Atlas of Endometrial Histopathology

Third Edition
This new edition differs from the preceding ones in that there has been extensive revision of most chapters. Recent advances in research gained by immunohistochemical, molecular biological, and cytogenetic methods are included, as far as they are applicable for daily diagnostic work. The chapter on neoplasms, in particular, has been greatly expanded in accordance with the new WHO International Histological Classification of genital tract tumors, covering all pertinent differential diagnostic aspects. The chapter on malignant lymphomas and hematopoetic neoplasms involving the endometrium is a new contribution to this edition (F.D., Ulm). We have revised the chapter on gestational diseases, incorporating recent advances in the differential diagnosis of gestational trophoblastic tumors. New discoveries and experiences in correlating structure and function in infertility and in hormone replacement therapy of peri- and postmenopausal patients are also included. Many new microphotographs have been added to illustrate the advances in tumor research and in immunohistochemical detection methods. We have updated the list of references including recent relevant publications.

To the correspondents and consultants who have contributed valuable observations and suggestions, bringing thereby to our attention omissions in the second edition, we acknowledge our cordial thanks. We thank Dr. Wolfram Klapper, Director of the Lymphom Register of the University of Kiel, for case material for making the microphotographic illustrations of lymphomas. The staff of Springer-Verlag has earned our gratitude for their skill in preparing this new edition.

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The cyclic changes in the function and histology of the endometrium and the rapidity with which they take place complicate the histopathologic diagnosis. Disease states of varying intensities which superimpose themselves on these fluctuating functional changes often produce complex admixtures of the functional state and the damage wrought by the pathologic processes.

The pathologist’s functional and morphologic diagnoses should represent the result of constant cooperation with the attending gynecologist. The prime purpose of this atlas is to help the pathologist to find, classify, and diagnose differentially the changes he sees under his microscope. We have kept the text brief. It provides a short description of the pathology of the disease, summarizes and differentiates the possible causes, and explains the clinical importance of the condition and thus how to treat it. Accordingly, this book’s approach to diseases is the opposite to that of a textbook, which systematically presents the histopathology of the endometrium according to etiology. This atlas proceeds from the histologic changes and relates these to the possible cause. It follows, as it were, the line of thought which the diagnostic pathologist uses, sparing him the search through different chapters on etiology in a textbook of comparable histologic states. In addition, it obviates the need to duplicate pictures of similar structural changes caused by different diseases.

The atlas is restricted to the endometrium. Neighboring parts of the uterus are included only where we considered them important in differential diagnoses. We have not included electron microscopic pictures, since they can be dispensed with in routine diagnostic studies.

This book is intended for all pathologists, to aid them in their routine diagnostic work and to explain as clearly as possible just how comprehensive, complex, and important the histopathology of the endometrium has become in the last few years. The book is also meant for the clinician, to help guide him through the broad spectrum of possibilities of histopathologic diagnosis of the endometrium, especially in functional disturbances. If this atlas, through its sharp distinction of histopathologic states, helps to improve dialogue between pathologists and clinicians so that optimal therapy of the patients can be provided, then it has fulfilled its purpose.

Gisela Dallenbach-Hellweg and Hemming Poulsen
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2.1 Procedures for Obtaining Endometrial Tissue

A complete curettage is the ideal method for optimal diagnostic evaluation of the endometrium. A fractionated curettage has additional advantages; it helps to localize the site and extent of malignancy and assists in evaluating endocervical changes that develop during hormonal therapy. For functional diagnosis in infertile patients, endometrial biopsies will usually suffice if properly taken with a single-stroke biopsy from the uterine fundus, and they offer the advantage that they can be repeated within the same menstrual cycle. Interpreting biopsies taken by brush or aspiration may prove quite difficult and inaccurate, except for advanced carcinomas.

2.2 Selection of the Proper Time for Curettage

In order to obtain optimal diagnostic results, the time for curettage must be carefully selected. In infertile patients, differential diagnosis of the various causes of sterility is best made shortly before the onset of menstruation. Only at this late time can the failure in endometrial differentiation be completely surveyed. In menorrhagia possibly due to irregular shedding, the best time for curettage is 5–10 days after the onset of menstruation, in order to recognize remnants of nonlysed mucosa. In metrorrhagia, curettage is best done without delay when much of the endometrium is still available for examination. With amenorrhea in a patient of reproductive age, pregnancy must be excluded before curettage is performed.

Clinical information is equally important to the pathologist, mainly about the patient’s age, menstrual history, any hormone therapy, contraceptive device, endocrinologic disorders, and size and cavity of the uterus, including sonographic and/or hysteroscopic reports.
2.3 Preparation of the Endometrial Specimen

For fixation, a 4% neutral solution of formaldehyde is commonly used and is ideal for most of the diagnostic procedures involved in endometrial examination. Routine staining of all specimens should include hematoxylin-eosin (H & E) and a connective tissue stain, for instance van Gieson’s solution. The latter is particularly important for recognizing endometrial polyps, or portions of them, and hyalinized placental villi. An additional periodic acid-Schiff (PAS) reaction may be helpful in detecting small amounts of glycogen or mucopolysaccharides in glandular epithelial cells. A reticulin impregnation can be useful for verifying tissue lysis or for distinguishing between various types of tumors. These four staining methods suffice for most questions arising in routine examination of the endometrial biopsy.

For subclassification of endometrial carcinomas, sarcomas, carcinosarcomas and other nonepithelial and nonmesenchymal tumors such as neuroectodermal tumors or lymphomas, immunohistochemical stainings may be helpful or needed. Among these, monoclonal antibodies against various cytokeratins, vimentin and carcinoembryonic antigen (CEA) are the most important for adenocarcinomas. In addition, immunostainings for the estrogen and progesterone receptor might be desired by the clinicians as a prerequisite for adjuvant hormonal treatment. In case of neuroendocrine differentiation of a carcinoma, chromogranin A, synaptophysin and CD56 are useful. Tumors of neuroectodermal origin (tumors of the PNET-group) typically express vimentin, CD99, NSE, Fli-1, and may partially express CD57, S-100, and neuroendocrine markers. Malignant lymphomas of the endometrium are preferentially B cell lymphomas (see p. 196 ff). Therefore, markers for lymphatic differentiation antigens such as CD3, CD5, CD10, CD11c, CD20, CD23, CD30, CD75 in combination with markers for BCL-2, BCL-6, Cyclin D1, Mum-1, IgM, IgD and immunoglobulin light chains should be applied. To clarify the dignity of a lymphatic proliferation, a molecular pathological clonality analysis with the polymerase chain reaction (PCR method) might be indicated. For all questional reactive processes and neoplastic lesions in the endometrium, the proliferation marker Ki-67 (MIB-1 antibody) and p53 should be applied to clarify the dignity and the clinical behavior. For the distinction between mucinous adenocarcinomas of the endocervix and those of the endometrium p16 may be a helpful additional marker (see p. 163).

2.4 Interpretation of Endometrial Specimens

Since the endometrial biopsy may contain admixtures of small pieces from various endometrial layers, its interpretation is more difficult than that of intact endometrium received with a surgically removed uterus. Only the functional layer is of diagnostic value in the recognition of functional disturbances. On the other hand, early adenomatous or carcinomatous changes may best be detected in the basal layer. The isthmus mucosa is unsuited for functional diagnosis; it may give a false impression of atrophy or functional deficiency (for differential diagnoses, see p. 43). Regions of necrosis in curettings may have various causes; they should always
be reported. Myomatous proliferations may indicate the presence of submucous leiomyomas as the cause of functional bleeding. If the endometrial biopsy contains only endocervical mucosa, the gynecologist may not have reached the endometrial cavity for various reasons, e.g., stenosis of the isthmus or endocervical canal. If fragments of tissue not occurring in the uterine cavity are found in the endometrial biopsy, e.g., fatty tissue, this finding should raise strong suspicions, if not being indicative, of a perforation of the uterine wall and should therefore always be reported (for further details, see Dallenbach-Hellweg 1987).

Artificial changes that should not be misinterpreted may be produced, e.g. when endometrial glands are squeezed by handling the curettage fragments and their lining epithelium intussuscepts to lie within the glandular lumen. Severe freezing artifacts occur when endometrial tissue in formalin solution is allowed to freeze slowly, as may happen when it is sent by mail in winter (Fig. 2.1).

In addition to a histopathologic diagnosis, the pathologist should always try to answer all questions raised by the gynecologist. In postmenopausal patients, there are generally two main reasons for the gynecologist to perform a curettage: either postmenopausal bleeding or a suspicious sonogram. Accordingly, he should explain why the patient was bleeding (e.g., polyps, hyperplasia, spotting from vascular breakage of atrophic vessels, or focal differences in receptor content). If the gynecologist has performed the curettage because of a suspicious sonogram with thickened endometrium and the endometrial specimen obtained is very small, it may not be representative of the lesion. The pathologist should discuss the discrepancy with the gynecologist and encourage him to repeat the curettage if the sonographic lesion is still present.

![Fig. 2.1 Freezing artifact of the endometrium. Irregular empty spaces produced by ice crystals separate glands and push stromal cells aside. These spaces have no cellular lining (from Dallenbach-Hellweg 1987)]
Before puberty, the endometrium is resting. The first cycles at the onset of puberty are anovulatory, resulting in deficient or irregular proliferation (see p. 63).

3.1 Normal Menstrual Cycle

The normal menstrual cycle consists of the proliferative and secretory phases, menstruation, and regeneration.

3.1.1 Proliferative Phase

The proliferative phase usually lasts 14 days, but under physiologic conditions may fluctuate between 10 and 20 days. It is therefore impossible to distinguish each day during this phase. Consequently, it is subdivided only into the early, middle, and late proliferative phases. For clinical purposes this subdivision suffices, since the relevant functional changes become evident only in the secretory phase.

Early Proliferative Phase

In the early proliferative phase (Figs. 3.1–3.3), the endometrium is low with sparse, narrow, and straight glands evenly distributed in a loose stroma of spindle-shaped cells. The glandular epithelial cells are low columnar cells; they contain small, rounded, or oval-shaped chromatin-dense nuclei in a sparse cytoplasm. The stromal cells are poorly differentiated and of equal size with small, dense nuclei in a scanty cytoplasm. Nucleoli are inconspicuous and mitoses are very rare. The cells are surrounded by a firm reticulin network. Spiral arterioles are undeveloped. The surface epithelium is flat and still regenerating.
Fig. 3.1 Early proliferative phase. H & E, ×25

Fig. 3.2 Early proliferative phase. H & E, ×100
3.1 Normal Menstrual Cycle

Clinical Possibilities. (a) fourth to seventh day of a normal menstrual cycle; (b) anovulatory cycle with follicular insufficiency; (c) deficient proliferation, if the patient is beyond the seventh day of her cycle, or is postmenopausal, with continuing, but deficient estrogen production or replacement therapy.

Distinction is possible only by precise statement of the day of the menstrual cycle and by recording (in anovulatory cycles) the basal body temperature curve. Distinction on morphologic grounds alone is not possible.

Midproliferative Phase

In the midproliferative phase (Figs. 3.4, 3.5), there is beginning tortuosity of glands due to their increase in length, which exceeds the growth in height of the endometrium. The glandular epithelial cells are tall columnar cells and contain slightly enlarged oval-shaped, chromatin-rich nuclei in a dense, sparse cytoplasm rich in RNA. Nucleoli are prominent, and mitoses frequent. The spindle-shaped stromal cells are still poorly differentiated, but rich in DNA and RNA with occasional mitoses and separated by interstitial edema (Harkin 1956). Spiral arterioles are not seen. The surface epithelium has increased in height and is now low columnar.

Clinical Possibilities. (a) Eighth to 11th day of a normal menstrual cycle; (b) anovulatory cycle.
Fig. 3.4 Midproliferative phase. H & E, ×25

Fig. 3.5 Midproliferative phase. H & E, ×100
3.1 Normal Menstrual Cycle

Distinction is possible only by precise statement of the day of the menstrual cycle and by recording (in anovulatory cycles) the basal body temperature curve.

Late Proliferative Phase

In the late proliferative phase (Figs. 3.6, 3.7), there is marked tortuosity of glands, which are lined by tall columnar epithelial cells piled up against one another with their nuclei at different levels, giving a pseudostratified appearance. The nuclei are enlarged still further, rich in DNA, and more elongated or oval shaped. Nucleoli are quite prominent, and mitoses are frequent. The cytoplasm is still sparse and poorly differentiated, but rich in RNA. The stromal cells are further enlarged and contain increased amounts of DNA and RNA. Stromal mitoses are frequent. The cells are still of uniform size and without signs of differentiation. The stromal edema has subsided. The reticulin network is dense. Spiral arterioles are still absent. The general height may be slightly lower than that of the midproliferative phase due to decrease of edema. The surface epithelium is distinctly columnar.

Clinical Possibilities. (a) Twelfth–14th day of a normal menstrual cycle; (b) anovulatory cycle with follicular persistence.

Distinction is possible only by precise statement of the day of the menstrual cycle and by recording (in anovulatory cycles) the basal body temperature curve.

Fig. 3.6 Late proliferative phase. H & E, ×100
In the presence of a normally developing and involuting corpus luteum, the secretory phase lasts precisely 14 days (±1 day; Rock and Hertig 1944). If the deviation from this limit is more than 2 days, a functional disturbance should be diagnosed. The daily changes induced in the endometrium by the influence of progesterone enable us to date the endometrium (Noyes et al. 1950; Moricard 1954; Philippe et al. 1965; Dallenbach and Dallenbach-Hellweg 1968; see Fig. 3.8). Because the glandular epithelium reacts more quickly to the influence of progesterone than do the stromal cells, histologic dating is based mainly on the changes of the glandular epithelium during the first week of the secretory phase and on the changes in the stromal cells during the second week. The histologic signs induced in the endometrium by hormones are never uniform. Differences are related to various factors, such as local blood supply, cellular nutrition, and various metabolic factors, e.g., receptors for estrogen and progesterone. For dating the endometrium, those regions showing the most advanced changes are relevant.

The first day after ovulation is morphologically mute and therefore designated as interphase because it takes 36–48 h before changes induced by the progesterone secretion can be detected with certainty under the light microscope. Sporadic vacuoles appear in some of the glandular cells even immediately before ovulation, but are unreliable as definite signs of ovulation.
On the second day after ovulation (Figs. 3.9, 3.10) numerous basal glycogen vacuoles have appeared in the glandular epithelium (at least in 50% of the glandular cells). They have pushed the nucleus towards the lumen. At their formation the previously pseudostratified appearance of the nuclei disappears; the nuclei again form a single row. They are still
Fig. 3.9  Second day after ovulation. H & E, ×100

Fig. 3.10  Second day after ovulation. H & E, ×250
3.1 Normal Menstrual Cycle

oval shaped and chromatin dense. With this new arrangement of the nuclei, the glands become increasingly tortuous and enlarge their surfaces at the glandular lumen. Basal vacuoles, however, fail to appear in the superficial portions of the gland where it merges with the surface epithelium (Fig. 3.9). The surface epithelium of the endometrium remains tall, with no signs of basal secretion. No remarkable changes can be detected in the stroma when compared with those of the late proliferative phase.

Based on morphology, ovulation can be confirmed when distinct basal vacuoles have appeared in most of the glandular epithelial cells.

The third day after ovulation (Figs. 3.11, 3.12) shows enlarged basal vacuoles now occupying all the glandular epithelial cells. This secretion has pushed all the nuclei toward the apical end of the cell, where they form a uniform row around the lumen. The nuclei remain dense and rich in chromatin, and only a few mitoses are seen. The cytoplasm still contains abundant RNA.

On the fourth day after ovulation (Figs. 3.13–3.15) the nuclei of the glandular epithelial cells have become distinctly rounded and less chromatin dense. A few of them start to return to the base of their cells, while tiny amounts of glycogen move towards the lumen.

A small rim of mucopolysaccharides can be observed at the luminal margin of the epithelial cells, but only with special stains (Fig. 3.15). This rim is characteristic of the fourth day after ovulation.

On the fifth day after ovulation (Fig. 3.16) the majority of the nuclei in the glandular epithelial cells have returned to the base of their cells and are distinctly rounded. The glycogen has moved or is moving towards the lumen of the cells on both sides of the nucleus,

Fig. 3.11 Third day after ovulation. H & E, ×100
Fig. 3.12  Third day after ovulation. H & E, ×250

Fig. 3.13  Fourth day after ovulation. H & E, ×25
3.1 Normal Menstrual Cycle

Fig. 3.14 Fourth day after ovulation. H & E, ×100

Fig. 3.15 Fourth day after ovulation. Pentachrome reaction, ×350
and secretion can be seen beginning as tiny globular caps at the free margin of the cells. The movement of the glycogen can best be followed with a PAS stain. Since the nuclei are now vesicular and pale upon staining, they can be easily distinguished from the dense, elongated, basally located nuclei of the cells before the glycogen vacuoles appeared. The nucleoli have now become greatly enlarged.

The sixth day after ovulation (Figs. 3.17, 3.18) shows a dilatation of the glandular lumina produced by the continued secretion of glycogen. All epithelial nuclei have now returned to the base of the cells. The glandular cells have decreased in height and their luminal margins appear shredded and hazy due to the excessive apocrine secretion. The RNA content of the cytoplasm is decreased.

On the seventh day after ovulation (Figs. 3.19–3.21) the dilated glandular lumen is filled with glycogen, which is partly dissolved in H & E-stained preparations, giving the lumen a false, empty appearance. As a sign of active secretion, the apical ends of the glandular cells appear frayed. No new glycogen is secreted from the cell base. The first stromal reaction after ovulation has occurred and becomes apparent on this day; there is a patchy reaccumulation of stromal edema.

On the eighth day after ovulation (Figs. 3.22–3.24) there is virtually no change in the glandular epithelium compared with the preceding day; however, stromal edema has reached its maximum for the secretory phase. It is now diffuse and quite intense, with the stromal cells widely separated from one another. These are still spindle shaped, but somewhat enlarged. Their differentiation becomes even more apparent from this day on (Fig. 3.24). The overall height of the endometrium has increased.
3.1 Normal Menstrual Cycle

Fig. 3.17 Sixth day after ovulation. H & E, ×100

Fig. 3.18 Sixth day after ovulation. H & E, ×250
Fig. 3.19 Seventh day after ovulation. H & E, ×100

Fig. 3.20 Seventh day after ovulation. H & E, ×250