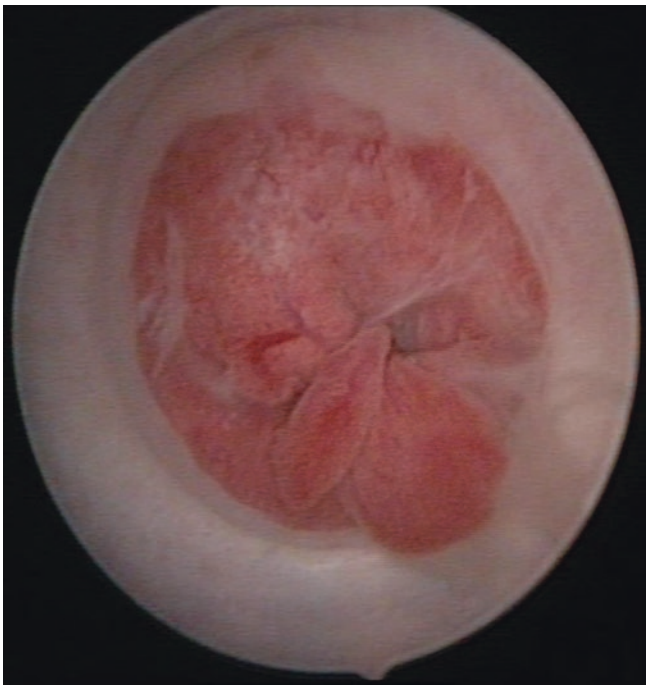

Fig. 1.6 Cervical erosions complicated by cervical polyps. The cervical polyps are seen lined by cervical epithelium and protruding from the external os

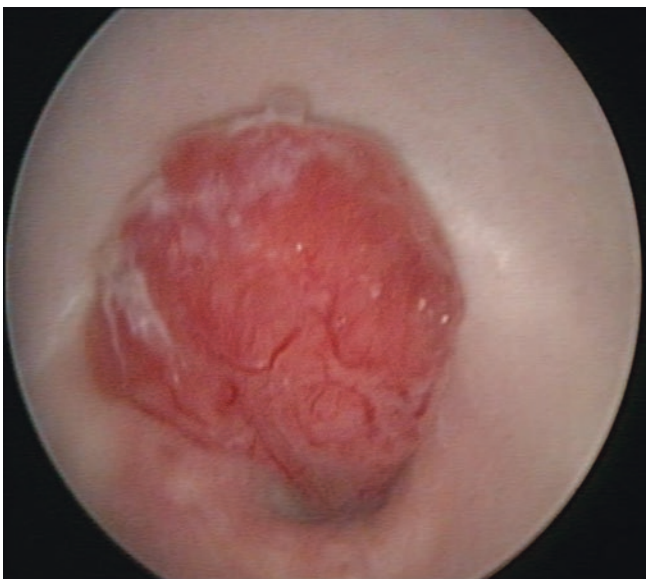

Fig. 1.7 Cervical fibroid prolapsed from the external os and into the vagina

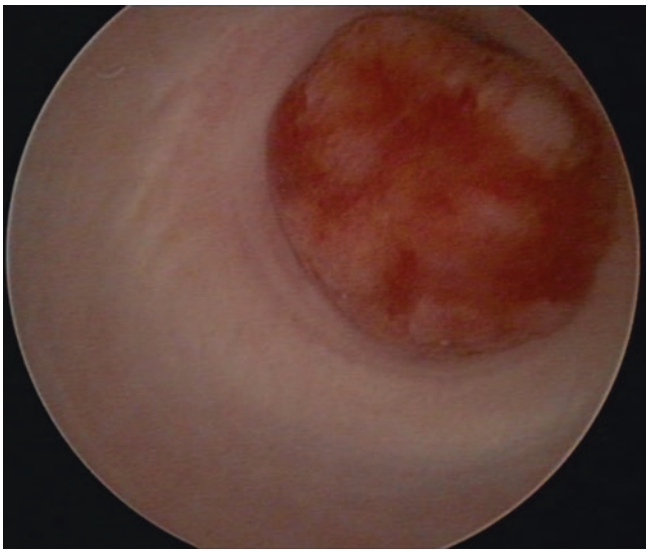

Fig. 1.8 Submucous fibroid prolapsed through the cervical canal and into the vagina. Characteristic appearance of endometrium on the fibroid indicates that the origin is from the uterine cavity

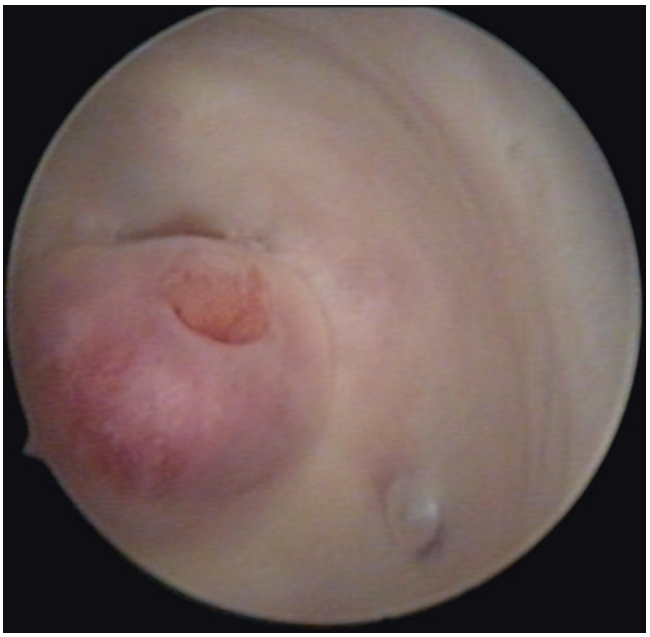

Fig. 1.10 Multiple spots of vaginal endometriosis seen in the anterior and lateral fornices. This pathology has been implicated in cases of dyspareunia and deep pelvic pain

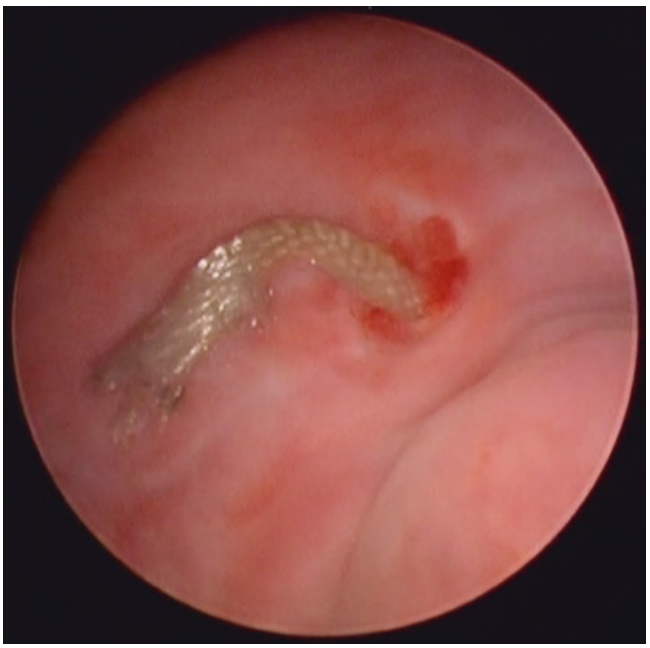

Fig. 1.9 Cerclage tape left near the anterior fornix following improper removal of vaginal cerclage tape

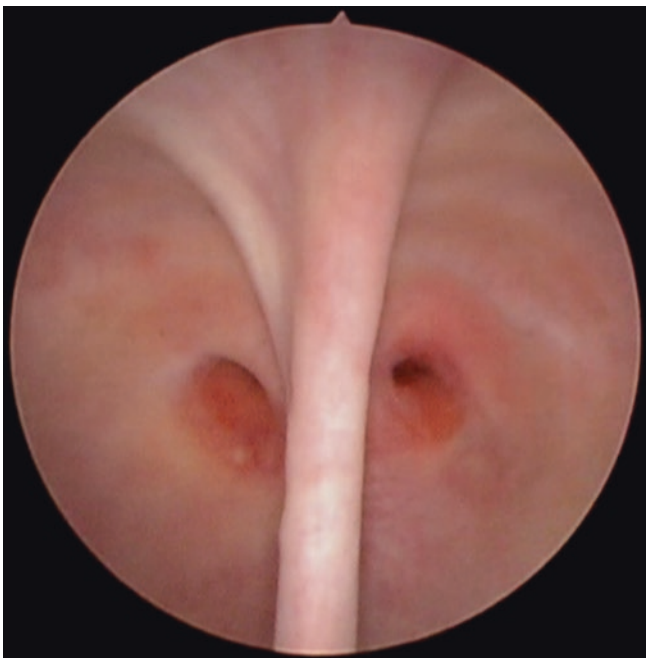

Fig. 1.11 Longitudinal non-obstructing vaginal septum, class V1 [5]. Two external cervical ostia are seen on either side of the septum

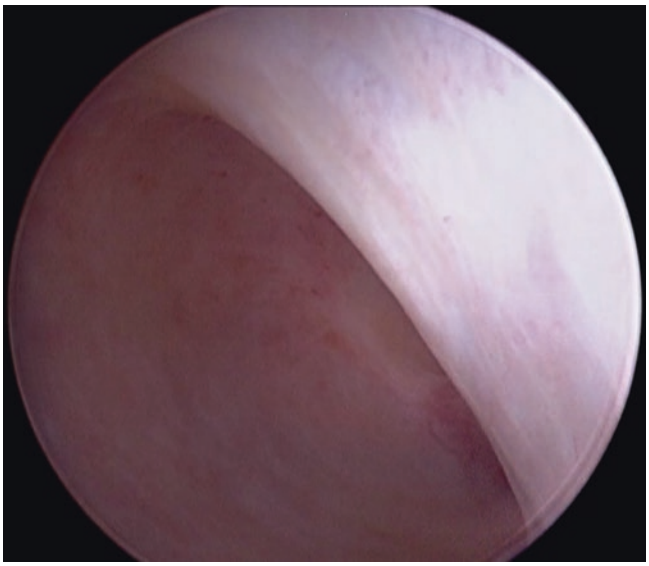

Fig. 1.12 Longitudinal obstructing vaginal septum, class V2 [5], in a case of didelphys uterus leading to an OHVIRA syndrome. Septum is obstructing the left hemi-vagina

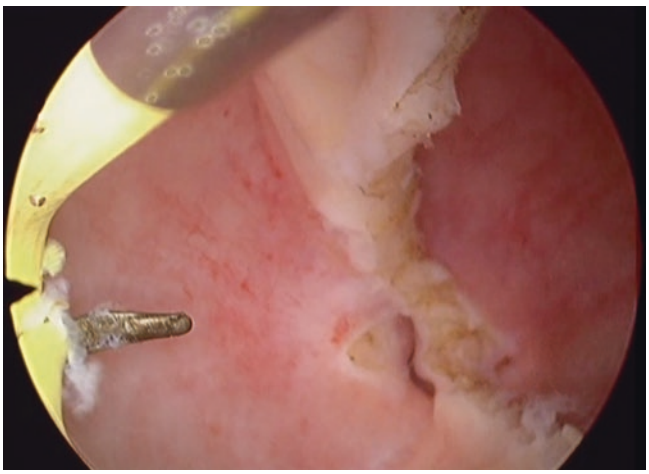

Fig. 1.13 Post-hysteroscopic correction of a longitudinal obstructing vaginal septum with the obstructed hemi-vagina now communicating with normal side

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Cyclic Endometrial Changes

2

Alfonso Arias and Alicia Úbeda

Although when students who learn hysteroscopy go in the look for organic and structural, benign, or malign pathology, this endoscopic technique allows much more. With the help of a video camera and increasing visual acuity (Figs. 2.1 and 2.2), hysteroscopy can describe either endometrium development or functional disturbances that may cause hypotrophy, hypertrophy, hyperplasia, and neoplasia [1]. This accuracy has shown to be higher than histologic examination, as the latter may miss nearly half of major intrauterine disorders [2].

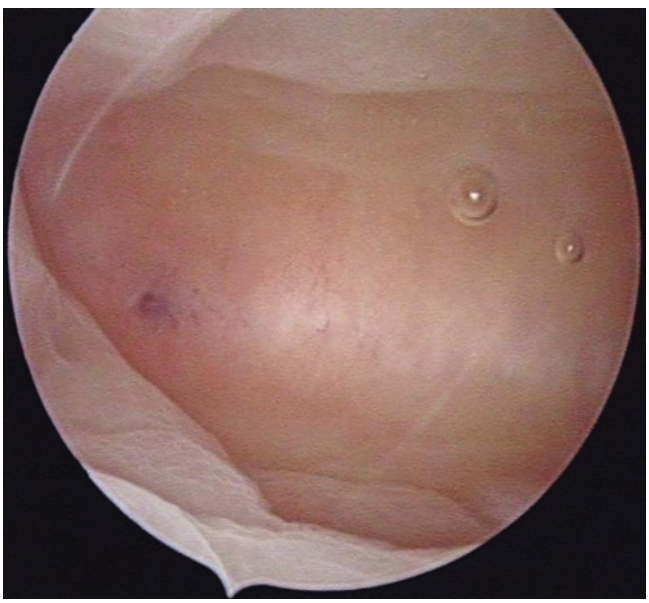

Fig. 2.1 Normal uterine cavity under fluid distention. Notice the right tubal ostium. Surrounding endometrium is in a secretory phase

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Endometrial changes are strongly related to ovarian stimulation in the absence of other external or internal stimuli. Hormone secretion is reflected in glandular and vascular changes, and therefore in visual appearance of this layer along a conventional cycle of 24–35 days. This evolution was yet first described in the early 1990s through CO_2 hysteroscopy [3] and strongly correlates with Netter's descriptions [4].

The endometrial tissue is made up of two layers: the basilar zone, which attaches the endometrium to the underlying myometrium, and the functional zone, which is the majority and the one that progressively changes under hormone influence. On the one hand, physiological changes in thickness and appearance are due to the ovarian hormones. On the other, other internal or external influences (hormonal, infectious, medication) may cause changes that will not be treated in this chapter.

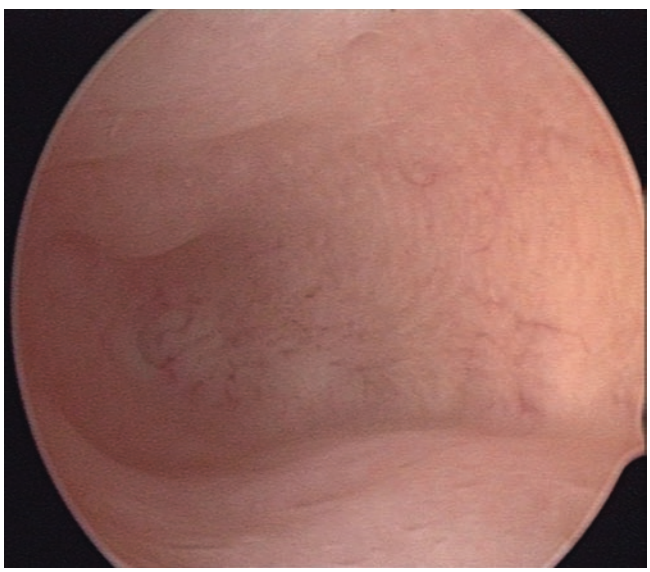

Fig. 2.2 Uterine right cornual region. Endometrium is in late secretory phase. Vessels start to be seen in the fundal area as menstrual phase is near to begin

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This chapter describes the most frequent changes after endogenous ovarian stimulation image by image. The endometrial cycle starts with the detachment of its upper layer. Later, there are two main phases:

1. The proliferative phase, which is a period of tissue regeneration and mucosal growth (Figs. 2.3, 2.4, and 2.5): Under estrogen influence, both glands and vessels increase in number, size, and width towards the surface until ovulation (Fig. 2.6).
2. The secretory phase (Figs. 2.7, 2.8, 2.9, and 2.10), where the maturation occurs after progesterone effect, and glands increase in shape, come together, and hide vessels underneath.

Both stages are separated by ovulation and start of progesterone secretion. As long as hysteroscopic knowledge moves along, early and late half of each can also be differentiated. At the end, as hormone secretion stops from the ovaries, an ischemic phase starts and endometrial cycle ends [5].

Performance of hysteroscopy in the secretory phase shows two main advantages:

- The endometrial mucosa expresses the summing effect of both estrogen and progesterone influences, and allows the diagnosis of hypertrophic (Fig. 2.11) or hypotrophic (Fig. 2.12) endometrium.

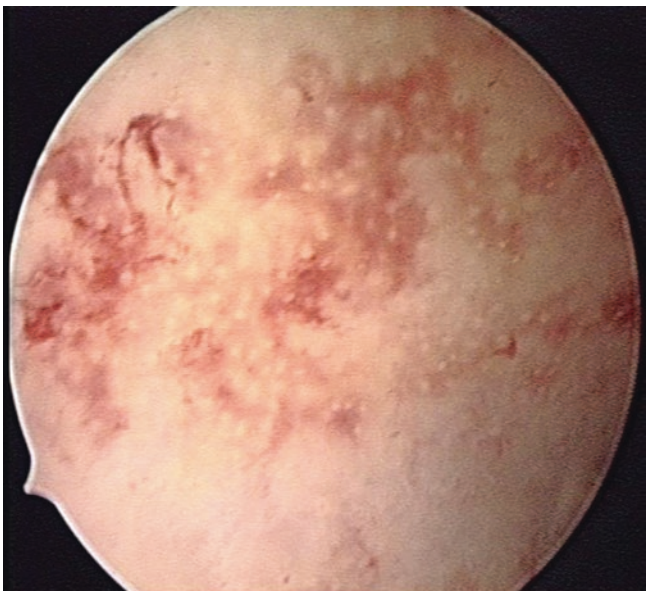

Fig. 2.3 Regenerative endometrium in the very early proliferative phase. Glands grow from the basal layer (out and inside the red plates) straight up to the surface and are seen as narrow white dots. Red areas belong to the basal layer which is still visible

- Endometritis will not be so easily missed because no red plates belonging to the proliferative phase will interact at the same time.

Final images of premenstrual (Figs. 2.13 and 2.14) and menstrual phases (Figs. 2.15 and 2.16) are infrequent but they show how the endometrium tears until being totally expelled.

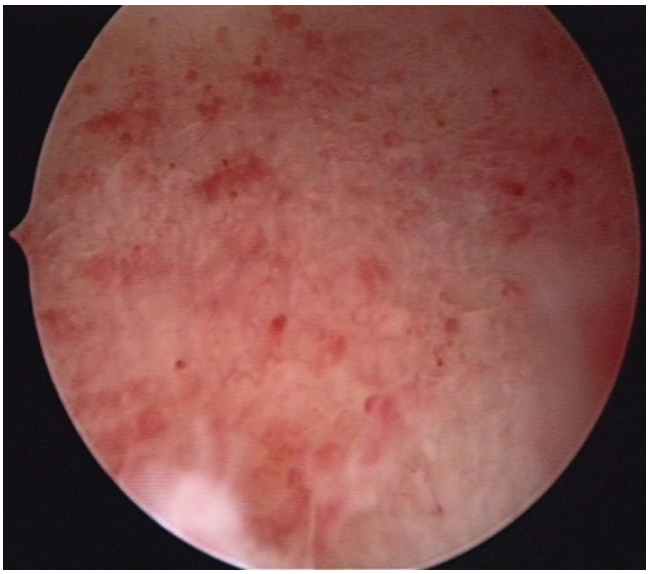

Fig. 2.4 Late proliferative phase: red plates are progressively hidden by glandular and vascular growth. Endometrial spiral arteries give a red color to the mucosa

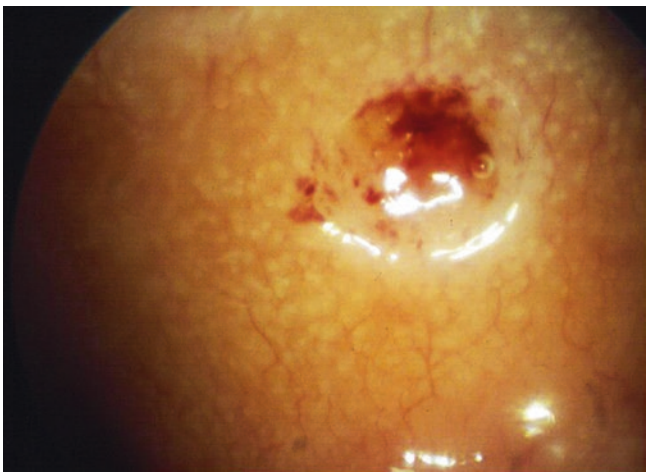

Fig. 2.5 Endometrial notch in the late proliferative phase. Small petechia appears under the hysteroscope's pressure. The procedure has been carried out under gas distention

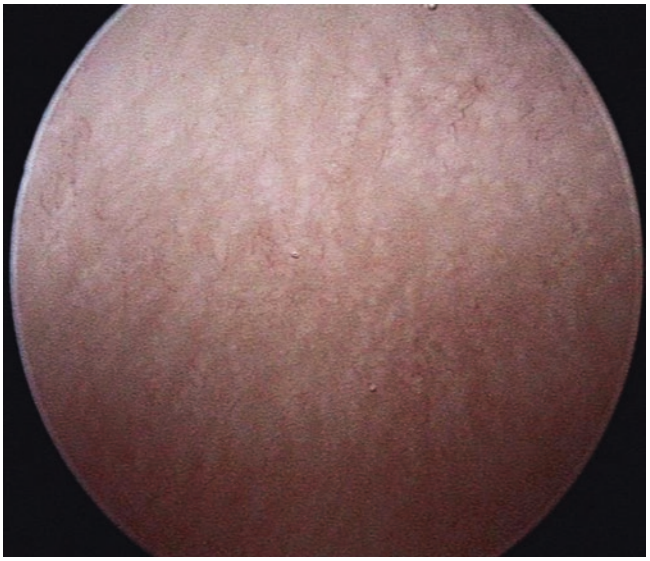

Fig. 2.6 The ovulatory phase: Under the progesterone stimuli glands end their growth and tend to come together, even though they are well distinguished. Different shapes are seen among white dots. Red color of the mucosa is substituted for a light yellow one

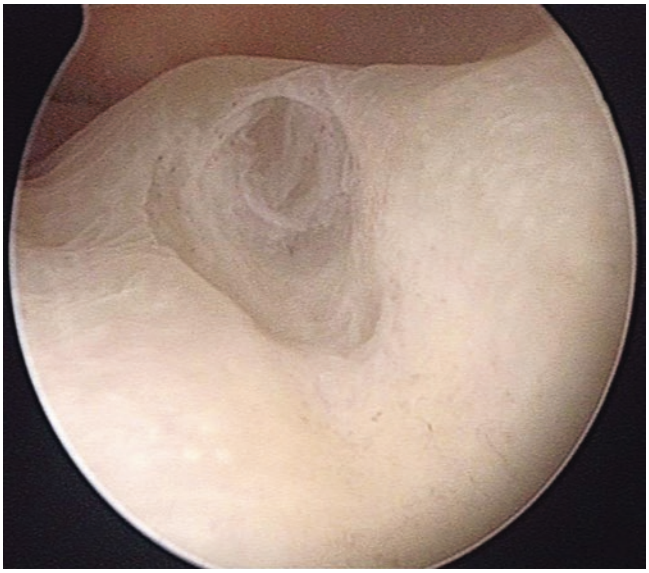

Fig. 2.8 Endometrial notch in the early secretory phase. Mucus secretion allows the hysteroscope's sinking as it is wide enough thanks to mucus secretion

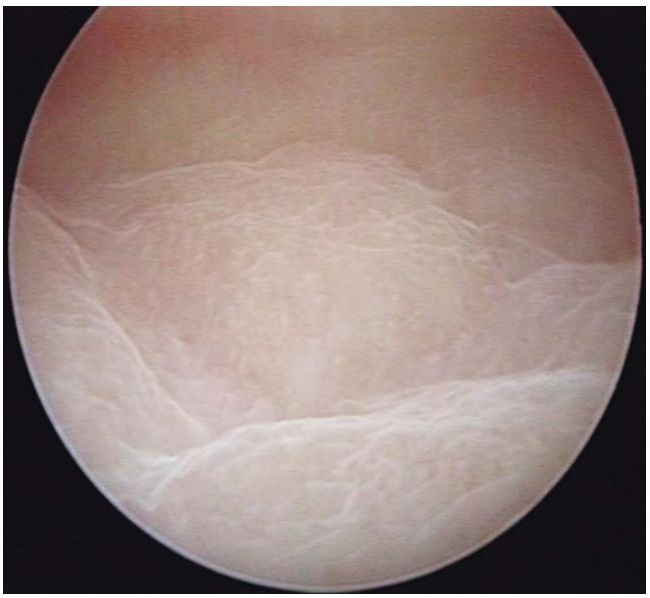

Fig. 2.7 Early secretory endometrium. A global whitish color starts to dominate after mucus glandular secretion in the mucosal surface. Still some glands are seen, though the majority have come together back to back. Vessels remain underneath and are no longer seen

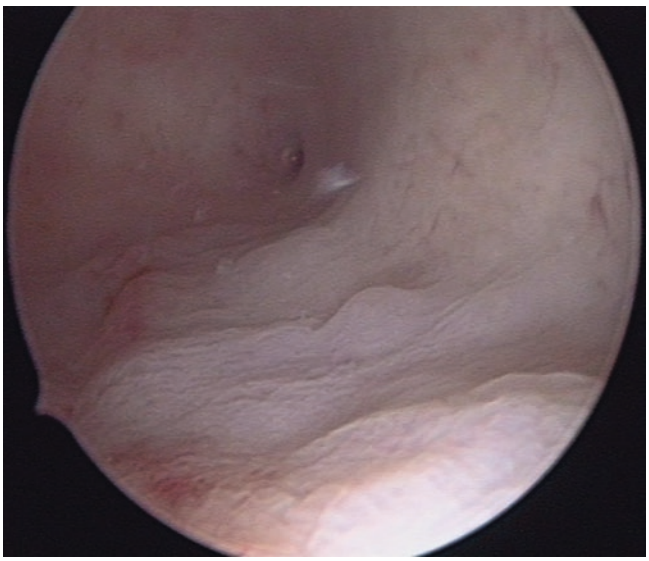

Fig. 2.9 Late secretory phase: Endometrium has flattened as glandular mucus has thickened. Color remains quite white. Endometrial glands are no longer seen as individuals

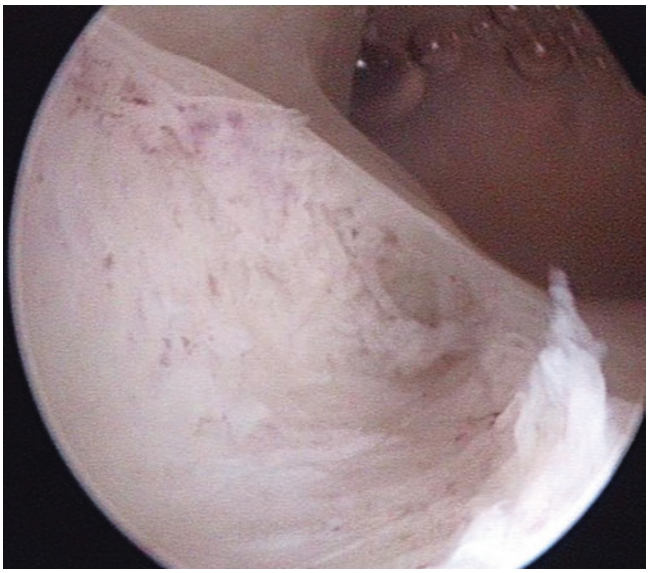

Fig. 2.10 Endometrial notch in the late secretory phase under the hysteroscope pressure. Thickening of the mucosa does not allow the hole sinking of the endoscope, as it is not as wide as in the early secretory phase

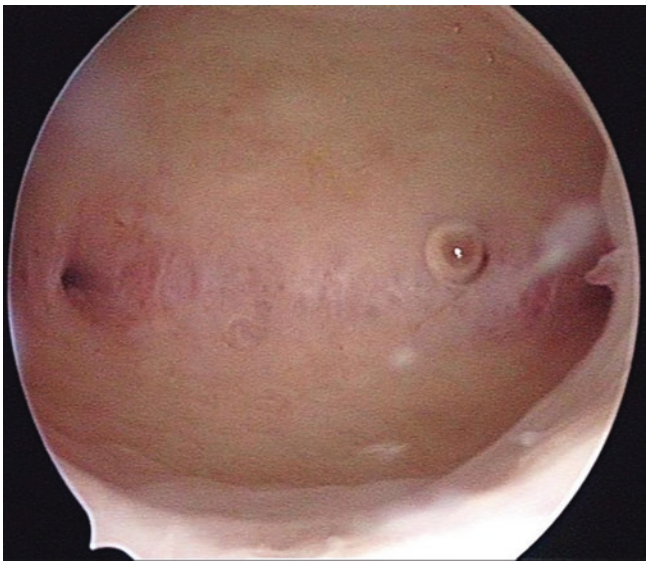

Fig. 2.12 Hypotrophic secretory endometrium in a 46-year-old woman. A decreased influence of both estrogens and progesterone makes the mucosa thinner compared to that in younger women. Myometrial fibers are seen in the fundal area

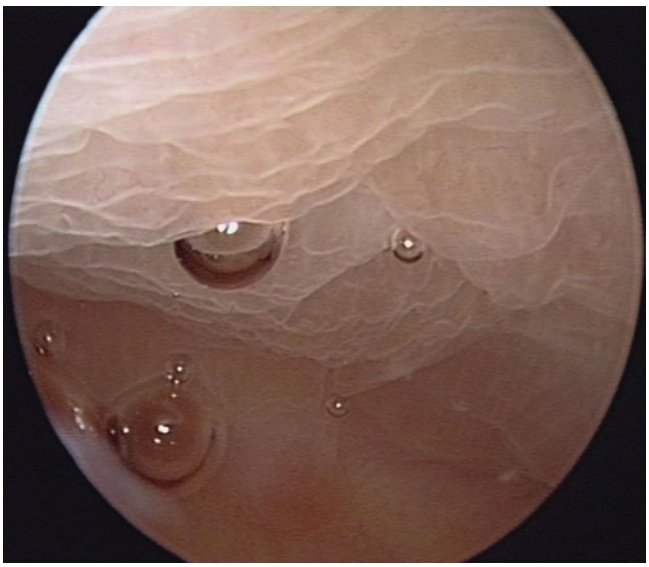

Fig. 2.11 Light endometrial hypertrophy under a disbalanced influence of estrogens over progesterone in the mid-secretory phase

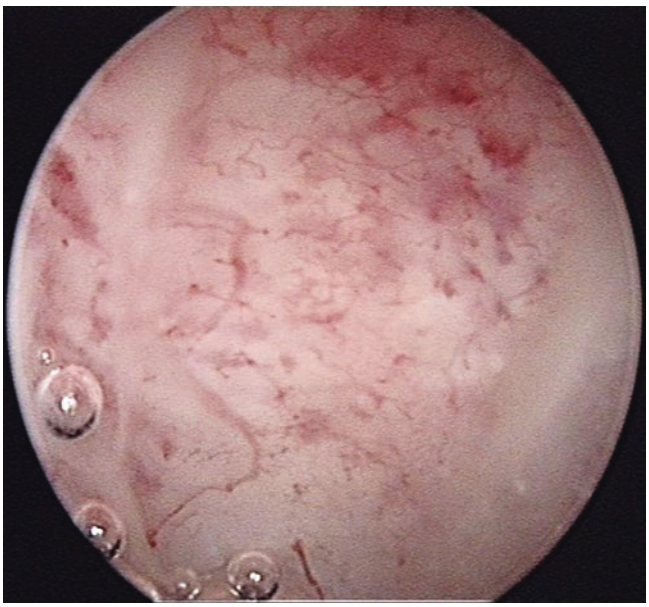

Fig. 2.13 Early premenstrual phase. Short vessels start to appear in the endometrial surface

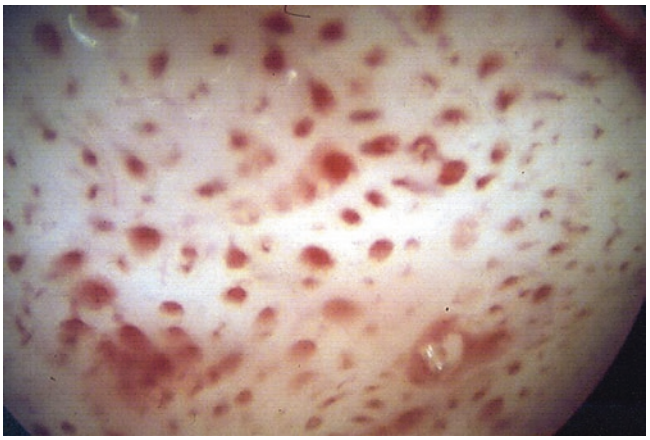

Fig. 2.14 Early menstrual phase. The mucosa starts its tearing as little bleeding holes. This image is not often seen. This hysteroscopy was carried out under CO_2 distention

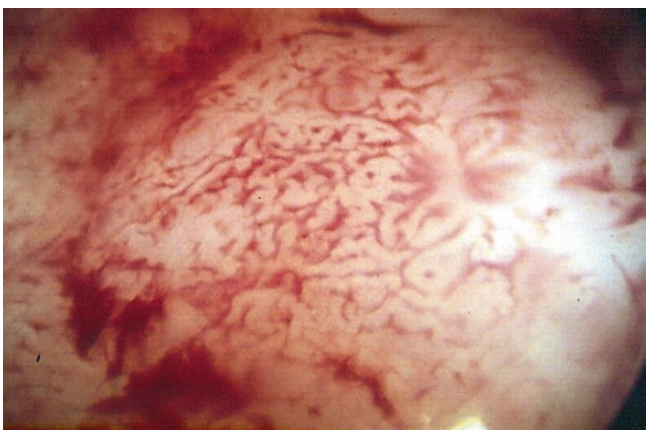

Fig. 2.15 Exceptional image of the immediate beginning of the menstrual phase. Tearing of the functional mucosa starts to take off from the basal layer and torn vessels clearly start to bleed

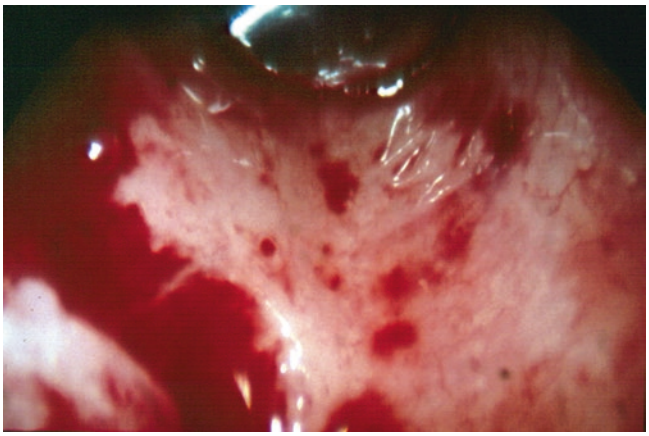

Fig. 2.16 Full menstrual phase. This image has been taken under CO_2 distention. Endometrial grooves result from the partially expelled upper mucosa

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The Atrophic Endometrium

3

Nash S. Moawad, Alejandro M. Gonzalez, and Santiago Artazcoz

3.1 Introduction

The endometrium is the hormonally responsive glandular tissue lining the uterine cavity. This tissue consists of:

1. Epithelium (endometrial glands)
2. Stroma (endometrial stroma)

The structure and activity of a functional endometrium reflect the pattern of ovarian hormone secretion. The histologic types of glandular cells are columnar or cuboid. The endometrium undergoes regular growth and maturation throughout the menstrual (endometrial) cycle, corresponding to the proliferative effects of estrogen and secretory effects of progesterone produced by the ovary during the hormonal (ovarian) cycle. In the absence of pregnancy, shedding of the thickened, vascular endometrial lining occurs in the form of a menstrual cycle, leading to a thin endometrium, followed by regeneration.

The endometrial tissue becomes atrophic after menopause as a result of cessation of ovulation and ovarian estrogen and progesterone secretion. At this time, there is loss of the functional layer and the endometrial glands take on a simple tubular or low cuboidal, often cystic, form, showing neither proliferative nor secretory activity, whereas the endometrial stroma becomes fibrotic.

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The diameter of the glands usually is 0.1 mm and the thickness of the endometrium on transvaginal ultrasound is less than 4 mm.

Microscopic examination shows the following:

Glands: small columnar cells:

- Moderate quantity of eosinophilic cytoplasm
- Ovoid (palisaded) nuclei, more or less nuclear pseudostratification
- No mitoses

Architecture:

- Cystic dilation

In the absence of sufficient estrogenic stimulation, the epithelium becomes quiescent and can appear as either weakly proliferative (inactive) or atrophic.

Weakly proliferative endometrium shows a pattern intermediate between normal proliferative and atrophic. The epithelium is columnar, with only a minor degree of pseudostratification. The nuclear chromatin is dense. Atrophic endometrial epithelium is low cuboidal to flattened, with a single row of dense nuclei. Mitotic activity is absent.

There are four histological types of atrophic endometrium, atrophic inactive, atrophic/weakly proliferative (non-inactive), mixed (inactive and non-inactive), and cystic atrophic [1, 2].

1. Atrophic and inactive endometria are defined as those deprived of functionalis and consisting exclusively of thin basalis with a few narrow tubular glands lined by cuboidal indeterminate epithelium showing neither proliferative nor secretory activity.
2. Atrophic/weakly proliferative (atrophic non-inactive) endometria are defined by the following criteria:
   (a) Shallow endometrium 2.2 mm thick (mean 2.2, range 1.0–3.5 mm) with the loss of distinction between the basal and functional layer

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(b) Proliferative-type epithelial glands, somewhat tortuous, with tall columnar pseudostratified epithelium, oval nuclei, and very infrequent mitoses
(c) A dense fibrotic endometrial stroma devoid of mitoses
3. Mixed: The mixed form of endometrium is defined as atrophic and inactive endometria showing focal areas of weakly proliferative glands.
4. Cystic atrophic endometrium.

Often, atrophic endometrium has cystically dilated glands. These may be the atrophic variants of cyclically dilated glands that are seen in the lower functionalis in women aged 35 years and above. Cystically dilated glands also are seen in cystic glandular hyperplasia with retrogressive atrophy. In this case the endometrial mucosa retains the thickness of an otherwise active hyperplasia, but the glandular epithelium is atrophic and the stroma is collagenized and no mitotic figures are seen. A third explanation is that fibrosis of the stroma blocks the glands that then become distended.

Cystic endometrial atrophy is a benign process that can occur as part of tamoxifen-associated endometrial changes. It is diagnosed histologically when multiple cystic spaces (dilated glands) lined with atrophic epithelium are present within a dense fibrous stroma. It is an unusual hysteroscopic finding.

At hysteroscopy, the endometrium appears white but hypervascularized, with scattered protuberances. This "tamoxifen-like" mucosa can be seen as early as 6 months after the start of tamoxifen therapy. At histopathologic examination, these protuberances are identified as cystic glandular dilatation [3, 4].

This condition is benign and is not associated with an increased risk of either endometrial adenocarcinoma or endometrial hyperplasia.

While atrophic glands are characteristic of premenopausal patients taking exogenous hormones, atrophy in any other setting in a premenopausal woman is an unexpected finding and its significance is not well understood.

Why is it so important to take this condition into account?

The most frequent cause of postmenopausal bleeding is endometrial atrophy, found in approximately 45–50% of the patients. The exact cause of bleeding from the atrophic endometrium is not known. It is postulated to be due to anatomic vascular variations or local abnormal hemostatic mechanisms. Thin-walled veins, and thin fragile stromal support of these superficial vessels [1].

An Italian prospective study that includes 18 centers and 930 patients showed that an endometrial thickness of ≤4.0 mm safely predicts endometrial atrophy and justifies expectant management of patients with postmenopausal bleeding, as long as the patient understands the need for proper follow-up should bleeding persist, recur, or worsen [5].

The cutoff of 4 mm is important for general gynecologists and hysteroscopic surgeons because sometimes with thin endometrium in ultrasound it is not necessary to perform diagnostic hysteroscopy or endometrial biopsy.

It is important for hysteroscopic surgeons to recognize the variable patterns of the normal atrophic endometrium, in order to direct guided biopsies and proper management of abnormal endometrial lesions, while avoiding overtreatment and unnecessary procedures when normal atrophic patterns are noted and confirmed by pathologic examination when needed [6, 7] (Figs. 3.1, 3.2, 3.3, 3.4, 3.5, 3.6, 3.7, 3.8, 3.9, 3.10, 3.11, 3.12, 3.13, 3.14, 3.15, 3.16, 3.17, 3.18, 3.19, 3.20, 3.21, 3.22, 3.23, 3.24, 3.25, 3.26, 3.27, 3.28, 3.29, 3.30, 3.31, 3.32, 3.33, 3.34, 3.35, 3.36, 3.37, 3.38, 3.39, 3.40, and 3.41).

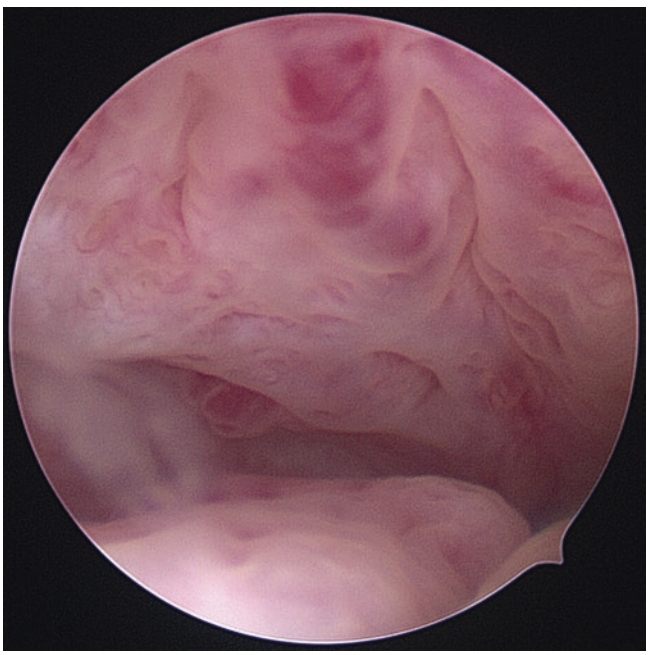

Fig. 3.1 Abnormally thickened postmenopausal endometrium in a patient with a history of endometrial ablation

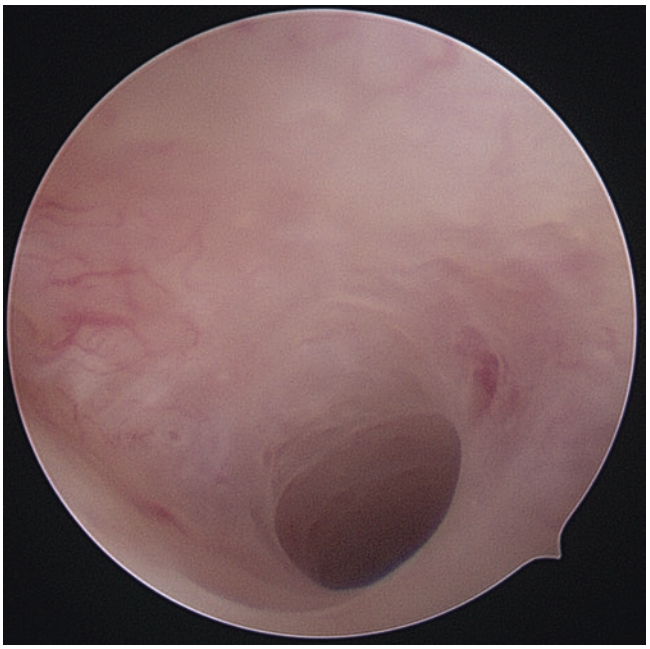

Fig 3.2 Atrophic cervical canal with cervical stenosis

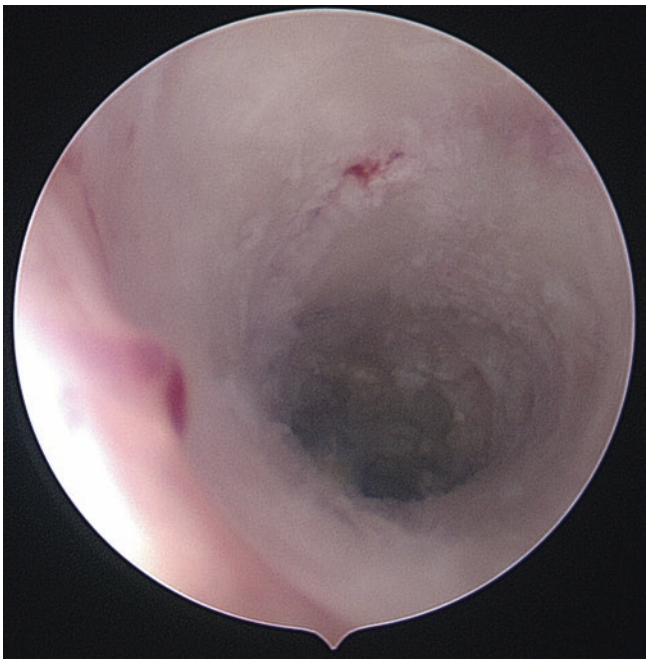

Fig. 3.3 Atrophic cervical canal, status post-cervical dilation of cervical stenosis. The typical features of the cervical canal are absent

Fig. 3.4 Pale atrophic endometrium of the left cornual region; tubal ostium noted