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Jeffcoate's
Principles of Gynaecology



Editor
Muralidhar V Pai

10th Edition

JAYPEE

Contents

1. A Clinical Approach to Gynaecology 1

Shripad Hebbar, Divyashree Bhat, Muralidhar V Pai

- Psychosomatic and Sociological Aspects of Gynaecology *1*
- Clinical Methods *2*
- Physical Examination *6*
- Special Tests and Accessory Aids to Diagnosis *12*
- Endometrial Sampling Procedures *13*
- Transvaginal Sonography *16*
- Transrectal Sonography *16*
- Colour Doppler *16*
- Endoscopy *17*
- Laparoscopy *17*
- Hysteroscopy *19*
- Robotic Surgery *20*
- Computed Tomography *21*
- Magnetic Resonance Imaging *22*
- Positron Emission Tomography Scan *23*

2. Anatomy 26

Argyia Desouza, Muralidhar V Pai

- Vulva *26*
- Vagina *30*
- Uterus *35*
- Fallopian Tubes *40*
- Ovary *41*
- Urethra and Bladder *43*
- Ureter *47*
- Sigmoid Colon *48*
- Rectum and Anus *48*
- Pelvic Peritoneum and Ligaments *49*
- Pelvic Musculature *51*
- Pelvic Fascia and Cellular Tissue *52*
- The Supports of the Genital Organs *52*
- Blood Vessels of the Pelvis *53*
- Lymphatic Drainage *57*
- Innervation of Pelvic Organs *57*

3. Ovarian Functions 61

Priya SP Patil, Muralidhar V Pai

- Production of OVA *62*
- Ovarian Hormones *75*
- Pituitary Hormones *81*

- Pituitary–Hypothalamic Relations *82*
- Pituitary–Ovarian Relations (Control of Ovulation) *85*
- Hormone Levels and Assays *86*

4. Menstruation and Other Cyclical Phenomena 88

Deepa Reddy, Muralidhar V Pai

- Normal Menstrual Cycle *88*
- Endometrial Cycle *88*
- Correlation of Endometrial and Ovarian Cycle *92*
- Uterine Bleeding *94*
- Myometrial Cycle *98*
- Cyclical Changes in the Tube *98*
- Cervical Cycle *99*
- Vaginal Cycle *99*
- Cyclical, Metabolic, Vascular and Psychological Changes *99*

5. Clinical Aspects of Menstruation and Ovulation 101

Nithesh Prabhu, Muralidhar V Pai

- Menstruation *101*
- The Menopause and the Climacteric *106*
- Abnormal Menopause *119*
- Ovulation *120*

6. Puberty and Adolescent Gynaecology 132

Roopa PS, Muralidhar V Pai

- Puberty and Adolescence *132*
- Abnormalities of Puberty and Adolescence *136*
- Prolactin *144*
- Puberty Menorrhagia *146*

7. Conception 150

Akhila Vasudeva, Muralidhar V Pai

- Process *150*
- Early Development of the Ovum *152*
- Formation of Foetus and Membranes *155*
- Hormonal Control of Early Pregnancy *158*

8. Spontaneous Abortions (Including Recurrent Loss) 162

Akhila Vasudeva, Muralidhar V Pai

- Spontaneous Abortions *162*
- Recurrent Early Pregnancy Loss *169*

9. Ectopic Pregnancy and Pregnancy of Unknown Location 179

Shripad Hebbar, Kavisha Bhat, Muralidhar V Pai

Ectopic Pregnancy 179

- Frequency of Ectopic Pregnancy *179*
- Sites of Ectopic Pregnancy *179*
- Aetiology of Ectopic Pregnancy *180*
- Ectopic Pregnancy in Fallopian Tubes *181*
- Management and Treatment Options of Ectopic Pregnancy *192*

- Expectant Management *193*
- Medical Management *194*
- Surgical Management *194*
- Complications of Ectopic Pregnancy *200*
- Ovarian Pregnancy *200*
- Cornual Pregnancy *201*
- Cervical Pregnancy *202*
- Caesarean Scar Pregnancy *202*
- Abdominal Pregnancy *203*
- Interstitial Pregnancy *204*
- Intraligamentary Pregnancy *205*

Pregnancy of Unknown Location 205
- Outcome of Ectopic Pregnancy *206*
- Diagnosis *207*
- Management of Pregnancy of Unknown Location *207*

10. Gestational Trophoblastic Disease 214

Priya Ballal K, Suprajna Shetty
- Types of Tumours *214*
- Aetiology *215*
- Pathology *215*
- Clinical Features *218*
- Evaluation and Treatment *218*
- Postmolar Surveillance *219*
- Persistent Trophoblastic Disease *219*
- Classification and Staging of Gestational Trophoblastic Disease *221*
- Treatment of Gestational Trophoblastic Tumours *222*
- Management of Metastasis *223*
- Postchemotherapy Surveillance for High-risk Gestational Trophoblastic Tumours *223*

11. Breast Function and its Disorders 225

Shripad Hebbar, Rajani Upadhyaya, Muralidhar V Pai
- Breast Development *225*
- Developmental Anomalies of the Breast *227*
- Breastfeeding and Lactation *231*
- Endocrine Disorders (Galactorrhoea and Breast Atrophy) *232*
- Benign Breast Condition *237*
- Screening for Breast Diseases *240*
- Benign Breast Disease *241*
- Breast Cancer *245*
- Epidemiology *245*

12. Development of the Urogenital System 252

Latha Kharka, Muralidhar V Pai
- Gonad *253*
- Wolffian System *253*
- Müllerian Ducts *255*

- Mesenteries and Ligaments *255*
- Development of the Vagina, Bladder and Urethra *257*
- Development of the Vulva *257*

13. Malformations and Maldevelopments of the Genital Tract 259

Priyankur Roy, Muralidhar V Pai

- Causes of Malformations *259*
- Cytogenetics *260*
- Uterine Anomalies *263*
- Ovary *276*
- Fallopian Tube *277*
- Uterus *277*
- Conical Cervix and Pinhole Ostium *278*
- Vagina *279*
- Vulva *282*
- Errors Arising in Connection with the Cloaca *283*
- Malformations of the Urinary Tract *284*

14. Disorders of Sex Differentiation 287

Rajeshwari G Bhat, Muralidhar V Pai

- Factors Responsible for Normal Sexual Patterns *287*
- Turner Syndrome 45, XO *292*
- Triple X Syndrome (47, XXX) (From Old Jeffcoate's) *293*
- Klinefelter Syndrome (47, XXY) (From Old Jeffcoate's) *294*
- YY Syndrome *294*
- Disorders of Sex Development with Normal Sex Chromosomes *295*
- Approach to a Neonate with Atypical Genitalia *300*
- Intersex Developing after Birth *303*

15. Injuries 312

Kavitha D Nayak, Muralidhar V Pai

- Different Types of Injuries *312*

16. Female Genital Mutilation 318

Rajeshwari G Bhat, Muralidhar V Pai

- Classification of Female Genital Mutilation *318*
- Prevalence of Female Genital Mutilation *318*
- Indications *320*
- Complications *320*
- Diagnosis *322*
- Management in Obstetric and Gynaecological Practice *322*
- Management of Short-term Complications *322*
- Management during the Antenatal Period *323*
- Management during the Intrapartum Period *323*
- Postnatal Care *324*
- Eradication *324*

17. Pelvic Organ Prolapse 327

Rajeshwari G Bhat, Muralidhar V Pai

- Uterine and Vaginal Prolapse *327*
- Prolapse of the Ovaries *347*

18. Other Displacements of the Uterus 348
Jyothi Shetty, Muralidhar V Pai
- Upward Displacement of the Uterus *348*
- Lateral Displacement of the Uterus *348*
- Forward Displacement of the Uterus *348*
- Backward Displacement of the Uterus *349*
- Retroverted Gravid Uterus *353*
- Inversion of the Uterus *355*
- Chronic Inversion *356*

19. Torsion of Pelvic Organs 359
Latha V Khakra, Muralidhar V Pai
- Torsion in Normal and Abnormal Uterus *359*
- Aetiology *361*
- Differential Diagnosis *362*
- Treatment *363*

20. Infections Including Sexually Transmitted Diseases 364
Jayaraman Nambiar M, Muralidhar V Pai
- Natural Defences of the Genital Tract *364*
- Variations in the Efficiency of Defence Mechanisms *364*
- Types of Infecting Organisms *365*
- Sexually Transmitted Diseases *366*
- Genital Tuberculosis *382*
- Sarcoidosis *389*
- Actinomycosis *389*
- Schistosomiasis (Bilharzia) *390*
- Amoebiasis *390*

21. Infections as They Affect Individual Organs 391
Jayaraman Nambiar M, Muralidhar V Pai
- Vulvitis *391*
- Bartholinitis *395*
- Vaginitis *396*
- Cervicitis *404*
- Endometritis *406*
- Metritis *407*
- Salpingo-Oophoritis *407*
- Oophoritis *413*
- Pelvic Peritonitis *413*
- Pelvic Cellulitis *414*
- Chronic Cellulitis *415*
- Pelvic Inflammatory Disease *416*
- Suppurative Thrombophlebitis of the Pelvic Veins *417*

22. Genital Tuberculosis 419
Pesona Grace Lucksom, Latha V Kharka, Muralidhar V Pai
- Clinical Profile *419*
- Classification *420*

- Diagnosis *421*
- Management *430*
- Pathology of Pelvic Organs *431*

23. Endometriosis and Chronic Pelvic Pain 433

Anjali Mundkur, Muralidhar V Pai

Endometriosis and Allied States 433

- Endometriosis and Adenomyosis *433*
- Adenomyosis *454*
- Endosalpingiosis *455*
- Cervical Endometriosis *455*
- Causes in the Genital Tract *456*
- Extragenital Causes *457*
- Management and Treatment *459*

Chronic Pelvic Pain 459

- General Considerations *459*
- Step-wise Evaluation of Chronic Pelvic Pain *460*
- Examination *460*
- Diagnostic Investigations *460*
- Management *460*

24. Polycystic Ovary Syndrome 463

Meghna A Nayak

- Oligomenorrhoea and Anovulation: Definition *464*
- Hyperandrogenism (Clinical and/or Biochemical) *464*
- Polycystic Ovaries on Ultrasound or Elevated Serum Anti-Müllerian Hormone *464*
- Pathophysiology *464*
- Ovarian and Other Endocrine Aspects of Polycystic Ovary Syndrome *465*
- Metabolic Component of Polycystic Ovary Syndrome *467*
- Pathology *469*
- Clinical, Biochemical and Metabolic Features of Polycystic Ovary Syndrome *469*
- Ultrasonographic Examination *470*
- Puberty and Polycystic Ovary Syndrome *471*
- Management *471*
- Infertility *473*
- Weight Loss *473*
- Summaries *476*

25. Hirsutism 478

Deepak Goenka, Shamantaka Somayaji, Muralidhar V Pai

- Types of Hair *478*
- Physiology of Hair Growth *478*
- Androgens Physiology *479*
- Aetiology *480*
- Diagnosis of Hyperandrogenism *482*
- Laboratory Evaluation *482*
- Treatment *484*

26. Epithelial Abnormalities of the Genital Tract 487
Shyamala Guruvare, Muralidhar V Pai
- Senile Atrophy *487*
- Lichen Sclerosus *488*
- Aetiology *489*
- Symptoms *490*
- Diagnosis *490*
- Treatment *490*
- Vulvar and Oral Epithelial Abnormalities *492*
- Vulvar Intraepithelial Neoplasia *492*
- Vagina *496*
- Cervix *497*
- Uterine Corpus *509*
- Fallopian Tube *512*

Annexures 514
- Annexure 1: Flowcharts for Screen-and-Treat Strategies (Negative or Unknown Human Immunodeficiency Virus Status) *514*
- Annexure 2: Flowcharts for Screen-and-Treat Strategies (Human Immunodeficiency Virus-Positive Status or Unknown Human Immunodeficiency Virus Status in Areas with High Endemic Human Immunodeficiency Virus Infection) *518*

27. Genital Cancers 522
Shyamala Guruvare, Muralidhar V Pai
- Significance of Genital Cancer *522*
- Treatment and Results *523*
- Prevention of Pelvic Cancer *523*
- Early Diagnosis *524*
- General Management of the Cancer Patient *528*
- Management of Advanced Pelvic Cancer *529*

28. Tumours of the Vulva 536
Rekha Upadhya, Muralidhar V Pai
- Swellings of the Vulva *536*
- Varicose Veins *537*
- Oedema *537*
- Retention Cysts *537*
- Benign Neoplasms *538*
- Malignant Neoplasms of Vulva *542*
- Tumours of Bartholin's Gland *550*
- Urethral Tumours *551*
- Tumours of the Inguinal Canal *555*

29. Tumours of the Vagina 557
Rekha Upadhya, Muralidhar V Pai
- Swellings of the Vagina *557*
- Vaginal Cysts *557*
- Benign Neoplasms *558*
- Malignant Neoplasms *563*

30. Tumours of the Cervix Uteri 570
Priya Ganeshkumar
- Benign Neoplasms of the Cervix *570*
- Benign Tumours of the Cervix *570*
- Carcinoma of the Cervix *572*
- Screening for Cervical Cancer *576*
- Other Malignant Tumours of the Cervix *595*

31. Tumours of the Corpus Uteri 596
Saritha Shamsunder, Sufian Zaheer, Shivani Verma
- Enlargement of the Uterus *596*
- Uterine Polyps *597*
- Benign Neoplasms *597*
- Malignant Neoplasms *616*
- Molecular Classification *624*
- Adjuvant Treatment *628*
- Endometrial Stromal Tumours *632*

32. Adenomyosis 636
Anjali Mundkur, Muralidhar V Pai
- Associated Pathology *636*
- Aetiology *636*
- Diagnosis *637*
- Differential Diagnosis *638*
- Management *638*
- Complications *640*

33. Tumours of the Fallopian Tubes 642
Sanjay Kumar KR, Muralidhar V Pai
- Benign Neoplasms *642*
- Primary Malignant Neoplasms *642*
- Secondary Malignant Neoplasms *648*
- Prevention of Fallopian Tube Cancer *648*
- Recurrent Disease *648*
- Coping with Disease *648*

34. Tumours of the Pelvic Ligaments 649
Sanjay Kumar KR, Muralidhar V Pai
- Cysts of the Broad Ligament and Associated Structures *649*
- Neoplasms of the Pelvic Ligaments and Connective Tissues *650*
- Neoplasms of the Peritoneum *652*

35. Tumours of the Ovary 654
Pralhad Kushtagi
- Clinical Diagnosis of Ovarian Tumours *654*
- Complications of Ovarian Tumours *655*
- Features Suggestive of Malignancy in Ovarian Tumour *656*
- Pathology of Ovarian Tumours *658*
- Non-neoplastic Ovarian 'Tumours' *658*

- Neoplastic Ovarian Tumours *663*
- Staging *668*
- Nonepithelial Ovarian Tumours *674*
- Germ Cell Tumours *677*
- Treatment of Malignant Germ Cell Tumours *681*

36. Chemotherapy in Gynaecological Malignancies 686

Sourjya Banerjee, Pratik Kumar Chatterjee

- Chemotherapy and the Cell Cycle *686*
- Therapeutic Agents Used in the Treatment of Gynaecological Cancer *687*
- Chemotherapy Toxicity *688*
- Systemic Therapy in Gynaecological Malignancies *688*
- Assessment of Response to Chemotherapy *693*

37. Radiotherapy in Gynaecological Malignancies 695

Sourjya Banerjee, Pratik Kumar Chatterjee

- Biological basis of Radiotherapy Treatment *695*
- Radiation Dosage *695*
- Therapeutic Ratio *696*
- Radiotherapy Machines *696*
- Brachytherapy *696*
- Radiotherapy in Carcinoma of the Cervix *696*
- Radiotherapy in Endometrial Cancer *697*
- Radiotherapy in Vulvar Cancer *698*
- Brachytherapy in Carcinoma of the Cervix *699*
- Chemoradiation in Locally Advanced Carcinoma of the Cervix *700*
- Radiotherapy in Vaginal Cancer *700*

38. Immunotherapy in Obstetrics and Gynaecology 701

Pralhad Kushtagi

- Immune System—Some Basics *701*
- Immunotherapy in Gynaecologic Cancers *702*
- Immunotherapy in Reproductive Medicine *709*
- Immunotherapy in Other Situations *714*
- Immunotherapy in Pregnancy *714*

39. Amenorrhoea, Hypomenorrhoea, and Oligomenorrhoea 718

Pratik Tambe, Muralidhar V Pai

- Amenorrhoea *718*
- Aetiology *718*
- Treatment *735*
- Hypomenorrhoea *738*
- Oligomenorrhoea *741*

40. Abnormal and Excessive Uterine Bleeding 743

Jayaraman Nambiar M, Muralidhar V Pai

- Terminology in Abnormal Uterine Bleeding *743*
- Palm-Coein Classification of Abnormal Uterine Bleeding *744*
- Causes of Abnormal Uterine Bleeding—Palm-Coein *744*

- Wamsteker Classification *746*
- STEPW Classification of Myoma *746*
- Approach to a Patient with Abnormal Uterine Bleeding *749*
- Special Clinical Types of Bleeding *750*

41. Dysmenorrhoea 754

Brihathi Pranavi Dasari, Muralidhar V Pai

- Primary Dysmenorrhoea *754*
- Secondary Dysmenorrhoea *758*
- Membranous Dysmenorrhoea *760*
- Other Conditions Simulating Dysmenorrhoea *760*

42. Premenstrual Syndrome and Other Menstrual Phenomena 762

Kaushik Naik V, Muralidhar V Pai

- Clinical Features *762*
- Natural History *762*
- Laboratory Findings *763*
- Aetiology *763*
- Differential Diagnosis *764*
- Treatment *764*
- Menstrual Migraine *766*
- Premenstrual Mastalgia *767*
- Recurrent (Cyclical) Buccal and Vulvar Ulceration *768*
- Pelvic Allergy *769*
- Vicarious Menstruation *770*
- Cyclical Haemothorax and Pneumothorax *770*
- Menstrual Epilepsy *771*

43. Hormone Therapy in Gynaecology 772

Vidyashree G Poojari, Muralidhar V Pai

- Oestrogens *772*
- Antioestrogens *778*
- Progestogens *779*
- Antiprogestogens *782*
- Androgens *783*
- Antiandrogens *785*
- Types of Gonadotrophins *785*
- Antigonadotrophins *787*
- Hypothalamic Hormones *790*

44. Vaginal Discharge 792

Sujatha BS, Muralidhar V Pai

- General Considerations *792*
- Causes of Physiological Discharge *792*
- Prognosis *797*
- Risk Factors *797*
- Diagnosis *797*
- Management *799*
- Therapy *800*
- Prevention *802*

45. Pruritus Vulvae and Vulvodynia 804
Sujatha BS, Muralidhar V Pai
- Definition and Incidence *804*
- Pathophysiology *804*
- Natural Defence Mechanisms *804*
- Aetiological Classification of Pruritus Vulvae *805*
- Causes of Pruritus According to Age Groups *805*
- Evaluation of Patient with Pruritus *805*
- Investigations *805*
- General Measures for Women with Pruritus *805*
- Differential Diagnosis of Pruritus *807*
- Treatment *812*
- Results *813*
- Vulvodynia *813*
- Management *815*

46. Problems of Sex and Marriage 818
Niraj Jadav, Poonam Mishra
- Physical Sex—Coitus *819*
- Consummation of Marriage *819*
- Annulment of Marriage *820*
- Unconsummated Marriages *820*
- Coital Difficulties in the Male *821*
- Homosexuality *825*
- Marriage and Sexual Problems in Females: Understanding, Challenges and Solutions *826*
- Female Sexual Interest/Arousal Disorder *826*
- Female Orgasmic Disorder *829*
- Genito-Pelvic Pain/Penetration Disorder *832*
- Vaginismus *833*

47. Male and Female Infertility Overview 840
Mahesh C Koregal, Muralidhar V Pai, Soumya Mahesh Koregal
- Infertility *840*
- Frequency *840*
- Concept of Fertility *840*
- Causes of Infertility *841*
- Investigation of Infertility *846*
- Treatment *858*

48. Assisted Reproductive Technology 869
Mahesh C Koregol, Soumya Mahesh Koregol, Muralidhar V Pai
- In Vitro Fertilisation and Embryo Transfer *869*
- Controlled Ovarian Hyperstimulation *869*
- Oocyte Retrieval *870*
- Insemination *870*
- Embryo Transfer *872*
- Outcome *872*
- Cryopreservation *873*
- Oocyte Donation *873*

- Embryo Donation *874*
- Surrogacy *874*
- Intrauterine Insemination *875*
- Donor Insemination *877*
- Other Assisted Reproductive Technology Methods *878*
- Results of Treating Infertility *879*
- Dangers of Investigating and Treating Infertility *880*
- Complications of Assisted Reproductive Technology *880*
- Adoption *881*

49. Instruments in Gynaecological Procedures 884

Shripad Hebbar, Pooja HN, Muralidhar V Pai

- Instruments *884*
- Gynaecological Procedures *892*

50. Ultrasonography in Gynaecology 896

Priti Venkatesh

- Indications for Ultrasound in Gynaecology *896*
- Techniques *896*
- Normal Female Pelvic Anatomy *897*
- Uterus *897*
- Myometrium *911*
- Adenomyosis *919*
- Endometrium *926*
- Cervix *939*
- Adnexa *943*
- Polycystic Ovary *953*
- Ovarian Malignant Lesions *953*
- Fallopian Tubes *959*
- Endometriosis *960*
- Ultrasound Imaging in Gynaec Emergencies *962*

51. Ultrasound in Infertility 970

Vidyashree G Poojari, Muralidhar V Pai

- Method *971*
- Uterine Abnormalities *971*
- Cycle Assessment Consisting of Baseline Scan, Preovulatory Scan and Secretory Scan *991*

52. Ultrasound in Menopause 1001

Priti Venkatesh

- Normal Uterus in Menopause *1001*
- Ultrasound Evaluation of the Endometrium in Postmenopausal Women *1002*
- Postmenopausal Bleeding *1002*
- Hormone Replacement Therapy and Tamoxifen *1003*
- Incidental Finding of Thick Endometrium *1005*
- Normal Atrophic Ovary *1006*
- Fluid in Pouch of Douglas *1006*
- Ovary and Ovarian Cancer Screening *1006*

53. Robotic Surgery 1011
Rani Akhil Bhat
- About da Vinci Surgical System *1011*
- Distinguishing Laparoscopy from Robotic Surgery: Unveiling Contrasts in Minimally Invasive Techniques *1014*
- Robotic Surgery Considerations for Patients *1016*
- Robotic Surgery Considerations for Doctors *1016*
- Introduction of da Vinci Robotic in Gynaecology *1016*
- Revolutionising Medicine: The Future Horizons of Robotic Surgery Innovation *1018*

54. Endoscopic Surgery in Gynaecology 1019
Vidya Bhat, Mythreyi Kadambi

Laparoscopic Surgery 1019
- Surgical Equipment *1019*
- Indications *1026*
- Contraindications to Laparoscopy *1030*
- Preoperative Preparation *1030*
- Surgical Steps *1032*
- Postoperative Care *1034*
- Advantages *1035*
- Disadvantages *1035*
- Complications *1035*

Hysteroscopic Surgery 1037
- Surgical Equipment *1037*
- Indications *1041*
- Preoperative Preparation *1044*
- Surgical Steps *1044*
- Postoperative Care *1046*
- Advantages *1047*
- Disadvantages *1048*
- Complications *1048*
- Conclusion *1052*
- Hysteroscopic Resectoscope *1052*
- Slim Resectoscope *1052*
- Hysteroscopic Morcellator *1052*

Laparoendoscopic Single-site Surgery 1053
- Surgical Equipment *1053*
- Indications *1055*
- Laparoendoscopic Single-site Surgical Techniques *1055*
- Advantages *1057*
- Complications *1057*
- Conclusion *1057*
- Robotic Surgery *1057*

55. Contraception 1061
Shobha N Gudi
- General Consideration *1061*
- Epidemiology *1061*
- Impact of Contraception Throughout the World *1062*
- Efficacy of Contraception *1062*
- Indications for Contraception *1063*
- Criteria for the Use of Various Contraceptive Methods *1064*
- Contraceptive Methods *1064*
- Natural Family Planning Method *1064*
- Barrier Methods *1068*
- Intrauterine Contraceptive Devices *1075*
- Hormonal Contraception Methods *1092*
- Progestogen-only Contraception *1105*
- Emergency Postcoital Contraception (Morning After Pills) *1115*
- Other Methods of Contraception *1117*
- Choice of Methods *1123*
- Contraception Choice for Older Women *1124*
- Contraception and Litigation *1124*

56. Sterilisation and Termination of Pregnancy 1128
Priya Ballal K, Nikil Shetty, Ektha M Shetty
- Sterilisation *1128*
- Female Sterilisation *1129*
- Male Sterilisation *1131*
- Termination of Pregnancy *1133*

57. Urinary Incontinence 1140
Shripad Hebbar, Muralidhar V Pai, Chandra Mithila
- Bladder Dysfunction *1140*
- Urethral Sphincter Dysfunction *1143*
- Investigation of Urinary Problems *1145*
- Treatment of Urinary Problems *1148*
- Incontinence of Urine *1149*
- Management *1153*
- Overview of Surgical Management *1155*
- Enuresis *1163*
- Urinary Retention and Difficulty in Micturition *1164*

58. Urinary Tract Infection 1170
Shripad Hebbar, Chandra Mithila, Muralidhar V Pai
- Aetiology *1173*
- Diagnosis *1173*
- Investigations *1173*
- Management *1174*
- Treatment *1175*
- Complications *1177*
- Clinical Pearls *1178*
- Evidence-Based Medicine *1179*

59. Menopause 1181
Shyjus P
- History *1181*
- Definitions and Staging of Menopause *1181*
- Physiology of Menopause *1183*
- Problems Associated with Menopause *1185*
- Effect of Oestrogen Deficiency *1186*
- Menstrual Problems *1195*
- Management of Menopause *1196*
- Symptoms of Menopause *1196*
- Various Types of Hormonal and Nonhormonal Pharmacological Agents Available *1198*
- Use of Progesterones in Menopausal Hormone Therapy *1200*
- Hormone Therapy in Special Circumstances *1201*
- Androgens in Menopause *1202*

60. Hysterectomy and its Aftermath 1204
Shashikala K Bhat, Muralidhar V Pai
- History *1204*
- Types of Hysterectomy *1204*
- Preoperative Preparation *1205*
- Abdominal Hysterectomy—Operative Steps *1207*
- Minimally Invasive Hysterectomy Procedures *1208*
- Total Laparoscopic Hysterectomy in Special Situations *1212*
- Vaginal Hysterectomy *1212*
- Complications of Hysterectomy *1214*

61. Conditions of the Lower Intestinal Tract 1216
Srirama Bhat M
- Rectal Prolapse *1216*
- Incontinence of Faeces and Flatus *1223*
- Diarrhoea *1224*
- Difficult Evacuation *1225*
- Irritable Bowel Syndrome *1226*
- Pruritus Ani *1227*
- Pilonidal Sinus (Jeep Bottom; Driver's Bottom) Pilus—Hair; Nidus—Nest *1228*
- Rectal and Anal Pain *1228*
- External Pile/Haemorrhoids *1234*
- Anorectal Abscess *1234*
- Fistula-In-Ano *1235*
- Proctitis *1238*
- Endometriosis *1238*
- Neoplasm of the Anal Canal *1239*

62. Preoperative and Postoperative Management and Postoperative Complications 1241
Srirama Bhat M
- Fluid and Electrolytes *1241*
- Preoperative Management *1246*
- Postoperative Management *1249*
- Fast-track Protocol *1254*

- Postoperative Examination *1254*
- Postoperative Complications *1255*
- Deep Venous Thrombosis *1262*
- Pulmonary Embolism *1268*
- Pulmonary Complications *1271*

63. Nutrition and Exercise in Women 1274

Vidya Thobbi

Nutrition in Women 1274

- Maternal, Infant and Young Child Nutrition and Sustainable Goals *1275*
- Nutrition Basics *1275*
- Micronutrients *1275*
- Macronutrients *1275*
- Energy *1280*
- Adolescents Nutrition *1280*
- Nutrition During the Preconception Period *1285*
- Nutritional Requirements in Midlife *1285*

Exercise and Physiotherapy in Gynaecology 1287

- Active Muscle Exercises *1287*
- Electrical Stimulation of Pelvic Muscles *1289*
- Short-wave Therapy *1289*
- Infrared Radiation *1290*
- Transcutaneous Electric Nerve Stimulation *1290*
- Ultrasound *1290*

64. Applications of Lasers in Gynaecology 1292

Vidya Bhat, Mythreyi Kadambi

- History *1292*
- Laser Physics *1292*
- Types of Lasers *1293*
- Laser Safety *1294*
- Extraperitoneal Laser Surgery *1294*
- Clinical Applications of Lasers in Intra-abdominal Gynaecological Surgery *1295*
- Other Clinical Applications of Lasers in Gynaecology and Reproductive Medicine *1297*
- Novel Concepts—Lasers in Cosmetic Gynaecology *1298*

65. Genetics in Gynaecology 1300

Krupa Hitesh Shah, Muralidhar V Pai

- Genetics of Gynaecological Cancers *1300*
- Genetics of Common Benign Condition *1302*
- Genetic Testing *1304*
- Genetic Counselling *1304*

66. Artificial Intelligence in Gynaecology 1306

Sonal Panchal, Chaitanya Nagori

- How Artificial Intelligence Works? *1307*
- Artificial Intelligence Applications in Gynaecology *1307*

- Imaging *1308*
- Role of Artificial Intelligence in Benign Gynaecology *1308*
- Role of Artificial Intelligence in Gynaecological Cancers *1309*
- Role of Artificial Intelligence in Gynaecological Surgery *1310*
- Artificial Intelligence in In Vitro Fertilisation *1310*
- Embryology *1311*
- Urogynaecology *1311*
- Drug Research *1312*
- Artificial Intelligence Limitations *1312*
- Challenges *1312*

Index *1315*

4

CHAPTER

Menstruation and Other Cyclical Phenomena

Deepa Reddy, Muralidhar V Pai

Chapter Outline

- Normal Menstrual Cycle
- Endometrial Cycle
- Correlation of Endometrial and Ovarian Cycle
- Uterine Bleeding
- Myometrial Cycle
- Cyclical Changes in the Tube
- Cervical Cycle
- Vaginal Cycle
- Cyclical, Metabolic, Vascular and Psychological Changes

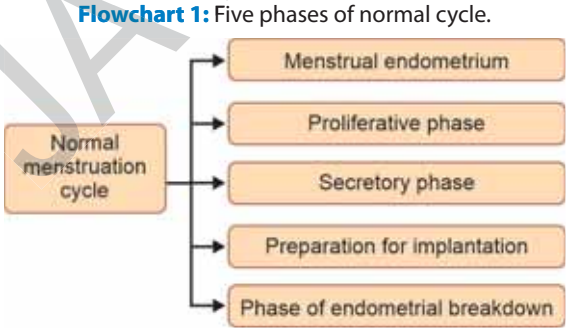
NORMAL MENSTRUAL CYCLE

Menstruation is the periodic and cyclical sloughing of uterine inner lining (endometrium) in response to interactions between hormones produced by the hypothalamus, pituitary and ovaries. A menstrual cycle takes place at approximately 28-day intervals although a normal cycle can be anywhere between 24 and 38 days, with 2–8 days of flow and blood loss of 20–60 mL. Menstruation begins at menarche and stops at menopause.

There are five phases in the normal cycle as mentioned in **Flowchart 1**.

The normal human menstrual cycle can be divided into two segments: (1) The ovarian cycle and (2) the uterine cycle. There is orderly cyclic hormone production accompanied by the simultaneous proliferation of uterine endometrium in the respective segments. The ovarian cycle may be further divided into follicular and luteal phases, whereas the uterine cycle is divided into corresponding proliferative and secretory phases.

Flowchart 1: Five phases of normal cycle.



ENDOMETRIAL CYCLE

The cyclic histologic changes in the adult human endometrium proceed in an orderly fashion in response to cyclic hormonal production by the ovaries. Histologic cycling of the endometrium can be best viewed in two parts: (1) The endometrial glands and (2) the surrounding stroma. The superficial two thirds of the endometrium is the zone that proliferates and is ultimately shed with each cycle if pregnancy does not occur. This cycling portion of the endometrium is known as the decidua functionalis and is composed of a deeply situated intermediate zone (stratum spongiosum) and a superficial compact zone (stratum compactum). The decidua basalis is the deepest region of the endometrium. It does not undergo significant monthly proliferation but, instead, is the source of endometrial regeneration after each menses.

In a 28-day cycle, the first 4 days are occupied with menstruation when two thirds to four fifths of the endometrium is shed. During the remaining 24 days, the histological cycle consists basically of a proliferative and a secretory phase.

Menstrual Endometrium

The first day of vaginal bleeding is conventionally dated as day 1 of menstruation. Menstrual endometrium is dense tissue composed of nonfunctioning basalis and variable stratum spongiosum. Menstrual endometrium represents a transitional state between proliferative and exfoliative endometrium. As much as two thirds of the functioning endometrium is lost during menstruation.

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Proliferative Phase

At the conclusion of menses or vaginal bleeding, the remaining endometrium is necrotic, disorganised and devoid of surface epithelium; sometimes only the basal layer remains. The glands are macerated and broken; the stroma is infiltrated with red and white blood corpuscles **(Figs. 1 and 2)**. Repair proceeds quickly, and the surface is re-covered with epithelium, which grows from the glands and the stroma, within hours of the completion of the menstrual phase. Vascular endothelial growth factor (VEGF) is a highly potent endothelial mitogen produced by the endometrium in response to oestrogen and hypoxia. It plays a role in angiogenesis and endometrial repair. By the end of 2 or 3 days, the surface is intact, new vessels are growing from the stumps of the old, and the glands and stroma are reformed. At this stage, the glands are small and lined by cuboidal epithelium; they have a regular outline and tend to lie parallel to the surface **(Figs. 3 and 4)**. Thereafter proliferation proceeds slowly at first but then quickly, the glands increasing in size and becoming

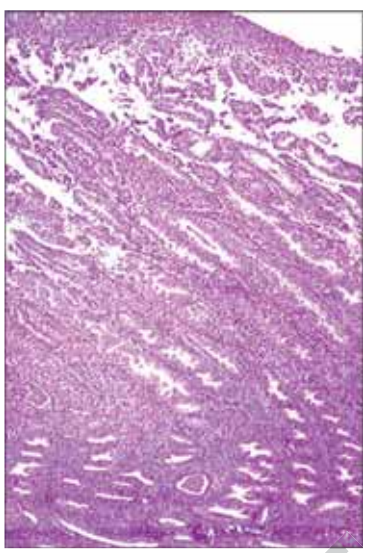


Fig. 1: A low-power view of a menstrual endometrium showing the unresponsive basalis in the lower part of the photomicrograph and the functionalis disintegrating near the surface.

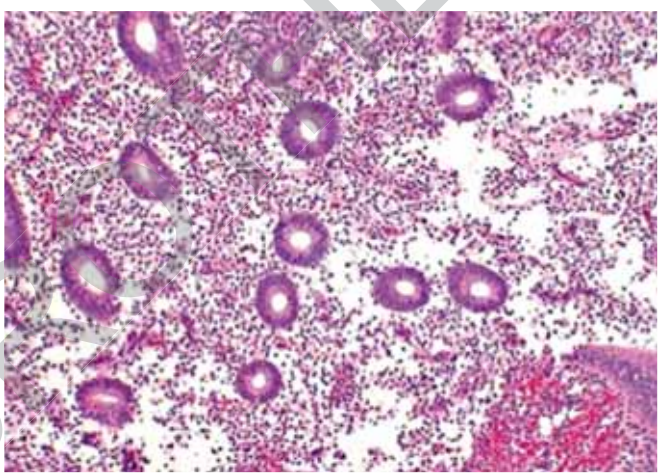


Fig. 3: Early proliferative endometrium. In cross section, the glands look like blue donuts.

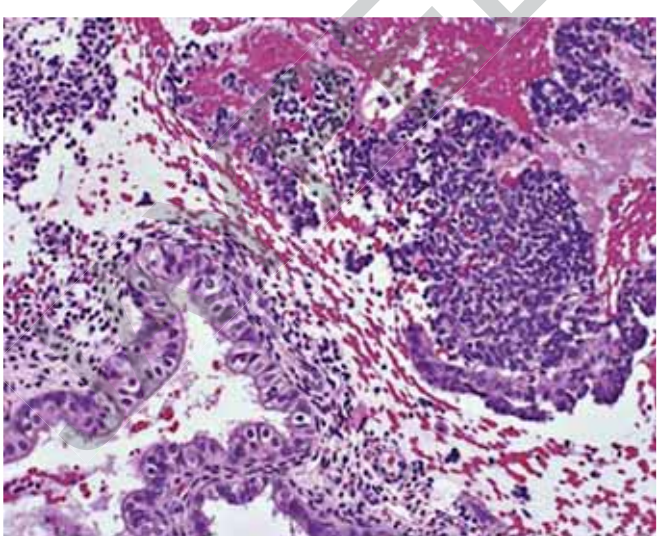


Fig. 2: Menstrual endometrium, secretory glands and detached stromal aggregate ('blue ball').

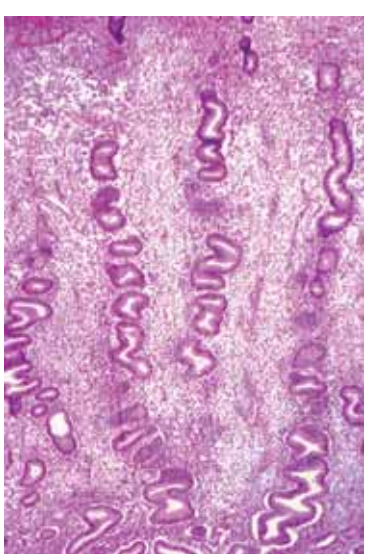


Fig. 4: Midproliferative endometrium. Note the early coiling and synchronously developed glands.

NOT TO BE REPRODUCED

perpendicular to the surface **(Figs. 5 to 7)**. Mitoses are common in epithelial and stromal cells. The epithelial cells become columnar with their nuclei situated basally, but they show no evidence of secretory activity. The stromal cells become spindle-shaped and assume a compact arrangement.

At the beginning of the proliferative phase, the endometrium is thin and measures about 1–2 mm, whereas at the end of it, i.e. on day 14, it measures around 8 mm. The predominant change seen at the beginning of the proliferative phase during this time is the evolution of the initially straight, narrow and short endometrial glands into longer, tortuous structures. Histologically, these proliferating glands have multiple mitotic cells and their organisation changes from a low columnar pattern in the early proliferative period to a pseudostratified pattern before ovulation. The stroma is a dense compact layer throughout this time.

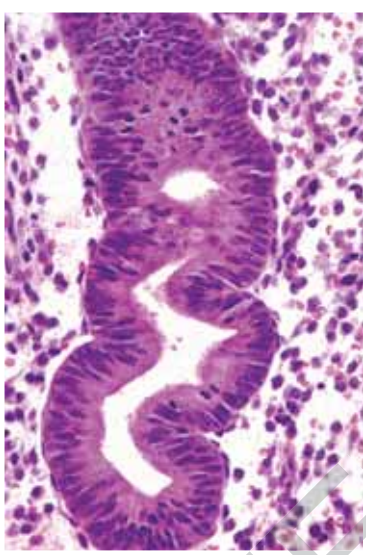


Fig. 5: High-power view of a proliferative phase gland. The constituent cells are pseudostratified, and the characteristically elongated glandular nuclei have dense chromatin.

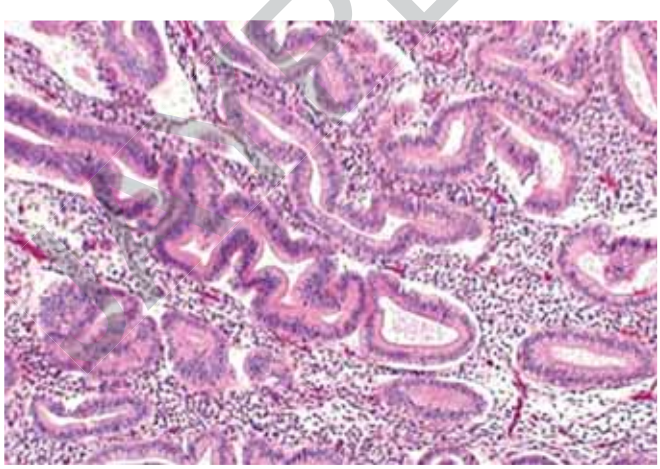


Fig. 6: Late proliferative endometrium showing extensive coiling of glands. This can appear as numerous glands in cross section and should not be interpreted as glandular crowding or hyperplasia.

Secretory Phase

The secretory phase begins at approximately the 14th day of a 28-day cycle. During this phase, the endometrium continues to grow to reach a thickness of 5–7 mm. During the first 3 days, the epithelial cells still undergo mitosis and show pseudostratification of their nuclei. Thereafter, mitosis ceases in the glandular epithelium but can be seen in the stromal cells. The glands increase in size and become actively secretory **(Figs. 7 to 9)**. The secretion collects first as a globule at the base of the cell to give rise to the appearance of subnuclear vacuolation. The globule is later seen on both sides of the nucleus but eventually collects near the lumen of the glands acinus and pushes the nucleus to the base of the cell. At the end of 5–8 days (19th to the 22nd day of the cycle), the secretion enters the gland lumen on its way to the uterine cavity, leaving the cells with their edges frayed **(Figs. 10A and B)**. This secretion, rich in glycogen, fructose and glucose, has a nutritive function for any fertilised ovum reaching the uterus. The secretory phase is also characterised by a change in the

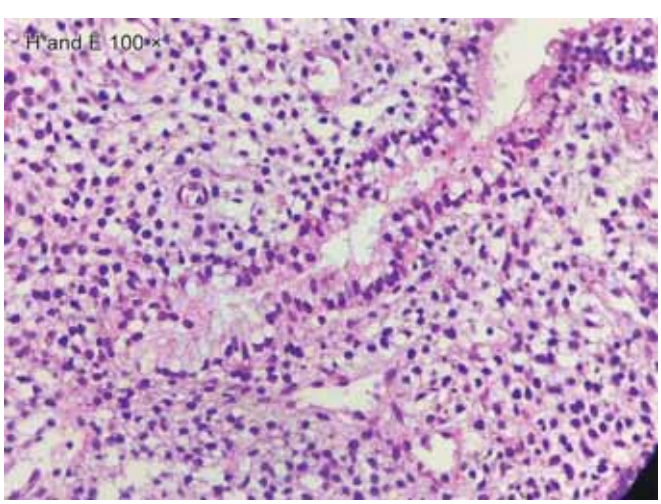
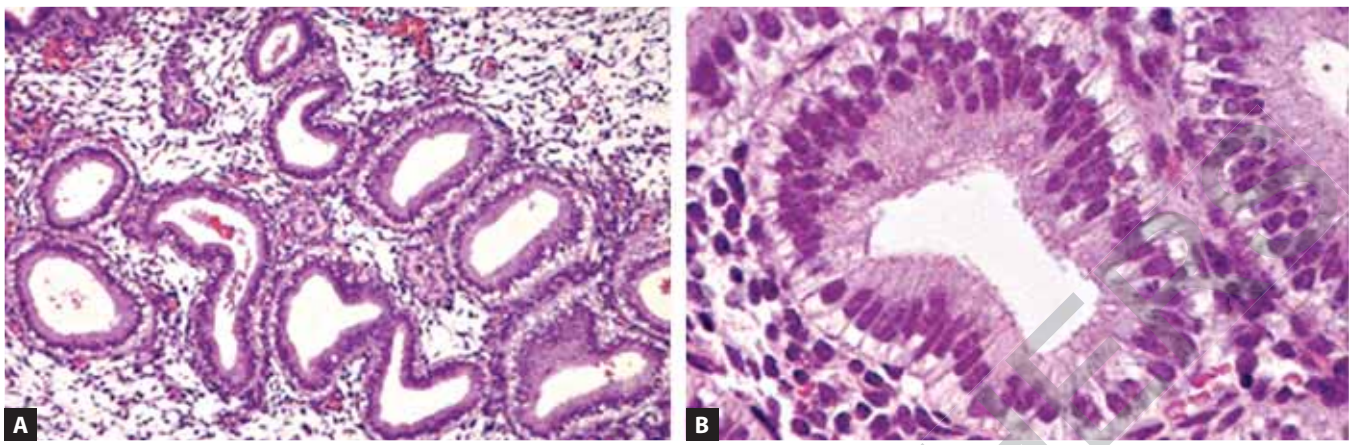
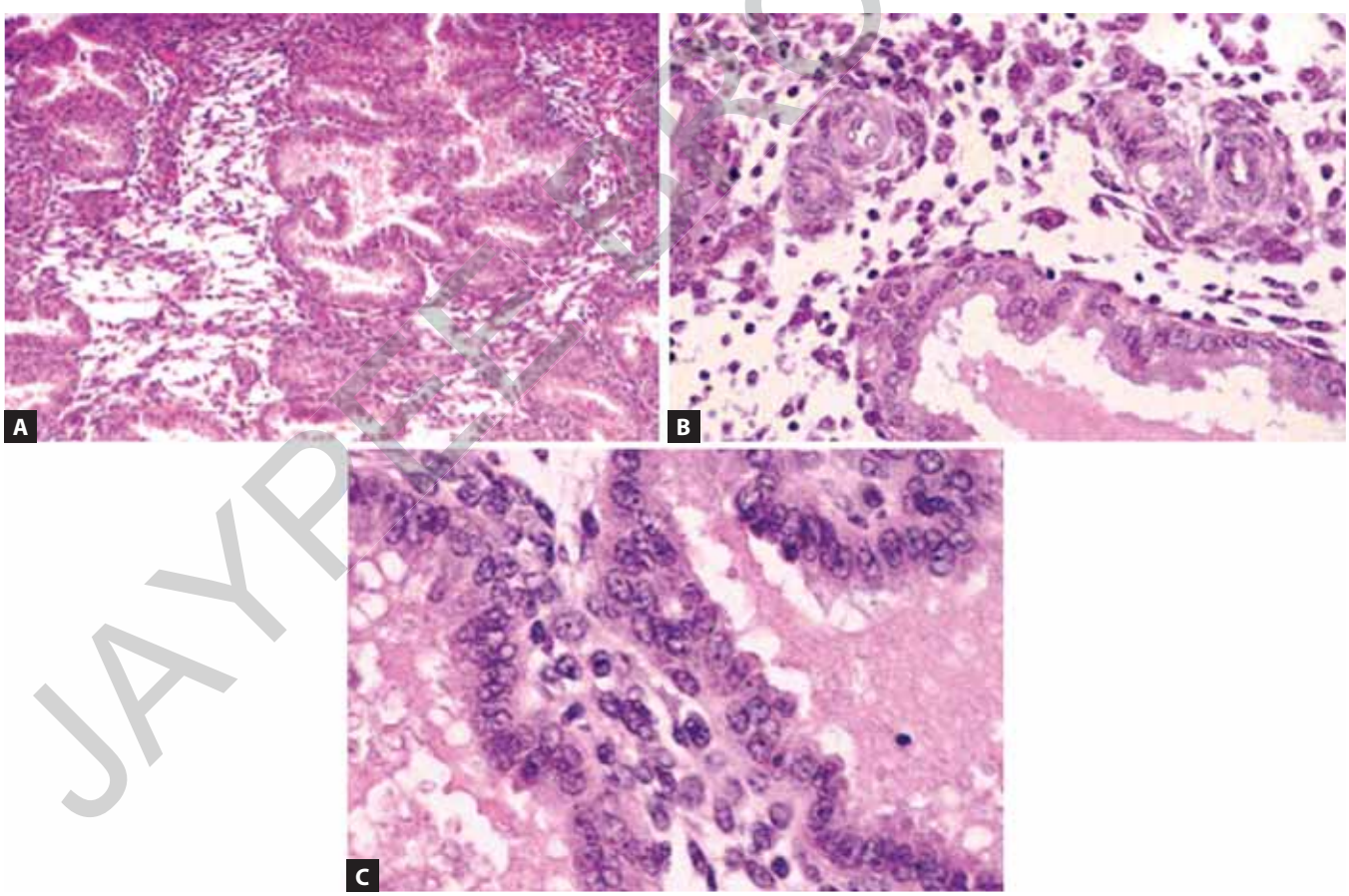


Fig. 7: The early secretory phase. The glands are larger and less regular in outline. The lining cells show globules of secretion lying basal to the nuclei, the so-called subnuclear vacuolation (photomicrograph 100×).
Courtesy: Dr Megha Murali, Assistant Professor, KMC, Manipal.

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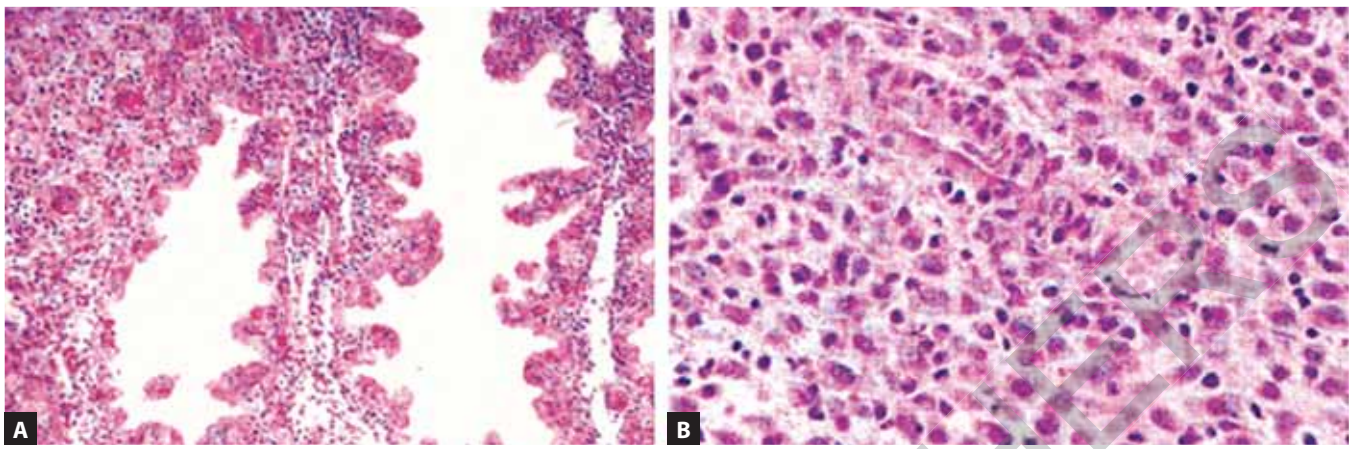


Figs. 8A and B: Early secretory endometrium with subnuclear vacuoles. The nuclei retain the dense chromatin of the proliferative phase. Uniformly present cytoplasmic vacuoles are the most useful marker of the first third of the secretory phase.



Figs. 9A to C: Midsecretory endometrium. The hallmarks of this period can be seen in these three photomicrographs. At low magnification (A), extreme glandular coiling and stromal oedema are apparent. At a somewhat higher magnification (B), the coiled spiral arteries are seen within an oedematous stroma. Perivascular predecidual reaction has not occurred. At yet higher magnification (C), the characteristically round vesicular nuclei of the midsecretory endometrium are apparent.

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Figs. 10A and B: Late secretory endometrium. At low magnification (A), the serrated appearance of the gland reflects its coiled state. The stroma cells have undergone predecidual reaction (B). Predecidual reaction begins around the spiral arteries, and this reaction serves to distinguish midsecretory endometria from late secretory endometria.

shape of the glands. As the glands and cells increase in size, and possibly also because of increased pressure from the surrounding stroma, the gland walls infold so that, on cross section, they are crenated instead of round, and on longitudinal section, they appear 'corkscrew' and finally 'saw-toothed'. Meanwhile, the stroma as a whole becomes more vascular and oedematous. These changes give the stroma a reticular appearance although near the surface it shows a decidual reaction which makes it compact. The latter keeps the necks of the glands narrow and straight. Decidual reaction also occurs in the vicinity of blood vessels **(Table 1 and Figs. 11A and B)**. During its secretory phase, the endometrium shows three layers:

1. A superficial compact zone consisting of stroma (with decidual reaction) surrounding the mouths of the glands
2. A middle spongy functional layer—spongy because of the numerous dilated glands and oedematous stroma
3. An inactive basal layer that shows little secretory response. This is not shed during menstruation and from it the new endometrium is formed after each period.

Preparation for Implantation

On days 21–22 of the cycle, oedema of endometrial stroma occurs secondary to oestrogen and progesterone-mediated increase in prostaglandin production by the endometrium. Implantation requires endometrial haemostasis, and the maternal uterus requires resistance to invasion.

Phase of Endometrial Breakdown

Endometrial growth ceases 5 or 6 days before menstruation (22nd or 23rd day), and within 1 or 2 days, shrinkage is apparent. This occurs as a result of dehydration of the stroma, decreased blood flow and discharge of secretion from the glands. The stroma becomes infiltrated with leucocytes and some red blood cells. Menstruation is heralded by extravasations of serum and blood, which collect in small pools near the endometrial surface. These fragment the new necrotic endometrium and break through the surface epithelium, carrying pieces of it away in the bloody discharge, which is the menstrual flow. Loss of tissue occurs mainly on the 2nd and 3rd days.

CORRELATION OF ENDOMETRIAL AND OVARIAN CYCLE

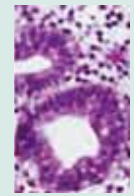
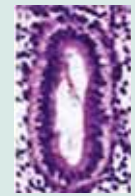
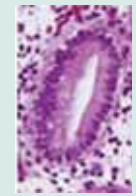
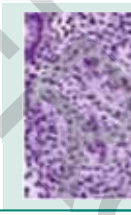
By the end of, if not just before the onset of, a menstrual period, a new follicle is beginning to ripen in the ovary; endometrial proliferation therefore occurs during the follicular phase in the ovary and is the direct result of a mounting oestrogen influence **(Figs. 11A and B)**.

Ovulation marks the changeover from the proliferative to the secretory phase in the endometrium. Secretory activity and decidual reaction are manifestations of the luteal phase in the ovary and are brought about by progesterone acting in the presence of oestrogens.

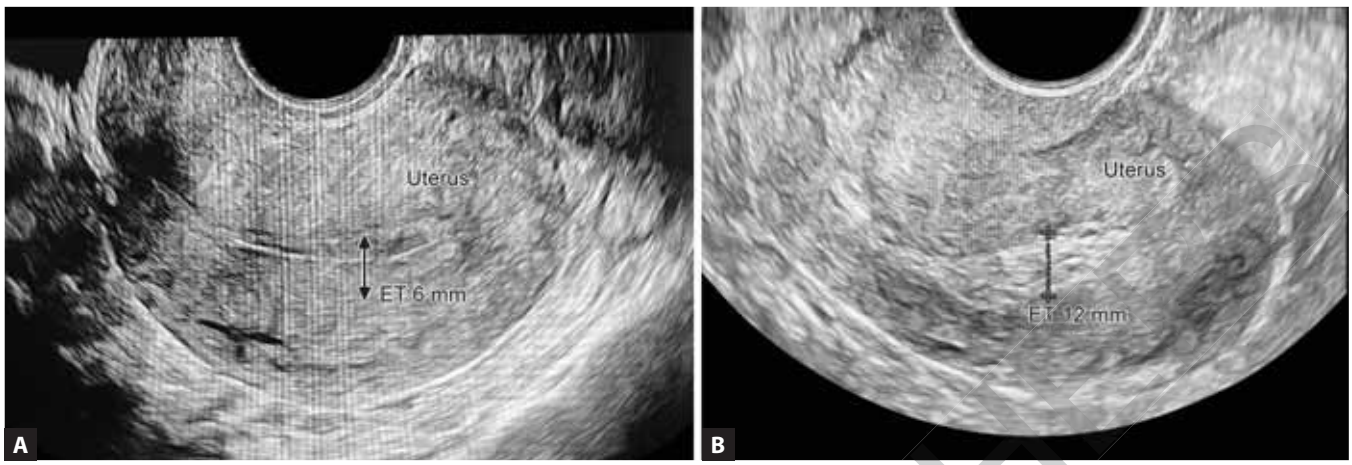
The shrinkage of the endometrium premenstrually coincides with the commencement of the failure of corpus luteum activity and is the direct result of the withdrawal of the supporting effect of oestrogens and progesterone.

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TABLE 1: Main histologic features of the menstrual cycle. Histologic classification stages, according to a 28-day idealised cycle in which day 1 is the first day of clinical menses and ovulation occurs on day 14.

Endometrial phase	***Menstrual***		***Proliferative***	***'Interval'***	***Secretory***				
					Early		***Mid***	***Late***	
Day of 28 days cycle (ovulation on D14)		1–3	4–15	-16	17	18	19–21	22–24	25–28
Key feature		Stromal crumbling	Mitoses	Subnuclear vacuoles, staggered, with mitoses	Subnuclear vacuoles, regular with mitoses	• Subnuclear and supranuclear vacuoles • No mitoses	Stromal oedema	Focal predecidua around spiral arteries	Predecidua involves surface
Microscopic features of functional zone	Stroma	• Stromal aggregates • "Blue balls" haemorrhage	• Loose stroma • Mitoses	• Loose stroma • Mitoses	Loose stroma, scanty mitoses	Loose stroma, scanty mitoses	Stromal oedema increases from D19–21	Perivascular "naked nuclei" (D22) from predecidual cuffs (D23) that bridge vessels (D24)	• Focal (D25) then extensive (D26) surface predecidua extends downward (D26-28) • Prominent granulated lymphocytes
	Glands	Collapsed epithelium and displaced secretory glands	• Straight to tightly coiled tubules • Mitoses	• Some staggered subnuclear vacuoles • Many mitoses	• "Piano key" subnuclear vacuoles • Some mitoses	• Some staggered subnuclear vacuoles • No mitoses	• Dilated glands with irregular outlines • Luminal secretion	"Saw tooth" glands	Prominent "saw tooth" glands
Appearances									

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Figs. 11A and B: Ultrasonography (USG) images of endometrium: (A) Proliferative phase and (B) secretory phase. (ET: endometrial thickness)

These are the fundamental correlations with ovarian activity, although the timing of the histological cycle does not exactly match the rise and fall of hormone levels in the plasma **(Fig. 12)**.

Hormonal Variations

The relative pattern of ovarian, uterine and hormonal variation along the normal menstrual cycle is seen.

At the beginning of each monthly menstrual cycle, levels of gonadal steroids are low and have been decreasing since the end of the luteal phase of the previous cycle.

With the demise of the corpus luteum, follicle-stimulating hormone (FSH) levels begin to rise, and a cohort of growing follicles is recruited. These follicles each secrete increasing levels of oestrogen as they grow in the follicular phase. This, in turn, is the stimulus for uterine endometrial proliferation. Rising oestrogen levels provide negative feedback on pituitary FSH secretion, which begins to reduce by the midpoint of the follicular phase. Late in the follicular phase the luteinising hormone (LH) level is increased dramatically (biphasic response). At the end of the follicular phase (just prior to ovulation), FSH-induced LH receptors are present on granulosa cells and, with LH stimulation, modulate the secretion of progesterone.

With the oestrogen rising to a peak, the pituitary LH surge is triggered, which induces ovulation 24–36 hours later. Ovulation is the beginning of the transition to the luteal phase of the ovary or the secretory phase of the endometrium. The oestrogen level decreases through the early luteal phase from just before ovulation until the midluteal phase, when it begins to rise again as a result of corpus luteum secretion. Progesterone levels rise precipitously after ovulation and can be used as a presumptive sign that ovulation has occurred. Both oestrogen and progesterone levels remain elevated through the lifespan of the corpus luteum and then wane with its demise, thereby setting the stage for the next cycle **(Fig. 12 and Table 2)**.

UTERINE BLEEDING

The phenomenon of uterine bleeding is best considered from the experimental aspect.

- If a woman without ovaries but possessing a normal uterus is given large doses of oestrogens for a few weeks and the administration is then suddenly discontinued, uterine haemorrhage always occurs 2–10 days later. This is termed *oestrogen withdrawal bleeding*.
- If progesterone is substituted for oestrogen in this experiment, then, provided the uterus has been previously primed with oestrogens, *progesterone withdrawal bleeding* occurs within 2–5 days of suspension of medication.
- A mixture of oestrogens and progesterone has a similar effect.
- If large doses of oestrogens are given first and then replaced by progesterone, no bleeding occurs on withholding the oestrogen. It is deferred until the progesterone is withdrawn. Progesterone is an adequate substitute for oestrogen but the converse is not true.
- If, in the first type of experiment, only a small dose of oestrogen is administered, no effect is seen when it is withdrawn.
- If, however, a moderate dose of oestrogen is given, then bleeding sometimes occurs intermittently, even while treatment is in progress.

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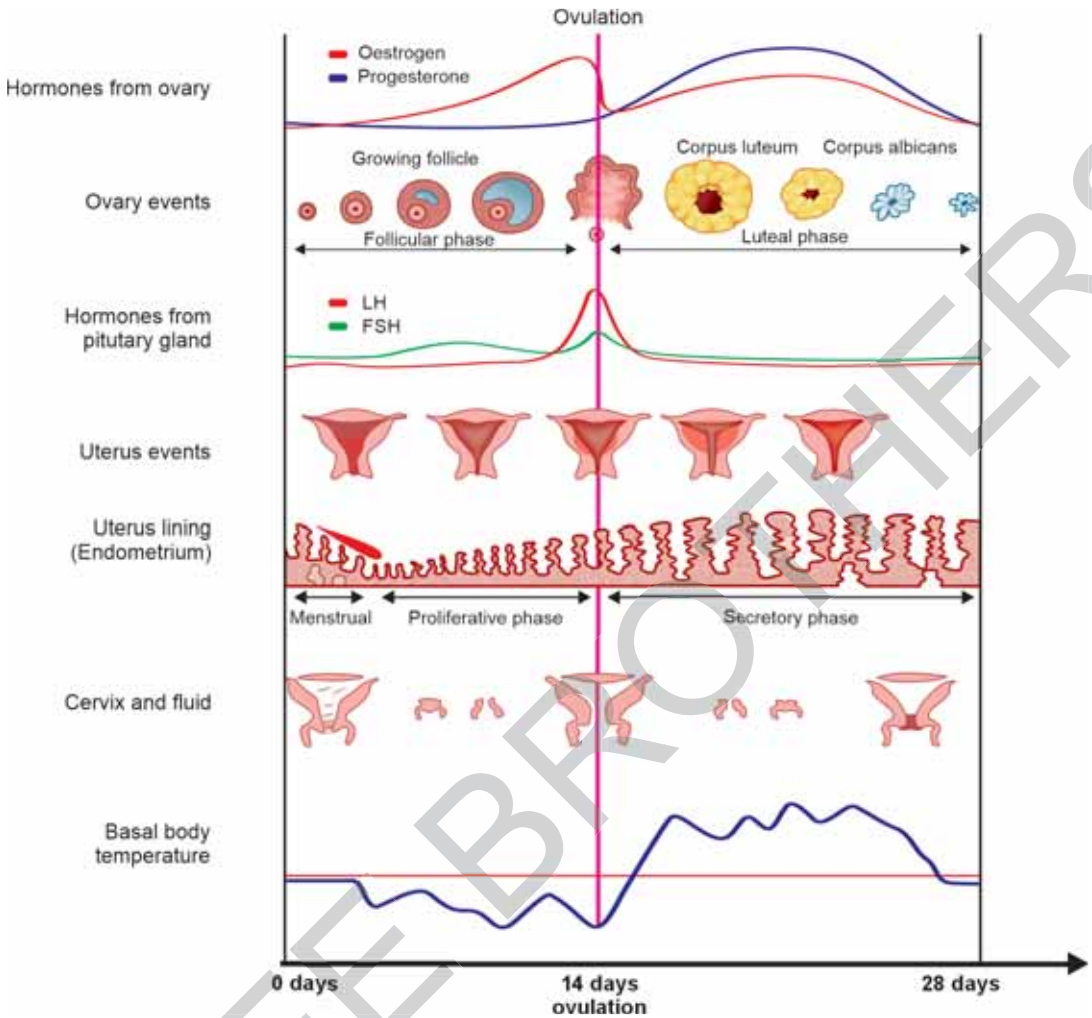


Fig. 12: Diagrammatic representation of the hormonal control of the ovarian and endometrial cycles. (FSH: follicle-stimulating hormone; LH: luteinising hormone)

Observations such as these have given rise to a concept of a *bleeding threshold* level of oestrogens in the bloodstream. The quantitative value of this level is unknown, and its existence is hypothetical. Nevertheless, the idea is of practical value and is illustrated in **Figure 13**. If the critical level is not reached, that is, if the amount of oestrogen administered is too small, no bleeding occurs. If the oestrogen level is pushed above the threshold, then no bleeding takes place so long as it remains above, but once it drops to and below the threshold, haemorrhage occurs. If, however, the amount of oestrogen administered is such as to keep the blood level at or about the threshold, then bleeding may occur even while oestrogen is being given. This is generally called 'breakthrough' bleeding.

The above observations explain why removal of an ovary, or of the corpus luteum, at operation is often followed by uterine bleeding 2 or 3 days later.

Menstruation

As a result of the ovarian cycle, the uterus is subjected to the influence first of oestrogens and then of a combination of oestrogens and progesterone. Both are withdrawn when the corpus luteum degenerates and menstruation occurs within a few days, that is, as soon as their levels fall to, and below, the bleeding threshold. There is some dispute as to whether the period represents oestrogen deprival haemorrhage or progesterone deprival haemorrhage, but this is an academic argument.

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TABLE 2: Female reproductive cycle.

Days	*Changes in the ovary*	*Changes in the endometrium*	*Hormonal changes*
1–5	*Follicular phase:* Follicles further develop and compete for dominance; one outgrows the others. The other follicles become atretic	*Menstruation phase:* The endometrium breaks and is shed as menstruation	Luteinising hormone (LH) and follicle-stimulating hormone (FSH) (hormones secreted from the anterior pituitary) act on ovaries to stimulate follicular growth. The follicles secrete oestrogen and progesterone
6–11	*Follicular phase:* A single dominant follicle continues to grow The oocyte inside is maturing and begins producing oestrogen	*Proliferative phase:* Oestrogen secreted from the growing follicle causes the endometrium to grow and thicken, thereby preparing for implantation	The follicles continue to secrete oestrogen and progesterone
12–13	*Follicular phase:* LH surge occurs. The rise in LH causes the follicle to rupture over the next 24–36 hours and release a mature oocyte	*Proliferative phase:* The endometrium continues to grow	Rising levels of oestrogen reach a point that causes a rapid rise in LH
14	*Ovulation phase:* The follicle wall ruptures and releases the mature oocyte from the ovary	*Proliferative phase:* The endometrium continues to grow	
15–20	*Luteal phase:* The remains of the follicle that released the oocyte form a structure called the *corpus luteum*	*Secretory phase:* Progesterone from *the corpus luteum* brings about secretory changes in the uterus and prepares it for implantation Progesterone also raises basal body temperature (BBT)	Corpus luteum secretes progesterone
21–28 If the oocyte was fertilised	*Luteal phase:* The corpus luteum continues to secrete progesterone	*Secretory phase:* After about 9 days, the embryo gets implanted	• The embryo releases human chorionic gonadotrophin (hCG) • Corpus luteum produces progesterone until 11 weeks of pregnancy, when the uteroplacental shift takes place
21–28 If the egg was NOT fertilised	*Luteal phase:* The corpus luteum transforms into the *corpus albicans*	*Secretory phase:* Without progesterone from the corpus luteum, the endometrium begins to break up and is eventually shed again (a new cycle begins)	Hormones fall back to their original levels

The **fundamental cause** of menstruation is degeneration of the **corpus luteum,** and this only degenerates if the ovum **is not fertilised.** Menstruation represents the breaking down and casting off of an endometrium prepared for a pregnancy, which does not materialise, and so is sometimes described as 'the weeping of a disappointed uterus.'

With the withdrawal of sex steroids, there is a profound spiral artery vascular spasm that ultimately leads to endometrial ischaemia. Simultaneously, there is a breakdown of lysosomes and a release of proteolytic enzymes, which further promote local tissue destruction. This layer of endometrium is then shed, leaving the decidua basalis as the source of subsequent endometrial growth. Prostaglandins are produced throughout the menstrual cycle and are at their highest concentration during menses. Prostaglandin $F_{2\alpha}$ ($PGF_{2\alpha}$) is a potent vasoconstrictor, causing further arteriolar vasospasm and endometrial ischaemia. $PGF_{2\alpha}$ also produces myometrial contractions that decrease local uterine wall blood flow and may serve to physically expel sloughing endometrial tissue from the uterus.

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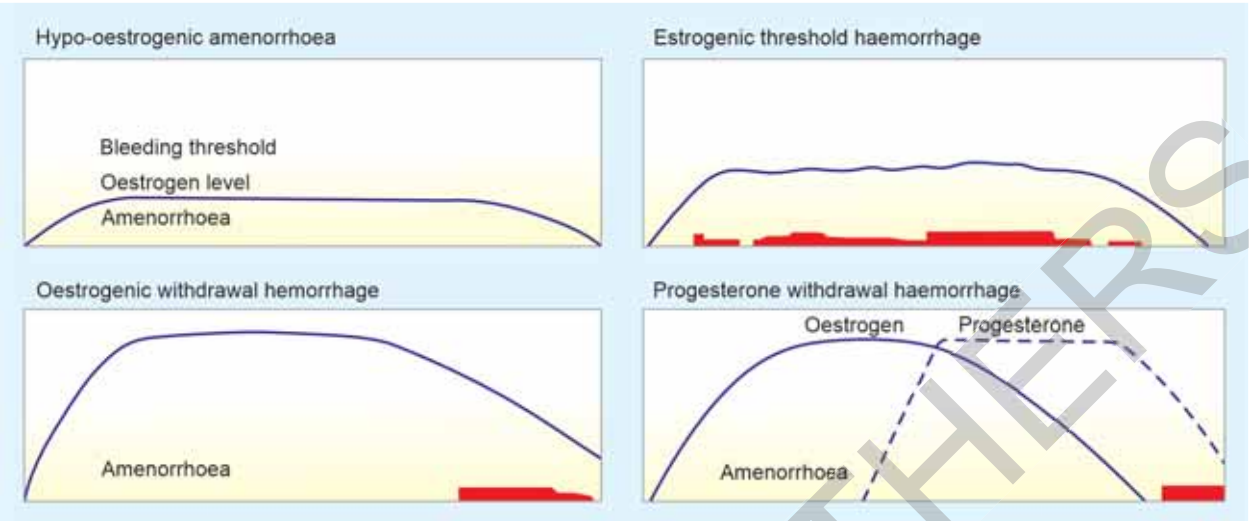


Fig. 13: The theoretical concept of a blood threshold level of hormones to account for different types of uterine bleeding. The black areas on the baseline indicate episodes of bleeding.

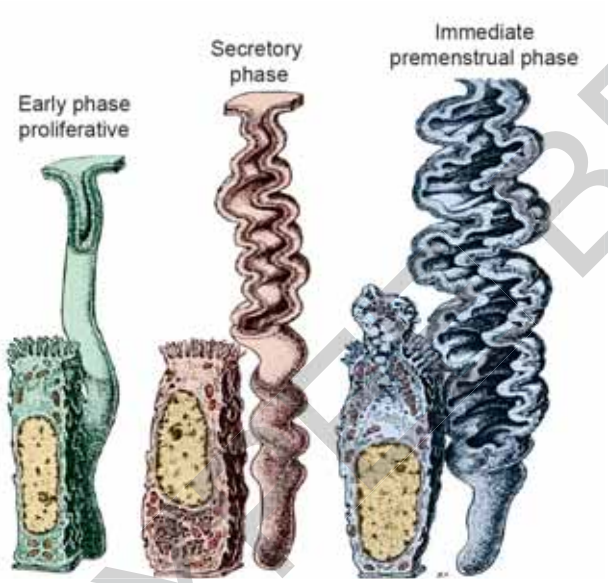


Fig. 14: Diagrammatic representation of the endometrial glands and arterioles throughout the menstrual cycle. The veins are omitted for simplicity. The endometrium deep to the dotted line is relatively insensitive to hormones and is not shed during menstruation; its viability is maintained by special branches from the straight portions of the arterioles.

Anovular Menstruation

Degeneration of the corpus luteum was suggested as the cause of menstruation long before ovarian hormones and their effects were discovered, but the idea was always questioned because it was noted that sometimes there is no sign of a recent corpus luteum in the ovary when the abdomen is opened during a menstrual period. This observation is now explained by the phenomenon of *anovular menstruation*. Sometimes, a follicle ripens but fails to rupture and is not converted to a corpus luteum. After a phase of activity, the follicle degenerates, and there is thus a rise and fall in oestrogen production, the latter giving rise to oestrogen withdrawal haemorrhage. This can be completely cyclical and is outwardly indistinguishable from normal menstruation. It can, however, be recognised by histological examination of the endometrium, which remains proliferative or hyperplastic but never secretory. Anovular menstruation is also always painless and unaccompanied by the usual menstrual molimina.

Anovular menstruation occurs occasionally in all women. It is probably not uncommon during the first 2 or 3 years of menstrual life and after the age of 40 years, the incidence may rise to as high as 1 in 4 cycles. In the active reproductive period, however, not more than 1 in 10 to 1 in 50 regular cycles is anovular. For practical purposes, regular menstruation in mature women means regular ovulation.

Mechanism of Bleeding from the Endometrium

The main arteries supplying the endometrium lie where the muscle mucosal elements of the uterus intermingle, and they run parallel to the endometrium. Arterioles from these basal arteries come off at right angles and are directed towards the uterine cavity **(Fig. 14)**. In the first part of their course, they are straight, but more superficially they are

JAYPEE BROTHERS

coiled. The coiling allows lengthening and shortening of the vessels and reduces intravascular pressure. These arterioles provide for the middle and superficial layers of the endometrium, each supplying a limited area and having very little communication with its fellows. Capillaries beneath the surface of the endometrium are drained by veins that run parallel to the arteries. Arteriovenous connections are also described.

When the hormonal support of the secretory endometrium is withdrawn consequent to the degeneration of the corpus luteum, the endometrium shrinks and the vessels have to shorten by increasing and tightening their coils. This in turn retards their circulation and results in ischaemic necrosis of the area of the endometrium for which they are responsible. Some of the capillary stasis and other changes such as leucocytic infiltration of the stroma are caused by haemodynamic factors, which are not understood, but some authorities believe that there is an arteriovenous shunt. Increased premenstrual permeability of the capillary reflects an oestrogen influence.

The breakdown does not occur simultaneously in all parts of the endometrium and each microscopic area bleeds intermittently for short periods, but the endometrium oozes from one part or another for a total of 3–5 days. The bleeding itself occurs from broken arteries, veins and capillaries, and from haematomas, which form in the stroma in the immediate premenstrual phase.

Having entered the uterine cavity, the blood coagulates, but the clot is mostly dissolved by thrombolysis released from the endometrium. Proactivator of plasminogen is present in the endometrium at all times and, with the approach of menstruation, is converted to activator, probably as a result of anoxia or breakdown of tissue. Fibrin degradation products therefore circulate in increased amounts during menstruation, and especially high levels are found in the uterine veins.

As a result of fibrinolysis, the effluent from the uterus consists of altered blood, rich in calcium but deficient in prothrombin and fibrinogen. The expulsion of this, together with fragments of endometrium, is brought about by uterine contractions.

The occurrence of spasm in the straight stems of the endometrial arterioles is very important. This is seen about the time that bleeding commences and may be the result of the action of the $PGF_{2\alpha}$ released from the degenerating surface of the endometrium. It was at one time thought that the spasm causes the ischaemic necrosis but it is now believed to be a later phenomenon whose purpose is to limit the amount of bleeding from the arteriole. Indeed, it is alleged that but for this protective spasm, a woman might bleed to death as a result of menstruation. The amount of loss may also be controlled by muscular compression of the larger arteries as they pass through the myometrium. It is also determined by the area of the endometrial bleeding surface and by the intensity of the previous hormone stimulus. Other vasoconstrictors, e.g. endothelium and platelet-activating factor (PAF) have been identified but their exact role is unclear.

The mechanism of bleeding is the same no matter whether menstruation is ovular or anovular in type, although, in the latter case, there is rather less tissue destruction.

When the endometrium breaks down, the basal layers are not involved because they are supplied by blood vessels, which come off the main arteriole proximal to the part affected by coiling and spasm **(Fig. 13)**. Postmenstrually, new arterioles grow from the old stumps just as the endometrium grows from its basal layer. They do so as a result of the oestrogen stimulus from the next ripening Graafian follicle.

MYOMETRIAL CYCLE

The activity of uterine muscle changes with the menstrual cycle. During the early follicular phase, the contractions are small and frequent and the response to oxytocics is limited. Activity increases with the oestrogen stimulus of approaching ovulation. In the premenstrual and menstrual phases, the contractions are less frequent but are of greater amplitude, and muscle sensitivity is increased. The strongest contractions occur during menstruation and may, to some extent, be stimulated by *prostaglandins* released from the endometrium.

CYCLICAL CHANGES IN THE TUBE

The muscle of the fallopian tube behaves like the myometrium in that it shows increased movement about the time of ovulation. This is an oestrogen effect, as is the increased cilial activity at that time. These changes are timed to propel the ovum towards the uterus.

The follicular phase is marked by slight proliferation, with minimal mitotic activity of the mucosa, and this continues up to the premenstrual phase when it regresses. During menstruation, there is further shrinkage and slight

JAYPEE BROTHERS

shedding of the surface epithelium; occasionally, the endosalpinx may even menstruate.

The secretory activity of the tube is also cyclical, being highest just before ovulation and in response to oestrogen. Progesterone may also play a part in this, but the consensus of opinion is that this hormone reduces the amount of secretion.

CERVICAL CYCLE

Progesterone raises the tone of the muscles of the isthmus and internal os, so the cervical 'sphincter' is tighter and more competent during the luteal than during the follicular phase. The glandular elements of the cervix proliferate during the follicular phase, and the epithelial cells become taller. Under the influence of oestrogens, the glands also actively secrete mucus which will stretch into threads measuring >6.5 cm, and even 10–15 cm, at the time of ovulation. This property of 'spinnbarkeit' is the basis of the thread test for oestrogen in circulation. During the follicular phase, the cervical mucus absorbs water and salts and, when allowed to dry, deposits crystals of sodium chloride and potassium chloride in a characteristic pattern that suggests the fronds of a fern (*see* **Fig. 3** in **Chapter 5**). At the time of ovulation, the secretion is so profuse that it may be noticeable as a vaginal discharge—the 'ovulation cascade.' Its special character at this time makes for its easy penetration by spermatozoa. This property is related to its low content of protein.

During the luteal phase, the cervical glands become more branched and their secretion changes its physical and chemical properties. The mucus becomes more viscous and forms a more secure cervical plug. It loses its ability to stretch without breaking and resists penetration by spermatozoa. These changes are brought about by progesterone and are related to an increase in the amount of protein in the mucus and to the presence of phospholipids.

Progesterone also reduces the electrolyte content of the mucus, so by the 22nd day of the cycle, its property of ferning or arborisation on drying is lost.

During menstruation, the cervical glands collapse and may show slight desquamation with loss of blood.

VAGINAL CYCLE

Cyclical histological changes occur in the vaginal epithelium, but they are not clearly defined in tissue sections. They are better seen in smears of desquamated cells. The unstimulated vagina shows relatively small basal-type cells with healthy nuclei. These and intermediate basophil forms are also seen in vaginal smears taken in the early follicular phase. The fully oestrogenic smear, evident during the late follicular phase, contains a preponderance of large cornified epithelial cells with pyknotic nuclei. These stain pink with eosin. During the luteal phase, the smear shows evidence of increased desquamation, many of the cells having rolled edges, and is characterised by the reappearance of clumps of intermediate cells and the presence of leucocytes.

The *maturation index*, which is the percentage of superficial, intermediate and parabasal cells in a vaginal smear, is used as a measure of the levels of hormones in circulation. It is a useful guide, but it is not as precise as assaying the hormones in blood.

CYCLICAL, METABOLIC, VASCULAR AND PSYCHOLOGICAL CHANGES

These are discussed in detail in **Chapter 5** (Section on Menstruation) and **Chapter 42**.

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Muralidhar V Pai MD FRCOG FICOG FKCOG FICMCH is currently serving as the Pro Vice Chancellor, Sikkim Manipal University, Sikkim, India; and the Dean and Professor, Department of Obstetrics and Gynaecology, Sikkim Manipal Institute of Medical Sciences, Gangtok, Sikkim. He previously held several key positions at Kasturba Medical College, Manipal, Karnataka, including that of Head of the Department, and he has accumulated over 37 years of combined teaching experience. Throughout his distinguished career, he has consistently excelled in education, winning the Best Teacher Award (ten times) and the Best Teaching Material Award (five times) at Kasturba Medical College. His exceptional research contributions have been recognised with both the Junior and Senior FOGSI Corion Award for Best Research.

An outstanding clinician, Dr Pai's contributions to the field of Obstetrics and Gynaecology have earned him international recognition. In 2023, he was honoured with the Honorary Fellowship of the Royal College of Obstetricians and Gynaecologists (Hon FRCOG), further cementing his stature in the global medical community. He has co-authored two widely respected textbooks: *Holland and Brew's Manual of Obstetrics (4th Edition)* and *Obstetrics and Gynaecology Prep Manual for Undergraduates (1st Edition)*, both published by Elsevier. In addition, he has contributed chapters to numerous textbooks in Obstetrics and Gynaecology.

His specialization lies in Reproductive Medicine and Surgery, fields in which he has received advanced training in the UK, USA, and Belgium. He is also a sought-after orator, having delivered over 850 guest lectures. His *Lecture Series on YouTube* being highly popular among teachers and students alike. In his professional capacity, he has served in several important roles within the Federation of Obstetric and Gynaecological Societies of India (FOGSI). He has been the Chairman of the Public Awareness Committee, a Governing Council Member of the Indian College of Obstetrics and Gynaecology (ICOG), and the National Corresponding Editor of the Journal of Obstetrics and Gynaecology of India. He is also the Founder Chairman of the Karnataka College of Obstetricians and Gynecologists, an institution, he established to advance the field in the region. His career is a testament to his dedication to teaching, clinical excellence, research, and contributions to the advancement of obstetrics and gynaecology.

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